


Rosen & Barkin's **5-Minute Emergency Medicine Consult**

5TH EDITION

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Roger M. Barkin
Stephen R. Hayden
Richard E. Wolfe
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 **Wolters Kluwer**



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5-Minute Emergency Medicine Consult

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Rosen and Barkin's *5-Minute Emergency Medicine Consult* continues to reflect the evolving nature of our clinical emergency medicine practices. Emergency medicine provides unique challenges to the clinician; the remarkable breadth of clinical conditions encountered, the time constraints of an acute illness, environmental considerations, and the logistical demands of busy emergency departments. Time is of the essence, and this book is truly designed to meet the needs of clinicians working in settings providing urgent and emergent care. To look for a diagnosis, one must think of it, and the very nature of the emergency department (ED) makes prolonged deliberation difficult. Nevertheless, it must become instinctive to think about the statistically rare, but clinically serious entity, rather than to just reach for the statistically probable but nonlife-threatening diagnosis.

Rosen and Barkin's 5-Minute Emergency Medicine Consult provides concise formatted information allowing the busy clinician to respond to each patient appropriately. It is meant to be readily available in the ED, and frequently used in the trenches. Written and edited by practicing clinicians for their colleagues, the book is designed to synthesize a mountain of information into tightly formatted chapters that stimulate analysis and subsequent assessment.

We have attempted to integrate our authors' expertise, experience, and knowledge base onto the pages of this book. The chapters are not meant to be a diagnostic engine, but rather a place to confirm a diagnosis supported by clinical judgment and the subjective and objective evidence of a patient's presentation. Our authors attempt to provide a precise and clinically relevant summary useful in caring for the patient while also equipping students and residents with the structure needed to approach individual disease processes. We are indebted to our contributors for their commitment to this task.

The book is intended to be accurate, focused, and readily integrated into practice, rather than being definitive and all encompassing. As in the past, this new edition incorporates new information and approaches to management, while allowing us to modify topics that reflect some of the new challenges we face.

Our goal is to make *Rosen and Barkin's 5-Minute Emergency Medicine Consult* useful to both novices in emergency medicine and experienced clinicians. The information and organization is designed to be easily used within the "chaos" that surrounds our clinical settings.

Clinical acumen, judgment, and experience are the foundations for our clinical practice. It is our hope that this book will serve students, nurses, emergency medical personnel, residents, and practicing emergency physicians as a readily used resource in excellent patient care and knowledge acquisition.

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It is also important to acknowledge and thank our colleagues nationally and internationally who continue to teach each of us on an ongoing basis and will hopefully find this book useful in their practices.

R.M.B.

To my wife Marina, and my children Connor, Maia, and Kenny; without your support and understanding, my involvement in academic emergency medicine and this book would be not possible. To my current residents and past graduates; I hope the information in this book helps guide your clinical decisions and prompts you to always maintain your intellectual curiosity.

S.R.H.

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Boerhaave Syndrome

Bowel Obstruction (Small and Large)

Caustic Ingestion

Cholangitis

Cholecystitis

Cholelithiasis

Cirrhosis

Constipation

Diarrhea, Adult

Diarrhea, Pediatric

Diverticulitis

Diverticulosis

Dysphagia
Feeding Tube Complications
Foreign Body, Esophageal
Foreign Body, Rectal
Gallstone Ileus
Gastric Outlet Obstruction
Gastritis
Gastroenteritis
Gastroesophageal Reflux Disease
Gastrointestinal Bleeding
Hemorrhoid
Hepatic Encephalopathy
Hepatitis
Hepatorenal Syndrome
Hernias
Hirschsprung Disease
Inflammatory Bowel Disease
Intussusception
Irritable Bowel Syndrome
Mallory–Weiss Syndrome
Malrotation
Meckel Diverticulum
Mesenteric Ischemia
Pancreatitis
Peptic Ulcer
Perforated Viscous
Perirectal Abscess
Rectal Prolapse
Spontaneous Bacterial Peritonitis
Varices
Volvulus
Vomiting, Adult
Vomiting, Cyclic
Vomiting, Pediatric

Genitourinary Emergencies

Chancroid

Epididymitis/Orchitis

Fournier Gangrene

Hematuria/Proteinuria

Herpes, Genital

Hydrocele

Lymphogranuloma Venereum

Paraphimosis

Penile Shaft Fracture

Phimosis

Priapism

Prostatitis

Syphilis

Testicular Torsion

Urethral Trauma

Urethritis

Urinary Retention

Urinary Tract Fistula

Urinary Tract Infections, Adult

Urinary Tract Infections, Pediatric

Gynecologic Emergencies

Amenorrhea

Bartholin Abscess

Dysfunctional Uterine Bleeding

Endometriosis

Fibrocystic Breast Changes

Gonococcal Disease

Herpes, Genital

Mastitis

Ovarian Cyst/Torsion

Pelvic Inflammatory Disease

Sexual Assault

Trichomonas

Vaginal Bleeding

Vaginal Bleeding in Pregnancy

Vaginal Discharge/Vaginitis

Head and Neck Emergencies

Angioedema

Aphthous Ulcers

Bell's Palsy

Blow-Out Fracture

Cavernous Sinus Thrombosis

Cervical Adenitis

Dental Trauma

Epiglottitis, Adult

Epiglottitis, Pediatric

Epistaxis

External Ear Chondritis/Abscess

Facial Fractures

Foreign Body, Ear

Foreign Body, Nasal

Head Trauma, Blunt

Head Trauma, Penetrating

Labyrinthitis

Laryngitis

Ludwig Angina

Mandibular Fractures

Mastoiditis

Ménière Disease

Nasal Fractures

Otitis Externa

Otitis Media

Otologic Trauma

Periodontal Abscess

Peritonsillar Abscess

Pharyngitis

Retropharyngeal Abscess

Sinusitis (Rhinosinusitis)

Temporal–Mandibular Joint Injury/Syndrome

Toothache

Torticollis

Tympanic Membrane Perforation

Uvulitis

Hematologic Emergencies

Anemia

Coagulopathy Reversal (Nonwarfarin Agents)

Disseminated Intravascular Coagulation

HELLP Syndrome

Hemophilia

Idiopathic Thrombocytopenic Purpura

Leukocytosis

Polycythemia

Sickle Cell Disease

Thrombotic Thrombocytopenic Purpura

Transfusion Complications

Von Willebrand Disease

Immune System Emergencies

Anaphylaxis

Angioedema

Granulocytopenia

HIV/AIDS

Idiopathic Thrombocytopenic Purpura

Immunosuppression

Opportunistic Infections

Pruritus

Serum Sickness

Systemic Lupus Erythematosus

Transplant Rejection

Vasculitis

Infectious Disease Emergencies

Abscess, Skin/Soft Tissue
Amebiasis
Babesiosis
Botulism
Cellulitis
Cervical Adenitis
Chancroid
Dengue Fever
Ehrlichiosis
Encephalitis
Endocarditis
Epididymitis/Orchitis
Epiglottitis, Adult
Epiglottitis, Pediatric
Fournier Gangrene
Giardiasis
Gonococcal Disease
Hand Infection
Hemorrhagic Fevers
Herpes Simplex
Herpes Zoster
Herpes, Genital
Immunizations
Impetigo
Influenza
Kawasaki Disease
Leukocytosis
Lyme Disease
Lymphadenitis
Lymphangitis
Lymphogranuloma Venereum
Malaria
Mastitis
Mastoiditis

Measles
Meningitis
Meningococemia
Mononucleosis
MRSA, Community Acquired
Mumps
Myocarditis
Necrotizing Soft Tissue Infections
Necrotizing Ulcerative Gingivitis
Needlestick
Osteomyelitis
Pelvic Inflammatory Disease
Periorbital and Orbital Cellulitis
Pertussis
Pharyngitis
Pneumocystis Pneumonia
Pneumonia, Adult
Pneumonia, Pediatric
Polio
Prostatitis
Rabies
Rash
Rheumatic Fever
Rocky Mountain Spotted Fever
Roseola
Rubella
Sepsis
Sinusitis (Rhinosinusitis)
Sporotrichosis
Staphylococcal Scalded Skin Syndrome
Stevens–Johnson Syndrome
Streptococcal Disease
Syphilis
Tetanus

Tick Bite

Tinea Infections, Cutaneous

Toxic Epidermal Necrolysis

Toxic Shock Syndrome

Toxoplasmosis

Trichomonas

Tuberculosis

Tularemia

Urethritis

Urinary Tract Infections, Adult

Urinary Tract Infections, Pediatric

Varicella

West Nile Virus

Metabolic Emergencies

Acidosis

Alcoholic Ketoacidosis

Alkalosis

Diabetic Ketoacidosis

Hypercalcemia

Hyperkalemia

Hypernatremia

Hyperosmolar Syndrome

Hypocalcemia

Hypoglycemia

Hypokalemia

Hyponatremia

Neurologic Emergencies

Amyotrophic Lateral Sclerosis

Bell's Palsy

Cauda Equina Syndrome

Cavernous Sinus Thrombosis

Cerebral Aneurysm

Cerebral Vascular Accident

Coma
Delirium
Dementia
Diplopia
Dystonic Reaction
Encephalitis
Epidural Abscess
Epidural Hematoma
Giant Cell Arteritis (GCA) (Temporal Arteritis)
Guillain–Barré Syndrome
Headache
Headache, Cluster
Headache, Migraine
Horner Syndrome
Hydrocephalus
Intracerebral Hemorrhage
Multiple Sclerosis
Myasthenia Gravis
Neuroleptic Malignant Syndrome
Oculomotor Nerve Palsy
Parkinson Disease
Periodic Paralysis
Peripheral Neuropathy
Polyneuropathy
Pseudotumor Cerebri
Reperfusion Therapy, Cerebral
Seizure, Adult
Seizure, Febrile
Seizure, Pediatric
Spinal Cord Syndromes
Subarachnoid Hemorrhage
Transient Global Amnesia
Transient Ischemic Attack (TIA)
Trigeminal Neuralgia

Ventricular Peritoneal Shunts
Vertebrobasilar Insufficiency
Weakness

Nontraumatic Musculoskeletal Emergencies

Ankylosing Spondylitis
Arthritis, Degenerative
Arthritis, Juvenile Idiopathic
Arthritis, Monoarticular
Arthritis, Rheumatoid
Arthritis, Septic
Back Pain
Bursitis
Carpal Tunnel Syndrome
Dermatomyositis/Polymyositis
Fibromyalgia
Gout/Pseudogout
Legg–Calvé–Perthes Disease
Osgood–Schlatter Disease
Osteogenesis Imperfecta
Osteomyelitis
Osteoporosis
Paget Disease
Paronychia
Reactive Arthritis
Rhabdomyolysis
Ring/Constricting Band Removal
Sciatica/Herniated Disc
Slipped Capital Femoral Epiphysis
Spondylolysis/Spondylolisthesis
Synovitis, Toxic
Tendonitis
Tenosynovitis

Obstetrical Emergencies

Abortion, Spontaneous
Cardiomyopathy, Peripartum
Cesarean Section, Emergency
Delivery, Uncomplicated
Ectopic Pregnancy
HELLP Syndrome
Hydatidiform Mole
Hyperemesis Gravidarum
Labor
Placental Abruption
Placenta Previa
Postpartum Hemorrhage
Postpartum Infection
Preeclampsia/Eclampsia
Pregnancy, Trauma In
Pregnancy, Uncomplicated
Vaginal Bleeding in Pregnancy

Oncologic Emergencies

Hyperviscosity Syndrome
Leukemia
Multiple Myeloma
Tumor Compression Syndromes

Ophthalmologic Emergencies

Central Retinal Artery Occlusion
Central Retinal Vein Occlusion
Conjunctivitis
Corneal Abrasion
Corneal Burn
Corneal Foreign Body
Dacryocystitis and Dacryoadenitis
Diplopia
Glaucoma
Globe Rupture

Hordeolum and Chalazion

Hyphema

Iritis

Optic Neuritis

Periorbital and Orbital Cellulitis

Red Eye

Retinal Detachment

Retro-Orbital Hematoma

Ultraviolet Keratitis

Uvulitis

Visual Loss

Vitreous Hemorrhage

Orthopedic Injuries

Acromioclavicular Joint Injury

Amputation Traumatic/Replantation

Ankle Fracture/Dislocation

Ankle Sprain

Carpal Fractures

Clavicle Fracture

Elbow Injuries

Epiphyseal Injuries

Femur Fracture

Foot Fracture

Forearm Fracture, Shaft/Distal

Fracture, Open

Hand Infection

Hip Injury

Humerus Fracture

Knee Dislocation

Knee injuries: ACL, PCL, MCL, Meniscus

Lunate Dislocation

Metacarpal Injuries

Nursemaid's Elbow

Patellar Injuries

Pelvic Fracture
Perilunate Dislocation
Phalangeal Injuries, Foot
Phalangeal Injuries, Hand
Reactive Arthritis
Sacral Fracture
Scaphoid Fracture
Shoulder Dislocation
Spine Injury: Cervical, Adult
Spine Injury: Cervical, Pediatric
Spine Injury: Coccyx
Spine Injury: Lumbar
Spine Injury: Thoracic
Sternoclavicular Joint Injury
Thumb Fracture
Tibial Plateau Fracture
Tibial/Fibular Shaft Fracture

Pediatric Emergencies

Abuse, Pediatric (Nonaccidental Trauma [NAT])
Apnea, Pediatric
Asthma, Pediatric
Bacterial Tracheitis
Bronchitis
Croup
Cystic Fibrosis
Diabetes Mellitus, Juvenile
Diaper Rash
Diarrhea, Pediatric
Epiglottitis, Pediatric
Epiphyseal Injuries
Failure to Thrive
Feeding Problems, Pediatric
Fever, Pediatric
Fractures, Pediatric

Henoch–Schönlein Purpura

Hirschsprung Disease

Hydrocele

Inborn Errors of Metabolism

Intussusception

Irritable Infant

Kawasaki Disease

Malrotation

Neonatal Jaundice

Neonatal Sepsis

Nursemaid’s Elbow

Patent Ductus Arteriosus

Pediatric Trauma

Pertussis

Pneumonia, Pediatric

Pyloric Stenosis

Rash, Pediatric

Resuscitation, Neonate

Resuscitation, Pediatric

Reye Syndrome

Seizure, Febrile

Seizure, Pediatric

Spine Injury: Cervical, Pediatric

Staphylococcal Scalded Skin Syndrome

Sudden Infant Death Syndrome (SIDS)

Urinary Tract Infections, Pediatric

Vomiting, Pediatric

Psychobehavioral Emergencies

Abuse, Elder

Abuse, Pediatric (Nonaccidental Trauma [NAT])

Agitation

Bipolar Disorder

Delirium

Depression

Domestic Violence

Dystonic Reaction

Eating Disorder

Hallucinations

Munchausen Syndrome

Panic Attack

Psychiatric Commitment

Psychosis, Acute

Psychosis, Medical vs. Psychiatric

Schizophrenia

Suicide, Risk Evaluation

Violence, Management of

Withdrawal, Alcohol

Withdrawal, Drug

Pulmonary-thoracic Emergencies

Airway Adjuncts

Airway Management

Asthma, Adult

Asthma, Pediatric

Bacterial Tracheitis

Bronchiolitis

Bronchitis

Chronic Obstructive Pulmonary Disease

Cor Pulmonale

Croup

Cystic Fibrosis

Dyspnea

Noncardiogenic Pulmonary Edema

Pleural Effusion

Pneumocystis Pneumonia

Pneumomediastinum

Pneumonia, Adult

Pneumonia, Pediatric

Pneumothorax

Pulmonary Edema
Pulmonary Embolism
Sarcoidosis
Sleep Apnea
Thoracic Outlet Syndrome
Tuberculosis
Ventilator Management

Renal Emergencies

Dialysis Complications
Glomerulonephritis
Hematuria/Proteinuria
Nephritic Syndrome
Nephrotic Syndrome
Pyelonephritis
Renal Calculus
Renal Failure (Acute Kidney Injury)
Rhabdomyolysis

Toxicologic Emergencies

Acetaminophen Poisoning
Alcohol Poisoning
Amphetamine Poisoning
Anticholinergic Poisoning
Antidepressant Poisoning
Arsenic Poisoning
Barbiturates Poisoning
Bath Salts – Synthetic Cathinones Poisoning
Benzodiazepine Poisoning
Beta-Blocker Poisoning
Biologic Weapons
Calcium Channel Blocker Poisoning
Carbamazepine Poisoning
Carbon Monoxide Poisoning
Caustic Ingestion

Chemical Weapons Poisoning
Cocaine Poisoning
Cyanide Poisoning
Digoxin, Poisoning
Disulfiram Reaction
Ethylene Glycol Poisoning
GHB Poisoning
Hallucinogen Poisoning
Hazmat
Hydrocarbon Poisoning
Hypoglycemic Agent Poisoning
Iron Poisoning
Irritant Gas Exposure
Isoniazid Poisoning
Isopropanol Poisoning
Lead Poisoning
Lithium Poisoning
MDMA Poisoning
Mercury Poisoning
Methanol Poisoning
Methemoglobinemia
Monoamine Oxidase Inhibitor Poisoning
Mushroom Poisoning
Neuroleptic Poisoning
Nonsteroidal Anti-inflammatory Poisoning
Opiate Poisoning
Organophosphate Poisoning
Phencyclidine Poisoning
Phenytoin Poisoning
Plant Poisoning
Poisoning
Poisoning, Antidotes
Poisoning, Gastric Decontamination
Poisoning, Toxidromes

Salicylate Poisoning
Serotonin Syndrome (Drug-Induced)
Smoke Inhalation
Snake Envenomation
Spider Bite, Black Widow
Spider Bite, Brown Recluse
Sting, Bee
Sting, Scorpion
Sympathomimetic Poisoning
Theophylline Poisoning
Toluene Poisoning
Tricyclic Antidepressant, Poisoning
Warfarin Complications

Traumatic Injuries

Abdominal Trauma, Blunt
Abdominal Trauma, Imaging
Abdominal Trauma, Penetrating
Abuse, Elder
Abuse, Pediatric (Nonaccidental Trauma [NAT])
Bite, Animal
Bite, Human
Bladder Injury
Blow-Out Fracture
Burns
Chest Trauma, Blunt
Chest Trauma, Penetrating
Colon Trauma
Compartment Syndrome
Dental Trauma
Diaphragmatic Trauma
Domestic Violence
Drowning
Duodenal Trauma
Epidural Hematoma

Esophageal Trauma
Extremity Trauma, Penetrating
Facial Fractures
Flail Chest
Geriatric trauma
Head Trauma, Blunt
Head Trauma, Penetrating
Hemothorax
Hepatic Injury
Intracerebral Hemorrhage
Laceration Management
Larynx Fracture
Lightning Injuries
Myocardial Contusion
Nasal Fractures
Neck Injury by Strangulation/Hanging
Neck Trauma, Blunt, Anterior
Neck Trauma, Penetrating, Anterior
Otologic Trauma
Pancreatic Trauma
Pediatric Trauma
Penile Shaft Fracture
Pneumothorax
Pregnancy, Trauma In
Pulmonary Contusion
Rectal Trauma
Renal Injury
Retro-Orbital Hematoma
Rib Fracture
Small-Bowel Injury
Spinal Cord Syndromes
Splenic Injury
Subarachnoid Hemorrhage
Subdural Hematoma

Taser Injuries

Tendon Laceration

Trauma, Multiple

Urethral Trauma

Wound Ballistics

Vascular Emergencies

Abdominal Aortic Aneurysm

Aortic Dissection, Thoracic

Aortic Rupture, Traumatic (TAI)

Arterial Occlusion

Cavernous Sinus Thrombosis

Cerebral Aneurysm

Deep Vein Thrombosis

Gangrene

Giant Cell Arteritis (GCA) (Temporal Arteritis)

Mesenteric Ischemia

Peripheral Vascular Disease

Venous Insufficiency

Rosen & Barkin's

5-Minute Emergency Medicine Consult

5TH EDITION

Rapid-Sequence Intubation*

1. Pre-oxygenate with 100% oxygen
2. Apply cricoid pressure
3. Induction: etomidate (0.3 mg/kg), propofol (0.5–2 mg/kg) or ketamine (2 mg/kg) IV push
4. Neuromuscular blockade: succinylcholine 1.5 mg/kg IV push
5. Wait 30–45 sec
6. Intubate when optimal conditions achieved

*Consider pretreatment with fentanyl (1–2 µg/kg) IV push (over 1–2 min) and lidocaine (1.5 mg/kg) IV push if concern for increased intracranial pressure or severe hypertension

*Consider defasciculating dose of paralytic if concern for increased intracranial pressure (see table for dosage)

*Atropine: 0.02 mg/kg IV push (for children < 1 y)

Neuromuscular Blocking Agents

Agent	RSI Dosage (paralytic)	Dosage (fas pro*)	Onset	Duration
Succinylcholine	RSI: 1–2 mg/kg		30–60 sec	4–6 min
Rocuronium	RSI: 0.6–1.2 mg/kg	0.06 mg/kg	2 min	30 min
Vecuronium	RSI: 0.015–0.25 mg/kg	0.01 mg/kg	2.5–5 min	25–40 min

*fas pro, fasciculation prophylaxis/defasciculating dose; RSI, rapid-sequence intubation.

Sedative and Induction Agents

Sedative	Dosage IVP	Onset	Duration
Etomidate	0.2–0.3 mg/kg	60 sec	3–5 min
Fentanyl	Induction: 2–10 µg/kg Sedation (titrate): 0.5–1 µg/kg	60 sec	30–60 min
Ketamine	2 mg/kg slow IVP (<0.5 mg/kg/min)	30–60 sec	15 min
Midazolam	Induction: 0.07–0.3 mg/kg Sedation (titrate): 0.02–0.04 mg/kg	2 min	1–2 h
Propofol	0.5–2 mg/kg IVP	30 sec	3–10 min
Thiopental	3–4 mg/kg	20–40 sec	5–10 min

Pediatric Vital Signs and Resuscitation Equipment Sizes

	Term	6 mo	1 yr	2 yr	5 yr	10 yr
Approximate weight	2-4 kg	8 kg	10 kg	13 kg	20 kg	35 kg
Vital signs						
BP (systolic) mm Hg	60 ± 10	89 ± 29	96 ± 30	99 ± 25	99 ± 20	112 ± 19
HR	125	130	125	115	100	75
RR	40 ± 10	38 ± 10	39 ± 11	28 ± 4	27 ± 6	21 ± 4
Resuscitation						
Defibrillation	8 J	16 J	20 J	26 J	40 J	70 J
Cardioversion	2-4 J	4-8 J	5-10 J	7-13 J	20-40 J	25-70 J
Suction catheter	8F	8-10F	8-10F	10F	10F	12F
Airway						
Laryngoscope blade	1 (st)	1-2 (st)	1-2 (st)	2 (st/c)	2 (st/c)	2-3 (st/c)
Endotracheal tube (mm)	3.0-3.5	3.5-4.0	4.0-4.5	4.5	5.0-5.5	6.5
Lip-tip length (mm)	10.5	12	12	13.5	16.5	19.5
Tubes						
Nasogastric tube	5/6	8	10	10	10-12	12
Urinary catheter	5 feeding tube	5-8 feeding tube	8 feeding tube	10 Foley	10 Foley	10 Foley
Chest tube (Fr)	10-12	14-20	16-20	14-24	20-28	28-32

Temperature Conversion: Celsius ↔ Fahrenheit

Celsius	Fahrenheit	Celsius	Fahrenheit
34.2	93.6	38.6	101.4
34.6	94.3	39.0	102.2
35.0	95.0	39.4	102.9
35.4	95.7	39.8	103.6
35.8	96.4	40.2	104.3
36.2	97.1	40.6	105.1
36.6	97.8	41.0	105.8
37.0	98.6	41.4	106.5
37.4	99.3	41.8	107.2
37.8	100.0	42.2	108.0
38.2	100.7	42.6	108.7

$$^{\circ}\text{F} = 9/5 \times ^{\circ}\text{C} + 32$$

Weight Conversion: Pounds ↔ Kilograms

10 lb	4.53 kg	110 lb	49.89 kg
20 lb	9.07 kg	120 lb	54.43 kg
30 lb	13.60 kg	130 lb	58.96 kg
40 lb	18.14 kg	140 lb	63.50 kg
50 lb	22.68 kg	150 lb	68.04 kg
60 lb	27.21 kg	160 lb	72.57 kg
70 lb	31.75 kg	170 lb	77.11 kg
80 lb	36.28 kg	180 lb	81.64 kg
90 lb	40.82 kg	190 lb	86.18 kg
100 lb	45.36 kg	200 lb	90.72 kg

$$\text{kg} = \text{lb} \times 2.2$$

ABDOMINAL AORTIC ANEURYSM

Daniel J. Henning • Jason C. Imperato • Carlo L. Rosen



BASICS

DESCRIPTION

- Focal dilation of the aortic wall with an increase in diameter by at least 50% (> 3 cm).
- 95% are infrarenal.
- Rapid expansion or rupture causes symptoms.
- Rupture can occur into the intraperitoneal or retroperitoneal spaces
- Intraperitoneal rupture is usually immediately fatal
- Average growth rate of 0.2–0.5 cm/yr
- Of ruptures:
 - 90% overall mortality
 - 80% mortality for patients who reach the hospital
 - 50% mortality for patients who undergo emergency repair

Geriatric Considerations

- Risk increases with advanced age.
- Present in:
 - 4–8% of all patients older than 65 yr
 - 5–10% of men 65–79 yr old
 - 12.5% of men 75–84 yr old
 - 5.2% of women 75–84 yr old

ETIOLOGY

- Risk factors:
 - Male gender
 - Age > 65 yr
 - Family history
 - Cigarette smoking
 - Atherosclerosis
 - HTN
 - Diabetes mellitus
 - Connective tissue disorders:
 - Ehlers–Danlos syndrome
 - Marfan syndrome
- Uncommon causes:
 - Blunt abdominal trauma
 - Congenital aneurysm

- Infections of the aorta
- Mycotic aneurysm secondary to endocarditis
- Rupture risk factors:
 - Size (annual rupture rates):
 - Aneurysms 5–5.9 cm = 4%
 - Aneurysms 6–6.9 cm = 7%
 - Aneurysms 6.9–7 cm = 20%
 - Expansion:
 - A small aneurysm that grows > 0.5 cm in 6 mo is at high risk for rupture.
 - Gender:
 - For aneurysms 4.0–5.5 cm, women have 4 × higher risk of rupture compared to men with similar sized aneurysms.

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Abdominal, back, or flank pain:
 - Vague, dull quality
 - Constant, throbbing, or colicky
 - Acute, severe, constant
 - Radiates to chest, thigh, inguinal area, or scrotum
 - Flank pain radiating to the groin in 10% of cases
- Lower extremity pain
- Syncope, near-syncope
- Unruptured are most often asymptomatic

Physical-Exam

- Unruptured:
 - Abdominal mass or fullness
 - Palpable, nontender, pulsatile mass
 - Intact femoral pulses
- Ruptured:
 - Classic triad (only 1/3 of the cases):
 - Pain
 - Hypotension
 - Pulsatile abdominal mass
 - Systemic:
 - Hypotension
 - Tachycardia

- Evidence of systemic embolization
- Abdomen:
 - Pulsatile, tender abdominal mass
 - Flank ecchymosis (Grey Turner sign) indicates retroperitoneal bleed.
 - Only 75% of aneurysms > 5 cm are palpable.
 - Abdominal tenderness
 - Abdominal bruit
 - GI bleeding
- Extremities:
 - Diminished or asymmetric pulses in the lower extremities
- Complications:
 - Large emboli: Acute painful lower extremity
 - Microemboli: Cool, painful, cyanotic toes (“blue toe syndrome”)
 - Aneurysmal thrombosis: Acutely ischemic lower extremity
 - Aortoenteric fistula: GI bleeding

ESSENTIAL WORKUP

- Unstable patients:
 - Bedside abdominal US
 - Explorative surgery without further ancillary studies
- Stable, symptomatic patients:
 - Abdominal CT

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Type and cross-match blood
- CBC
- Creatinine
- Urinalysis
- Coagulation studies

Imaging

- Plain radiographs:
 - Abdominal or lateral lumbar radiographs
 - Only if other tests are unavailable
 - Curvilinear calcification of the aortic wall or a paravertebral soft-tissue mass indicates abdominal aortic aneurysm (AAA) in 75% of patients.
 - Cannot identify rupture
 - Negative study does not rule out AAA.
- Abdominal ultrasound:
 - 100% sensitive and 92–99% specific for detecting AAA prior to rupture
 - In emergent setting, useful to determine presence of AAA.

- Ultrasound findings consistent with AAA are enlarged aorta >3 cm or focal dilatation of the aorta.
- Sensitivity has been reported as low as 10% following rupture.
- Indicated in the unstable patient
- Abdominal CT scan:
 - Contrast is not necessary to make the diagnosis but CT angiogram is required for surgical planning for an endovascular approach
 - Will demonstrate both aneurysm and site of rupture (intraperitoneal vs. retroperitoneal)
 - Allows more accurate measurement of aortic diameter

DIFFERENTIAL DIAGNOSIS

- Other abdominal arterial aneurysms (i.e., iliac or renal)
- Aortic dissection
- Renal colic
- Biliary colic
- Musculoskeletal back pain
- Pancreatitis
- Cholecystitis
- Appendicitis
- Bowel obstruction
- Perforated viscus
- Mesenteric ischemia
- Diverticulitis
- GI hemorrhage
- Aortic thromboembolism
- Myocardial infarction
- Addisonian crisis
- Sepsis
- Spinal cord compression



TREATMENT

PRE HOSPITAL

- Establish 2 large-bore IV lines
- Rapid transport to the nearest facility with surgical backup
- Alert ED staff as soon as possible to prepare the following:
 - Operating room
 - Universal donor blood
 - Surgical consultation

INITIAL STABILIZATION/THERAPY

- 2 large-bore IV lines
- Crystalloid infusion
- Cardiac monitor
- Early blood transfusion

ED TREATMENT/PROCEDURES

For patients suspected of symptomatic AAA:

- Avoid over aggressive fluid resuscitation; this leads to increased bleeding
- Emergent surgical consult and operative intervention
- Laparotomy versus endovascular aortic repair (EVAR) by vascular surgeon
- Diagnostic tests should not delay definitive treatment.



FOLLOW-UP

DISPOSITION

Admission Criteria

All patients with symptomatic AAA require emergent surgical intervention and admission.

Discharge Criteria

Asymptomatic patients only

FOLLOW-UP RECOMMENDATIONS

- Close vascular surgery follow-up must be arranged prior to discharge
- Instructions to return immediately for:
 - Any pain in the back, abdomen, flank, or lower extremities
 - Any dizziness or syncope

PEARLS AND PITFALLS

- AAA should be on the differential for any patient presenting with pain in the abdomen, back, or flank.
- Symptomatic AAA requires immediate treatment. Do not delay definitive care for extra studies.
- A hemodynamically unstable (i.e., hypotensive) patient should not be taken for CT scan.

ADDITIONAL READING

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- Tibbles C, Barkin A. The aorta. In: Cosby K, Kendall J. *Practical Guide to Emergency Ultrasound*. Philadelphia, PA: Lippincott Williams & Wilkins; 2006:219–236.



See Also (Topic, Algorithm, Electronic Media Element)

- Aortic Dissection
- Peripheral Artery Disease



CODES

ICD9

ICD9

- 441.3 Abdominal aneurysm, ruptured
- 441.4 Abdominal aneurysm without mention of rupture

ICD10

- I71.3 Abdominal aortic aneurysm, ruptured
- I71.4 Abdominal aortic aneurysm, without rupture

ABDOMINAL PAIN

Saleh Fares



BASICS

DESCRIPTION

- **Parietal pain:**
 - Irritating material causing peritoneal inflammation
 - Pain transmitted by somatic nerves
 - Exacerbated by changes in tension of the peritoneum
 - Pain is sharp, well localized with abdominal, rebound tenderness and involuntary guarding
- **Visceral pain:**
 - Afferent impulses result in poorly localized pain based on the embryologic origin rather than true location of an organ.
 - Pain of foregut structures to the epigastric area
 - Pain from midgut structures to the periumbilical area
 - Pain from hindgut structures to the suprapubic region
 - Distention of a viscous or organ capsule or spasm of intestinal muscularis fibers
 - Pain is constant and colicky
 - **Inflammation:**
 - Focal tenderness develops once the inflammation extends to the peritoneum
 - **Ischemia from vascular emergencies:**
 - Pain is severe and diffuse
- **Referred pain:**
 - Felt at distant location from diseased organ
 - Due to an overlapping supply by the affected neurosegment
- **Abdominal wall pain:**
 - Constant, aching with muscle spasm
 - Involvement of other muscle groups

ETIOLOGY

- **Peritoneal irritants:**
 - Gastric juice, fecal material, pus, blood, bile, pancreatic enzymes
- **Visceral obstruction:**
 - Small and large intestines, gallbladder, ureters and kidneys, visceral ischemia, intestinal, renal, splenic
- **Visceral inflammation:**
 - Appendicitis, inflammatory bowel disorders, cholecystitis, hepatitis, peptic

ulcer disease, pancreatitis, pelvic inflammatory disease, pyelonephritis

- Abdominal wall pain
- Referred pain: (e.g., intrathoracic disease)



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Pain
 - Nature of onset of pain
 - Time of onset and duration of pain
 - Location of pain initially and at presentation
 - Extra-abdominal radiations
 - Quality of pain (sharp, dull, crampy)
 - Aggravating or alleviating factors
 - Relation of associated finding to pain onset
- Anorexia
- Nausea
- Vomiting (bilious, coffee-ground emesis)
- Malaise
- Fainting or syncope
- Cough, dyspnea, or respiratory symptoms
- Change in stool characteristics (e.g., melena)
- Hematuria
- Changes in bowel or urinary habits
- History of trauma or visceral obstruction
- Gynecologic and obstetric history
- Postoperative (e.g., cause ileus)
- Family history (e.g., familial aortic aneurysm)
- Alcohol use and quantity
- Medications: (e.g., aspirin and NSAIDs)

Physical-Exam

- General:
 - Anorexia
 - Tachycardia
 - Tachypnea
 - Hypotension
 - Fever
 - Yellow sclera (icterus)
 - Distal pulses and pulse amplitudes between lower and upper extremities

- Abdominal:
 - Distended abdomen
 - Abnormal bowel sounds:
 - High-pitched rushes with bowel obstruction
 - Absence of sound with ileus or peritonitis
 - Pulsatile abdominal mass
 - Rebound tenderness, guarding, and cough test for peritoneal irritation (e.g., appendicitis, peritonitis)
 - Rovsing sign, suggestive of appendicitis:
 - Palpation of left lower quadrant causes pain in right lower quadrant (RLQ).
 - Psoas sign suggests appendicitis (on right)
 - Pain on extension of thigh
 - Obturator sign suggests pelvic appendicitis (on the right only)
 - Pain on rotation of the flexed thigh, especially internal rotation
 - McBurney point tenderness associated with appendicitis:
 - Palpation in RLQ 2/3 distance between umbilicus and right anterior superior iliac crest causes pain.
 - Murphy sign, suggestive of cholecystitis:
 - Pause in inspiration while examiner is palpating under liver
 - Carnett sign indicates abdominal wall pain
 - Pain when a supine patient tenses the abdominal wall by lifting the head and shoulders.
 - Tender or discolored hernia site
 - Rectal and pelvic examination:
 - Tenderness with pelvic peritoneal irritation
 - Cervical motion tenderness
 - Adnexal masses
 - Rectal mass or tenderness
 - Guaiac positive stool
- Genitourinary:
 - Flank pain
 - Dysuria
 - Costovertebral angle tenderness
 - Suprapubic tenderness
 - Tender adnexal mass on pelvis
 - Testicular pain:
 - May be referred from renal or appendiceal pathology
- Referred pain:
 - Kehr sign (diaphragmatic irritation due to blood or other irritants) causes shoulder pain.
- Extremities:
 - Pulse deficit or unequal femoral pulses

- Skin:
 - Jaundice
 - Liver disease (caput medusa)
 - Hemorrhage
 - Grey Turner sign of flank ecchymosis
 - Cullen sign is ecchymotic area round the umbilicus
 - Herpes zoster
 - Cellulitis
 - Rash (Henoch–Schönlein purpura [HSP])

ESSENTIAL WORKUP

- For a woman in reproductive age group a pregnancy test is essential
- Where applicable for majority of cases, ultrasonography should be done with CT used in cases of negative or inconclusive ultrasonography.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC
- Serum electrolytes, creatinine, and glucose
- ESR
- LFTs
- Lactic acid
- Serum lipase:
 - More sensitive and specific than amylase
- Urinalysis
- Stool analysis and culture
- Pregnancy testing (age reproductive women)

Imaging

- EKG:
 - Consider if risk factors for coronary artery disease are present
- Abdominal radiograph: Supine and upright
 - CT is superior for suspected visceral perforation and bowel obstruction.
- Upright CXR:
 - Pneumoperitoneum
 - Intrathoracic disease causing referred abdominal pain
- US:
 - Biliary abnormalities
 - Hydronephrosis
 - Intra-peritoneal fluid
 - Aortic aneurysm
 - Intussusception

- US (Doppler ultrasonography)
 - Volvulus and malrotation
 - Testicular and ovarian torsion
 - Hepatitis, cirrhosis, and portal vein thrombosis
- Abdominal CT:
 - Spiral CT without contrast:
 - Renal Colic
 - Retroperitoneal hemorrhage
 - Appendicitis
 - CT with intravenous contrast only:
 - Vascular rupture suspected in a stable patient (e.g., acute abdominal aortic aneurysm [AAA], aortic dissection)
 - Ischemic bowel
 - Pancreatitis
 - CT with IV and oral contrast:
 - Indicated when there is a suspicion of a surgical etiology involving bowel
 - History of inflammatory bowel disease
 - Thin patients (low BMI)
 - Diverticulitis
 - CT angiography:
 - Mesenteric ischemia
 - AAA
- IVP:
 - CT has replaced the use of intravenous urography in detection of ureteral stones
- Barium enema:
 - Intussusception
 - Treatment and confirmation of intussusception is with air contrast enema.
- MRI:
 - If concerns for radiation exposure or nephrotoxicity
 - Contraindicated in patients with metallic implants

Pregnancy Considerations

Ultrasonography and MRI should be preferred to prevent exposure of ionizing radiation to the fetus.

DIFFERENTIAL DIAGNOSIS

- AAA
- Abdominal epilepsy or abdominal migraine
- Boerhaave syndrome
- Adrenal crisis
- Early appendicitis

- Bowel obstruction
- Cholecystitis
- Constipation +/- fecal impaction
- Diabetic ketoacidosis
- Diverticulitis
- Dysmenorrhea
- Ectopic pregnancy
- Esophagitis
- Endometriosis
- Fitz-Hugh–Curtis syndrome
- Gastroenteritis
- Hepatitis
- Incarcerated hernia
- Infectious gastroenteritis
- Inflammatory bowel disease
- Irritable bowel syndrome
- Ischemic bowel
- Meckel diverticulitis
- Neoplasm
- Ovarian torsion
- Ovarian cysts (hemorrhagic)
- Pancreatitis
- Pelvic inflammatory disease
- Peptic ulcer disease
- Renal/ureteral calculi
- Renal Infarction
- Sickle cell crisis
- Spider bite (Black widow)
- Splenic infarction
- Spontaneous abortion
- Testicular torsion
- Tubo-ovarian abscess
- UTI
- Volvulus
- Referred pain:
 - Myocardial infarction
 - Pneumonia
- Abdominal wall pain:
 - Abdominal wall hematoma or infection
 - Black widow spider bite
 - Herpes zoster

Pediatric Considerations

- Under 2 yr:
 - Hirschsprung disease
 - Incarcerated hernia
 - Intussusception
 - Volvulus
 - Foreign body ingestion
- 2–5 yr:
 - Appendicitis
 - Incarcerated hernia
 - Meckel diverticulitis
 - Sick cell crisis
 - HSP
 - Constipation



TREATMENT

ED TREATMENT/PROCEDURES

- Nasogastric tube decompression and bowel rest
- IV fluids and electrolyte repletion
- Antiemetics are important for comfort.
- Narcotics or analgesics should not be withheld.
- Send for blood type and cross-match for unstable patient
- Surgical consultation based on suspected etiology

MEDICATION

- Fentanyl: 1–2 $\mu\text{g}/\text{kg}$ IV qh
- Morphine sulfate: 0.1 mg/kg IV q4h PRN
- Ondansetron: 4 mg IV
- Prochlorperazine: 0.13 mg/kg IV/PO/IM q6h PRN nausea; 25 mg PR q6h in adults
- Promethazine: 25–50 mg/kg IM/PO/PR



FOLLOW-UP

DISPOSITION

Admission Criteria

- Surgical intervention
- Peritoneal signs
- Patient unable to keep down fluids
- Lack of pain control
- Medical cause necessitating in-house treatment (MI, DKA)
- IV antibiotics needed

Discharge Criteria

No surgical or severe medical etiology found in patient who is able to keep fluid down, has good pain control, and is able to follow detailed discharge instructions

FOLLOW-UP RECOMMENDATIONS

The patient should return with any warning signs:

- Vomiting
- Blood or dark/black material in vomit or stools
- Yellow skin or in the whites of the eyes
- No improvement or worsening of pain within 8–12 hr
- Shaking chills, or a fever > 100.4°F (38°C)

PEARLS AND PITFALLS

- Elderly patients are more likely to present with atypical presentations and life threatening etiologies requiring admission.
- Do not consider constipation if stool is absent in the rectal vault.
- Etiology requiring surgical intervention is less likely when vomiting precedes the onset of pain.

ADDITIONAL READING

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- Yeh EL, McNamara RM. Abdominal pain. *Clin Geriatr Med*. 2007;23(2):255–270.



CODES

ICD9

- 789.00 Abdominal pain, unspecified site
- 789.06 Abdominal pain, epigastric
- 789.07 Abdominal pain, generalized

ICD10

- R10.9 Unspecified abdominal pain
- R10.13 Epigastric pain
- R10.84 Generalized abdominal pain

ABDOMINAL TRAUMA, BLUNT

Stewart R. Coffman



BASICS

DESCRIPTION

- Injury results from a sudden increase of pressure to abdomen.
- Solid organ injury usually manifests as hemorrhage.
- Hollow viscus injuries result in bleeding and peritonitis from contamination with bowel contents.

ETIOLOGY

- 60% result from motor vehicle collisions.
- Solid organs are injured more frequently than hollow viscus organs.
- The spleen is the most frequently injured organ (25%), followed by the liver (15%), intestines (15%), retroperitoneal structures (13%), and kidney (12%).
- Less frequently injured are the mesentery, pancreas, diaphragm, urinary bladder, urethra, and vascular structures.

Pediatric Considerations

- Children tend to tolerate trauma better because of the more elastic nature of their tissues.
- Owing to the smaller size of the intrathoracic abdomen, the spleen and liver are more exposed to injury because they lie partially outside the bony rib cage.



DIAGNOSIS

SIGNS AND SYMPTOMS

- Spectrum from abdominal pain, signs of peritoneal irritation to hypovolemic shock
- Nausea or vomiting
- Labored respiration from diaphragm irritation or upper abdominal injury
- Left shoulder pain with inspiration (Kehr sign) from diaphragmatic irritation owing to bleeding
- Delayed presentation possible with small-bowel injury

ESSENTIAL WORKUP

- Evaluate and stabilize airway, breathing, and circulation (ABCs).
- Primary objective is to determine need for operative intervention.
- Examine abdomen to detect signs of intra-abdominal bleeding or peritoneal irritation.
- Injury in the retroperitoneal space or intrathoracic abdomen is difficult to assess

by palpation.

- Remember that the limits of the abdomen include the diaphragm superiorly (nipples anteriorly, inferior scapular tip posteriorly) and the intragluteal fold inferiorly and encompass entire circumference.
- Abrasions or ecchymoses may be indicators of intra-abdominal injury:
 - Roll the patient to assess the back.
 - Lap-belt abrasions can be indicative of significant intra-abdominal injuries.
- Bowel sounds may be absent from peritoneal irritation (late finding).
- Foley catheter (if no blood at the meatus, no perineal hematoma, and normal prostate exam) to obtain urine and record urinary output
- Plain film of the pelvis:
 - Fracture of the pelvis and gross hematuria may indicate genitourinary injury.
 - Further evaluation of these structures with retrograde urethrogram, cystogram, or IV pyelogram
- CT most useful in assessing need for operative intervention and for evaluating the retroperitoneal space and solid organs:
 - Patient must be stable enough to make trip to scanner.
 - Also useful for suspected renal injury
- Focused abdominal sonography for trauma (FAST) to detect intraperitoneal fluid:
 - US is rapid, requires no contrast agents, and is noninvasive.
 - Operator dependent
- Diagnostic peritoneal lavage (useful for revealing injuries in the intrathoracic abdomen, pelvic abdomen, and true abdomen) primarily indicated for unstable patients:
 - Positive with gross blood, RBC count of $>100,000/\text{mm}^3$, WBC count of $500/\text{mm}^3$, or presence of bile, feces, or food particles

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Hemoglobin/hematocrit, which initially may be normal owing to isovolemic blood loss
- Type and screen is essential. Cross-match PRBC units for unstable patients.
- Urinalysis for blood:
 - Microscopic hematuria in the presence of shock is an indication for genitourinary evaluation.
- ABG:
 - Base deficit may suggest hypovolemic shock and help guide the resuscitation.

Imaging

See “Essential Workup.”

Diagnostic Procedures/Surgery

See “Essential Workup”

DIFFERENTIAL DIAGNOSIS

Lower thoracic injury may cause abdominal pain.



TREATMENT

PRE HOSPITAL

- Titrate fluid resuscitation to clinical response. Target SBP of 90–100 mm Hg
- Normal vital signs do not preclude significant intra-abdominal pathology.

INITIAL STABILIZATION/THERAPY

- Ensure adequate airway:
 - Intubate if needed.
 - O₂ 100% by nonrebreather face mask
- 2 large-bore IV lines with crystalloid infusion
- Begin infusion of PRBCs if no response to 2 L of crystalloid.
- If patient is in profound shock, consider immediate transfusion of O-negative blood.

ED TREATMENT/PROCEDURES

- Continue stabilization begun in field.
- Nasogastric tube to evacuate stomach, decrease distention, and decrease risk of aspiration:
 - May relieve respiratory distress if caused by a herniated stomach through the diaphragm

MEDICATION

- Tetanus toxoid booster: 0.5 mL IM for patients with open wounds
- Tetanus immunoglobulin: 250 U IM for patients who have not had complete series
- IV antibiotics: Broad-spectrum aerobic with anaerobic coverage such as a 2nd-generation cephalosporin

Pediatric Considerations

- Crystalloid infusion is 20 mL/kg if patient is in shock.
- PRBC dose is 1 mL/kg.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Postoperative cases
- Equivocal findings on diagnostic peritoneal lavage, FAST exam, or CT
- Many blunt abdominal trauma patients benefit from admission, monitoring, and serial abdominal exams.

Discharge Criteria

No patient in whom you suspect intra-abdominal injury should be discharged home without an appropriate period of observation, despite negative exam or imaging studies.

PEARLS AND PITFALLS

- Do not delay blood products when patient is in obvious shock despite normal Hct.
- Avoid overaggressive resuscitation with crystalloids.
- Obtain a pregnancy test in all females of childbearing age.
- Do not transport unstable patients to CT for diagnostic imaging.

ADDITIONAL READING

- Amoroso TA. Evaluation of the patient with blunt abdominal trauma: An evidence based approach. *Emerg Med Clin North Am.* 1999;17:63–75.
- Holmes JF, Offerman SR, Chang CH, et al. Performance of helical computed tomography without oral contrast for the detection of gastrointestinal injuries. *Ann Emerg Med.* 2004;43(1):120–128.
- Kendall JL, Faragher J, Hewitt GJ, et al. Emergency department ultrasound is not a sensitive detector of solid organ injury. *West J Emerg Med.* 2009;10(1):1–5.
- Stengel D, Bauwens K, Sehouli J, et al. Systematic review and meta-analysis of emergency ultrasonography for blunt abdominal trauma. *Br J Surg.* 2001;88:901–912.



CODES

ICD9

- 459.0 Hemorrhage, unspecified
- 865.00 Injury to spleen without mention of open wound into cavity, unspecified injury
- 868.00 Injury to other intra-abdominal organs without mention of open wound into cavity, unspecified intra-abdominal organ

ICD10

- R58 Hemorrhage, not elsewhere classified
- S36.00XA Unspecified injury of spleen, initial encounter
- S36.90XA Unspecified injury of unspecified intra-abdominal organ, initial encounter

ABDOMINAL TRAUMA, IMAGING

Alfred A. Joshua



BASICS

DESCRIPTION

Diagnostic procedures: Use of these imaging and procedure modalities will be based on history and physical exam.



DIAGNOSIS

SIGNS AND SYMPTOMS

- Abdominal trauma can be seen in a variety of patients ranging from those with isolated abdominal injury to multisystem trauma.
- Abdominal trauma is divided into blunt and penetrating injuries. Penetrating abdominal injuries can further be divided into stab wounds and gunshot wounds.
- Hemodynamic status should be the primary initial focus of evaluation. Most unstable patients will require early surgical management, while many stable patients with abdominal trauma may be managed nonoperatively.

History

- History should include mechanism of injury, restraint use and type, airbag or helmet use, prehospital vital signs, initial mental status, and change in mental status.
- AMPLE history (*allergies-to-medications and radiographic contrast agents, medications taken, past medical and surgical history, last meal, events leading up to the injury*)

Physical-Exam

- A comprehensive physical exam should start with ABCDE survey and include full exposure of the patient and careful palpation of all abdominal quadrants.
- Abdominal injury in only 45–50% of cases.
- The abdominal physical exam is frequently misleading in intoxicated, uncooperative, and multisystem trauma patients.

ESSENTIAL WORKUP

- See “Abdominal Trauma (Blunt)” and “Abdominal Trauma (Penetrating).”
- All trauma patients initially managed with:
 - ABCDE survey (Airway, Breathing, Circulation, Disability, Exposure)

DIAGNOSIS TESTS & NTERPRETATION

General approach to imaging in abdominal trauma:

- Unstable trauma patients:
 - Unstable patients should have a bedside ultrasound performed immediately as part of the primary survey (circulation). A positive FAST suggests that intra-abdominal bleeding is the source of hypotension. A negative FAST suggests either a retroperitoneal bleed, blood loss in the field, bleeding from an unstable pelvic fracture, or hemorrhage into another body cavity.
 - A surgeon should be consulted immediately to prepare for definitive operative care of the patient.
 - Stable trauma patients: The 3 main modes of diagnosing the extent of injury in hemodynamically stable abdominal trauma patients include:
 - US: Initial screening test of choice for hemodynamically stable patients. A positive US in the stable trauma patient warns the clinician about the possibility of impending hemodynamic deterioration. CT scan and surgical consult should be rapidly facilitated in this group of patients.
 - CT scan: The definitive test for stable abdominal trauma patients. CT scanning will diagnose both solid organ and retroperitoneal injuries better than ultrasound. CT imaging allows a determination of whether an embolization procedure is warranted for hemorrhage control. It is indicated in all stable patients with stab wounds. It is also indicated in patients with gross hematuria, to look for renal injury.
 - Diagnostic peritoneal lavage: Currently used infrequently.
- Local wound exploration: While frequently used in the past in penetrating abdominal trauma to look for violation of the fascia, it has now also been replaced with CT scanning in the majority of patients (see Diagnostic Tests & Interpretation).

Lab

- Blood type and screen
- CBC
- Electrolytes and creatinine
- Lipase
- UA
- EKG

Imaging

- Ultrasound: FAST exam focuses on dependent intraperitoneal areas where blood can accumulate which include: Hepatorenal space (Morison pouch), splenorenal space, suprapubic region (bladder and pouch of Douglas), pericardium
 - Advantages:
 - Rapid

- Noninvasive
- Can be performed at the patient's bedside concurrent with evaluation and initial resuscitation
- Does not require contrast agents or ionizing radiation
- Can be repeated in the case of changes in the patient's hemodynamic status or physical exam

– Disadvantages:

- Operator dependent
- Does not reliably identify solid organ (e.g., spleen and liver lacerations) or retroperitoneal injuries. May be negative with pelvic fractures despite significant hemorrhage. Not sensitive for bowel injury.

– Contraindications:

- Absolute: None
- Relative: Obesity; subcutaneous emphysema

– Positive test:

- Demonstration of free fluid or obvious solid organ injury. ~ 600 mL free fluid required in adults for a positive right upper quadrant Morison pouch view. ~ 150 mL is required for a positive pelvic/suprapubic view (optimally performed prior to Foley placement).
- Adequate exam includes visualization of the right upper quadrant, left upper quadrant, suprapubic/pelvis, and cardiac areas.

● CT scan:

– Advantages:

- Sensitivity of 85–98%, PPV (for detecting need for laparotomy) of 85%
- Provides specific and detailed organ injury information
- May aid in a nonoperative approach to solid organ injuries, which may be managed with observation or interventional radiology mediated embolectomy.
- Allows imaging of adjacent spinal structures to diagnose fracture.

– Disadvantages:

- Costly
- Possible risk: Up to 1 in 2,000 increase in risk of fatal cancer from radiation
- Requires IV contrast (with risk of acute contrast reactions and renal toxicity).
- Isolated diaphragmatic, pancreatic, bowel injuries may be missed, especially if performed immediately after injury.

– Indications:

- Hemodynamically stable patients

– Contraindications:

- Absolute: Pre-existing indication for exploratory laparotomy; hemodynamic instability; previous contrast reaction
- Considerations:
 - Many institutions now manage multisystem trauma patients with the “pan-scan,” which includes CT imaging of the head, C-spine, chest, and abdomen/pelvis in 1 session.
 - IV contrast is sufficient in the abdominal trauma patient. Oral and rectal contrast is not needed.
 - Angiography
 - Unstable patients and pelvic fractures
 - This approach can embolize vessels from pelvis, spleen, etc.

Diagnostic Procedures/Surgery

- Diagnostic peritoneal lavage:
 - Advantages:
 - Rapid
 - Helpful in detecting mesenteric and hollow organ injuries
 - May be considered in patients with pelvic fractures and hemorrhage
 - Relatively simple to perform
 - Sensitivity 87–92%, specificity 82%, PPV 52%, NPV 87%
 - Low complication rate
 - Disadvantages:
 - Invasive
 - Largely replaced by bedside US
 - Does not identify specific organ injury
 - 1–2% complication rate
 - May miss retroperitoneal injuries and intraperitoneal bladder rupture
 - High false-positive rates
- Possible indications:
 - Hemodynamically unstable patients
 - Patients requiring emergent surgery for other conditions (e.g., craniotomy for epidural hematoma)
 - Pelvic fractures
- Contraindications:
 - Absolute: Pre-existing indication for exploratory laparotomy
 - Relative: Previous abdominal surgery, severe abdominal distention, pregnancy, pediatric patients
- Considerations:
 - Foley catheter and nasogastric tube placement is recommended before beginning the procedure.
- Contraindications:
 - Blood at urethra
 - High riding prostate

- Positive test:
 - Aspiration of > 10 mL of blood, bile, bowel contents, or urine
 - Diagnostic peritoneal lavage fluid in the urine or chest tube
 - Blunt trauma with > 100,000 erythrocytes/mm³
 - Penetrating trauma > 1,000 erythrocytes/mm³

DIFFERENTIAL DIAGNOSIS

See “Abdominal Trauma (Blunt)” and “Abdominal Trauma (Penetrating).”



TREATMENT

PRE HOSPITAL

All patients with a significant mechanism of injury or suspicion of major trauma should be triaged to a designated trauma center (preferably a Level 1 Center)

Pediatric Considerations

- Pediatric patients should be triaged to a pediatric trauma center or to an adult trauma center equipped to manage children.
- CT scan should be considered the diagnostic test of choice in children.

INITIAL STABILIZATION/THERAPY

- In unstable patients, management of the airway, breathing, and circulation with resuscitation of hypovolemic shock and rapid control of major hemorrhage must take precedence.
- See “Abdominal Trauma (Blunt)” and “Abdominal Trauma (Penetrating).”

ED TREATMENT/PROCEDURES

- See “Abdominal Trauma (Blunt)” and “Abdominal Trauma (Penetrating).”
- Crystalloid IV therapy is generally warranted with significant abdominal injury.
- 2 large-bore IV catheters should be placed.
- Blood transfusion is indicated for all hemodynamically unstable abdominal trauma patients. O negative or O positive blood can be used in men/women beyond childbearing age.
- Hemodynamically unstable trauma patients with altered mental status and inability to protect airway will usually need endotracheal intubation prior to transfer to operating suite.



FOLLOW-UP

DISPOSITION

Admission Criteria

- All unstable trauma patients require admission to the hospital and most will require surgical management.
- Most multisystem trauma patients who also have abdominal trauma will need admission.
- Pregnant women > 24 wk gestation should be admitted for fetal–maternal monitoring.
- Stable trauma patients are divided into 3 classes:
 - Gun shot wounds to abdomen: Almost all will require admission. Rate of surgical exploration is high in this category due to elevated risk of organ injury.
 - Stab wounds to abdomen: Patients with penetration of fascia will require admission. US, CT, or physical exam will define patients who need operative management.
 - Blunt abdominal trauma: US, CT, or exam will define patients who need admission.

Discharge Criteria

- Patients with stable hemodynamics during their ED course with a negative evaluation and reliable follow-up may be considered for discharge.
- Patients with inability to travel back to the hospital or to contact EMS for aid in case of deterioration must be considered for admission.

FOLLOW-UP RECOMMENDATIONS

A small subset of discharged patients may have an undiagnosed injury (most commonly intestinal or pancreatic). Patients must be instructed to return to the ED with worsening abdominal pain, distention, vomiting, or rectal bleeding.

PEARLS AND PITFALLS

- US can be immediately performed at the bedside concurrent with initial stabilization.
- Consider serial US exams. This is especially important if there is a change in the patient's hemodynamic status or physical exam.
- Many stable adult and pediatric trauma patients are now being managed nonoperatively based on CT findings.
- “Pan CT scan” decreases missed injury rate but increases lifetime risk of cancer.
- With increased use of US and CT, DPL and local wound exploration have become less useful in the evaluation of abdominal trauma.
- Pitfalls include:
 - Not immediately sending type and screen or checking a pregnancy test
 - Sending pregnant women > 24 wk gestation home without fetal–maternal monitoring

ADDITIONAL READING

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- Rose JS. Ultrasound in abdominal trauma. *Emerg Med Clin North Am*. 2004;22(3):581–599.



See Also (Topic, Algorithm, Electronic Media Element)

- Abdominal Trauma, Blunt
- Abdominal Trauma, Penetrating



CODES

ICD9

- 88.02 Other abdomen tomography
- 88.19 Other x-ray of abdomen
- 88.76 Diagnostic ultrasound of abdomen and retroperitoneum

ICD10

- BW00ZZZ Plain Radiography of Abdomen
- BW40ZZZ Ultrasonography of Abdomen
- S39.91XA Unspecified injury of abdomen, initial encounter

ABDOMINAL TRAUMA, PENETRATING

Stewart R. Coffman • Stephen R. Hayden



BASICS

DESCRIPTION

- Solid organ injury usually results in hemorrhage.
- Hollow viscus injury can lead to spillage of bowel contents and peritonitis.
- Associated conditions:
 - Injury to both thoracic and abdominal structures occurs in 25% of cases.

ETIOLOGY

80% of gunshot wounds and 20–30% of stab wounds result in significant intra-abdominal injury. Commonly injured structures include:

- Liver (37%)
- Small bowel (26%)
- Stomach (19%)
- Colon (17%)
- Major vessel (13%)
- Retroperitoneum (10%)
- Mesentery/omentum (10%)
- Other:
 - Spleen (7%)
 - Diaphragm (5%)
 - Kidney (5%)
 - Pancreas (4%)
 - Duodenum (2%)
 - Biliary (1%)



DIAGNOSIS

SIGNS AND SYMPTOMS

- Penetrating wound from knife, gun, or other foreign object
- Spectrum of presentation ranging from localized pain to peritoneal signs:
 - High-velocity projectile can cause extensive direct tissue damage.
 - Secondary missiles and temporary cavitation of effected structures
 - Exit wound may be larger than entrance wound, but small entrance and exit wounds can conceal massive internal damage.
- Remember the borders of the abdomen: Superior from the nipples (anteriorly) or inferior tip of scapula (posteriorly) to inferior gluteal folds.

ESSENTIAL WORKUP

- Diagnosis of intra-abdominal injury from gunshot wounds to the abdomen are made by laparotomy in the operating room.
- Locally explore stab wounds to anterior abdomen:
 - If the wound penetrates the anterior fascial layer, the patient should undergo diagnostic peritoneal lavage or bedside US.
- Diagnostic laparoscopy is useful in diagnosing diaphragmatic injury and spleen and liver lacerations:
 - May help avoid unnecessary surgery.
- CT is useful in the evaluation of patients with a suspected retroperitoneal injury:
 - Not reliable for detection of hollow viscus or diaphragmatic injuries
- If 10,000 RBC/mm³ or more are found in the diagnostic peritoneal lavage fluid, the patient should undergo laparotomy.
- If <10,000 RBC/mm³ are present, the patient should be observed for 8–24 hr for the development of peritoneal signs.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Hemoglobin or hematocrit:
 - Repeated measurements to assess for ongoing hemorrhage
- Urinalysis for blood to assess for possible genitourinary tract damage
- ABG:
 - Base deficit may be helpful in assessing hypovolemia and guide volume resuscitation.
- Type and cross-match for all patients with significant intra-abdominal injuries.

Imaging

- Plain films:
 - Obtain after placement of markers for localization of foreign bodies, missiles, associated fractures, and free air.
- IV pyelogram:
 - For possible renal injury
- Bedside abdominal US (FAST: Focused abdominal sonography for trauma):
 - May reveal intraperitoneal blood or fluid
- CT with IV contrast in experienced facilities and with stable patients:
 - For possible retroperitoneal and solid organ injuries

DIFFERENTIAL DIAGNOSIS

- In cases of upper abdominal wounds, consider the possibility of intrathoracic injury.
- In cases of wounds to the lower thoracic area, consider the possibility of intra-

abdominal injury.



TREATMENT

PRE HOSPITAL

- Controversies:
 - Military antishock trousers (MAST) should not be used.
 - Titrate fluid resuscitation to clinical response.
- Caution:
 - Apply sterile dressings to open wounds and moistened sterile dressings to eviscerated bowel.
 - Secure impaled foreign objects in place; do not remove them.

INITIAL STABILIZATION/THERAPY

- 2 large-bore IV lines with crystalloid infusion
- If no response to 2 L of crystalloid, infuse 2–4 units packed red blood cells:
 - May use O negative blood initially if patient is unstable
 - Type-specific and cross-matched blood when it becomes available
- 100% oxygen by nonrebreather face mask

Pediatric Considerations

- Children in hypovolemic shock should receive 20 mL/kg boluses of crystalloid.
- Children in severe hypovolemic shock should receive 1 mL/kg of packed red blood cells.
- Age < 8 yr is a relative contraindication for diagnostic peritoneal lavage.

ED TREATMENT/PROCEDURES

- Nasogastric tube placement:
 - Will decrease aspiration risk
 - Place nasogastric tube before performing diagnostic peritoneal lavage to decompress stomach and reduce risk of iatrogenic injury.
 - May relieve respiratory distress in cases of diaphragmatic injury with herniated abdominal contents in the thorax
- Foley catheter placement:
 - Insert after ruling out urethral injuries
 - Facilitates rapid assessment of genitourinary injury
 - Assists in monitoring of urinary output
- Tetanus if appropriate; tetanus immunoglobulin if primary tetanus series not administered

MEDICATION

- Tetanus: 0.5 mL IM

- Tetanus immunoglobulin: 250 units IM for patients who have not had a complete series
- IV antibiotics: Antibiotics with coverage against gram-negative and anaerobic organisms:
 - Ampicillin/sulbactam:
 - Adults: 3 g q6h IV (peds: 50 mg/kg IV)
 - Cefotetan:
 - Adults: 2 g q12h IV (peds: 40 mg/kg IV)
 - Cefoxitin:
 - Adults: 2 g q6h IV (peds: 80 mg/kg q6h IV)
 - Piperacillin/tazobactam:
 - Adults: 3.375 g IV (peds: 75 mg/kg IV)
 - Ticarcillin/clavulanate:
 - Adults: 3.1 g IV (peds: 75 mg/kg IV)
- Additional anaerobic coverage:
 - Clindamycin:
 - Adults: 600–900 mg IV (peds: 10 mg/kg IV)
 - Metronidazole:
 - Adults: 1 g IV (peds: 15 mg/kg IV)
- Combination therapy:
 - Adults: Ampicillin 500 mg IV q6h, gentamicin 1–1.7 mg/kg IV, and metronidazole 1 g IV
 - Peds: Ampicillin 50 mg/kg IV q6h, gentamicin 1–1.7 mg/kg IV, and metronidazole 15 mg/kg IV



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients requiring abdominal surgery
- Observe the following patients for at least 8 hr:
 - Patients with negative findings on diagnostic peritoneal lavage, CT, or US. During hospitalization, the following are necessary:
 - Frequent abdominal exam
 - Repeated hematocrit levels at regular intervals

Discharge Criteria

Patients with stab wounds without fascial penetration may be discharged after observation in the ED.

PEARLS AND PITFALLS

Permissive hypotension is gaining support as a resuscitative principle:

- Avoid normal or near normal BP.
- Avoid overaggressive resuscitation with crystalloids.
- Completely exposing the patient will minimize overlooking an injury.
- Spinal immobilization is unnecessary unless there is an obvious spinal cord injury.

ADDITIONAL READING

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- Kirkpatrick AW, Sirois M, Ball CG, et al. The hand-held ultrasound examination for penetrating abdominal trauma. *Am J Surg.* 2004;187:660–665.
- Oyo-Ita A, Ugare UG, Ikpeme IA. Surgical versus non-surgical management of abdominal injury. *Cochrane Database Syst Rev.* 2012;14(11):CD007383.
- Sebesta J. Special lessons learned from Iraq. *Surg Clin North Am.* 2006;86(3):711–726.



CODES

ICD9

868.10 Injury to other intra-abdominal organs with open wound into cavity, unspecified intra-abdominal organ

ICD10

- S31.609A Unsp opn wnd abd wall, unsp quadrant w penet perit cav, init
- S31.639A Pnctr w/o fb of abd wall, unsp Quadrant w penet perit cav, init

ABORTION, SPONTANEOUS

Ivette Motola • Aviva Jacoby Zigman



BASICS

DESCRIPTION

- Spontaneous termination of a < 20 wk intrauterine pregnancy
- Synonyms: Early pregnancy loss, miscarriage
- Occurs in up to 15–20% of recognized pregnancies (most common complication of early pregnancy)
- Vaginal bleeding in the 1st trimester seen in about 25% of pregnant patients:
 - 50% of these women will eventually mis-carry
- Definitions:
 - *Threatened abortion*: Vaginal bleeding, cervical os is closed, viable intrauterine pregnancy confirmed:
 - 50% of women seen in the ED for threatened abortion will eventually miscarry
 - *Inevitable abortion*: Vaginal bleeding, cervical os is open; products of conception (POC) have not been expelled
 - *Incomplete abortion*: Vaginal bleeding, cervical os is open with partial passage of some POC and some retained POC
 - *Complete abortion*: Vaginal bleeding, cervical os closed, complete passage of POC; no surgical or medical intervention
 - *Missed abortion*: Fetal demise with no uterine activity to expel
 - *Septic abortion*: Spontaneous abortion complicated by intrauterine infection
 - *Recurrent spontaneous abortion*: 3 or more consecutive pregnancy losses

ETIOLOGY

- Chromosomal abnormalities of the fetus
- Uterine abnormalities
- Risk factors include:
 - Increased age of both the mother and father
 - Increased parity
 - Alcohol use
 - Cigarette smoking
 - Cocaine use
 - Conception within 3–6 mo after delivery
 - Chronic maternal disease:
 - Poorly controlled diabetes
 - Autoimmune disease
 - Celiac disease

- Intrauterine device
- Maternal BMI < 18 or > 25 kg/m²
- Maternal infections:
 - Bacterial vaginosis
 - Mycoplasmosis
 - Herpes simplex
 - Toxoplasmosis
 - Listeriosis
 - Chlamydia/gonorrhea
 - HIV
 - Syphilis
 - Parvovirus B19
 - Malaria
 - CMV
 - Rubella
- Medications:
 - Misoprostol
 - Methotrexate
 - NSAIDs
- Multiple previous elective abortions
- Previous early pregnancy loss
- Toxins
- Uterine abnormalities (e.g., leiomyoma, uterine adhesions, congenital anomalies)

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Last menstrual period (LMP)
- Obstetric history:
 - Parity
 - Risk factors for pregnancy loss
 - Prenatal care
- Abdominal pain, cramping
- Vaginal bleeding:
 - Duration
 - Amount of bleeding (quantify by number of pads used, compare with normal menstrual period for patient)
 - Passage of clots
- Dizzy, syncope

Physical-Exam

- Determine hemodynamic status of patient:
 - Pregnant patients in late 1st trimester have an increased blood volume
 - Can lose substantial amount of blood before having abnormal vital signs
- Pelvic exam:
 - Determine whether the internal cervical os is opened or closed
 - Amount of bleeding
 - Presence of POC
 - Presence of adnexal tenderness or peritoneal irritation can be consistent with an ectopic pregnancy
- Bimanual exam to determine the size of the uterus:
 - Size of an orange: 6–8 wk
 - Fundus at the symphysis pubis: 12 wk
 - Fundus at the umbilicus: 16–20 wk

ESSENTIAL WORKUP

- Pregnancy test as below
- Imaging as below

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Confirm pregnancy with a urine or serum test:
 - Urine pregnancy test: Most are positive at β -hCG levels of 25–50 mIU/mL ~ 1 wk gestational age and remain positive 2–3 wk after induced or spontaneous abortions
- CBC
- Rapid hemoglobin determination: Type and Rh
- Type and cross-match for woman with low Hct or signs of active blood loss
- Quantitative β -hCG
- Any POC passed should be sent to pathology for confirmation

Imaging

- Transvaginal ultrasound (TVS):
 - Gestational sac seen at 5 wk
 - Cardiac activity seen at 6.5 wk
- Transabdominal ultrasound (TAS):
 - Gestational sac at 6 wk
 - Cardiac activity seen at 8 wk
- Discriminatory zone: Level of β -hCG where a normal IUP should be detected:
 - 1,500–2,000 for TVS
 - 6,500 for TAS

DIFFERENTIAL DIAGNOSIS

- Positive pregnancy test with vaginal bleeding:
 - Cervicitis
 - Ectopic pregnancy
 - Molar pregnancy
 - Pregnancy of unknown location (PUL)
 - Septic abortions
 - Subchorionic hemorrhage
 - Trauma
- 2nd- and 3rd-trimester vaginal bleeding:
 - Placenta previa
 - Placental abruption



TREATMENT

PRE HOSPITAL

- IV fluids, oxygen, and cardiac monitor
- Monitor vital signs and transport
- Cautions:
 - Patients with spontaneous abortion/vaginal bleeding can have severe hemorrhage and present in shock, especially at > 12 wk
 - BP drops during the 2nd trimester of pregnancy with an average of 110/70

INITIAL STABILIZATION/THERAPY

- Stable patients:
 - IV
 - Pelvic exam
- Unstable patients:
 - Oxygen, IV fluids via 2 large-bore IVs, cardiac monitor
 - Transfuse PRBC if patient does not stabilize after 2–3 L of crystalloid
 - Gynecologic consultation immediately
 - Oxytocin or methylergonovine may be necessary to control hemorrhage
 - These patients are at high risk for having ruptured ectopic pregnancies and may need emergent operative intervention

ED TREATMENT/PROCEDURES

- Threatened abortion:
 - Pelvic rest, close follow-up with obstetrics
 - Patients < 6.5 wk pregnant with no documented cardiac activity by vaginal US need to be followed with serial β -hCG to assess the viability of the fetus and to rule out ectopic pregnancy
- Inevitable and incomplete abortions:

- Expectant management:
 - o Successful in up to 85%
 - o Increased risk of unplanned surgical intervention and blood loss as compared to surgical management
- Medical management:
 - o Misoprostol
 - o Successful in up to 85%
- Surgical management:
 - o Dilation and curettage (D&C) or evacuation, removal of POC at the cervical os to help decrease bleeding and cramping
 - o Less unplanned hospital admissions, curettages, and blood transfusions
 - o The confirmation of POC by pathology rules out ectopic pregnancy
- Complete abortion:
 - May treat with methylergonovine or oxytocin if bleeding is heavy
 - If quantitative β -hCG is $< 1,000$ and the US is negative, may follow-up with obstetrics for serial β -hCG to confirm the levels are decreasing
- Missed abortion:
 - These patients are at risk for disseminated intravascular coagulation (DIC), especially if fetus is retained $> 4-6$ wk
 - Obtain CBC, PT/PTT, fibrin-split products (FSP), and fibrinogen levels
 - These patients may be followed closely as outpatients if stable with an early, confirmed IUP and no evidence of DIC
 - Patients may choose to have a D&C at a later date or miscarry at home with medication or no intervention; this decision should be made in consultation with OB/GYN

MEDICATION

First Line

- RHO immunoglobulin in Rh-negative women:
 - 50 μ g for women with threatened or complete abortion at < 12 wk
 - 300 μ g for women with threatened or complete abortion at ≥ 12 wk
- Patients need RhoGAM administration within 72 hr to prevent future isoimmunization
- Misoprostol 800 μ g vaginally if medical management is chosen in consultation with OB/GYN
- Repeat dose required in 48 hr

Second Line

Usually given in consultation with OB/GYN:

- Oxytocin: 20 IU in 1,000 mL of NS at a rate of 20 mIU/min titrated to decrease bleeding; may repeat for a max. dose of 40 mIU/min

- Methylergonovine: 0.2 mg IM/PO QID for bleeding



FOLLOW-UP

DISPOSITION

Admission Criteria

- Suspected unstable ectopic pregnancy (see “Ectopic Pregnancy”)
- Hemodynamically unstable patients with hypovolemia or anemia
- DIC
- Septic abortions
- Suspected gestational trophoblastic disease

Discharge Criteria

- D&Cs can be done in the ED for incomplete and inevitable abortions, and patients may be discharged home if stable after 2–3 hr
- Some early inevitable miscarriages can be discharged to complete their miscarriages at home without a D&C
- Discharge with pain medications and close OB/GYN follow-up
- Patients with threatened abortions should be told to avoid strenuous activity
- Pelvic rest (i.e., “nothing in the vagina” during active bleeding; may increase risk of infection)
- Patients should be instructed to return to the ED for any increase in bleeding, dizziness, or temperature $> 100.4^{\circ}\text{F}$
- Patients and their partners should be counseled that early pregnancy loss is common and that it is not anyone’s fault

FOLLOW-UP RECOMMENDATIONS

Patients with positive pregnancy tests and vaginal bleeding with or without abdominal pain should be followed by OB/GYN.

PEARLS AND PITFALLS

- Recognize the possibility of ectopic pregnancy
- Patients with spontaneous abortion may have clinically significant blood loss

ADDITIONAL READING

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- Prine LW, MacNaughton H. Office management of early pregnancy loss. *Am Fam Physician*. 2011;84:75–82.



See Also (Topic, Algorithm, Electronic Media Element)

- Ectopic Pregnancy
- Vaginal Bleeding



CODES

ICD9

- 634.90 Spontaneous abortion, without mention of complication, unspecified
- 634.91 Spontaneous abortion, without mention of complication, incomplete
- 634.92 Spontaneous abortion, without mention of complication, complete

ICD10

- O02.1 Missed abortion
- O03.4 Incomplete spontaneous abortion without complication
- O03.9 Complete or unspecified spontaneous abortion without complication

ABSCESS, SKIN/SOFT TISSUE

Neal P. O'Connor



BASICS

DESCRIPTION

- A localized collection of pus surrounded and walled off by inflamed tissue. Abscesses can occur on any part of the body
- Furuncle:
 - Arises from infected hair follicle
 - Most common on back, axilla, and lower extremities
- Carbuncle:
 - Larger and more extensive than furuncle
- Dog/cat bite:
 - Usually polymicrobial
- Breast:
 - Puerperal:
 - Usually during lactation
 - Located in peripheral wedge
 - Usually staphylococci
 - Duct ectasia:
 - Caused by ectatic ducts
 - Periareolar location
 - Usually polymicrobial
- Hidradenitis suppurativa:
 - Chronic abscess of apocrine sweat glands
 - Groin and scalp
 - *Staphylococcus aureus* and *staphylococcus viridans* are common
 - *Escherichia coli* and *Proteus* may be present in chronic disease
- Pilonidal abscess:
 - Epithelial disruption of gluteal fold over coccyx
 - Staphylococcal species are most common
 - May be polymicrobial
- Bartholin abscess:
 - Obstruction of Bartholin duct
- Perirectal abscess:
 - Originates in anal crypts and extends through ischiorectal space
 - Inflammatory bowel disease and diabetes are predisposing factors
 - *Bacteroides fragilis* and *E. coli* are most common
 - Requires operative drainage
- Muscle (pyomyositis):

- Typically in the tropics
- *S. aureus* is most common
- IV drug abuse:
 - Staphylococcal species are most common
 - MRSA is common
 - May be sterile
- Paronychia:
 - Infection around nail fold
 - Usually *S. aureus*
- Felon:
 - Closed space abscess in distal pulp of finger
 - Usually *S. aureus*

ETIOLOGY

- Abscess formation typically occurs due to a break in the skin, obstruction of sebaceous or sweat glands, or inflammation of hair follicles. The collection may be classified as bacterial or sterile:
- Bacterial: Most abscesses are bacterial with the microbiology reflective of the microflora of the involved body part:
 - *S. aureus* is the most common causative organism
 - Community-acquired MRSA (CA-MRSA) common
- Sterile: More associated with IV drug abuse and injection of chemical irritants
- Risk factors for abscess formation:
 - Immunosuppression
 - Soft tissue trauma
 - Mammalian/human bites
 - Tissue ischemia
 - IV drug use
 - Chron's disease (perirectal)



DIAGNOSIS

SIGNS AND SYMPTOMS

- Local:
 - Erythema
 - Tenderness
 - Heat
 - Swelling
 - Fluctuance
 - May have surrounding cellulitis
 - Regional lymphadenopathy and lymphangitis may occur
- Systemic:
 - Often absent

- Patients with extensive soft tissue involvement, necrotizing fasciitis, or underlying bacteremia may present with signs of sepsis including:
 - Fever
 - Rigors
 - Hypotension
 - Altered mentation

History

- Previous episodes: Raise concern for CA-MRSA
- Immunosuppression
- Medications:
 - Chronic steroids, chemotherapy
- IVDU
- History of mammalian bite

Physical-Exam

- Location and extent of infection
- Presence of:
 - Associated cellulitis
 - Subcutaneous air
 - Deep structure involvement
- Involvement of specialty area:
 - Perirectal
 - Hand
 - Face/neck

ESSENTIAL WORKUP

- History and physical exam
- Gram stain unnecessary for simple abscesses in healthy patients
- Wound cultures:
 - Not indicated in simple abscesses
 - May help guide therapy if systemic treatment is planned
 - May be useful in confirming CA-MRSA in patients with recurrent abscesses
 - May guide specific therapy in a compromised host, abscesses of the central face or hand, and treatment failures

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Routine laboratory tests are not typically indicated.
- Glucose determination may be useful if:
 - Underlying undiagnosed diabetes is a concern
 - There is a concern for associated DKA

- For febrile patients who appear septic, systemically ill, or have recent IVDU the following labs are indicated:
 - Blood cultures
 - Lactate
 - Renal function
 - CK if myositis suspected

Imaging

- Bedside US can be helpful in distinguishing cellulitis from abscess
- CT/MRI can be helpful in determining deep tissue involvement
- Plain films may reveal gas in tissue planes

DIFFERENTIAL DIAGNOSIS

- Cellulitis
- Necrotizing fasciitis
- Aneurysm (especially with IV drug abusers)
- Cysts
- Hematoma



TREATMENT

PRE HOSPITAL

Caution: Septic patients may require rapid transport with IV access and volume resuscitation.

INITIAL STABILIZATION/THERAPY

Septic patient:

- Immediate IV access
- Oxygen
- Crystalloid volume resuscitation
- Blood cultures/lactate
- Early antibiotic therapy—broad spectrum to include MRSA coverage.
- Rapid source control (abscess drainage)
- If patient remains hypotensive after volume resuscitation consider:
 - Central venous pressure monitoring
 - Mixed venous sampling

ED TREATMENT/PROCEDURES

- Incision and drainage are the mainstays of treatment.
 - Incision should be deep enough to allow adequate drainage
 - Elliptical incision prevent early closure
 - Break loculations with gentle exploration

- Irrigate cavity after expressing all pus
- Loose packing of abscess cavity when:
 - Larger than 5 cm
 - Comorbid medical conditions
 - HIV
 - Diabetes
 - Malignancy
 - Chronic steroid use
 - Immunosuppressed
 - Abscess location: face, neck, scalp, hands/feet, perianal, perirectal, genital
 - Promote drainage and prevent premature closure
- For simple cutaneous abscesses (<5 cm) packing may not be routinely indicated.
- Routine antibiotics are not indicated.
- Antibiotics are indicated for the following conditions:
 - Sepsis/systemic illness
 - Facial abscesses drained into the cavernous sinus
 - Concurrent cellulitis (see “Medication”)
 - Mammalian bites
 - Immunocompromised hosts
- Perirectal abscess requires treatment in the operating room
- Hand infections that may require surgical intervention:
 - Deep abscesses
 - Fight bite abscesses
 - Associated tenosynovitis/deep fascial plane infection
- Loop drainage technique:
 - Less invasive
 - Simplifies wound care
 - Procedure:
 - Anesthetize locally
 - Incision made at outer margin of abscess
 - Use a hemostat to break loculations and manually express pus
 - Use hemostat to localize distal margin of abscess and use as guide for a second incision
 - Grasp silicone vessel loop with hemostat and pull through and then gently tie
 - Patient should move loop daily to promote drainage
 - No repeat ED visits generally required
 - Removal in 7–10 days is painless

Pediatric Considerations

Incision and drainage are painful procedures that often require procedural sedation and analgesia.

MEDICATION

ALERT

- Know your local susceptibility patterns
- Oral antibiotics (moderate associated cellulitis):
 - _ Amoxicillin/clavulanate:
 - Use: Mammalian bites/MSSA/Streptococcus species
 - Adult dose: 500–875 mg (peds: 40–80 mg/kg/d div q12h) PO q12h
 - _ TMP-SMX:
 - Use: MRSA
 - Adult dose: 160/800 mg (peds: 4–5 mg/kg) PO BID
 - _ Clindamycin:
 - Use: MRSA
 - Adult dose: 300–450 mg (peds: 4–8 mg/kg) PO q6h
 - _ Doxycycline:
 - Use: MRSA
 - Adult dose: 100 mg (peds: over 8 yr: 1.1 mg/kg) PO q12h
 - _ Cephalexin:
 - Use: MSSA/Strep species
 - Adult dose: 250 mg PO q6h or 500 mg PO q12h (peds: 25–50 mg/kg/d div q12h)
 - _ Erythromycin:
 - Use: MSSA/Streptococcus species
 - Adult dose: 250–500 mg (peds: 10 mg/kg) PO q6–8h
- IV antibiotics (systemic illness or extensive associated cellulitis):
 - _ Ampicillin/sulbactam
 - Uses: Human/mammalian bites and facial cellulitis
 - Adult dose: 1.5–3 g (peds: < 40 kg, 75 mg/kg; ≥ 40 kg, adult dose) IV q6h (max = 12 g/d)
 - _ Vancomycin:
 - Use: MRSA
 - Adult dose: 15 mg/kg IV q12h (peds: 10–15 mg/kg/d div q6–8 h) (max. = 2,000 mg/d)
 - _ Daptomycin:
 - Use MRSA
 - Adult dose: 4 mg/kg IV q24h
 - _ Linezolid:
 - Use: MRSA
 - Adult dose: 600 mg IV/PO q12h (peds: 30 mg/kg/d div q8h)
 - _ Clindamycin:
 - Use: MRSA
 - Adult dose: 600 mg (peds: 10–15 mg/kg) IV q8h



FOLLOW-UP

DISPOSITION

In accordance with abscess type and severity of infection

Admission Criteria

- Sepsis/systemic illness
- Immunocompromised host with moderate/large cellulitis
- Perirectal involvement
- Any abscess requiring incision and debridement in the operating room

Discharge Criteria

Most patients with uncomplicated abscesses can be treated with incision and drainage and close follow-up.

FOLLOW-UP RECOMMENDATIONS

- Recheck in 24–48 hr for packing removal and wound check.
- Warm soaks for 2–3 days after packing removal

PEARLS AND PITFALLS

- Consider CA-MRSA in recurrent abscesses
- Pain control is essential during incision and drainage of abscesses
- Beware of tenosynovitis and deep fascial space infections

ADDITIONAL READING

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- Tsoraides SS, Pearl RH, Stanfill AB, et al. Incision and loop drainage: A minimally invasive technique for subcutaneous abscess management in children. *J Pediatr Surg*. 2012;45:606–609.



See Also (Topic, Algorithm, Electronic Media Element)

- Bartholin Abscess
- Bite, Animal
- Cellulitis
- CA-MRSA
- Hand Infection
- Mastitis
- Paronychia



CODES

ICD9

- 566 Abscess of anal and rectal regions
- 682.9 Cellulitis and abscess of unspecified sites
- 685.0 Pilonidal cyst with abscess

ICD10

- K61.0 Anal abscess
- L02.91 Cutaneous abscess, unspecified
- L05.01 Pilonidal cyst with abscess

ABUSE, ELDER

Helen Straus

BASICS

DESCRIPTION

Elder abuse may include the following:

- Emotional abuse:
 - Insults
 - Humiliation
 - Threats to institutionalize or abandon
- Physical and/or sexual abuse:
 - Hitting
 - Slapping
 - Pushing
 - Burning
 - Inappropriate restraining
 - Forced sexual activity
- Material exploitation:
 - Stealing or coercion involving patient money or property
- Neglect:
 - Behaviors by a patient or caregiver that compromise the patient's health or safety
 - Failure to provide adequate food, shelter, hygiene, and/or medical attention

EPIDEMIOLOGY

Incidence and Prevalence Estimates

- In the US., 1–2 million cases age 65 or older mistreated by someone on whom they depend (these numbers likely will increase in the near term as US age demographics shift):
 - 55% neglect
 - 14.6% physical mistreatment
 - 12.3% financial exploitation
 - 7.7% emotional mistreatment
 - 0.3% sexual abuse
 - 6.1% all other types
 - 4% unknown
- Family members, including partners and adult children, are perpetrators in approximately 90% of cases
- For every case of financial exploitation reported, 25 cases likely unreported

- Elder abuse (even modest abuse) is associated with a 300% greater risk of death as well as increased rates of additional health problems such as chronic pain, bone/joint, digestive or psychological disorders (compared to the non-abused)

ETIOLOGY

- Caregiver stress, dependency, or psychopathology
- Victim dependency or diminishment of ability to perform activities of daily living



DIAGNOSIS

SIGNS AND SYMPTOMS

Variable, possibly inconsistent, history or physical findings

History

- Not willing or able to obtain adequate food/clothing/shelter
- Not providing for personal hygiene/safety
- Delay in obtaining medical care/previously untreated medical condition
- Vague (or implausible/inappropriate) explanations
- Disparities between histories given by patient and caregiver
- Caregiver who insists on giving the patient's history
- Medication difficulties:
 - Incorrect doses
 - Lost medications
 - Unfilled prescriptions
- Altered interpersonal interactions:
 - Withdrawn
 - Indifferent
 - Demoralized
 - Fearful
 - Substance abuse
- Caregiver with:
 - Financial dependence on patient
 - Substance abuse or psychiatric or violence history
 - Controlling behavior (may refuse to leave elder alone with physician) or poor knowledge
 - Significant life stressors
 - Relationship issues
 - Financial difficulties
 - Legal problems

Physical-Exam

- Inconsistent findings:

- Patterns or variable-age bruises, burns, lacerations/abrasions
- Unusual sites of bruising (inner arm, torso, buttocks, scalp)
- Poor hygiene (inadequate care of skin, nails, teeth)
- Unexplained injuries:
 - Bruised or bleeding genital or rectal area
 - Wrist or ankle lesions suggestive of restraint use
- Findings that may be consistent with neglect or delay in seeking/obtaining medical attention:
 - Dehydration
 - Weight loss
 - Decubitus ulcer
 - Malnutrition

DIAGNOSIS TESTS & INTERPRETATION

Perform any exam and lab or radiographic studies as indicated by the patient's condition.

ESSENTIAL WORKUP

- Obtain history without family members/caregivers present:
 - Abused elders may fear institutionalization if they report caregivers.
 - Many may feel embarrassment and responsibility for abuse.
 - Frequently will not volunteer information
 - Ask patient specifically about abuse or neglect (in private)
- Patient's medical condition may influence quality of history obtained
- Obtain history from caregivers/other relatives/friends/neighbors
- Document a clear and detailed description of findings including the following:
 - Statements of the patient as they pertain to the abuse
 - Psychosocial history:
 - Family and other social relationships
 - Caregiver burdens/coping mechanisms
 - Drug/ethanol (Etoh) use
 - Prior adult protective services reports
 - Skin and other physical findings:
 - Photographic documentation
 - Safety assessment

DIAGNOSIS TESTS & INTERPRETATION

As appropriate for medical condition(s)

Imaging

As appropriate for medical condition(s)

Diagnostic Procedures/Surgery

As appropriate for medical condition(s)

DIFFERENTIAL DIAGNOSIS

- Patient may present with any chief complaint:
 - Potential differential diagnosis is nonspecific.
 - Abuse best identified by asking patient directly in a setting apart from caregivers/family and correlating with risk factors and provider findings
- Differentiate findings consistent with other disease entities from abuse/neglect:
 - Dehydration
 - Ill-fitting dentures
 - Burns
 - Ecchymosis
 - Insomnia
 - Medication noncompliance
 - Dementia
 - Depression



TREATMENT

PRE HOSPITAL

Observe details of the patient's environment that may not be immediately available to the hospital care team, including the following:

- Interpersonal interactions at the scene:
 - Embarrassment
 - Shame
 - Fear of reprisal, abandonment, and/or institutionalization
- Conditions in the physical environment that present a potential danger

INITIAL STABILIZATION/THERAPY

- ABCs
- Treat life-threatening medical/traumatic conditions as appropriate.

ED TREATMENT/PROCEDURES

- May require separation of the patient and the caregiver or family member
- Social work referral:
 - Safety planning
 - Respite planning for caregiver
 - Adult protective services referral
- Competent elder patients are free to accept or decline treatment or disposition despite risks they may incur.
- General measures appropriate to the medical/traumatic conditions identified, including:
 - Fluids

- Medications
- Surgery
- Diet
- Activity
- Nursing care
- Physical therapy



FOLLOW-UP

DISPOSITION

Admission Criteria

Disposition determined by medical condition and home environment:

- Medical condition requiring admission
- Abuse or neglect renders home conditions unsafe.
- Need for more information or time to enhance objective decision making and patient management

Discharge Criteria

- Medical condition(s) addressed
- Safe environment available
- Abuse or neglect successfully countered by social services and/or law enforcement

Issues for Referral

- Many states have mandatory reporting requirements:
 - Comply with area legal requirements.
- Alcohol/drug treatment as appropriate
- Notify adult protective services.

FOLLOW-UP RECOMMENDATIONS

As appropriate for medical condition(s)

PEARLS AND PITFALLS

- Entertaining the possibility of abuse or neglect in an elder patient offers the best possibility of diagnosis and successful intervention.
- Only ~ 1/3 of healthcare providers identified a case of elder abuse in the past year.
- Current data are inconclusive about the effectiveness of interventions for diminishing recurrence of elder abuse.
- Obtain the aid of social worker, physicians trusted by the patient, even an ethics consultant, should a vulnerable competent elder seek to decline an elder

abuse/neglect investigation.

ADDITIONAL READING

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- Lachs MS, Pillemer K. Elder abuse. *Lancet.* 2004;364:1263–1272.
- Laumann EO, Leitsch SA, Waite LJ. Elder mistreatment in the United States; prevalence estimates from a nationally representative study. *J Gerontol B Psychol Sci Soc Sci.* 2008;63(4):S248–S254.
- Ploeg J, Fear J, Hutchison B, et al. A systematic review of interventions for elder abuse. *J Elder Abuse Negl.* 2009;21(3):187–210.



CODES

ICD9

- 995.80 Adult maltreatment, unspecified
- 995.81 Adult physical abuse
- 995.82 Adult emotional/psychological abuse

ICD10

- T74.11XA Adult physical abuse, confirmed, initial encounter
- T74.31XA Adult psychological abuse, confirmed, initial encounter
- T74.91XA Unspecified adult maltreatment, confirmed, initial encounter

ABUSE, PEDIATRIC (NONACCIDENTAL TRAUMA [NAT])

Suzanne Z. Barkin

BASICS

DESCRIPTION

- Child abuse impacts up to 14 million or 2–3% of US children each year.
- 1,200–1,400 children die of maltreatment each year in the US. Of these, 80% < 5 yr and 40% < 1 yr.
- Mandated reporters of suspected abuse or neglect include all health care workers.
- Risk factors:
 - Child: Usually < 4 yr, often handicapped, retarded, or special needs (“vulnerable child”), premature birth, or multiple birth
 - Abusive parent: Low self-esteem, abused as child, violent temper, mental illness history, rigid and unrealistic expectations of child, or young maternal age
 - Family: Monetary problems, isolated and mobile, or marital instability
 - Poor parent–child relationship, unwanted pregnancy
 - Abuse crosses all religious and socioeconomic groups

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- History and mechanism inconsistent with the injury or illness:
 - Unexplained death, apnea, and injury
 - Unexplained ingestion or toxin exposure
 - Recurrent injury
 - Parent/caregiver reluctant to give information or denies knowledge of how injury occurred
 - Begin with open-ended questions about injury and mechanism
 - Discrepancy or inconsistencies among different caregivers
 - Developmentally, child unable to experience mechanism
 - Inappropriate response of care provider to injury or illness; delay in seeking care
 - If alleged anogenital/sexual abuse, history credible
- Munchausen by proxy:
 - Recurrent illness without medical explanation
 - Unexplained metabolic disorder suspicious for poisoning
- Failure to thrive:

- Inadequate caloric intake secondary to poor maternal bonding/neglect
- Review of past ED encounters and contact with the patient's primary care physician may be helpful.

Physical-Exam

- Injury not consistent with history
- Cutaneous bruising/contusions:
 - Regular pattern, straight line of demarcation, regular angles, slap marks from fingers, dunking burns (stocking or glove burns or doughnut shaped on buttock), bites, strap, buckle, cigarette burns
 - Location: Buttocks, hips, face (not forehead), arms, back, thighs, genitalia, or pinna
 - Aging:
 - Often different ages of bruises
 - Yellow bruises are older than 18 hr
 - Red, blue and purple, or black color may occur from 1 hr after injury to resolution
 - Red may be present irrespective of age
 - Bruises of identical age and cause on the same person may appear to be different.
- Skeletal trauma:
 - Usually multiple, unexplained, various stages of healing
 - Metaphyseal or corner (classic metaphyseal lesions) fractures (pathognomonic)
 - Skull fractures that cross suture lines
 - Posterior rib fractures (rib fractures almost never occur in infants from CPR)
 - Spiral fractures of long bones
 - Subperiosteal new bone formation
 - Uncommon fractures (vertebrae, sternum, scapula, spinous process) without significant mechanism
- CNS:
 - Altered mental status or seizure
 - Head trauma is leading cause of death in child abuse.
 - Skull fracture: Must consider child abuse in children <1 yr
 - Subdural hematoma, subarachnoid hemorrhage
 - Shaken baby syndrome with shearing and rotational injury
- Ocular findings:
 - Retinal hemorrhage or detachment:
 - 53–80% of abusive head injury has retinal hemorrhage (commonly bilateral) while present in only 0–10% severe accidental trauma
 - Rare in the absence of evidence of head trauma and normal neuroimaging
 - Hyphema

- Corneal abrasion/conjunctival hemorrhage
- Oral trauma
- Abdominal injuries:
 - Lacerated liver, spleen, kidney, or pancreas
 - Intramural hematoma (duodenal most common)
 - Retroperitoneal hematoma
- Anogenital/sexual abuse:
 - Contusion, erythema, open wounds, scarring, or foreign material (hair, debris, or semen)
 - Presence of STD or pregnancy in child <12 yr
- Death:
 - Unexplained death

ESSENTIAL WORKUP

- Formal oral and written report to appropriate child welfare agency
- Family and environmental evaluation, usually in cooperation with responsible child welfare agency
- Diagram or photograph of bruises is helpful.

ALERT

When suspected, health professionals have a legal obligation to report their suspicion to the appropriate authorities.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Bleeding screen if there is a history of recurrent bruising or bruising is the prominent manifestation; may usually be done electively: CBC, platelets, PT/PTT, or bleeding time (or PFA collagen epinephrine)
- If significant blunt trauma, CBC, LFT, amylase, and urinalysis
- Toxicology, chemistry, and metabolic screens in children with altered mental status
- Consider other differential considerations.

Imaging

- Global assessment:
 - Indicated for children <2 yr to exclude unsuspected injuries
 - In children 2–5 yr, in selected cases where physical abuse is strongly suspected
 - In older children, radiographs of individual sites of injury suspected on clinical grounds
 - Radiographic skeletal survey:
 - Anteroposterior (AP) and lateral skull
 - Lateral cervical spine

- AP and lateral thoracic and lumbar spine
- AP and obliques of chest
- AP pelvis
- AP humerus, forearm, and hands (bilateral)
- AP femur, tibia, and feet (bilateral)
- If fracture identified, get at least 2 views, 90° to original view.
- May need coned-down view of joints for visualization of classic metaphyseal lesions
- Skeletal scintigraphy provides adjunctive screening if suspicion exists beyond skeletal survey.
- Visceral imaging:
 - Suspected thoracoabdominal injury:
 - Abdominal CT scan with IV and possibly oral contrast
- Neuroimaging:
 - Nonenhanced head CT with brain, subdural, and bone windowing
 - MRI:
 - Adjunctive in evaluation of acute, subacute, and chronic intracranial injury; useful for shear injuries, evolving hemorrhage, contusion, or secondary hypoxic/ischemic injury

DIFFERENTIAL DIAGNOSIS

- General:
 - Trauma—accidental or birth/obstetrical
- Cutaneous:
 - Burn—accidental
 - Infection
 - Impetigo/cellulitis
 - Staphylococcal scalded skin syndrome
 - Henoch–Schönlein purpura
 - Purpura fulminans/meningococemia
 - Sepsis
 - Dermatitis: Contact or photo
 - Hematologic/oncologic disorder (idiopathic thrombocytopenic purpura [ITP], leukemia)
 - Bleeding diathesis (hemophilia, von Willebrand)
 - Nutritional deficiency: Scurvy
 - Cultural healing practices (coining, cupping)
- Skeletal:
 - Osteogenesis imperfecta
 - Nutritional (rickets, copper deficiency, or scurvy)
 - Menkes syndrome
 - Peripheral sensory impairment (indifference to pain)
- Ocular:

- Conjunctivitis
- Abdomen and GU tract:
 - GI disease (obstruction, peritonitis, or inflammatory bowel disease)
 - GU tract infection/anomaly
- CNS:
 - Intoxication, ingestion (CO, lead, or mercury)
- Infection:
 - Metabolic: Hypoglycemia
 - Epilepsy
- Death:
 - SIDS, apparent life-threatening event (ALTE)



TREATMENT

PRE HOSPITAL

- Diagnosis relies on physical evidence in child and inconsistency with the history and mechanism.
- Examination of the scene may be useful:
 - Evaluate validity of mechanisms
 - General appearance of home
 - Consistency of history by multiple caregivers
 - Evaluation of parent–child interaction

INITIAL STABILIZATION/THERAPY

As indicated by specific injury

ED TREATMENT/PROCEDURES

- Medical and trauma management as required
- Mandatory reporting to local child welfare agency of any suspected child abuse to determine appropriate social disposition:
 - This does *not* imply or require 100% certainty of abuse.
 - Expedited family, environmental, and social evaluation
 - Essential to be nonjudgmental
- Communication with family about report and primary concern is responsibility of child welfare.
 - Security may be required to protect child and staff.
- Siblings and other household children must be examined in appropriate time frame.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Observation and intervention for traumatic injury
- Concerns about disposition or lack of availability of child welfare receiving site, if required
- Goal must always be to ensure safety of child and siblings.

Discharge Criteria

- Adequate ED evaluation and medical follow-up
- Safe setting for child must determine disposition
- An abused child has a significant chance of further abuse so disposition must be determined in collaboration with social services and family evaluation
- Child (and siblings) may require placement in foster care.

Issues for Referral

- All patients require referral to the appropriate child welfare agency.
- Other family members may require evaluation before disposition is determined.

PEARLS AND PITFALLS

- A history inconsistent with the physical findings should lead to a suspicion of NAT.
- When child abuse is suspected, it must be reported.

ADDITIONAL READING

- American Academy of Pediatrics, Committee on Child Abuse and Neglect. Evaluation of suspected child physical abuse. *Pediatrics*. 2007;119:1232.
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- Hudson M, Kaplan R. Clinical response to child abuse. *Pediatr Clin North Am*. 2006;53:27–39.
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- Lane WG, Dubowitz H, Langenberg P. Screening for occult abdominal pain in children with suspected physical abuse. *Pediatrics*. 2009;129:1595.
- Lindberg DM, Shapiro RA, Laskey AL, et al: Prevalence of abusive injuries in siblings and household contacts of physically abused children. *Pediatrics*. 2012;130:193.
- Togioka BM, Arnold MA, Bathurst MA, et al. Retinal hemorrhages and shaken baby syndrome: An evidence-based review. *J Emerg Med*. 2009;37:98–106.
- Vandeven AM, Newton AW. Update on child physical abuse, sexual abuse and prevention. *Curr Opin Pediatr*. 2006;18:201



See Also (Topic, Algorithm, Electronic Media Element)

Trauma, Multiple



CODES

ICD9

- 995.50 Child abuse, unspecified
- 995.53 Child sexual abuse
- 995.54 Child physical abuse

ICD10

- T74.12XA Child physical abuse, confirmed, initial encounter
- T74.22XA Child sexual abuse, confirmed, initial encounter
- T74.92XA Unspecified child maltreatment, confirmed, initial encounter

ACETAMINOPHEN POISONING

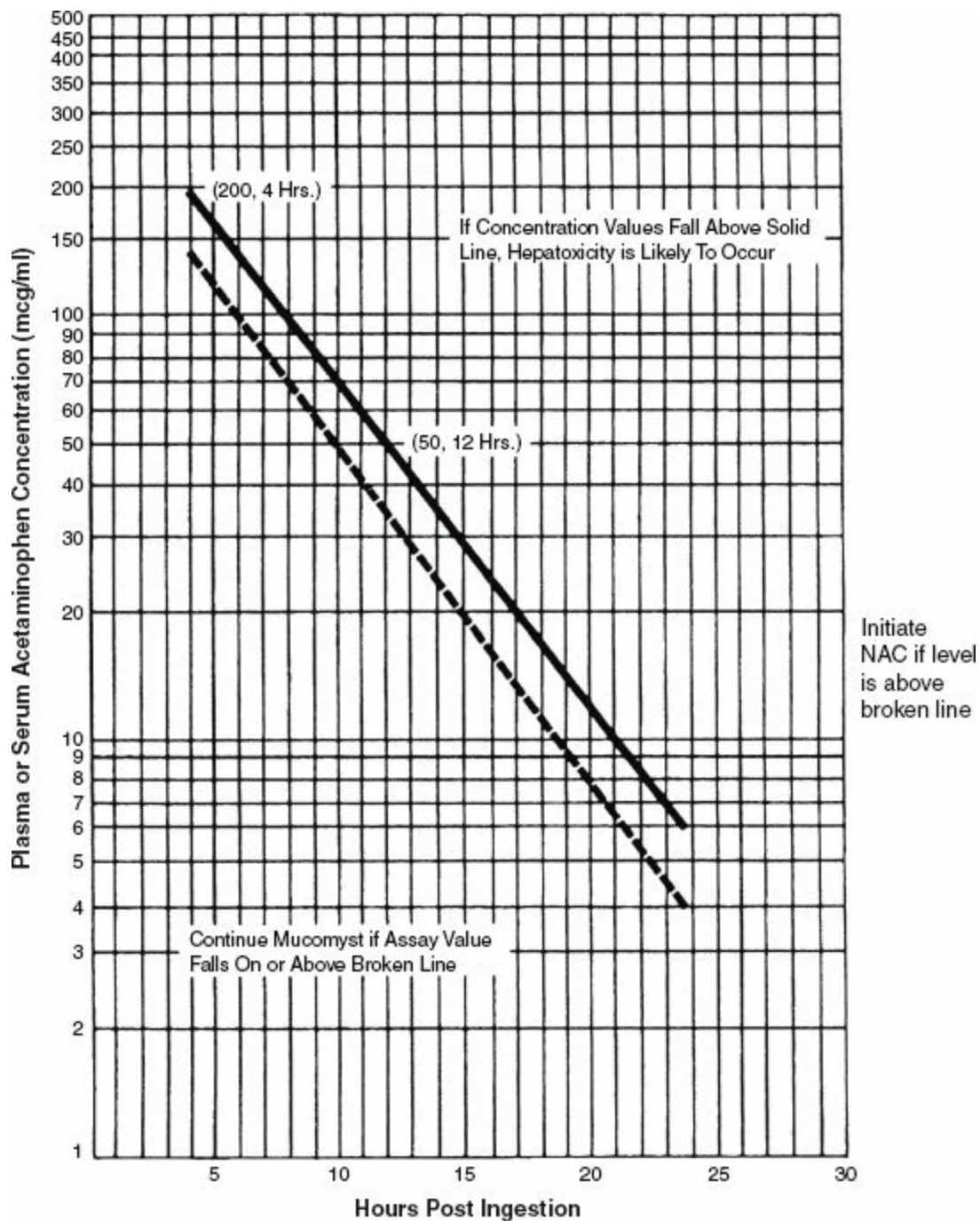
Mark B. Mycyk



BASICS

DESCRIPTION

- Acetaminophen (APAP) is available alone, in combination with oral opiate, and in > 200 OTC cold remedies:
 - One of the most common drugs implicated in intentional and unintentional poisonings
 - The number 1 reason for hepatic transplantation in the US
- *N*-acetyl-*p*-benzoquinoneimine (NAPQI) produced when APAP metabolized by cytochrome P-450:
 - NAPQI normally detoxified by glutathione
 - In overdose, glutathione is quickly depleted and NAPQI causes hepatic damage.
 - *N*-acetylcysteine (NAC) replenishes the liver's glutathione stores.
- Increased risk of toxicity:
 - Patients with poor nutrition have decreased glutathione stores.
- Pharmacokinetics:
 - APAP half-life:
 - 2.5–4 hr in a nonoverdose setting
 - > 4 hr in overdose
- Toxic dose > 150 mg/kg acutely
- Probable toxic level is 140 $\mu\text{g}/\text{mL}$ at 4 hr postingestion (see Fig. 1 nomogram for acute intoxication).
- Therapeutic plasma concentration is 5–20 $\mu\text{g}/\text{mL}$.



Rumack–Matthew nomogram. (Adapted from Rumack BH, Matthew H. Acetaminophen poisoning and toxicity. *Pediatrics*. 1975;55:871–876.)

DIAGNOSIS

SIGNS AND SYMPTOMS

Acute overdose:

- Phase 1: 0.5–24 hr postingestion:
 - Nausea, vomiting, malaise
 - Occurs with large overdoses
 - May not be present with smaller toxic doses
- Phase 2: 24–72 hr postingestion:
 - Decreased GI symptoms

- Hepatic damage is occurring.
- Right upper quadrant pain and tenderness
- Elevation of liver enzymes, PT/INR, bilirubin
- Oliguria
- Prolonged (> 4 hr) APAP half-life implies hepatic toxicity.
- Phase 3: 72–96 hr postingestion:
 - Critical time period in the prognosis
 - Peak liver function abnormalities
 - Hepatic encephalopathy develops.
 - If the PT/INR continues to rise and/or renal insufficiency develops beyond the 3rd day postingestion, there is high likelihood that the patient will require hepatic transplantation.
- Phase 4: 96 hr to 10 days postingestion:
 - Resolution of hepatic injury or progression to complete hepatic failure

ESSENTIAL WORKUP

- Ingestion history of all APAP-containing products
- Time of ingestion
- APAP level:
 - Obtain 4 hr postingestion level or immediately on presentation if > 4 hr postingestion.
 - Use Rumack–Matthew nomogram as therapeutic guide for single acute overdose (see Fig. 1).
 - In chronic or very late ingestions (> 24 hr), obtain level, but do not use nomogram for therapeutic guidance.
- Call poison center ([800] 222-1222) or toxicologist.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- APAP level
- Electrolytes, BUN, creatinine, and glucose
- Liver enzymes:
 - Elevated AST is the first abnormality detected.
 - AST/ALT levels may rise > 10,000 in stage III of toxicity.
 - Bilirubin
- PT/INR
- Pregnancy test
- Toxicology screen

DIFFERENTIAL DIAGNOSIS

- Suspect APAP as coingestant with other drugs in overdose.
- Causes of acute onset hepatotoxicity:

- Infectious hepatitis
- Reye syndrome
- *Amanita* sp. mushrooms toxicity
- Herbal and dietary supplements
- Other drug ingestions



TREATMENT

PRE HOSPITAL

- Transport all pill bottles/pills involved in overdose for identification in ED.
- OTC cold remedies often contain APAP.

INITIAL STABILIZATION/THERAPY

- Airway, breathing, circulation (ABCs)
- Administer supplemental oxygen.
- Administer naloxone, thiamine, D₅₀ (or Accu-Chek) for altered mental status.

ED TREATMENT/PROCEDURES

- Supportive care:
 - IV fluids
 - Antiemetics
- Gastric decontamination:
 - Administer a single dose of activated charcoal if recent ingestion.

N-acetylcysteine (NAC) Administration

- Administer if toxic level detected as defined by Rumack–Matthew nomogram.
- NAC virtually 100% hepatoprotective if initiated within 8 hr of an acute overdose
- NAC available in oral form or IV form
- < 8 hr postingestion:
 - Check APAP level.
 - Initiate NAC if APAP level will not be available within 8 hr of ingestion and toxic ingestion suspected.
 - Discontinue NAC if APAP level nontoxic.
- ≥ 8 hr postingestion:
 - Initiate NAC immediately if suspected toxic ingestion.
 - Check APAP level.
 - Discontinue NAC if APAP level is nontoxic.
- > 24 hr postingestion or chronic repeated APAP ingestion
 - Initiate NAC if:
 - Ingestion > 150 mg/kg APAP
 - Symptomatic
 - Abnormal hepatic screening panel

- Discontinue NAC if APAP falls to nondetectable level and no AST elevation occurs by 36 hr postingestion.
- Call poison center ([800] 222-1222) or toxicologist for help.

NAC Preparations

- Oral NAC:
 - _ Poor taste and odor:
 - Dilute to 5% with fruit juice or soft drink to increase palatability.
 - _ Use antiemetics (metoclopramide or ondansetron) liberally to facilitate PO administration.
 - _ If the patient vomits NAC within 1 hr of administration, repeat the dose.
 - _ Administer NAC as a drip through nasogastric (NG) tube if vomiting continues.
 - _ Given q4h
- IV NAC (2 options):
 - _ Acetadote[®] infusion given per manufacturer's instructions
 - _ Oral NAC given by IV route if:
 - Oral form not tolerated because of vomiting
 - Acetadote[®] not available
 - Contact local poison center or toxicologist for help.

Pregnancy Considerations

- No teratogenicity with NAC
- NAC may be effective in protecting fetal liver:
 - _ Fetal liver metabolizes APAP to toxic NAPQI after 14 wk gestation.

ALERT

A shortened oral NAC protocol may be considered with poison center or toxicology consultation.

MEDICATION

- NAC: 140 mg/kg PO loading (adult and pediatric) followed by 70 mg/kg q4h for 17 additional doses
- Acetadote: 21 hr IV infusion: 150 mg/kg over 60 min, then 50 mg/kg over 4 hr, then 100 mg/kg over 16 hr for total dose 300 mg/kg (see package insert for additional guidance, especially for pediatric infusion dosing)
- Activated charcoal: 1–2 g/kg PO
- Dextrose: D₅₀W 1 amp (50 mL or 25 g; peds: D₂₅W 2–4 mL/kg) IV
- Metoclopramide: Start with 10 mg (peds: 1 mg/kg) IV (1 mg/kg max.)
- Naloxone (Narcan): 0.4–2 mg (peds: 0.1 mg/kg) IV or IM initial dose
- Ondansetron: > 80 kg, 12 mg; 45–80 kg, 8 mg (peds: 0.15 mg/kg) IV
- Thiamine (vitamin B₁): 100 mg (peds: 50 mg) IV or IM

Pregnancy Considerations

Treating the mother maximizes treatment for the fetus. NAC crosses the placenta and is considered safe PO or IV.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Hepatotoxic level of APAP requiring full course of NAC therapy (see “Treatment”)
- LFT abnormalities in the setting of chronic ingestion or late presentation
- Nontoxic suicide attempt requiring psychiatric treatment

Discharge Criteria

Asymptomatic patients with nontoxic ingestions not requiring full course of NAC therapy

Issues for Referral

Evidence of significant hepatotoxicity at time of ED arrival warrants early evaluation by hepatology and/or transplant service.

FOLLOW-UP RECOMMENDATIONS

- Substance abuse referral for patients with oral opiate abuse
- Patients with unintentional (accidental) poisoning require poison prevention counseling.
- Patients with intentional (e.g., suicide) poisoning require psychiatric evaluation.

PEARLS AND PITFALLS

- Consider occult APAP poisoning in patients evaluated for oral opiate abuse.
- Do not use the nomogram for patients with chronic ingestion or late presentation.
- Do not stop NAC therapy until nondetectable APAP level *and* improvement (or resolution) of laboratory and clinical evidence of hepatotoxicity.

ADDITIONAL READING

- Brok J, Buckley N, Gluud C. Interventions for paracetamol (acetaminophen) overdose. *Cochrane Database Syst Rev.* 2006;19(2):CD003328.
- Heard K. Acetylcysteine for acetaminophen poisoning. *N Engl J Med.* 2008;359(3):285–292.
- Larson AM, Polson J, Fontana R, et al. Acetaminophen-induced acute liver failure: results of a United States multicenter, prospective study. *Hepatology.* 2005;42(6):1364–1372.

- Rumack BH. Acetaminophen misconceptions. *Hepatology*. 2004;40(1):10–15.
- Williamson K, Wahl MS, Mycyk MB. Direct Comparison of 20-Hour IV, 36-Hour Oral, and 72-Hour Oral Acetylcysteine for Treatment of Acute Acetaminophen Poisoning. *Am J Ther*. 2013;20(1):37--40.



CODES

ICD9

965.4 Poisoning by aromatic analgesics, not elsewhere classified

ICD10

- T39.1X1A Poisoning by 4-Aminophenol derivatives, accidental, init
- T39.1X2A Poisoning by 4-Aminophenol derivatives, self-harm, init
- T39.1X4A Poisoning by 4-Aminophenol derivatives, undetermined, init

ACIDOSIS

Matthew T. Robinson

BASICS

DESCRIPTION

Respiratory acidosis.

- Reduced pH owing to alveolar hypoventilation with elevated PaCO₂
- Defined as PaCO₂ >45 mm Hg or higher than expected for calculated respiratory compensation for metabolic acidosis
- Divided into 3 broad categories:
 - _ Primary failure in CNS drive to ventilate:
 - Sleep apnea
 - Anesthesia
 - Sedative overdose
 - _ Primary failure in transport of CO₂ from alveolar space:
 - COPD
 - Myasthenic crisis
 - Severe hypokalemia
 - Guillain–Barré syndrome
 - _ Primary failure in transport of CO₂ from tissue to alveoli:
 - Severe heart failure/pulmonary edema

Metabolic acidosis

- Process that reduces serum pH by decreasing plasma bicarbonate levels
- Primarily caused by:
 - _ Accumulation of a strong acid through ingestion or metabolism
 - _ Loss of bicarbonate from the body
- Metabolic acidosis is clinically evaluated by dividing into 2 main groups:
 - _ Elevated anion gap metabolic acidosis:
 - Bicarbonate reduced through buffering of added strong acid
 - Anion gap is increased due to retention of the unmeasured anion from the titrated strong acid.
 - _ Normal anion gap metabolic acidosis due to:
 - Kidneys fail to reabsorb or regenerate bicarbonate.
 - Losses of bicarbonate from GI tract (diarrhea)
 - Ingestion or infusion of substances that release hydrochloric acid
 - _ No anion gap is observed owing to the absence of any unmeasured anion of a titrated acid and secondary chloride retention with HCO₃⁻ loss.

ETIOLOGY

- Respiratory acidosis:
 - Inhibition of respiratory center:
 - Cardiac arrest
 - Drugs (opiates, benzodiazepines, etc.)
 - Meningitis/encephalitis
 - CNS lesions (mass, CVA)
 - Impaired gas exchange:
 - Pulmonary edema
 - Asthma/COPD
 - Pneumonia
 - Interstitial lung disease
 - Obesity
 - Pulmonary contusion
 - Neuromuscular disease:
 - Diaphragmatic paralysis
 - Guillain–Barré syndrome
 - Myasthenia gravis
 - Muscular dystrophy
 - Spinal cord injury
 - Hypokalemia/hypophosphatemia
 - MS
 - Obstructive:
 - Congenital lesions (laryngomalacia)
 - Foreign body aspiration
 - Vascular ring
 - Infectious (epiglottitis, croup, abscess)
- Anion gap acidosis: Mnemonic *A CAT PILES MUD*:
 - Alcohol ketoacidosis
 - Carbon monoxide or cyanide
 - Aspirin
 - Toluene
 - Paraldehyde
 - Iron/isoniazid
 - Lactic acidosis
 - Ethylene glycol
 - Starvation
 - Methanol
 - Uremia
 - Diabetic ketoacidosis
- Increased osmolar gap: Mnemonic *ME DIE*:
 - Methanol
 - Ethylene glycol
 - Diuretics (mannitol; no acidosis)

- Isopropyl alcohol (no acidosis)
- Ethanol
- Nonanion gap metabolic acidosis:
 - GI losses of bicarbonate:
 - Diarrhea
 - Villous adenoma
 - Removal of small bowel, pancreatic or biliary secretions
 - Tube drainage
 - Small bowel/pancreatic fistula
 - Anion exchange resins (i.e., cholestyramine)
 - Ingestion of calcium chloride or magnesium chloride
 - Type I renal tubular acidosis (distal): Hypokalemic hyperchloremic metabolic acidosis:
 - Decreased ability to secrete hydrogen
 - Serum $\text{HCO}_3^- < 15 \text{ mEq/L}$ when untreated
 - Potassium low
 - Renal stones common
 - Type II renal tubular acidosis (proximal): Hypokalemic hyperchloremic metabolic acidosis:
 - Decreased proximal reabsorption of HCO_3^-
 - Acidosis limited by reabsorptive capacity of proximal tubule for HCO_3^-
 - Serum HCO_3^- typically 14–18 mEq/L
 - Low/normal potassium
 - Type IV renal tubular acidosis (hypoaldosteronism): Hyperkalemic hyperchloremic acidosis:
 - Aldosterone deficiency or resistance causing decreased H^+ secretion
 - Serum bicarb $> 15 \text{ mEq/L}$
 - Normal/elevated potassium
 - Carbonic anhydrase inhibitors
 - Tubulointerstitial renal disease
 - Hypoaldosteronism
 - Addition of hydrochloric acid such as:
 - Ammonium chloride
 - Arginine hydrogen chloride
 - Lysine hydrogen chloride

DIAGNOSIS

SIGNS AND SYMPTOMS

- Nonspecific findings
- Vital signs:
 - Tachypnea or Kussmaul respirations with metabolic acidosis
 - Hypoventilation with respiratory acidosis
 - Tachycardia
- Somnolence
- Confusion
- Altered mental status (CO₂ narcosis)
- Myocardial conduction and contraction disturbances (dysrhythmias)

ESSENTIAL WORKUP

- Electrolytes, BUN, creatinine, and glucose:
 - Decreased bicarbonate with metabolic acidosis
 - Hyperkalemia and hypercalcemia with severe metabolic acidosis
- Arterial blood gases:
 - pH
 - CO₂ retention in respiratory acidosis
 - CO level

Check the degree of compensation by calculating the expected values and comparing them to the observed laboratory values as follows:

- Respiratory acidosis:
 - Acute: Expected HCO₃⁻ increased by 1 mEq/L for every 10 mm Hg increase in PaCO₂
 - Chronic: Expected HCO₃⁻ increased by 4 mEq/L for every 10 mm Hg increase in PaCO₂
- Calculate anion gap: Na⁺ – (HCO₃⁻ + Cl⁻):
 - Correct anion gap for hypoalbuminemia:
 - For every 1 g/dL decrease in albumin (from 4 g/dL), add 2.5 points to calculated anion gap.
 - Do not correct sodium concentration when calculating the anion gap in the setting of marked hyperglycemia because hyperglycemia affects the concentration of chloride and bicarbonate, as well as sodium.
 - Normal range = 5 – 12 ± 3 mEq/L
 - Anion gap > 25 mEq/L is seen only with:
 - Lactic acidosis
 - Ketoacidosis
 - Toxin-associated acidosis
- Calculate the degree of compensation:
 - Expected PaCO₂ = 1.5[HCO₃⁻] + 8

- If PaCO₂ inappropriately high, patient has a concomitant respiratory acidosis and/or inadequate compensation.
- Evaluate the delta gap (Δ Gap):
 - For every 1-point increase in anion gap, HCO₃⁻ should decrease by ~1 mEq/L in simple acid–base disorder.
 - As the volumes of distribution of the unmeasured anions and serum HCO₃⁻ are not in unity, a Δ Gap > 6 signifies a mixed acid–base disorder
- Evaluate Δ Gap by comparing the change in the anion gap (Δ AG) with the change in the HCO₃⁻ (Δ HCO₃⁻) from normal:
 - If Δ AG > Δ HCO₃⁻, then patient has a concomitant metabolic alkalosis.
 - If Δ HCO₃⁻ > Δ AG, then patient has concomitant nonanion gap acidosis.

DIAGNOSIS TESTS & NTERPRETATION

Lab

- ABG: See interpretation above.
- VBG:
 - Obvious benefit is less patient discomfort and ease in acquiring sample
 - pH varies by <0.04 units when compared to arterial sampling.
 - Correlation between venous pCO₂ lacking
 - Limited role in screening for hypercapnia. pCO₂ > 45 mm Hg is sensitive (but not specific) for detection of arterial pCO₂ > 50 mm Hg in hemodynamically stable patients
 - Useful in simple acid–base disorders
- Urinalysis for glucose and ketones
- Measure serum osmolality:
 - Calculated serum osmolality = 2 Na + glucose/18 + BUN/2.8
- Osmolar gap = difference between calculated and measured osmolality:
 - Normal = <10
 - Elevated osmolar gap may indicate toxic alcohol as etiology of acidosis.
 - Absence of an osmolar gap should never be used to rule out toxic ingestions:
 - Osmolar gap imprecisely defined
 - Delayed presentations may have normal gap
 - Large variance in gap among normal patients
- Toxicology screen:
 - Methanol, ethylene glycol, ethanol, and isopropyl alcohol if increased osmolality gap
 - Aspirin or iron levels for suspected ingestion
- Co-oximetry for CO exposure

- Serum ketones or β -hydroxybutyrate level
- Serum lactate

Imaging

CXR:

- May identify cardiomyopathy or CHF
- Underlying pneumonia

Diagnostic Procedures/Surgery

ECG:

- May identify regional wall motion abnormalities or valvular dysfunction
- Evaluate for conduction disturbances

DIFFERENTIAL DIAGNOSIS

- Anion gap acidosis:
 - _ Mnemonic *A CATPILES MUD*
- Increased osmolar gap:
 - _ Mnemonic *ME DIE*



TREATMENT

INITIAL STABILIZATION/THERAPY

Airway, breathing, and circulation (ABCs):

- Early intubation for severe metabolic acidosis with progressive/potential weakening of respiratory compensation
- Naloxone, D₅₀W (or Accu-Chek), and thiamine if mental status altered

ED TREATMENT/PROCEDURES

- Respiratory acidosis:
 - _ Treat underlying disorder
 - _ Provide ventilatory support for worsening hypercapnia
 - _ Identify and correct aggravating factors (pneumonia) in chronic hypercapnia.
- Metabolic acidosis:
 - _ Identify if concurrent osmolar gap.
 - _ Treat underlying disorder:
 - Diabetic ketoacidosis
 - Lactic acidosis
 - Alcohol ketoacidosis
 - Ingestion
 - _ Correct electrolyte abnormalities.
- IV fluids:

- Rehydrate with 0.9% normal saline if patient hypovolemic.
- Consider hemodialysis

MEDICATION

- Dextrose: D₅₀W 1 amp (50 mL or 25 g); (peds: D₂₅W 4 mL/kg) IV
- Naloxone (Narcan): 2 mg (peds: 0.1 mg/kg) IV or IM initial dose
- Thiamine (vitamin B₁): 100 mg (peds: 50 mg) IV or IM



FOLLOW-UP

DISPOSITION

Admission Criteria

Consider ICU admission if:

- pH < 7.1
- Altered mental status
- Respiratory acidosis
- Hemodynamic instability
- Dysrhythmias
- Electrolyte abnormalities

Discharge Criteria

Resolving or resolved anion gap metabolic acidosis

PEARLS AND PITFALLS

- Failure to appreciate acidosis in mixed acid–base disorders
- Failure to appreciate inadequate respiratory compensation for metabolic acidosis and need for ventilatory support
- Clues to the presence of a mixed acid–base disorder are normal pH with abnormal PCO₂ or HCO₃[−], when the HCO₃[−] and PCO₂ move in opposite directions, or when the pH changes in the direction opposite that expected from a known primary disorder.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Alkalosis



CODES

ICD9

276.2 Acidosis

ICD10

E87.2 Acidosis

ACROMIOCLAVICULAR JOINT INJURY

Aleksandr M. Tichter • Wallace A. Carter



BASICS

DESCRIPTION

- The acromioclavicular (AC) joint is formed by the articulation of the distal clavicle and the scapular acromion
- It is stabilized by the AC ligament, coracoclavicular (CC) ligament, and attachments from deltoid and trapezius muscles
 - AC ligament is responsible for horizontal stability
 - CC ligament is responsible for vertical stability
- Rockwood classification (sequential injury pattern):
 - Type I:
 - Sprained AC ligament (AC joint tender)
 - No CC ligament injury
 - No deltoid or trapezius injury
 - No radiographic abnormality (clinical diagnosis)
 - Type II:
 - Ruptured AC ligament (AC joint tender) (distal clavicle horizontally unstable)
 - Sprained CC ligament (CC ligament tender)
 - Minimal deltoid and trapezius injury
 - Radiographs show slight widening of AC joint (normal < 5 mm)
 - Normal CC space (11–13 mm)
 - Type III:
 - Ruptured AC ligament (AC joint tender) (distal clavicle horizontally unstable)
 - Ruptured CC ligament (CC ligament tender) (distal clavicle vertically unstable)
 - Detached deltoid and trapezius
 - Radiographs show widening of AC joint.
 - Increased CC space, with distal clavicle above superior aspect of acromion (100% displaced)
 - Types IV, V, and VI:
 - Cause more significant pain than Types I, II, and III.
 - Best visualized on lateral/axillary radiographs
 - All require operative treatment.
 - Greater risk for prolonged disability
 - Type IV:
 - Identical ligamentous/muscular injury pattern to Type III

- Clavicle is displaced *posteriorly* into trapezius muscle
- Posteriorly displaced clavicle may be palpable on exam
- May cause tenting of skin posteriorly
- Type V:
 - Rare
 - Identical ligamentous/muscular injury pattern to Type III
 - Clavicle is displaced *superiorly* above the trapezius (100–300% increase in CC space)
 - Shoulder droops severely
 - Clavicle may be palpated subcutaneously
 - May cause tenting, ischemia, or disruption of skin
- Type VI:
 - Usually associated with severe trauma
 - Identical ligamentous/muscular injury pattern to Type III
 - Clavicle is displaced *inferiorly* into subacromial or subcoracoid location.
 - Shoulder appears flattened
 - Associated neurovascular injury is common

ETIOLOGY

- Injury most commonly seen in young, active males during contact sports
- Most common mechanism is direct trauma to superior or lateral shoulder while arm is adducted, usually in the setting of a fall
 - acromion is displaced inferomedially
 - clavicle remains stabilized by sternoclavicular ligaments
- May also occur indirectly via a fall on an outstretched hand or elbow, with transmission of force to the AC joint

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Pain to anterior or superior aspect of the shoulder following trauma
- Pain exacerbated by moving arm across the chest, behind the back, or overhead
- Mechanism/force will dictate suspicion for and pattern of injury
- Associated neurovascular symptoms
- Cervical spine symptoms

Physical-Exam

- Exam in standing or sitting position, as supine position negates force of gravity which can mask joint instability

- Inspection: Ecchymosis, abrasion, swelling, symmetry, deformity of AC joint, skin tenting or laceration
 - prominence of clavicle with sagging of the acromion indicates rupture of AC joint (Rockwood Type II injury or greater)
- Palpation: Sequential exam of sternoclavicular joint, length of clavicle, AC joint, CC ligament, coracoid process, scapular spine, and proximal humerus
 - tenderness over AC joint indicates AC ligament injury (Rockwood Type I injury or greater)
 - horizontal instability of distal clavicle indicates AC ligament rupture (Rockwood Type II injury or greater)
 - tenderness over CC ligament indicates CC ligament injury (Rockwood Type II injury or greater)
 - vertical instability of distal clavicle indicates CC ligament rupture (Rockwood Type III injury or greater)
- Special tests
 - Cross-body adduction test:
 - Arm elevated to 90° with elbow flexed at 90°, and adducted across chest
 - Pain confirms AC injury by specifically compressing the joint
 - Sensitivity 77%, specificity 79%
 - O'Brien test
 - Arm elevated to 90° with elbow in extension, adduction of 10–15° and maximum forearm pronation
 - Examiner applies downward force against resistance
 - Pain over top of shoulder confirms AC injury
 - Sensitivity 16–93%, specificity 90–95%
- Complete distal neurovascular exam, including brachial plexus
- Careful cervical spine exam

ESSENTIAL WORKUP

- History to seek mechanisms that commonly cause AC joint injury and associated force
- Physical exam to evaluate for injury pattern, neurovascular compromise and exclude other causes of pain
- Radiographic evaluation as outlined below

DIAGNOSIS TESTS & INTERPRETATION

Imaging

- Specific AC joint radiograph
 - Recommended if AC injury suspected
 - Should include bilateral AC joints (for comparison)
 - Standard shoulder views will over penetrate AC joint and may obscure

subtle injuries

- Stress views no longer recommended
- Zanca view (10–15° cephalic tilt) for limited initial views
- Axillary view for Type III–VI injuries to determine position of distal clavicle
- CT or MRI for further evaluation of surgical cases (Rockwood Types IV–VI)
 - Angiography may be used to evaluate associated neurovascular injuries
 - US if CT/MRI is not available

DIFFERENTIAL DIAGNOSIS

- Shoulder dislocation
- Fractures of acromion or clavicle
- Rotator cuff injury
- Tendinitis
- Capsulitis
- Cervical radiculopathy
- Osteoarthritis
- Osteomyelitis

Pediatric Considerations

- Pediatric clavicle encased in periosteal tube:
 - CC ligament within tube
 - AC ligament external to tube (more vulnerable)
- AC joint injury rarely occurs in isolation in the pediatric population
- When injury does occur, it is more often Type I or II
- Distal clavicular fractures through physis are more common than Type III AC joint dislocations



TREATMENT

PRE HOSPITAL

- Ice packs
- Sling immobilization
- Cervical spine immobilization if indicated

INITIAL STABILIZATION/THERAPY

- Ice packs
- Sling immobilization
- Cervical spine immobilization if indicated
- Analgesia (NSAIDs, other analgesics)

ED TREATMENT/PROCEDURES

- Types I and II:

- Rest, ice, analgesics
- Brief sling immobilization (typically 3–7 days)
- Range of motion (ROM) and strengthening exercises as soon as can be tolerated
- Resume normal activities once painless ROM and strength have returned (2–4 wk)
- Type III:
 - Rest, ice, analgesics
 - Sling immobilization and early (within 72 hr) orthopedic referral
 - Treatment plan is controversial
 - Insufficient evidence exists to favor one management strategy over the other (conservative vs. surgical)
 - Which approach is chosen may depend on general health of patient, level of activity, occupation, hand dominance, and risk for reinjury
- Types IV, V, and VI:
 - Rest, ice, analgesics
 - Sling immobilization and immediate orthopedic referral
 - Require early surgical intervention
- Special circumstance: Potential future complication of AC joint injury is arthritis of the joint

Pediatric Considerations

- Types I and II:
 - Conservative management (rest, ice, analgesics, sling)
 - Should heal without major sequelae
- Type III:
 - Age < 15 yr, conservative management
 - Age ≥ 15 yr may require more aggressive treatment.
- Types IV, V, and VI:
 - Operative repair

MEDICATION

- Ibuprofen: 600 mg (peds: 4–10 mg/kg) PO QID
- Ketorolac: 30 mg (peds: 0.5 mg/kg up to 30 mg if > 6 mo) IM/IV q6h (15 mg IM/IV q6h if > 65 yr or < 50 kg)



FOLLOW-UP

DISPOSITION

Admission Criteria

- Open injury

- Types IV, V, and VI require admission for operative repair

Discharge Criteria

- Types I and II can be discharged with orthopedic referral
- Type III should have urgent orthopedic referral

FOLLOW-UP RECOMMENDATIONS

- Type I and II: Orthopedic follow-up within 2–4 wk
- Type III: Early (within 72 hr) orthopedic follow-up
- Type IV–VI: Immediate orthopedic referral
- All pediatric injuries should have prompt orthopedic follow-up, with Type IV–VI injuries requiring immediate referral

PEARLS AND PITFALLS

- Type I and II AC injuries:
 - No increase in CC space
 - Conservative management with rest, ice, sling, and ROM/strength exercises
- Type III injuries:
 - 100% superior displacement of distal clavicle
 - Management somewhat controversial
 - Require early orthopedic follow-up
- Type IV–VI injuries:
 - Identical ligamentous and muscular injuries to Type III
 - Difference according to position of distal clavicle
 - Operative management is standard of care

ADDITIONAL READING

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- Tamaoki MJS, Belloti JC, Lenza M, et al. Surgical versus conservative interventions for treating acromioclavicular dislocation of the shoulder in adults.



See Also (Topic, Algorithm, Electronic Media Element)

- [Clavicle Fracture](#)
- [Shoulder Dislocation](#)
- [Sternoclavicular Joint Injury](#)



CODES

ICD9

- [831.04 Closed dislocation of acromioclavicular \(joint\)](#)
- [840.0 Acromioclavicular \(joint\) \(ligament\) sprain](#)
- [840.8 Sprains and strains of other specified sites of shoulder and upper arm](#)

ICD10

- [S43.50XA Sprain of unspecified acromioclavicular joint, initial encounter](#)
- [S43.80XA Sprain of other specified parts of unspecified shoulder girdle, initial encounter](#)
- [S43.109A Unsp dislocation of unsp acromioclavicular joint, init](#)

ACUTE CORONARY SYNDROME: ANGINA

Shamai A. Grossman • Margaret J. Lin



BASICS

DESCRIPTION

- Chest discomfort, due to imbalance of myocardial blood supply and oxygen requirements
- Canadian Cardiovascular Society classification for angina:
 - Class I: No angina with ordinary physical activity
 - Class II: Slight limitation of normal activity with angina occurring with walking, climbing stairs, or emotional stress
 - Class III: Severe limitation of ordinary physical activity with angina when walking 1–2 blocks on level surface or climbing 1 flight of stairs
 - Class IV: Inability to carry on any physical activity without discomfort or angina symptoms occur at rest
- Typically categorized as either stable or unstable
- Stable angina: Predictable, with exertion, and improves with rest
- Unstable angina (UA):
 - New onset
 - Increase in frequency, duration or lower threshold for symptoms
 - At rest
- UA associated with increased risk of transmural myocardial infarction and cardiac death

ETIOLOGY

- Cardiac risk factors:
 - Age
 - Men > 35 yr
 - Postmenopausal in women
 - Hypercholesterolemia
 - DM
 - HTN
 - Smoking
- Atherosclerotic narrowing of coronary vessels
 - Stable angina: Chronic and leads to imbalance of blood flow during exertion
 - UA: Acute disruption of plaque which can lead to worsening symptoms with exertion or at rest
- Vasospasm: Prinzmetal angina, drug related (cocaine, amphetamines)
- Microvascular angina or abnormal relaxation of vessels if diffuse vascular disease
- Arteritis: Lupus, Takayasu disease, Kawasaki disease, rheumatoid arthritis

- Anemia
- Hyperbarism, carboxyhemoglobin elevation
- Abnormal structure of coronaries: Radiation, aneurysm, ectasia



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Chest pain:
 - Substernal pressure, heaviness, tightness, burning or squeezing
 - Radiates to neck, jaw, left shoulder, or arm
- Poorly localized, visceral pain
- Anginal equivalents include:
 - Dyspnea
 - Epigastric discomfort
 - Weakness
 - Diaphoresis
 - Nausea/vomiting
 - Abdominal pain
 - Syncope
- Symptoms usually reproduced by exertion, eating, cold exposure, emotional stress
- Symptoms not usually positional or pleuritic
- Usually relieved with rest or nitroglycerin
 - Relief with nitroglycerin in nondiagnostic
- Lasts more than a few minutes but < 20 min
- Considered stable angina if no changes in pattern of frequency of symptoms

Geriatric Considerations

- Women, diabetics, ethnic minorities, and those > 65 yr often present with atypical symptoms
- Prognosis is worse for people with atypical symptoms

Physical-Exam

- “Levine Sign”: Clenched fist over chest, classic finding
- BP often elevated during symptoms
- Physical exam often uninformative
 - occasional S3/S4,
 - mitral regurgitation or new murmur (papillary muscle dysfunction)
 - diminished peripheral pulses

ESSENTIAL WORKUP

ECG:

- Standard 12 lead
 - Ideally should be obtained and read within 10 min of presentation for patients with acute chest pain
- Mostly helpful in detecting acute MI, less so UA
- Compare to prior ECG if available
 - If normal or unchanged, serial ECGs every 10–30 min
- New ST changes or T-wave inversion suspicious for UA
 - T-wave flattening or biphasic T-waves
 - ≤ 1 mm ST depression 80 msec from the J point, is characteristic in UA
 - Can see evidence of old ischemia, strain or infarct, such as old TWI, Q-wave, ST depression
 - Single ECG for acute MI is about 60% sensitive and 90% specific
- ECG can also be helpful to diagnose other causes of chest pain
 - Pericarditis: Diffuse ST elevations, then TW inversions and pulse rate depression
 - Pulmonary embolus S1Q3T3 pattern, unexplained tachycardia and signs of right heart strain

ALERT

- Patients with normal or nonspecific ECGs have a 1–5% incidence of AMI and 4–23% incidence of UA

DIAGNOSIS TESTS & INTERPRETATION

Lab

- For stable angina, cardiac enzymes not indicated, but if history suspicious for acute MI, should obtain.
- CK-MB and troponin I or T
 - $< 50\%$ of patient with UA will have low level troponin elevations
 - CK-MB peaks 12–24 hr, return to baseline in 2–3 days
 - Troponin peaks in 12 hr, return to baseline 7–10 days
- Hematocrit (anemia increases risk of ischemia)
- Coagulation profile
- Electrolytes, especially Cr and K⁺

Imaging

- CXR:
 - Usually nonrevealing
 - May show cardiomegaly, or pulmonary edema, CHF suggests UA or MI
 - May be helpful in identifying other etiologies such as pneumonia, pneumothorax, or aortic dissection
- Coronary CTA:

- Good for low-risk patients with no known CAD to rule out ischemia as cause of pain in patient if no coronary stenosis
- “Triple rule-out” for ACS, PE, and aortic dissection
- Bedside echo: To detect wall motion abnormalities and other etiologies of shock, pericardial effusion, pneumothorax
- Technetium Tc-99 sestamibi (rest): Radionucleotide whose uptake by myocardium is dependent on perfusion

Diagnostic Procedures/Surgery

- Exercise stress testing:
 - Not appropriate if active chest pain with moderate to high likelihood of ischemia
 - Imaging stress test (sestamibi, thallium, or echo) if baseline ECG abnormalities
 - Early positive (within 3 min) concerning for UA
- Coronary angiography:
 - Gold standard of diagnosis for CAD

DIFFERENTIAL DIAGNOSIS

- Anxiety and panic disorders
- Aortic dissection
- Biliary colic
- Costochondritis
- Esophageal reflux
- Esophageal spasm
- Esophagitis
- GERD
- Herpes zoster
- Hiatal hernia
- Mitral valve prolapse
- Musculoskeletal chest pain
- MI
- Myocarditis
- Nonatherosclerotic causes of cardiac ischemia
 - Coronary artery spasm
 - Coronary artery embolus
 - Congenital coronary disease
 - Coronary dissection
 - Valvular disease: AS, AI, pulmonary stenosis, mitral stenosis
 - Congenital heart disease
- Peptic ulcer disease
- Pericarditis
- Pneumonia

- Psychogenic
- Pneumothorax
- Pulmonary embolism



TREATMENT

PRE HOSPITAL

- IV access
- Aspirin
- Oxygen
- Vital signs and oxygen saturation
- Cardiac monitoring
- 12-lead ECG, if possible
- Sublingual nitroglycerin

INITIAL STABILIZATION/THERAPY

- IV access
- Oxygen
- Cardiac monitoring
- Vital signs and continuous oxygen saturation

ED TREATMENT/PROCEDURES

- All patients with chest pain in which cardiac ischemia is a consideration should receive an aspirin upon arrival to the ED
- Sublingual nitroglycerin: If symptoms persist after 3 sublingual doses, suggestive of UA, AMI, or noncardiac etiology
- Pain control
- Anticoagulation

MEDICATION

First Line

- Aspirin: 325 mg PO (chewed) or 81 mg × 4 (chewed)
- In patients with aspirin allergy: Clopidogrel (Plavix) 300--600 mg PO, also consider prasugrel 60 mg PO or 180 mg PO ticagrelor
- Dual antiplatelet therapy should be given to patients with UA at medium to high risk who have been selected to have invasive strategy such as catheterization or surgery
- Nitroglycerin:
 - 0.4 mg sublingual
 - 5–10 µg/min IV USE NON-PVC tubing, titrating to effect
 - 1–2 in of nitro paste

- Hold for low BP (can severely drop BP)
- Beware if pt has history of erectile dysfunction and use of phosphodiesterase inhibitors like sildenafil (Viagra) or tadalafil (Cialis) can last 48 hr
- Morphine
 - 4 mg IV, titrate to relief of pain assuming no respiratory depression and SBP > 90
- Consider beta blocker
 - Metoprolol: 25—50 mg PO or 5 mg IV q5–15min for refractory HTN and tachycardia
 - Contraindicated in reactive airway disease, active CHF, bradycardia, hypotension, heart block, cocaine use
 - Does not necessarily need to be given while patient is in ED, suggested benefit within 24 hrs of AMI

Second Line

Anticoagulation

- Does not alter mortality
 - Consider conferring with cardiology prior to anticoagulation
 - Heparin: 60 U/kg IV bolus, then 12 U/kg/hr (goal PTT 50–70)
 - Enoxaparin: 1 mg/kg SC q12 or q24 if Cr clearance < 30mL/min
- Glycoprotein IIb/IIIa inhibitors: Primary benefit en route to cath
 - Eptifibatide (Integrilin): 180 µg/kg bolus IV over 1–2 min, then 2 µg/kg/min up to 72 hr
 - Tirofiban (Aggrastat): 0.4 µg/kg/min for 30 min, then 0.1 µg/kg/min for 48—108 hr
 - Abciximab (Reopro): 0.25 mg/kg IV bolus, then 0.125 µg/kg/min, maximum dose 10 µg/min for 12 hr
 - Bilvalirudin, fondaparinux
- Patients at risk for high risk for bleeding include the elderly, female, anemic, chronic renal failure



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients with UA require admission to the hospital
- Early intervention with cardiac catheterization likely decreases mortality in patients with elevations in cardiac enzymes, persistent angina or hemodynamic instability
- Patients with unclear diagnosis likely would benefit from admission to ED observation unit or hospitalization for serial cardiac enzymes, ECG and stress

Discharge Criteria

- Patients with stable angina
- Patients who are enzyme/stress testing or cath negative

FOLLOW-UP RECOMMENDATIONS

Patients with stable angina or workup negative chest pain should follow up with their PCP or cardiologist within several days of ED visit.

PEARLS AND PITFALLS

- History is the most important factor in differentiating unstable from stable angina or noncardiac pain
- All patients with chest pain or symptoms concerning for a cardiac etiology should have an immediate ECG
- If the initial ECG is normal or unchanged, do serial ECGs 10–30 min apart
- A single set of negative cardiac enzymes may not rule out ACS in a patient with chest pain
- Women, diabetics, ethnic minorities, and patients > 65 yr require a low threshold for ACS workup as they often have atypical presentations

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- ACS Myocardial Infarction
- ACS Coronary Vasospasm
- Cardiac Testing



CODES

ICD9

- 411.1 Intermediate coronary syndrome
- 413.1 Prinzmetal angina
- 413.9 Other and unspecified angina pectoris

ICD10

- I20.0 Unstable angina
- I20.1 Angina pectoris with documented spasm
- I20.9 Angina pectoris, unspecified

ACUTE CORONARY SYNDROME: CORONARY VASOSPASM

John W. Hardin • Shamai A. Grossman

BASICS

DESCRIPTION

- Spontaneous episodes of chest pain due to coronary artery vasospasm in the absence of increase in myocardial oxygen demand in either normal or diseased coronary vessels
- Also known as Prinzmetal angina or variant angina, originally described in 1959
- Most common in younger patients and men
- Usually occurs in patients without cardiac risk factors or coronary artery disease
- Risk factors:
 - Smoking (up to 75% of cases)
 - Hypertension
 - Hypercholesterolemia
 - Diabetes mellitus
 - Cocaine use

ETIOLOGY

- Abnormal vasodilator function in coronary arteries typically endothelial in origin
- High prevalence of microvascular and epicardial vessel involvement
- Defined by 3 types
 - Focal: Localized, often at or near a site of stenosis of a single artery
 - Multifocal: 2 or more segments of the same artery
 - Multivessel: Involving different coronary arteries
- Unopposed α sympathetic stimulation
- Sympathetic stimulation by endogenous hormones may cause vasoconstriction.
- Conversely, also associated with increased vagal tone or withdrawal from vagal tone as proven with acetylcholine provocative testing
- Hypersensitivity of coronary arteries due to mediators of vasoconstriction
- Endothelial dysfunction possibly from genetic mutations in nitric oxide synthase
- Newer research suggests potential increase ρ -kinase activity in smooth muscle cells

DIAGNOSIS

SIGNS AND SYMPTOMS

- Chest pain:
 - Retrosternal
 - Radiates to neck, jaw, left shoulder, or arm
 - Occurs at rest

– Occurs more frequently at night or in the morning

- Palpitations
- Presyncope or syncope
- Associated with migraine headaches and Raynaud disease in a minority of patients
- May occur during cold weather or stress
- May be prolonged in duration compared to typical angina
- May be elicited by hyperventilation
- May be elicited by exercise
- Circadian pattern, typically at night or early morning

History

May mimic angina, occurrences in the early morning should raise suspicion for vasospasm, but also ask about relationship to stress, exercise, and cold weather

Physical-Exam

Physical exam is typically nondiagnostic.

ESSENTIAL WORKUP

- Must include an ECG
- Use of other tests depends on history.

DIAGNOSIS TESTS & NTERPRETATION

- ECG:
 - Transient ST-segment elevation is characteristic, is typically quite pronounced
 - Often with reciprocal changes
 - May be followed by ST depression or T-wave inversion
 - May have associated dysrhythmia during coronary spasm
 - Heart block with right coronary artery spasm
 - Ventricular tachycardia with LAD spasm
 - In rare cases can present with sudden death during prolonged vasospasm period

Lab

- Troponin
- CK/CKMB fraction
- Toxicology screen:
 - Helpful if cocaine is suspected as etiology of chest pain

Imaging

- CXR:
 - May be helpful to rule out other etiologies such as pneumonia, pneumothorax, or aortic dissection

- Noninvasive coronary imaging (nuclear perfusion, coronary CTA, coronary MR):
 - Typically only helpful when combined with provocative testing

Diagnostic Procedures/Surgery

- Exercise stress testing:
 - Usually not helpful, but can help define those with true ischemic disease
- Noninvasive provocative hyperventilation
 - Highly specific, moderately sensitive, tends to favor those with increased disease activity
 - Paired with either EKG or EKG plus perfusion imaging
- Holter monitor:
 - Can be helpful in silent cases or dysrhythmia
- Coronary angiography:
 - Mild atherosclerosis is often the norm
 - Provocative test with acetylcholine is the gold standard

DIFFERENTIAL DIAGNOSIS

- Angina pectoris
- Anxiety and panic disorders
- Aortic dissection
- Cocaine chest pain
- Esophageal rupture
- Esophageal spasm
- Esophagitis
- GERD
- Mitral valve prolapse
- Musculoskeletal chest pain
- MI
- Peptic ulcer disease
- Pericarditis
- Pneumothorax
- Pulmonary embolism
- Takotsubo cardiomyopathy



TREATMENT

PRE HOSPITAL

Treat as any other acute coronary syndrome.

INITIAL STABILIZATION/THERAPY

- IV access
- Oxygen

- Cardiac monitoring
- Vital signs and oxygen saturation

ED TREATMENT/PROCEDURES

- All patients with chest pain in which cardiac ischemia is a consideration should receive an aspirin upon arrival to the ED:
 - Paradoxically may increase severity of Prinzmetal angina due to inhibition of biosynthesis of naturally occurring coronary vasodilator—prostacyclin
- Nitroglycerin should still be administered and usually will help relieve both ischemic and vasospastic chest pain.
- A trial of calcium-channel blockers is indicated if clinical history is consistent with coronary vasospasm.
- Heparin and β -blockers are not helpful in true coronary vasospasm:
 - β -blockers may be detrimental due to unopposed α -mediated vasoconstriction and should be avoided in suspected cocaine chest pain.

MEDICATION

- Aspirin: 325 mg PO
- Diltiazem: 30–60 mg PO (immediate release)
- Nitroglycerin, either:
 - 0.4 mg sublingual
 - 10–20 μ g/min IV USE NON-PVC tubing, titrating to effect
 - 1–2 in of nitro paste
- Verapamil: 40–80 mg PO (immediate release)

First Line

Diltiazem/verapamil:

- > 40% of patients will have recurrence of vasospastic angina despite calcium-channel blocker therapy

Long-acting nitrates

Second Line

- α -blocking agents
- Statin therapy
- Percutaneous intervention with stenting of fixed lesions in area of vasospasm controversial; can lead to spasm in other areas of coronary tree
- Pacemaker placement for patients with recurrent syncope or AV nodal block from vasospastic angina



FOLLOW-UP

DISPOSITION

Admission Criteria

- New-onset chest pain
- Rule-in with positive biochemical markers or provocative testing
- Rule-in with positive biochemical markers or stress testing
- Many patients previously admitted to the hospital can now be effectively evaluated in a chest pain observation unit or clinical decision unit

Discharge Criteria

- Stable (chronic chest pain)
- Negative ischemic workup

FOLLOW-UP RECOMMENDATIONS

Cardiology follow up within 7 days of ED evaluation

PEARLS AND PITFALLS

- 95% survival at 5 yr
- Typical patient will have no traditional coronary risk factors other than smoking.
- Calcium-channel blockers are 1st-line therapy.
- 30–40% of patients are refractory to treatment and will have repeat episodes.
- May present as STEMI, however true infarction is almost always relegated to patient with pre-existing coronary atherosclerotic disease
- β -blockers can lead to worsening of vasospasm due to unopposed α vasoconstriction.
- Patients with prolonged vasospasm can present with ST elevation MI, ventricular arrhythmias, and sudden death.

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See Also (Topic, Algorithm, Electronic Media Element)

- [ACS Unstable Angina](#)
- [ACS Myocardial Infarction](#)



CODES

ICD9

- [411.1 Intermediate coronary syndrome](#)
- [413.1 Prinzmetal angina](#)

ICD10

- [I20.1 Angina pectoris with documented spasm](#)
- [I24.9 Acute ischemic heart disease, unspecified](#)

ACUTE CORONARY SYNDROME: MYOCARDIAL INFARCTION

Josh W. Joseph • Shama A. Grossman

BASICS

DESCRIPTION

- Imbalance in myocardial blood supply and oxygen requirement
- Acute coronary syndrome (ACS) encompasses a spectrum of disease processes:
 - Unstable angina pectoris
 - Acute myocardial infarction (AMI)
 - ST elevation myocardial infarction (STEMI)
 - Non-STEMI

ETIOLOGY

- Atherosclerotic narrowing of coronary vessels
- Vasospasm (Prinzmetal or variant angina)
- Microvascular angina or abnormal relaxation of vessels with diffuse vascular disease
- Plaque disruption
- Thrombosis
- Arteritis:
 - Lupus
 - Takayasu disease
 - Kawasaki disease
 - Rheumatoid arthritis
- Prolonged hypotension
- Anemia/stress ischemia:
 - Hemoglobin < 8 g/dL
- Carbon monoxide/elevations in carboxyhemoglobin
- Coronary artery gas embolus
- Thyroid storm
- Structural abnormalities of coronary arteries:
 - Radiation fibrosis
 - Aneurysms
 - Ectasia
- Cocaine- or amphetamine-induced vasospasm
- Cardiac risk factors include:
 - Hypercholesterolemia
 - DM
 - HTN

- Smoking
- Family history in a 1st-degree relative < 55 yr old
- Men, age > 55 yr
- Postmenopausal women



DIAGNOSIS

SIGNS AND SYMPTOMS

- Chest pain:
 - Most common presentation of MI
 - Substernal pressure
 - Heaviness
 - Squeezing
 - Burning sensation
 - Tightness
- Anginal equivalents (MI without chest pain):
 - Abdominal pain
 - Syncope
 - Diaphoresis
 - Nausea or vomiting
 - Weakness
- May localize or radiate to arms, shoulders, back, neck, or jaw
- Associated symptoms:
 - Dyspnea
 - Syncope
 - Fatigue
 - Diaphoresis
 - Nausea
 - Vomiting
- Symptoms are usually reproduced by exertion, eating, exposure to cold, or emotional stress.
- Symptoms commonly last 30 min or more.
- Symptoms may occur with rest or exertion.
- Often preceded by crescendo angina
- May be improved/relieved with rest or nitroglycerin
- Symptoms generally unchanged with position or inspiration
- Positive Levine sign or clenched fist over chest is suggestive of angina.
- BP is usually elevated during symptoms.

Physical-Exam

- Physical exam is usually unrevealing.
- Occasional physical findings include:

- S3 or S4 due to left ventricular systolic or diastolic symptoms
- Mitral regurgitation due to papillary muscle dysfunction
- Diminished peripheral pulses
- Physical findings of decompensated CHF

ESSENTIAL WORKUP

History is critical in differentiating MI from noncardiac etiologies.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Electrolytes
- Calcium, magnesium
- Cardiac enzymes
- Digoxin level

Imaging

- CXR:
 - May identify cardiomyopathy or CHF
 - Often abnormal in aortic dissection

Diagnostic Procedures/Surgery

- ECG:
 - Differentiate from nonischemic causes of ST elevation
 - Pericarditis
 - Benign early repolarization
 - Left ventricular hypertrophy with strain
 - Prior MI with left ventricular aneurysm
 - Hyperkalemia
- ECG criteria for STEMI
 - New ST elevation at J point in at least 2 contiguous leads of 2 mm (0.2 mV) in men or 1.5 mm (0.15 mV) in women in leads V2–V3 and/or of 1 mm (0.1 mV) in other contiguous chest leads or the limb leads
 - ST depression in leads V1–V2 may indicate posterior injury
 - New or presumably new LBBB has been considered an STEMI equivalent. Most cases of LBBB at time of presentation, are not old but prior ECG is unavailable
 - Sgarbossa criteria for MI in LBBB are diagnostic
 - Concordant ST elevation > 1 mm in leads with a positive QRS complex
 - Concordant ST depression > 1 mm V1–V3
 - Excessively discordant ST elevation > 5 mm in leads with a negative QRS complex

- Echo:
 - May identify regional wall motion abnormalities or valvular dysfunction

DIFFERENTIAL DIAGNOSIS

- Aortic dissection
- Anxiety
- Biliary colic
- Costochondritis
- Esophageal spasm
- Esophageal reflux
- Herpes zoster
- Hiatal hernia
- Mitral valve prolapse
- Peptic ulcer disease
- Psychogenic symptoms
- Panic disorder
- Pericarditis
- Pneumonia
- Pulmonary embolus



TREATMENT

PRE HOSPITAL

- IV access
- Aspirin
- Oxygen
- Cardiac monitoring
- Sublingual nitroglycerin for symptom relief
- 12-lead ECG, if possible, with transmission or results relayed to receiving hospital

INITIAL STABILIZATION/THERAPY

- IV access
- Oxygen
- Cardiac monitoring
- Oxygen saturation
- Continuous BP monitoring and pulse oximetry

ED TREATMENT/PROCEDURES

- STEMI requires reperfusion therapy as soon as possible:
 - Percutaneous coronary intervention (PCI) is preferred diagnostic and therapeutic modality if available.
- Goal is primary PCI within 90 min of 1st medical contact.
 - Thrombolytics should be given if PCI is not available within 120 min of 1st

medical contact (see “Reperfusion Therapy, Cardiac”).

- Aspirin should be administered 1st to all patients with suspected MI unless known allergy.
- Glycoprotein IIb/IIIa inhibitors (e.g., Abciximab) may be started at time of PCI
- Prasugrel or Clopidogrel should be started at the time of PCI
- Prasugrel should not be given to patients with history of prior stroke or TIA
- Clopidogrel is the recommended ADP receptor inhibitor for patients given fibrinolytics
 - Dose is reduced (age <75 yr: 300 mg, >75 yr: 75 mg)
- If BP is >90–100 mm Hg systolic, administer sublingual nitroglycerin, nitropaste, or IV nitroglycerin assuming no ECG criteria or clinical evidence of right ventricular infarct:
 - Symptoms that persist after 3 sublingual nitroglycerin tablets are strongly suggestive of AMI or noncardiac etiology.
- β -blockers should be initiated within 1st 24 hr if not contraindicated (e.g., heart block, heart rate <60, signs of heart failure, hypotension, or obstructive pulmonary disease) are present
 - No benefit of administration prior to PCI or in ED
- Morphine may be given to relieve pain, anxiety, and increase oxygen carrying capacity.
- Heparin (UFH) or Bivalirudin should be used in patients undergoing primary PCI. Bivalirudin is indicated in patients at high risk for bleeding.
- In patients undergoing thrombolysis, Heparin (UFH), Enoxaparin, or Fondaparinux are appropriate.
- If patient is in cardiogenic shock, patient should be transported to a cardiac catheterization laboratory for angioplasty and intra-aortic balloon pump as soon as possible (see “Congestive Heart Failure”).
- Ventricular dysrhythmias:
 - See “Ventricular Tachycardia”
- Bradydysrhythmia associated with hypotension should be treated with atropine or external pacing.
- Conduction disturbances:
 - 1st-degree atrioventricular (AV) block and Mobitz I (Wenckebach) are often self-limited and do not require treatment.
 - Mobitz II, complete heart block, new right bundle branch block (RBBB) in anterior MI, RBBB plus left anterior branch block or left posterior fascicular block, left bundle branch block plus 1st-degree AV block may require a temporary transvenous pacemaker.
- Accelerated idioventricular rhythm (AIVR) may present after reperfusion, appearing as a ventricular rhythm with rate below 120 bpm
 - Only if sustained treat with electrical cardioversion or sodium bicarbonate
 - Lidocaine and other antidysrhythmics may cause asystole

MEDICATION

- Aspirin: 162–325 mg PO
- ADP receptor inhibitors
 - Clopidogrel (Plavix): 600 mg PO
 - Prasugrel (Effient): 60 mg PO
 - Ticagrelor (Ticlid): 180 mg PO
- Bivalirudin: 0.75 mg/kg IV bolus, then 1.75 mg/kg/h infusion
- Enoxaparin (Lovenox): 1 mg/kg SC q12h
 - Fondaparinux: 2.5 mg IV
- Glycoprotein IIb/IIIa inhibitors:
 - Abciximab (ReoPro) for use prior to PCI only: 0.25 mg/kg IV bolus
 - Eptifibatide (Integrilin): 180 µg/kg IV over 1–2 min, then 2 µg/kg/min up to 72 hr
 - Tirofiban (Aggrastat): 0.4 µg/kg/min for 30 min, then 0.1 µg/kg/min for 48–108 hr
- Heparin: 60 units/kg IV bolus (max. 4,000 U), then 12 U/kg/h (max. 1,000 U/h)
- Metoprolol: 5 mg IV q5–15min followed by 25–50 mg PO starting dose as tolerated (note: β-blockers contraindicated in cocaine chest pain)
- Morphine: 2 mg IV, may titrate upward in 2 mg increments for relief of pain assuming no respiratory deterioration and SBP >90 mm Hg
- Nitroglycerin: 0.4 mg sublingual q5min for max. 3 doses
- Nitroglycerin: IV drip at 5–10 µg/min, USE NON-PVD tubing
- Nitropaste: 1–2 in transdermal
- Thrombolytics: See “Reperfusion Therapy, Cardiac,” for dosing



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients with an AMI require hospital admission.
- If the diagnosis is unclear, admission to the hospital or an ED observation unit may be useful for serial cardiac enzymes, ECGs, and exercise stress testing and/or cardiac catheterization if needed.

Discharge Criteria

No patient with an AMI should be discharged from the ED.

Issues for Referral

- If PCI is unavailable at the treating institution, particularly if the patient is in cardiogenic shock, he should be transported to another hospital if PCI can be initiated within 120 min of 1st medical contact.

- Patients with failed reperfusion should be transported urgently to a PCI-capable facility
- Patients undergoing reperfusion therapy may benefit from transfer to a PCI-capable facility within 3–24 hr as part of an invasive strategy

PEARLS AND PITFALLS

- Goal of thrombolytic therapy is a 30 min door to needle time if PCI not possible.
- New or presumably new LBBB at presentation occurs infrequently, and should not be considered diagnostic of AMI in isolation

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See Also (Topic, Algorithm, Electronic Media Element)

- Cardiac Testing
- Reperfusion Therapy
- Unstable Angina



CODES

ICD9

- 410.90 Acute myocardial infarction, unspecified site, episode of care unspecified
- 410.91 Acute myocardial infarction of unspecified site, initial episode of care
- 410.92 Acute myocardial infarction of unspecified site, subsequent episode of care

ICD10

- I21.3 ST elevation (STEMI) myocardial infarction of unspecified site
- I21.29 STEMI involving oth sites
- I24.9 Acute ischemic heart disease, unspecified

ACUTE CORONARY SYNDROME: NON-Q-WAVE (NON-ST-ELEVATION) MI

David F. M. Brown • Kenneth R. L. Bernard

BASICS

DESCRIPTION

- Non-ST-elevation myocardial infarction (NSTEMI) is a part of a clinical syndrome that also includes unstable angina and ST-elevation MI (STEMI).
- Caused by subtotal occlusion of coronary blood flow:
 - _ Often indicates an incomplete ischemic event
- Coronary plaque disruption:
 - _ Endothelial disruption exposes subendothelial collagen and other platelet-adhering ligands, von Willebrand factor (vWF), and fibronectin.
 - _ Release of tissue factors activates factor VII and extrinsic pathway.
- Thrombus generation:
 - _ Platelet adhesion via glycoprotein (GP) Ia/IIa to collagen; GP Ib to vWF:
 - Platelet activation: Release of ADP, thromboxane A₂, and serotonin alters the platelet GP IIb/IIIa receptor; also causes local vasoconstriction
 - Platelet aggregation: GP IIb/IIIa receptor binds fibrinogen, cross-links platelets, forming local platelet plug
 - _ Platelet stabilization: Thrombin converts fibrinogen to fibrin, provides fibrin mesh, stabilizes platelet aggregate
- Microembolization to downstream coronary arterioles may occur

ETIOLOGY

- Coronary thrombosis
- Coronary vasospasm, idiopathic or cocaine induced
- In situ thrombosis/hypercoagulable states
- Embolic event (e.g., endocarditis, paradoxical emboli through PFO)
- Arteritis

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Pain:
 - _ Pressure or tightness or heaviness

- Substernal, epigastric
- +/- radiation to arm, jaw, back
- More likely nonpositional, nonpleuritic, nonreproducible on palpation
- Nausea, vomiting
- Diaphoresis
- Cough
- Dyspnea
- Anxiety
- Light-headedness
- Syncope
- Recent cocaine or amphetamine use
- Family history of coronary disease
- Atypical presentations common, especially in women, diabetics, and the elderly

Geriatric Considerations

Geriatric patients may present with atypical symptoms or silent ischemia.

Physical-Exam

- Pallor or diaphoresis
- Hypertension or hypotension
- Arrhythmias
- S4 gallop
- Physical exam is often normal

ESSENTIAL WORKUP

ECG, cardiac biomarkers, CXR

DIAGNOSIS TESTS & NTERPRETATION

Lab

- Cardiac markers:
 - Troponins: Specific indicators of myocardial infarction, rises within 3–6 hr after MI, peaks at 9–10 days
 - Creatine kinase (CK): Rises within 4–8 hr, peaks at 18–24 hr, subsiding at 3–4 days; isoenzyme CK-MB more specific for cardiac origin
 - Myoglobin: Rises within 2–6 hr, returns to baseline within 24 hr, highly sensitive but very nonspecific
 - LDH: Rises within 24 hr, peaks at 3–6 days, returns to baseline at 8–12 days
- CBC
- Serum electrolytes including magnesium
- PT/PTT/INR for patients on warfarin
- NT-proBNP: Higher levels correlate with increased mortality in NSTEMI patients.

Imaging

- ECG:
 - ST-segment depression or transient elevation indicates increased risk.
 - T-wave inversion in regional patterns does not increase risk but helps differentiate cardiac pain from noncardiac pain.
 - Deep (> 2 mm) precordial T-wave inversion suggests cardiac ischemia.
- CXR:
 - To assess heart size, pulmonary edema/congestion or identify other causes of chest pain
- ECHO (generally not part of ED evaluation):
 - To identify wall motion abnormalities and assess ventricular function
- Radionuclide studies (if conservative management; generally not part of ED evaluation):
 - Sestamibi scan: Identify viable myocardium
 - Technetium 99: Identify recently infarcted myocardium

Diagnostic Procedures/Surgery

Coronary angiography (+/- PCI), typically as an inpatient, depending on patient's risk profile and comorbidities

DIFFERENTIAL DIAGNOSIS

- STEMI
- Pulmonary embolus
- Aortic dissection
- Acute pericarditis/myocarditis
- Pneumothorax
- Pancreatitis
- Pneumonia
- Esophageal spasm/gastroesophageal reflux
- Esophageal rupture
- Musculoskeletal pain/costochondritis



TREATMENT

PRE HOSPITAL

- IV access
- Oxygen administration
- 12-lead EKG, cardiac monitoring, and treatment of arrhythmias
- Aspirin, analgesia, anxiolytics

INITIAL STABILIZATION/THERAPY

- Oxygen administration

- IV access
- 12-lead EKG, cardiac monitoring, and treatment of arrhythmias

ED TREATMENT/PROCEDURES

- Anti-ischemic therapy to reduce demand and increase supply of oxygen to myocardium:
 - β -blockers: IV only if hypertensive with ongoing pain, else use orally within 24 hr; contraindicated in heart failure
 - Nitrates: Contraindicated with critical AS, suspicion of RV infarct or recent use of phosphodiesterase inhibitors (e.g., sildenafil)
 - Oxygen
 - Morphine sulfate
 - Calcium-channel blockers (nondihydropyridines—e.g., diltiazem, verapamil) may be used in patients with ongoing ischemia and contraindications to β -blockade. Contraindicated in heart failure
- Dual antiplatelet therapy to decrease platelet aggregation:
 - Aspirin: Only withhold if prior anaphylaxis
 - ADP Inhibitor: Clopidogrel (substitute for ASA if hypersensitivity), ticagrelor or prasugrel (if low bleeding risk, CABG unlikely, no history of CVA, age <75 yr)
- GP IIb/IIIa inhibitors (eptifibatide, tirofiban):
 - Only if ongoing ischemia, positive cardiac markers and PCI planned; can defer to inpatient administration
 - May omit if loading dose of clopidogrel administered at least 6 hr prior to PCI or bivalirudin used for anticoagulation
- Anticoagulation therapy to prevent thrombus propagation:
 - Unfractionated heparin or enoxaparin are 1st-line therapies.
 - Fondaparinux (factor Xa inhibitor) is a reasonable alternative, especially for medically managed patients; may have reduced bleeding risk.
 - Reserve bivalirudin (direct thrombin inhibitor) for patients with known heparin-induced thrombocytopenia
- Anxiolytics to suppress sympathomimetic release

MEDICATION

First Line

- Aspirin 162–325 mg PO per day
- β -blockers:
 - Atenolol: Start 5 mg IV over 5 min, then 5 mg IV 10 min later, then 50–100 mg PO per day (1–2 hr after IV doses)
 - Esmolol: 100 μ g/kg/min IV infusion (titrate by increasing 50 μ g/kg/min q15min until effect—to max. dose 300 μ g/kg/min)
 - Metoprolol: Start 5 mg IV q5min \times 3, after 15 min begin 25–50 mg PO BID

- Propranolol: 0.5–1 mg IV then 40–80 mg PO q6–8h
- Clopidogrel: 300–600 mg PO × 1, then 75 mg/d
- Heparins:
 - Enoxaparin: 1 mg/kg SC q12h, can give 30 mg IV bolus before SC dose (beware of enoxaparin in patients with renal dysfunction) *or*
 - Unfractionated heparin: 60 U/kg IV bolus then 12 U/kg/hr infusion (max. bolus 4,000 U, max. infusion rate 1,000 U/hr (goal is a PTT 50–75 s))
- Morphine sulfate: 1–5 mg IV q5–30min PRN pain
- Nitroglycerin: 0.3–0.6 mg SL or 0.4 mg by spray q5min followed by IV infusion beginning at 10–20 µg/min if pain persists (max. dose 200 µg/min)
- GP IIb/IIIa inhibitors:
 - Eptifibatide: 180 µg/kg IV bolus then 2 µg/kg/min infusion for 72–96 hr
 - Tirofiban: 0.4 µg/kg/min IV × 30 min, then 0.1 µg/kg/min infusion for 12–24h

Second Line

- Calcium-channel blockers:
 - Diltiazem: Start 0.25 mg/kg IV bolus, then 0.35 mg/kg IV after 15 min if needed then 30 mg PO q6h: immediate release
 - Verapamil: Start 5–10 mg IV, repeat after 30 min if needed, then 80–160 mg PO q8h: immediate release
- ADP blocker:
 - Ticagrelor 180 mg PO × 1 at time PCI or no later than 1 hr post-PCI then 90 mg PO BID
 - Prasugrel 60 mg PO × 1 at time of PCI or no later than 1 hr post-PCI then 10 mg/d
- Lorazepam: 1–2 mg IV PRN anxiety
- Anticoagulation (instead of unfractionated heparin or enoxaparin):
 - Fondaparinux: 2.5 mg SC once a day *or*
 - Bivalirudin (only prior to PCI): 0.75 mg/kg IV bolus, then 1.75 mg/kg/hr IV for up to 4 hr, then 0.2 mg/kg/hr IV for up to 20 hr



FOLLOW-UP

DISPOSITION

Admission Criteria

- All patients with positive cardiac biomarkers, high risk for adverse outcomes by clinical prediction rules (TIMI, GRACE, PURSUIT), or significant clinical probability of acute coronary syndrome undergoing consideration for urgent or early invasive management 12–24 hr after presentation.
- Intensive care unit for monitoring unstable patients

Discharge Criteria

Only those who are ruled out for acute coronary syndrome/non-Q-wave infarction can be safely sent home.

FOLLOW-UP RECOMMENDATIONS

Only patients ruled out for acute coronary syndrome can be safely discharged:

- Discharged patients should follow up in 1–2 days with their primary care physician or cardiologist.
- Outpatient stress tests should be done within 72 hr.

PEARLS AND PITFALLS

- EKG should be done in all patients with chest pain on arrival to the ED, preferably within 10 min.
- Early medical therapy can reduce mortality in NSTEMI.
- Pitfalls:
 - Do not rule out infarction based on initial or single set of cardiac markers, particularly if the time from symptom onset is <4–6 hr.
 - Do not fail to ask about amphetamine or cocaine use.
 - Do not fail to ask about use of sildenafil, vardenafil, or tadalafil before giving nitroglycerin.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Acute Coronary Syndromes
- Cardiac Testing
- Chest Pain



CODES

ICD9

- 410.70 Subendocardial infarction, episode of care unspecified
- 410.71 Subendocardial infarction, initial episode of care
- 410.72 Subendocardial infarction, subsequent episode of care

ICD10

- I21.4 Non-ST elevation (NSTEMI) myocardial infarction
- I24.9 Acute ischemic heart disease, unspecified

ADRENAL INSUFFICIENCY

Rita K. Cydulka • Joseph P. Tagliaferro



BASICS

DESCRIPTION

- Inadequate hydrocortisone secretion to meet body's stress requirement
- Adrenal deficiency:
 - Inadequate cortisol
 - Unresponsive to stimulation with adrenocorticotrophic hormone (ACTH)
- Functional hypoadrenalism:
 - Inadequate cortisol
 - Partial response to stimulation with ACTH
- Addisonian crisis (acute adrenal insufficiency):
 - Life-threatening emergency
 - Precipitated by intensification of:
 - Chronic adrenal insufficiency
 - Acute adrenal hemorrhage
 - Rapid steroid withdrawal
 - Treatment of hypothyroidism with unrecognized adrenal disease
 - Steroid-dependent patient under stress owing to pregnancy, surgery, trauma, infection, or dehydration

ETIOLOGY

Primary Adrenal Failure

- Adrenal dysgenesis/impaired steroidogenesis:
 - Congenital hypoplasia
 - Allgrove syndrome:
 - ACTH resistance
 - Achalasia
 - Alacrima
 - Glycerol kinase deficiency:
 - Psychomotor retardation
 - Hypogonadism
 - Muscular dystrophy
- Congenital adrenal hyperplasia:
 - 21-hydroxylase deficiency accounts for 95% of cases
- Aldosterone synthetase deficiency
- Mitochondrial disease
- Adrenal destruction:

- Autoimmune:
 - Autoimmune polyglandular syndrome types 1 and 2 (alopecia universalis, chronic mucocutaneous candidiasis, hypoparathyroid, thyroid autoimmunity, diabetes, celiac disease, pernicious anemia)
 - Adrenoleukodystrophy
- Infectious:
 - Granulomatous: TB
 - Protozoal and fungal: Histoplasmosis, coccidioidomycosis, and candidiasis
 - Viral: Cytomegalovirus, herpes simplex virus, and HIV
 - Bacterial
 - Fungal
- Metastatic tumor
- Infiltration:
 - Sarcoid
 - Hemochromatosis
 - Amyloidosis
 - Iron depletion
- Bilateral adrenalectomy
- Hemorrhage:
 - Sepsis: Particularly meningococemia (Waterhouse–Friderichsen syndrome), Pseudomonas infection
 - Birth trauma/anoxia
 - Pregnancy
 - Seizures
 - Anticoagulants
 - Rhabdomyolysis
- Pharmacologic inhibition:
 - Etomidate
 - Herbal medications
 - Ketoconazole
 - Metyrapone
 - Suramin

Secondary Adrenal Failure

- Pituitary insufficiency
- Sepsis
- Head trauma
- Hemorrhage
- Infarction (Sheehan syndrome)
- Infiltration: Neoplasm, amyloid, sarcoid, and hemochromatosis
- ACTH deficiency
- Pharmacologic: Glucocorticoid administration, herbal medications

Tertiary Adrenal Failure

- Hypothalamus insufficiency
- Sepsis
- Infiltrative: Neoplasm, amyloid, sarcoid, and hemochromatosis
- Head trauma

DIAGNOSIS

SIGNS AND SYMPTOMS

- Symptoms:
 - Depression
 - Weakness, tiredness, fatigue
 - Anorexia
 - Abdominal pain (can mimic acute abdomen)
 - Nausea or vomiting
 - Salt craving
 - Postural dizziness
 - Muscle or joint pains
 - Dehydration (found in primary adrenal insufficiency only)
- Signs:
 - Fever or hypothermia
 - Mental status changes
 - Hypotension (< 110 mm Hg systolic)
 - Tachycardia
 - Orthostatic BP changes or frank shock
 - Weight loss
 - Goiter
 - Hypogonadism
 - Hyperkalemia
 - Hypercalcemia
 - Sodium depletion
 - Azotemia
 - Eosinophilia
 - Hyperpigmentation (found in primary adrenal insufficiency only)
 - Vitiligo
- Addisonian crisis:
 - Hypotension and shock
 - Hyponatremia
 - Hyperkalemia
 - Hypoglycemia

ESSENTIAL WORKUP

- Lab confirmation of diagnosis not possible in emergency department

- Adrenal crisis: Life-threatening condition:
 - High degree of suspicion should prompt initiation of therapy before definitive diagnosis.
- Plasma cortisol level $< 20 \mu\text{g/dL}$ accompanied by shock suggests adrenal insufficiency.
- Stat electrolytes:
 - Potassium, sodium
- BUN, creatinine:
 - Elevated owing to dehydration
- Serum glucose levels may be low.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC with differential:
 - Anemia
 - Eosinophilia
 - Lymphocytosis
- Arterial blood gases:
 - Hypoxemia
 - Acidosis
- Cosyntropin stimulation test:
 - Adrenal deficiency:
 - Random serum cortisol $< 20 \mu\text{g/dL}$ (while stressed)
 - ACTH stimulation unresponsive
 - Functional hypoadrenalism:
 - Random serum cortisol $= 20 \mu\text{g/dL}$ (while stressed)
 - 60 min post ACTH stimulation $< 30 \mu\text{g/dL}$ or delta cortisol (60 min – baseline) $= 9 \mu\text{g/dL}$
- Normal anion gap metabolic acidosis due to aldosterone deficiency
- Search for underlying infection

Imaging

CXR:

- Look for infection or edema

Diagnostic Procedures/Surgery

ECG:

- Evaluate for electrolyte disturbances

DIFFERENTIAL DIAGNOSIS

- Sepsis
- Shock (any cause)

- Acute abdominal emergency



TREATMENT

INITIAL STABILIZATION/THERAPY

- Airway, breathing, and circulation management (ABCs)
- Cardiac monitor
- BP support for hypotension:
 - _ Normal saline (0.9%) IV fluids 500 mL–1 L (peds: 20 mL/kg) bolus
 - _ Avoid pressors (if possible):
 - May precipitate dysrhythmias
- Supplemental oxygen to meet metabolic needs
- Correct hyperthermia:
 - _ Initiate cooling measures.

ED TREATMENT/PROCEDURES

- Glucocorticoid replacement:
 - _ IV hydrocortisone or dexamethasone immediately
 - _ Use IM route if no IV access
 - _ Dexamethasone will not interfere with results of cosyntropin stimulation tests.
- Volume expansion:
 - _ NS (0.9%) or D₅NS at rate of 500–1,000 mL/hr for 1st 3–4 hr
 - _ Care should be taken to note patient's age, volume, and cardiac and renal function.
- For hypoglycemia:
 - _ D₅₀W
- Treat life-threatening dysrhythmias secondary to hyperkalemia with calcium, bicarbonate, and insulin/glucose.
- Identification and correction of underlying precipitant
- Should see BP improvement within 4–6 hr of therapy

MEDICATION

- Dexamethasone: 6–10 mg (peds: 0.15 mg/kg per dose) q12h
- Dextrose: 50–100 mL D₅₀ (peds: 2 mL/kg of D₁₀ over 1 min) IV
- Hydrocortisone: 100 mg (peds: 1–2 mg/kg per dose) IV q6h
- Insulin (regular): 10 U by IV push (for hyperkalemia)
- Sodium bicarbonate: 1–2 mEq/kg IV (for hyperkalemia)



FOLLOW-UP

DISPOSITION

Admission Criteria

- All patients with acute adrenal insufficiency
- ICU admission for patients with unstable or potentially unstable cases

Discharge Criteria

- Normal lab evaluation with treated adrenal insufficiency
- Should speak with endocrinologist before discharge with chronic patients

FOLLOW-UP RECOMMENDATIONS

- Should have primary care physician follow-up within a few weeks depending on symptoms.
- May benefit from endocrinology referral.

PEARLS AND PITFALLS

- Acute adrenal insufficiency is a life-threatening emergency, and treatment should not be delayed in the ED while waiting for definite lab diagnosis.
- Cancer of any type can present with adrenal insufficiency; the most common being lung, melanoma, and breast.
- The benefit from steroids for relative adrenal insufficiency in septic shock is limited to the treatment of shock refractory to vasopressive (mortality benefit and clinical effect is questionable).
- The clinical consequence of a single dose of etomidate for rapid sequence intubation is controversial. Studies do show biochemical adrenal suppression which must be weighed against agents with other undesirable properties while performing a critical, life-saving procedure.

ADDITIONAL READING

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- Williams RH, Melmed, S, eds. *Williams Textbook of Endocrinology* 12th ed. Philadelphia, PA: Elsevier/Saunders, 2011.



See Also (Topic, Algorithm, Electronic Media Element)

Cushing Syndrome



CODES

ICD9

- 255.41 Glucocorticoid deficiency
- 255.5 Other adrenal hypofunction

ICD10

- E27.1 Primary adrenocortical insufficiency
- E27.2 Addisonian crisis
- E27.40 Unspecified adrenocortical insufficiency

AGITATION

Maura Kennedy

BASICS

DESCRIPTION

Agitation, a state of extreme restlessness

- Characterized by increased verbal and motor activity
- Can be the presenting symptom of a variety of medical (organic) and psychiatric (functional) disorders
- Broad spectrum of severity
 - _ From excessive talkativeness to threatening or violent behavior
 - _ Includes excited delirium syndrome
 - Characterized by agitation, acidosis, hyperadrenergic autonomic dysfunction
 - Associated with sudden cardiac death, particularly after a violent struggle

EPIDEMIOLOGY

Incidence and Prevalence Estimates

- 6% of emergency visits are for behavioral disturbances
- ~1.7 million emergency visits annually in US involve agitated patients

ETIOLOGY

Medical (organic) etiologies:

- Infectious:
 - _ CNS infections
 - Encephalitis
 - Meningitis
 - Neurosyphilis
 - Abscess
 - _ Hyperactive or mixed delirium secondary to sepsis
- Metabolic derangements:
 - _ Electrolyte derangement
 - Hyponatremia
 - Hypocalcemia
 - Hypoglycemia
 - _ Renal failure
 - _ Acid/base disturbances
 - _ Hepatic encephalopathy

- Wernicke encephalopathy
- Wilsons disease
- Endocrinopathies:
 - Thyroid storm
 - Hyperparathyroidism
- Pulmonary etiologies:
 - Hypoxemia
 - Hypercarbia
- Toxicologic causes
 - Toxidromes:
 - Sympathomimetic
 - Anticholinergic
 - Cholinergic
 - Alcohol intoxication
 - Alcohol withdrawal
 - Neuroleptic malignant syndrome (NMS)
 - Serotonin syndrome (SS)
- Neurologic causes:
 - Tumors
 - CNS infections (see above)
 - Huntington disease
 - Ischemic cerebrovascular accident
 - Traumatic intracranial hemorrhage
 - Subarachnoid hemorrhage
 - Postseizure
- Psychiatric (functional) etiologies:
- Mania/agitated depression
- Psychotic illnesses such as schizophrenia
- Anxiety disorders



DIAGNOSIS

SIGNS AND SYMPTOMS

A detailed history and physical exam are critical in differentiating between medical and psychiatric causes of agitation.

History

- The HPI has a sensitivity of 94% in detecting medical illness in psychiatric patients.
 - If a detailed HPI is unattainable from the patient seek collateral information from family, friends, and pre-hospital providers
- Inquire about:

- Trauma
- Recent illness and fever
- Headache, loss of consciousness, neurologic deficits, or visual hallucinations
- Current medications
- History of:
 - Psychiatric illness
 - Substance abuse
 - HIV/immunosuppressed state
 - Cancer
 - Neurologic disorders, including epilepsy

Physical-Exam

A thorough exam is critical to differentiate between organic and functional causes

- Vital sign abnormalities should prompt a full evaluation for an organic cause
 - Hyperthermia may indicate an infectious etiology, NMS, SS, or excited delirium syndrome
- Perform a toxidrome-oriented exam, including
 - Pupillary assessment
 - Skin evaluation for diaphoresis or absence of sweat
 - Evaluation for urinary retention
- A detailed neurologic exam is mandatory
 - Any neurologic deficit requires a full evaluation for an underlying medical illness
 - Orientation, memory, and attention should be intact for patients with a functional cause of agitation
 - Alterations in orientation and memory are seen in delirium and dementia
 - Inattention, such as inability to recite the days of the week backward, should raise suspicion of delirium
 - Muscle tone and reflexes should be assessed
 - Muscle rigidity may indicate NMS
 - Hyperreflexia and clonus may indicate SS

DIAGNOSIS TESTS & INTERPRETATION

The diagnostic work up is directed by the history, physical exam, and underlying suspicion of for an organic etiology of the agitation.

ESSENTIAL WORKUP

At minimum all patients should have:

- A full set of vital signs
- A complete physical exam, including a detailed neurologic exam and tests of cognition and attention
- Blood glucose testing

DIAGNOSIS TESTS & INTERPRETATION

Diagnostic tests should be directed on the basis of the suspicion of an organic etiology for the patient's agitation, and history and physical exam findings.

Imaging

Head CT should be considered in trauma patients or those with neurologic deficits.

Diagnostic Procedures/Surgery

- Lumbar puncture should be considered in patients
 - with meningeal signs
 - where infection is suspected as etiology of agitation but no source is identified

DIFFERENTIAL DIAGNOSIS

Agitation may be the presenting symptom of an underlying medical illness, substance abuse or withdrawal, or a psychiatric illness.



TREATMENT

PRE HOSPITAL

Pre-hospital providers frequently encounter agitated or violent patients and should:

- Follow regional protocols regarding physical and chemical restraints
- Provide prenotification when transporting an agitated or violent patient so that the receiving hospital can mobilize necessary resources
- Obtain a fingerstick glucose if feasible

INITIAL STABILIZATION/THERAPY

- ABCs
- Treat life-threatening medical/traumatic conditions as appropriate
- Severely agitated patients may become violent and pose harm to staff and other patients
 - Patients should change into hospital gowns and be searched for weapons
 - Physical and parenteral chemical restraints should be used when necessary to ensure safety of patient(s) and staff

ED TREATMENT/PROCEDURES

- When an organic etiology is suspected or diagnosed
- Treatment should be directed at underlying cause
- When a functional etiology is suspected or diagnosed
- Emergency psychiatric referral is indicated
- Management of agitation
- Verbal de-escalation techniques are 1st line for mild or moderate agitation

- Chemical restraint options include
 - Benzodiazepines
 - 1st line therapy for alcohol withdrawal
 - May precipitate or worsen delirium in geriatric patients
 - Antipsychotics
 - 1st line for patients with functional etiology of agitation/psychosis
 - Low dose can be used in delirious geriatric patient when verbal de-escalation is unsuccessful
 - Monitor for QTc prolongation and extrapyramidal symptoms
 - Combination therapy of parenteral benzodiazepines and haloperidol
 - May produce more rapid sedation than monotherapy
 - Should be consider in highly agitated/violent patient
- Physical restraint use:
- Chemical sedation should be used to facilitate early discontinuation of physical restraints
- Physician and nurses must document use and rationale for usage
- Prolonged use can result in:
 - Hyperthermia
 - Rhabdomyolysis
 - Nerve injury if extremities are kept in same position for prolonged time
 - Excited delirium syndrome



FOLLOW-UP

DISPOSITION

Admission Criteria

Disposition is ultimately determined by the underlying cause of the agitation and whether the condition resolves.

Admission Criteria

Admission is warranted if there is an underlying medical or psychiatric condition that requires inpatient treatment

Discharge Criteria

Discharge should be limited to those individuals where the underlying cause resolves (e.g., substance use/abuse) and/or can be safely treated as an outpatient

Issues for Referral

- Psychiatric referral as appropriate
- Alcohol/drug treatment as appropriate

FOLLOW-UP RECOMMENDATIONS

Follow-up is determined by the causative medical or psychiatric condition(s).

PEARLS AND PITFALLS

Search for potential medical illnesses causing the agitation

- Factors suggestive of organic causes include:
 - New onset at age > 45
 - Abnormal vital signs
 - Focal neurologic abnormalities
 - Acute onset
 - Visual hallucinations
 - Abnormalities of memory or attention on cognitive testing
 - Trauma with evidence of head injury

Pitfalls:

- Not assessing for underlying organic cause of agitation
- Not undressing patients and searching for weapons
- Inadequate dosing of sedatives/antipsychotics
- Failure to adjust extremity position in restraints to prevent nerve complications
- Inadequate documentation of the need for restraint

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- Rossi J, Swan MC and Isaacs ED. The violent or agitated patient. *Emerg Med Clin N Am.* 2010;28:235–256.
- Vilke GM, DeBard ML, Chan TC, et al. Excited delirium syndrome (EXDS): Defining based on a review of the literature. *J Emerg Med.* 2012;43:897–905.



See Also (Topic, Algorithm, Electronic Media Element)

- Alcohol Withdrawal
- Poisoning, Toxidromes
- Delirium



CODES

ICD9

307.9 Other and unspecified special symptoms or syndromes, not elsewhere classified

ICD10

R45.1 Restlessness and agitation

AIRWAY ADJUNCTS

David W. Schoenfeld



BASICS

DESCRIPTION

- Airway adjuncts are devices used for management of the upper airway
- Often used as rescue techniques/devices when unable to intubate with standard techniques and difficult to mask ventilate
- Oral and nasopharyngeal airways:
 - Lift tongue off hypopharynx
 - Combined with positioning aid in airway patency
 - Nasopharyngeal airway may be used when gag reflex intact
 - Oropharyngeal airway placement requires absent gag reflex
- Extraglottic devices (EGD):
 - Supraglottic (SGD) class (i.e., LMA, PAXpress, CobraPLA, iGel, etc.)
 - These sit above and surround the glottis
 - Retroglottic (RGD) or infraglottic (IGD) class (i.e., Combitube, King tube, Ruch EasyTube, etc.)
 - RGD/IGD ventilate at the hypopharynx and occlude the esophagus
- Blind insertion technique (specific to device)
- Less protection from aspiration compared to ET tube
- High success rates for placement of EGDs

EPIDEMIOLOGY

- 95% success with 1st method of airway management
- 98% overall success of intubation
- 4% of ED airways are difficult



DIAGNOSIS

SIGNS AND SYMPTOMS

Physical Exam

- Predictors of difficult to bag-mask ventilate (MOANS)
 - M – Mask seal (beards/structural abnormality)
 - O – Obese or obstructed
 - A – Advanced age (> 55 yr)
 - N – No teeth
 - S – Stiff
- Predictors of difficult laryngoscopy and intubation (LEMON)

- L – Look externally
 - Micrognathia
 - Buck teeth
 - Large tongue
 - Short neck
- E – Evaluate 3-3-2
 - Mouth opens < 3 fingerbreadths
 - Horizontal length of mandible < 3 fingerbreadths
 - Thyromental distance < 2 fingerbreadths
- M – Mallampati score (increasing difficulty)
 - Class I: Soft palate, uvula, fauces, pillars visible
 - Class II: Soft palate, uvula, fauces visible
 - Class III: Soft palate visible
 - Class IV: Hard palate only visible
- O – Obstruction
 - Vocal changes/muffled voice
 - Difficulty managing secretions
 - Stridor
- N – Neck mobility (limited)
- Predictors of difficult cricothyrotomy (SHORT)
 - S – Surgery or disrupted airway
 - H – Hematoma or infection
 - O – Obese (access problem)
 - R – Radiation
 - T – Tumor
- Predictors of difficult EGD (RODS)
 - R – Restricted mouth opening
 - O – Obstruction
 - D – Disrupted or distorted airway anatomy
 - S – Stiff lungs or cervical spine

DIAGNOSIS TESTS & NTERPRETATION

- Pulse oximetry should rise or remain at high level with successful airway management
- Confirming correct placement:
- Fiberoptic bronchoscopy (gold standard)
- End tidal capnometry/capnography (> 99% reliable)
- Physical exam (common but unreliable)
- Chest rise/fall
- Auscultation of breath sounds with absence of sound over epigastrium
- Condensation inside the ETT
- Arterial blood gas is used to guide ventilator settings once airway established.

Imaging

CXR: Useful only in patients following endotracheal intubation to exclude mainstem bronchus intubation or pneumothorax and to adjust the position of the tube

ALERT

CXR does not rule out esophageal intubation



TREATMENT

PRE HOSPITAL

- Options for patients requiring prehospital airway management vary by region and include:
 - _ Bag-valve-mask ventilation \pm OPA or NPA
 - _ Orotracheal intubation (\pm RSI)
 - _ Nasotracheal intubation
 - _ EGD placement
 - _ Surgical airway

INITIAL STABILIZATION/THERAPY

- Maintain in-line cervical spine immobilization in trauma patients
- Oxygen (high flow via nonrebreather or BVM)
- Vascular access (for resuscitation and medication administration) IV or IO

ED TREATMENT/PROCEDURES

- Rapid sequence intubation
- Prepare
 - _ Suction, BVM, ETT, primary airway management modality, rescue airway management modality, medications
- Preoxygenate
 - _ NRB or BVM with 100% FiO₂ for 3 min
- Pretreatment
 - _ Minimize adverse responses to airway management
 - _ Suspected elevated ICP
 - _ Ischemic heart disease or major vessel dissection/rupture
 - _ Adults with significant reactive airways disease
 - _ Children up to 10 yr of age
- Paralysis with induction
 - _ Administration of induction agent
 - _ Rapid sequential administration of paralytic agent

ALERT

Paralysis is relatively contraindicated in anticipated difficult airway

- Positioning
 - _ Head extension
 - _ Cricoid pressure (Sellick maneuver)
- Placement of tube
- Postintubation
 - _ Confirm ETT placement
 - _ Sedation with benzodiazepines, opiates, propofol, or other agents
 - _ Continued paralysis as needed combined with adequate sedation
- Failed intubation
- Consider other intubation techniques in failed airway algorithm or use of airway adjunct
- Surgical airway as last resort

MEDICATION

- Induction
 - _ Etomidate: 0.3 mg/kg IV
 - _ Ketamine: 1--2 mg/kg IV or 4--7 mg/kg IM
 - _ Midazolam: 0.07--0.3 mg/kg IV
 - _ Propofol: 2--2.5 mg/kg IV
 - _ Thiopental: 3 mg/kg IV
- Paralysis
 - _ Succinylcholine: 1--1.5 mg/kg (peds: 2 mg/kg) IV, 2.5 mg/kg IM/SC
 - _ Rocuronium: 1 mg/kg IV (paralyzing dose); 0.1 mg/kg IV (defasciculating dose)
 - _ Pancuronium: 0.1 mg/kg IV (paralyzing dose); 0.01 mg/kg IV (defasciculating dose)
 - _ Vecuronium: 0.1 mg/kg IV (paralyzing dose); 0.01 mg/kg IV (defasciculating dose)



FOLLOW-UP

DISPOSITION

Admission Criteria

Almost all intubated patients should be admitted to an ICU or OR

Discharge Criteria

Rarely, ED patients who have been intubated may be extubated in the ED and discharged after a period of observation.

PEARLS AND PITFALLS

- Failure to ventilate is a life-threatening condition
- Assess every patient for the possibility of difficult mask ventilation or intubation
- Always formulate a back-up plan in case of a failed attempt
- Do not fixate on intubation but rather successful ventilation and oxygenation
- Move to alternate airway management techniques and consider surgical airway if unable to intubate or ventilate despite use of airway adjuncts

Pediatric Considerations

- Oro- and nasopharyngeal airways are available in infant+ sizes
- LMAs are available in infant+ sizes
- Combitube is only designed for patients >48 in in height
- Nasotracheal intubation is contraindicated in children under 10 yr of age

ADDITIONAL READING

- Murphy MF. Airway management. In: Wolfson AB, Hendey G, Ling L, et al., eds. *Harwood-Nuss' Clinical Practice of Emergency Medicine*. 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2009.
- Walls RM (ed). *Manual of Emergency Airway Management*. 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2012



CODES

ICD9

- 96.01 Insertion of nasopharyngeal airway
- 96.02 Insertion of oropharyngeal airway
- 96.05 Other intubation of respiratory tract

ICD10

0CHY7BZ Insertion of Airway into Mouth and Throat, Via Natural or Artificial Opening

AIRWAY MANAGEMENT

Scott G. Weiner • Carlo L. Rosen



BASICS

DESCRIPTION

- Techniques that ensure adequate oxygenation and ventilation
- Oral and nasopharyngeal airways:
 - Lift tongue off hypopharynx
 - Facilitate bag-valve-mask (BVM) ventilation
 - Insert when gag reflex is absent
- RSI:
 - Preferred method for ED oral intubation (minimizes aspiration risk)
 - Rapid induction of anesthesia and paralysis
 - Contraindicated in patients who should not be paralyzed
 - A preformulated backup strategy with alternative airway techniques is essential
 - Use of fiberoptic techniques maximizes success
- Oral awake intubation:
 - Oral intubation with sedation only
 - Use when paralysis is contraindicated
 - Ketamine is most commonly used
 - Use with benzodiazepines
- Gum elastic bougie:
 - Airway adjunct used when vocal cords are not well visualized
 - Placement confirmed by feeling bougie bump against tracheal rings
 - Slide endotracheal tube (ET) over bougie, then remove bougie
- Alternative airway devices:
 - Extraglottic devices:
 - Inserted blindly into oropharynx and inflated
 - Laryngeal mask airway (LMA) forms a seal around glottic structures in hypopharynx.
 - LMA offers less protection against aspiration than ET tube
 - Intubating LMA can be used to place an ET tube
 - Esophageal–tracheal tubes (e.g., Combitube, King LT) occlude the esophagus and ventilate the hypopharynx
 - Video laryngoscopes:
 - Fiberoptic camera on the tip of laryngoscope blade (e.g., Glidescope, C-MAC) or LMA to visualize tube placement
 - Fiberoptic intubating stylets:
 - Fiberoptic camera on the tip of a stylet which holds ET tube (e.g.,

Shikani)

- Classic fiberoptic intubation:
 - _ ET tube placed over bronchoscope
 - _ Nasotracheal or orotracheal approach
 - _ Indications:
 - Anatomic limitations to glottis visualization
 - Limited mobility of mandible or cervical spine
 - Unstable cervical spine injury
 - _ Contraindications:
 - Need for immediate airway management
 - Significant oropharyngeal blood
- Nasotracheal intubation:
 - _ Indications:
 - Oral access impaired
 - Unsuccessful oral intubation
 - Paralysis is contraindicated
 - Limited cervical mobility
 - _ Contraindications:
 - Apnea (only absolute contraindication)
 - Anticoagulation
 - Massive facial, nasal, or head trauma
 - Upper airway abscess
 - Epiglottitis
 - Penetrating neck trauma
- Cricothyrotomy:
 - _ Definitive treatment for a failed airway
 - _ Incision in cricothyroid membrane
 - _ Tracheostomy tube inserted percutaneously into the airway
 - _ Indications:
 - Crash airway when other airway attempts have failed
 - Massive facial trauma
 - Total upper airway obstruction
 - _ Contraindications:
 - Laryngeal crush injury
 - Tracheal transection
 - Relative: Expanding zone II or III hematoma
- Percutaneous translaryngeal ventilation (PTV):
 - _ Percutaneous placement of 12G or 14G catheter through cricothyroid membrane
 - _ Intermittent ventilation via high-pressure oxygen source
 - _ Indications:
 - Failed oral or nasal intubation until cricothyrotomy is complete
 - _ Contraindications:

- Upper airway obstruction preventing expiration



DIAGNOSIS

SIGNS AND SYMPTOMS

Clinical conditions requiring airway management:

- Failure to maintain or protect the airway:
 - Oropharyngeal swelling
 - Absent gag reflex
 - Inability to clear secretions, blood
 - Stridor
- Hypoxia or ventilatory failure:
 - Shortness of breath
 - Altered mental status
 - Status epilepticus
- Anticipated clinical course:
 - Ventilatory control for head injury or tricyclic overdose
 - Sedation for diagnostic or therapeutic procedures
 - Early management if the airway might become compromised

ESSENTIAL WORKUP

- Always be prepared with a difficult airway algorithm prior to beginning the procedure.
- Recognition of a difficult airway (LEMON)
 - LOOK for anatomic considerations:
 - Short mandible, thick neck, narrow mouth, large tongue, and protruding teeth
 - Congenital syndromes, acromegaly
 - Obesity
 - EVALUATE 3-3-2 rule (difficult airway if met):
 - Mouth opens < 3 fingerbreadths
 - Horizontal length of mandible < 3 fingerbreadths
 - Thyromental distance < 2 fingerbreadths
 - MALLAMPATI criteria (increasing difficulty):
 - Class I: Soft palate, uvula, fauces, pillars visible
 - Class II: Soft palate, uvula, fauces visible
 - Class III: Soft palate visible
 - Class IV: Hard palate only
 - OBSTRUCTION from underlying disease states:
 - Angioedema
 - Goiter
 - Laryngeal–tracheal tumors
 - History of radiation therapy to the neck

- Infections (epiglottitis, supraglottitis, croup, intraoral or retropharyngeal abscess, Ludwig angina)
- Profuse upper gastrointestinal hemorrhage
- Trauma (facial, neck, cervical spine, laryngeal–tracheal, burns)
- NECK mobility limitation:
 - Rheumatoid arthritis and other arthropathies that decrease cervical spine mobility
 - Spinal immobilization for trauma
- Verification of correct tube placement:
 - Visualization of tube passing through the vocal cords
 - Tracheal tube depth (tube tip to upper incisors):
 - 21 cm (women)
 - 23 cm (men)
 - Age (yr)/2 + 12 (children)
 - End-tidal CO₂ colorimetric device:
 - Changes color if CO₂ is present, indicating tracheal placement
 - Color change may not be seen in cardiac arrest
 - Auscultate over stomach, axillae, and anterior lung fields
 - Observe chest wall movement
 - Condensation in the tube during ventilation

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Pulse oximetry should rise after tracheal intubation
 - Continuous capnography used as adjunct
- Arterial blood gas to manage ventilator settings after intubation

Imaging

CXR:

- To exclude mainstem bronchus intubation or pneumothorax
- Does not rule out esophageal intubation

Diagnostic Procedures/Surgery

Direct visualization of the ET tube through the cords is gold standard.

DIFFERENTIAL DIAGNOSIS

- Esophageal intubation
- Right or left mainstem bronchus intubation
- Extratracheal placement through tear in pyriform sinus or trachea
- Pneumothorax



TREATMENT

PRE HOSPITAL

Options for patients in respiratory arrest for advanced life support (ALS) providers:

- Bag–valve management (BVM) ventilation followed by definitive airway management in the ED
- Orotracheal intubation
- Esophageal–tracheal tubes
- LMA

INITIAL STABILIZATION/THERAPY

- Maintain in-line cervical spine immobilization in trauma
- Oxygen, monitor, IV

ED TREATMENT/PROCEDURES

- RSI
 - Simultaneous administration of sedation (induction agent) and paralysis to provide optimal conditions for emergency airway management
- Prepare equipment:
 - Suction, BVM, various sizes of ET tubes and laryngoscope blades, stylets, medications, and backup devices.
- Preoxygenation:
 - 100% FIO₂ for 3 min
- Pretreatment:
 - Prevents physiologic sequelae of intubation
 - Performed 3 min prior to paralytic
 - Defasciculating dose of nondepolarizing agent
 - Fentanyl and lidocaine may minimize ICP rise and hemodynamic response to intubation in head-injured patients
 - Lidocaine and albuterol in reactive airway disease
- Paralysis with induction:
 - Administration of induction agent (e.g., etomidate, thiopental, midazolam, ketamine)
 - Rapidly followed by administration of paralytic agent (e.g., succinylcholine, rocuronium)
 - Succinylcholine is relatively contraindicated with anticipated difficult oral intubation, open globe injury, organophosphate poisoning, burns > 3 days old, denervation syndromes, myopathies, and suspected hyperkalemia
 - Nondepolarizing agents (e.g., rocuronium) can be used as an alternative to succinylcholine
- Positioning:

- Head extension, with midline cervical stabilization if trauma patient
- Cricoid pressure (Sellick maneuver) is controversial and optional
- Placement of tube:
 - After muscle tone is lost (45–60 sec after succinylcholine)
 - Use a stylet with the ET tube
 - Place tube through vocal cords
 - Inflate cuff
 - Begin ventilation
 - Confirm correct ET tube placement
- Postintubation:
 - Benzodiazepines, opiates, or propofol used for continued sedation
 - Vecuronium may be used for continued paralysis

Pediatric Considerations

- Estimation of ET tube size: $4 + \text{age}/4$
- Uncuffed ET tubes may be used in patients < 8 yr old
- Straight Miller blade is preferred in patients < 3 yr old
- Cricothyrotomy contraindicated in patients < 12 yr old; PTV is preferred
- Use atropine as pretreatment to reduce secretions and attenuate vagal effect
- A defasciculating neuromuscular blocking agent not necessary for children < 5 yr old

MEDICATION

- Atracurium: 0.4–0.5 mg/kg IV
- Atropine: 0.02 mg/kg IV
- Diazepam: 2–10 mg (peds: 0.2–0.3 mg/kg) IV
- Etomidate: 0.3 mg/kg IV
- Fentanyl: 3 µg/kg IV
- Ketamine: 1–2 mg/kg IV or 4–7 mg/kg IM
- Lidocaine: 1.5 mg/kg IV
- Midazolam: 1–5 mg IV (0.07–0.30 mg/kg for induction)
- Propofol: 2–2.5 mg/kg IV
- Pancuronium: 0.01 mg/kg IV (defasciculating dose); 0.1 mg/kg IV (paralyzing dose)
- Rocuronium: 1 mg/kg IV
- Succinylcholine: 1.5 mg/kg (peds: 2 mg/kg) IV; 2.5 mg/kg IM/SC
- Thiopental: 3 mg/kg IV
- Vecuronium: 0.01 mg/kg IV (defasciculating dose); 0.1 mg/kg IV (paralyzing dose)



FOLLOW-UP

DISPOSITION

Admission Criteria

Almost all intubated patients should be admitted to an ICU.

Discharge Criteria

Rarely, certain ED patients who have been intubated for airway protection or to facilitate diagnostic workup may be extubated in the ED after a period of observation and then discharged.

PEARLS AND PITFALLS

Respect the airway. Failure to intubate and ventilate is a life-threatening condition:

- Assess each patient for the possibility of difficult intubation.
- Prepare and familiarize yourself with all needed equipment and medications (including contraindications and side effects).
- ALWAYS formulate your backup plan in the case of a crash airway or failed standard orotracheal intubation before beginning the procedure.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Rapid Sequence Intubation



CODES

ICD9

- 96.01 Insertion of nasopharyngeal airway
- 96.02 Insertion of oropharyngeal airway
- 96.05 Other intubation of respiratory tract

ICD10

0CHY7BZ Insertion of Airway into Mouth and Throat, Via Natural or Artificial Opening

ALCOHOL POISONING

Timothy J. Meehan



BASICS

DESCRIPTION

- Alcohol is the most commonly abused recreational agent among emergency department patients
- Alcohol is frequently associated with traumatic injuries

ETIOLOGY

- Alcohol intoxication:
 - Directly depresses CNS function
 - Blood alcohol levels drop by 15–40 mg/dL/hr depending on individual variables and chronicity of alcohol use
- Alcohol withdrawal:
 - Occurs in chronic alcohol abusers after partial or complete alcohol abstinence
 - May occur despite a serum alcohol level > 100 mg/dL (e.g., “intoxicated”)
 - Primarily due to loss of chronic CNS inhibition:
 - Profound CNS excitation
 - Increased catecholamine release and adrenergic tone



DIAGNOSIS

SIGNS AND SYMPTOMS

Acute Alcohol Intoxication

- CNS effects occur on a spectrum:
 - Relaxation
 - Euphoria
 - Sedation
 - Memory loss
 - Impaired judgment
 - Ataxia
 - Slurred speech
 - Obtundation/coma
- May also cause GI upset

Alcohol Withdrawal Syndrome

- Early or minor withdrawal:

- < 8 hr after last drink:
 - Symptoms of a hangover
 - Headache
 - Nausea/vomiting
- 12 hr after last drink:
 - Mild tremors/anxiety
 - Anorexia, nausea, vomiting
 - Weakness
 - Myalgias
 - Vivid dreams/nightmares
- 12–36 hr after last drink:
 - Irritability/agitation
 - Tachycardia/HTN
 - Tremors in hands and tongue
- 24–48 hr after last drink: Alcoholic hallucinosis:
 - Visual hallucinations most common (bug crawling)
 - Auditory hallucinations (buzz, clicks)
 - Present in minor and major withdrawal
- Alcoholic withdrawal seizures:
 - 8–12 hr after last drink
 - Brief, spontaneously abating tonic–clonic activity
 - Precedes delirium tremens (DTs)
- Late alcohol withdrawal or major withdrawal:
 - 48 hr after last drink
 - DTs:
 - Clouded consciousness and delirium
 - Confusion/disorientation
 - Agitation/combativeness
 - Tachycardia/HTN
 - Hyperpyrexia
 - Diaphoresis

History

- Often provided by EMS, family, or friends
- Beware the “frequent flyer” in the ED:
 - Can sometimes have other causes of AMS:
 - Hepatic disease/encephalopathy
 - Seizures (postictal)
 - Hypoglycemia
 - Head injury or intracranial bleeding

Physical-Exam

- Vital signs:
 - Acute intoxication: Normal or depressed
 - Withdrawal: Usually elevated
- Mental status:
 - Acute intoxication: Somnolent, obtunded, or comatose
 - Withdrawal: Hyperalert, agitated
- Signs of hepatic injury:
 - Jaundice
 - Icterus
 - Spider angiomas
 - Asterixis
 - Hepatomegaly
- Signs of malnutrition:
 - Alopecia
 - Poor dentition
 - Poor muscle mass
 - Abdominal wasting
 - Temporal wasting

ESSENTIAL WORKUP

- Obtain accurate alcohol ingestion and abstinence history
- Investigate for life-threatening causes of seizures:
 - Hypoglycemia (get rapid bedside glucose)
 - Intracranial hemorrhage
 - CNS infection
 - Electrolyte abnormalities
- Evaluate for occult trauma
- Monitor all vital signs frequently:
 - Hyperthermia predicts poorer outcomes

DIAGNOSTIC TESTS & INTERPRETATION

Lab

- Alcohol level if abnormal mental status
- Urine toxicology panel to screen for coingestants
- Electrolytes, BUN, creatinine, and glucose
- CBC
- Magnesium, calcium, and phosphate
- PTT, PT/INR if coagulopathy suspected
- LFTs if liver disease suspected
- Ammonia level if hepatic encephalopathy suspected
- Urinary ketones or serum acetone if alcoholic ketoacidosis suspected

Imaging

- CT of head if:
 - Alteration in mental status is out of proportion to expected AMS based on serum alcohol level
 - Suspected head trauma
 - Signs of increased intracranial pressure or focal findings on neurologic exams
 - New-onset seizure
 - Unimproved or deteriorating level of consciousness
- EEG differentiates alcohol withdrawal seizures from idiopathic epilepsy
- Chest radiograph if suspected aspiration or pneumonia

DIFFERENTIAL DIAGNOSIS

- Acute alcohol intoxication:
 - Hypoglycemia
 - Carbon dioxide narcosis
 - Mixed-drug overdose
 - Ethylene glycol, methanol, or isopropanol poisoning
 - Hepatic encephalopathy
 - Psychosis
 - Severe vertigo
 - Psychomotor seizure
- Alcohol withdrawal and seizures:
 - Sedative–hypnotic withdrawal
 - Acute intoxication or poisoning:
 - Carbon monoxide
 - Isoniazid (especially if prolonged seizures not responding to standard therapy)
 - Amphetamine
 - Anticholinergic
 - Cocaine
 - Secondary seizure disorders:
 - Infection
 - Meningitis
 - Encephalitis
 - Brain abscess
 - Trauma
 - Intracranial hemorrhage
 - CVA
 - Tumor
 - Anticonvulsant noncompliance
 - Thyroid disorder



TREATMENT

PRE HOSPITAL

- Administer benzodiazepines for seizures
- Give naloxone, oxygen, and dextrose for comatose individuals
- Intubate as necessary for airway protection to prevent aspiration
- C-spine immobilization if suspected trauma

INITIAL STABILIZATION/THERAPY

- Airway, breathing, circulation (ABCs)
- Evaluate C-spine if suspected trauma
- Initial IV rehydration with 0.9 NS, then D5 0.45 NS
- Administer naloxone, thiamine, and glucose (or Accu-Chek) if altered mental status
- Benzodiazepines if seizing (may require large doses)

Pediatric Considerations

- Young children have decreased hepatic glycogen reserves
- Cannot mount an appropriate response to increased glucose needs
- Rapid bedside glucose (Accu-Chek) is ESSENTIAL:
 - Administer dextrose if indicated with D5 (10 mL/kg), D10 (5 mL/kg), or D25 (2 mL/kg) depending on age and size

ED TREATMENT/PROCEDURES

- Alcohol intoxication:
 - Rehydrate with IV fluids
 - Correct electrolyte abnormalities:
 - Magnesium
 - Potassium
 - Folate
 - Thiamine
 - Multivitamins
- Alcoholic ketoacidosis:
 - Aggressive rehydration with D5 0.9 NS
 - Exclude other causes of wide anion-gap metabolic acidosis
- Alcohol withdrawal syndrome:
 - CIWA-Ar
 - Validated scale for assessing withdrawal severity
 - Guides initial pharmacotherapy
 - Gauges response to therapy and needs for repeat dosing (“symptom-triggered” therapy)
 - Benzodiazepines are the agent of choice:
 - Cross-tolerant with alcohol

- Increases GABA_A-mediated transmission
- Anticonvulsant effect
- Large, frequent doses required with significant withdrawal
- May halt progression to DTs
- Barbiturates (phenobarbital):
 - Useful if severe withdrawal or DTs refractory to large doses of benzodiazepines
- Propofol:
 - Agent of choice for intubated patients
 - Completely suppresses seizure activity
 - Requires intubation/ventilation
 - Caution if hypotensive
- β -blocker (labetalol, esmolol, or metoprolol):
 - Normalizes vital sign abnormalities
 - Does *not* treat CNS complications of alcohol use or withdrawal
- α -agonist (clonidine):
 - Centrally acting α_2 -adrenergic agonists
 - Normalizes vital sign abnormalities
 - Do *not* treat CNS complications of alcohol use or withdrawal
- Phenytoin:
 - Not indicated in seizures primarily due to alcohol withdrawal
 - Indicated if seizures secondary to idiopathic epilepsy, posttraumatic, or status epilepticus

MEDICATION

- Dextrose: D₅₀W 1 amp (50 mL or 25 g; peds: D₂₅W 2–4 mL/kg) IV
- Diazepam (Valium): 5–10 mg IV q5–10min until patient calm
- Lorazepam (Ativan): 0.5–4 mg IV/IM q5–10min until patient calm
- Naloxone (Narcan): 0.4–2 mg (peds: 0.1 mg/kg) IV or IM initial dose
- Phenobarbital: 10–20 mg/kg IV (loading dose) monitor for respiratory depression
- Phenytoin: 15–18 mg/kg not to exceed 25 mg/min:
 - May give Fosphenytoin at 15–20 mgPE/kg at a maximum rate of 150 mgPE/min
- Propofol: 25-75 μ /kg/min IV (loading dose) then 5–50 μ g/kg/min (maintenance dose)
- Thiamine (vitamin B₁): 100 mg (peds: 50 mg) IV or IM



FOLLOW-UP

DISPOSITION

Admission Criteria

- Inability to control seizures or withdrawal symptoms with oral medications
- Hepatic failure, infection, dehydration, malnutrition, cardiovascular collapse, cardiac dysrhythmia, or trauma
- Hallucinations, abnormal vital signs, severe tremors, or extreme agitation
- Wernicke encephalopathy
- Confusion or delirium

Discharge Criteria

- Clinically sober
- Seizure free for 6 hr (with negative workup if 1st seizure)

Issues for Referral

Discuss with social worker and/or police and/or department of family services for pediatric patients.

FOLLOW-UP RECOMMENDATIONS

Substance abuse referral for patients with recurrent alcohol intoxication/use

PEARLS AND PITFALLS

- Failure to appreciate AMS due to nonalcoholic causes in chronic alcoholics:
 - Serum levels should drop by 15–40 mg/dL/hr
 - If mental status not improving (or worsening) need to investigate further
- Failure to adequately treat with benzodiazepines:
 - May require massive doses (e.g., 200–300 mg of diazepam) to control
 - If unable to control, consider other GABAergic agents (phenobarbital, propofol)
- Failure to appreciate hypoglycemia as a common entity in these patients:
 - Can masquerade as “intoxication”
 - Can result in poor outcomes
 - Frequently occurs in chronic alcoholics and children

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- Nelson LS, Gold JA. Chapter 78. Ethanol Withdrawal. In: Hoffman RS, Nelson LS, Goldfrank LR, et al., eds. *Goldfrank's Toxicologic Emergencies*. 9th ed. New York, NY: McGraw-Hill; 2011.
- Pitzele HZ, Tolia VM. Twenty per hour: Altered mental state due to ethanol abuse and withdrawal. *Emerg Med Clin N Am*. 2010;28:683–705.



See Also (Topic, Algorithm, Electronic Media Element)

- Ethylene Glycol, Poisoning
- Methanol, Poisoning



CODES

ICD9

- 303.00 Acute alcoholic intoxication in alcoholism, unspecified
- 305.00 Alcohol abuse, unspecified
- 980.0 Toxic effect of ethyl alcohol

ICD10

- T51.0X1A Toxic effect of ethanol, accidental (unintentional), init
- T51.0X1D Toxic effect of ethanol, accidental (unintentional), subs
- T51.0X1S Toxic effect of ethanol, accidental (unintentional), sequela

ALCOHOLIC KETOACIDOSIS

Charles Garcia

BASICS

DESCRIPTION

- Increased production of ketone bodies due to:
 - Dehydration (nausea/vomiting, ADH inhibition) leads to increased stress hormone production leading to ketone formation
 - Depleted glycogen stores in the liver (malnutrition/decrease carbohydrate intake)
 - Elevated ratio of NADH/NAD due to ethanol metabolism
 - Increased free fatty acid production
- Elevated NADH/NAD ratio leads to the predominate production of β -hydroxybutyrate (BHB) over acetoacetate (AcAc)

ETIOLOGY

- Malnourished, chronic alcohol abusers following a recent episode of heavy alcohol consumption:
 - Develop nausea, vomiting, or abdominal pain
 - Leading to the cessation of alcohol ingestion
- Presentation usually occurs within 12–72 hr

DIAGNOSIS

SIGNS AND SYMPTOMS

- Dehydration
- Fever absent unless there is an underlying infection
- Tachycardia (common) due to:
 - Dehydration with associated orthostatic changes
 - Concurrent alcohol withdrawal
- Tachypnea:
 - Common
 - Deep, rapid, Kussmaul respirations frequently present
- Nausea and vomiting
- Abdominal pain (nausea, vomiting, and abdominal pain are the most common symptoms):
 - Usually diffuse with nonspecific tenderness
 - Epigastric pain common
 - Rebound tenderness, abdominal distension, hypoactive bowel sounds uncommon

- Mandates a search for an alternative, coexistent illness
- Decreased urinary output from hypovolemia
- Mental status:
 - Minimally altered as a result of hypovolemia and possibly intoxication
 - Altered mental status mandates a search for other associated conditions such as:
 - Head injury, cerebrovascular accident (CVA), or intracranial hemorrhage
 - Hypoglycemia
 - Alcohol withdrawal
 - Encephalopathy
 - Toxins
- Visual disturbances:
 - Reports of isolated visual disturbances with AKA common

History

Chronic alcohol use:

- Recent binge
- Abrupt cessation

Physical-Exam

- Findings of dehydration most common
- May have ketotic odor
- Kussmaul respirations
- Palmar erythema (alcoholism)

ESSENTIAL WORKUP

- Presence of an increased anion gap metabolic acidosis secondary to the presence of ketones
- Differentiate from toxic alcohol ingestion and other causes of anion gap metabolic acidosis.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Acid–base disturbance:
 - Increased anion gap metabolic acidosis hallmark
 - Mixed acid–base disturbance common:
 - Respiratory alkalosis
 - Metabolic alkalosis secondary to vomiting and dehydration
 - Hyperchloremic acidosis
 - Mild lactic acidosis common
 - Due to dehydration and the direct metabolic effects of ethanol

- Profound lactic acidosis should prompt a search for other disorders such as seizures, hypoxia, and shock.
- Positive urine and serum nitroprusside reaction tests for ketoacids
 - May not reflect the severity of the underlying ketoacidosis, since BHB predominates and is not measured by this test.
 - May become misleadingly more positive during treatment as more AcAc is produced.
- Electrolytes:
 - Decreased serum bicarbonate
 - Hypokalemia due to vomiting
 - Hypocalcemia
 - Hypophosphatemia may worsen with Tx
 - Hypomagnesemia
 - Initially, can see hyperkalemia and/or hyperphosphatemia which will correct with treatment of the acidosis
- Glucose:
 - Usually mildly elevated
 - Should be monitored frequently as per DKA
 - Hypoglycemia may be present
- Alcohol level may be negative
- BUN and creatinine mildly elevated due to dehydration unless underlying renal disease.
- CBC:
 - Mild leukocytosis—neither sensitive nor specific
 - Thrombocytopenia and anemia commonly due to chronic alcoholism
- Urinalysis:
 - Ketonuria without glucosuria
- Amylase/lipase:
 - Elevated with associated pancreatitis
- LFTs:
 - May have mildly elevated LFTs
- Osmolal gap:
 - May be elevated
 - Elevation > 20 mOsm/kg should prompt evaluation for other ingestions (methanol and ethylene glycol)
 - Correct for ethanol level in osmolal gap by dividing ethanol level by 4.6

Imaging

- CXR if suspect associated pneumonia
- Abdominal films for free air if an acute abdomen is present
- CT scan of the head if associated trauma or unexplained altered mental status

DIFFERENTIAL DIAGNOSIS

- Elevated anion gap metabolic acidosis: *ACAAT MUDPILES*:
 - *Alcoholic* ketoacidosis
 - *Cyanide*, CO, H₂S, others
 - *Acetaminophen*:
 - Rare in acute ingestion
 - Rare in chronic ingestion
 - Fulminant hepatic failure
 - *Antiretrovirals* (NRTI)
 - *Toluene*
 - *Methanol*, metformin
 - *Uremia*
 - *Diabetic* ketoacidosis
 - *Paraldehyde*, phenformin, propylene glycol
 - *Iron*, INH
 - *Lactic* acidosis
 - *Ethylene glycol*
 - *Salicylate*, acetylsalicylic acid (ASA; aspirin), starvation ketosis
- Hypovolemia:
 - GI bleeding
 - Sepsis
- Abdominal pain, nausea, vomiting:
 - Pancreatitis
 - GI bleeding
 - Gastritis
 - Hepatitis
 - Perforated ulcer
 - Alcohol withdrawal
 - DKA
 - Viral illness
 - Obstruction/Ileus



TREATMENT

PRE HOSPITAL

- Supportive measures including IV access with 0.9 NS, oxygen, and cardiac monitoring
- Search for historical clues that may suggest other etiologies such as toxic ingestions or diabetic history, consider scene search
- Attend to other possible coexistent illnesses such as GI bleeding.

INITIAL STABILIZATION/THERAPY

- Cardiac monitor and supplement oxygen

- Naloxone, thiamine, and dextrose if altered mental status
- Initiate 0.9 NS IV fluids
 - _ 500 mL–1 L bolus
 - _ Fluid resuscitation as necessary
 - _ Promotes renal excretion of ketone bodies

ED TREATMENT/PROCEDURES

- Antiemetic for vomiting—ondansetron, promethazine, or prochlorperazine
- Benzodiazepines for symptoms of alcohol withdrawal
- Start dextrose containing solutions (D₅NS):
 - _ More rapid resolution of the metabolic abnormalities than saline alone
 - _ Rate higher than maintenance as tolerated until acidosis resolves
 - _ Avoid with significant hyperglycemia
 - _ Help replete glycogen stores
 - _ Decreases production of ketone bodies by stimulating the production of endogenous insulin
- Thiamine repletion (IV) prior to glucose administration to avoid precipitating Wernicke encephalopathy
- Sodium bicarbonate rarely indicated:
 - _ Consider in severe acidosis with associated cardiovascular dysfunction or irritability
- Electrolyte replacement:
 - _ Hypokalemia occurs with treatment and should be anticipated.
 - _ Hypophosphatemia may occur with treatment.
 - _ Magnesium replacement as indicated for both hypomagnesemia and hypokalemia
- Insulin is not indicated and may precipitate hypoglycemia.

MEDICATION

- D₅₀W: 1 ampule of 50% dextrose (25 g) IVP
- Lorazepam (benzodiazepine): 2 mg IV and titrate to effect
- Narcan: 2 mg IVP
- Ondansetron: 4–8 mg IVP
- Prochlorperazine: 5–10 mg IVP slowly (not > 5 mg/min)
- Promethazine: 12.5–25 mg IVP
- Thiamine: 100 mg IVP



FOLLOW-UP

DISPOSITION

Admission Criteria

- Persistent metabolic acidosis
- Persistent signs of hypovolemia
- Persistent nausea and vomiting
- Abdominal pain of uncertain etiology
- Comorbid illness requiring admission for treatment
- Need for monitored bed due to electrolyte abnormalities requiring continued treatment

Discharge Criteria

- Many patients can be managed in observation unit over 12–24 hr.
- Tolerating oral fluids well
- Resolution of metabolic abnormalities
- No other associated illnesses requiring additional therapy
- Most will warrant at least observation

FOLLOW-UP RECOMMENDATIONS

Counseling regarding alcohol cessation

PEARLS AND PITFALLS

- Aggressive volume repletion with dextrose containing fluid is key.
- Volume resuscitate with NS as necessary
- Thiamine repletion
- Monitor electrolytes before and after treatment.
- Unrecognized increased osmolal gap
- Inadequate monitoring of glucose levels
- Failure to recognize initial electrolyte abnormalities and electrolyte shifts caused by treatment.
- Must be placed on monitor:
 - Cases of sudden death in AKA:
 - Possible alcoholic cardiomyopathy
 - Dysrhythmias
 - Electrolyte derangements

ADDITIONAL READING

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- Yanagawa Y, Sakamoto T, Okada Y. Six cases of sudden cardiac arrest in alcoholic ketoacidosis. *Intern Med*. 2008;47(2):113–117.



See Also (Topic, Algorithm, Electronic Media Element)

- Acidosis
- Diabetic Ketoacidosis



CODES

ICD9

276.2 Acidosis

ICD10

E87.2 Acidosis

ALKALOSIS

Matthew T. Robinson

BASICS

DESCRIPTION

- Respiratory alkalosis:
 - Elevated serum pH secondary to alveolar hyperventilation and decreased PaCO₂
 - Hyperventilation occurs through stimulation of 2 receptor types:
 - Central receptors—located in the brainstem and respond to decreased CSF pH
 - Chest receptors—located in aortic arch and respond to hypoxemia
 - Increased alveolar ventilation secondary to:
 - Disorders causing acidosis
 - Hypoxemia *or*
 - Nonphysiologic stimulation of those receptors by CNS or chest disorders
 - Rarely life threatening with pH typically <7.50
- Metabolic alkalosis:
 - Primary increase in serum HCO₃⁻ secondary to loss of H⁺ or gain of HCO₃⁻
 - Pathogenesis requires an initial process that generates the metabolic alkalosis with a secondary or overlapping process maintaining the alkalosis.
- Generation occurs through 1 of the following mechanisms:
 - Gain of alkali through ingestion or infusion
 - Loss of H⁺ through the GI tract or kidneys
 - Shift of hydrogen ions into the intracellular space
 - Contraction of extracellular fluid (ECF) volume with loss of HCO₃⁻-poor fluids
- Renal maintenance is required to sustain a metabolic alkalosis secondary to the kidney's enormous ability to excrete HCO₃⁻. This occurs through the following:
 - Decreased GFR (renal failure, ECF depletion)
 - Elevated tubular reabsorption of HCO₃⁻ secondary to hypochloremia, hyperaldosteronism, hypokalemia, ECF depletion
- Mortality 45% if pH >7.55 and 80% if pH >7.65

ETIOLOGY

- Respiratory alkalosis:
 - _ CNS:
 - Hyperventilation syndrome
 - Pain
 - Anxiety/psychosis
 - Fever
 - Cerebrovascular accident (CVA)
 - CNS infection (meningitis, encephalitis)
 - CNS mass lesion (tumor, trauma)
 - _ Hypoxemia:
 - Altitude
 - Anemia
 - Shunt
 - _ Medications/drugs:
 - Progesterone
 - Methylxanthines
 - Salicylates
 - Catecholamines
 - Nicotine
 - _ Endocrine:
 - Hyperthyroidism
 - Pregnancy
 - _ Chest stimulation:
 - Pulmonary embolism
 - Pneumonia
 - Pneumothorax
 - _ Other:
 - Sepsis
 - Hepatic failure
 - Heat exhaustion
- Metabolic alkalosis:
 - _ GI loss of H⁺:
 - Vomiting
 - Nasogastric (NG) suctioning
 - Bulimia
 - Antacid therapy
 - Chloride-losing diarrhea (villous adenoma)
 - _ Renal loss:
 - Diuretics (loop and thiazide)
 - Post (chronic) hypercapnia
 - Mineralocorticoid excess
 - Hyperaldosteronism

- Drug/medication (carbenicillin)
- Glucocorticoid excess (Cushing disease)
- Gitelman syndrome
- Hypercalcemia
- Milk-alkali syndrome
- Low chloride intake
- Bartter syndrome
- Intracellular H^+ shift:
 - Hypokalemia
 - Refeeding
- Contraction alkalosis:
 - Diuretics
 - Sweat loss in CF
 - Gastric losses
- HCO_3^- retention:
 - $NaHCO_3$ infusion
 - Blood transfusions



DIAGNOSIS

SIGNS AND SYMPTOMS

- Signs and symptoms secondary to:
 - Arteriolar vasoconstriction
 - Hypocalcemia secondary to decreased ionized calcium from increased calcium binding to albumin
 - Associated hypokalemia
 - Underlying cause
- Weakness
- Seizures
- Altered mental status
- Tetany
- Chvostek sign
- Trousseau sign
- Arrhythmias
- Myalgias
- Carpal-pedal spasm
- Perioral tingling/numbness
- Hypoxemia
- Dehydration

ESSENTIAL WORKUP

- Electrolytes:
 - Elevated HCO_3^- with metabolic alkalosis
 - Evaluate for hypokalemia and hypocalcemia.
- BUN/creatinine:
 - Evaluate for renal failure or dehydration.
- Blood gas (arterial/venous):
 - pH
 - PCO_2 decreased in respiratory alkalosis
 - PO_2 for hypoxemia
 - Venous versus arterial blood gas
 - pH—good correlation within 0.03–0.04 units
 - pCO_2 —good correlation, although VBG may not correlate with severe shock
 - HCO_3^- —good correlation
 - Base excess—good correlation
- Calculate compensation to identify mixed acid–base disorders:
 - Acute respiratory alkalosis:
 - HCO_3^- decreases secondary to intracellular shift and buffering within 10–20 min.
 - Expected HCO_3^- decreased by 2 mEq/dL for each 10 mm Hg decrease in PCO_2 .
 - Chronic respiratory alkalosis:
 - HCO_3^- decreased secondary to renal secretion of HCO_3^-
 - Requires 48–72 hr for maximal compensation
 - Expected HCO_3^- decreased by 5 mEq/dL for each 10 mm Hg decrease in PCO_2 .
 - If HCO_3^- greater than predicted, concomitant metabolic alkalosis
 - If HCO_3^- less than predicted, concomitant metabolic acidosis
 - Metabolic alkalosis:
 - Expected $\text{PCO}_2 = 0.9 [\text{HCO}_3^-] + 9$
 - If PCO_2 greater than predicted, concomitant respiratory acidosis
 - If PCO_2 less than predicted, concomitant respiratory alkalosis
- Urine chloride:
 - More accurate marker than urine Na^+ for patient's volume status:
 - $\text{UCl}^- < 20$ mEq/L in volume depletion
 - $\text{UCl}^- > 40$ mEq/L in euolemia or edematous states

- Useful in therapy for determining saline-responsive vs. saline-resistant causes of metabolic alkalosis

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Glucose
- Ionized calcium
- Magnesium level
- Urine pregnancy
- Additional labs to evaluate underlying cause:
 - CBC, blood cultures for sepsis
 - LFT for hepatic failure
 - Aspirin level
 - Urine toxicology screen
 - Urine diuretics screen (bulimia)
 - Urine diuretic screen (surreptitious diuretic abuse)
 - Renin level
 - Cortisol level
 - Aldosterone level
 - TSH, T4
 - D-dimer

Imaging

CXR:

- May identify cardiomyopathy or CHF
- Underlying pneumonia

Diagnostic Procedures/Surgery

ECG:

- May identify regional wall motion abnormalities or valvular dysfunction
- Evaluate for conduction disturbances.

DIFFERENTIAL DIAGNOSIS

- Respiratory alkalosis:
 - It is essential to rule out organic disease prior to diagnosing hyperventilation syndrome or anxiety states.
- Metabolic alkalosis:
 - Saline responsive (urine $\text{Cl}^- < 20$ mEq/dL):
 - Loss of gastric secretions
 - Chloride-losing diarrhea
 - Diuretics

- Post (chronic) hypercapnia
- CF
- Saline resistant:
 - Hyperaldosteronism
 - Cushing syndrome
 - Bartter syndrome
 - Exogenous mineralocorticoids or glucocorticoids
 - Gitelman syndrome
 - Hypokalemia
 - Hypomagnesemia
 - Milk–alkali syndrome
 - Exogenous alkali infusion/ingestion
 - Blood transfusions



TREATMENT

INITIAL STABILIZATION/THERAPY

Airway, breathing, circulation (ABCs):

- Early intubation and airway control for altered mental status
- IV, oxygen, and cardiac monitor
- Naloxone, D₅₀W (or Accu-Chek), and thiamine for altered mental status

ED TREATMENT/PROCEDURES

- Respiratory alkalosis:
 - Treat underlying disorder.
 - Rarely life threatening
 - Sedation/anxiolytics for anxiety, psychosis, or drug overdose
 - Rebreathing mask bag for hyperventilation syndrome (used cautiously)
- Metabolic alkalosis: Examination of the urine chloride allows etiologies to be divided into saline-responsive or saline-resistant causes:
 - Urine chloride < 20 mEq/L indicates volume depletion:
 - Rehydration with 0.9% saline lowers serum HCO₃[–] by increasing renal HCO₃[–] excretion
 - Saline-responsive causes are associated with volume depletion.
 - Urine chloride > 20 mEq/L indicates saline-resistant etiology. Treat underlying disorder:
 - Potassium supplementation in hypokalemic states
 - Antagonism of aldosterone with spironolactone
 - Acetazolamide to increase renal HCO₃[–] excretion in edematous states
 - Other:

- Infusion of dilute HCl in severe cases of metabolic alkalosis
- Antiemetics for vomiting
- Proton pump inhibitors for patients with NG suction
- Follow ventilatory status closely.
- Correct electrolyte abnormalities.
- Consider hemodialysis for severe electrolyte abnormalities.

MEDICATION

- Dextrose: D₅₀W 1 amp (50 mL or 25 g; peds: 2% dextrose and water 2–4 mL/kg) IV
- KCl (K-Dur, Gen-K, Klor-Con): 20–120 mEq PO daily
- Naloxone: 2 mg (peds: 0.1 mg/kg) IV or IM initial dose
- Thiamine (vitamin B₁): 100 mg (peds: 50 mg) IV or IM
- 0.1–0.2 N HCl (100–200 mEq/L): Infuse over 24–48 hr at a rate not faster than 0.2 mmol/kg/h and through a central line to prevent sclerosing vein



FOLLOW-UP

DISPOSITION

Admission Criteria

- ICU admission if:
 - pH >7.55 or altered mental status
 - Dysrhythmias
 - Severe electrolyte abnormalities
 - Hemodynamic instability
- Coexisting medical illness requiring admission

Discharge Criteria

Resolving or resolved alkalosis

PEARLS AND PITFALLS

- Increased minute ventilation is the primary cause of respiratory alkalosis, characterized by decreased PaCO₂ and increased pH.
 - Metabolic alkalosis is usually caused by an increase in HCO₃⁻, reabsorption secondary to volume, potassium, or Cl⁻ loss.
 - Contraction alkalosis can result from extracellular volume reduction, with a consequent increase in the plasma HCO₃⁻ concentration.
 - Clues to the presence of a mixed acid–base disorder are normal pH with

abnormal PCO_2 or HCO_3^- , when the HCO_3^- and PCO_2 move in opposite directions, or when the pH changes in the direction opposite that expected from a known primary disorder.

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See Also (Topic, Algorithm, Electronic Media Element)



CODES

ICD9

- 276.3 Alkalosis
- 276.4 Mixed acid-base balance disorder

ICD10

- E87.3 Alkalosis
- E87.4 Mixed disorder of acid-base balance

ALTERED MENTAL STATUS

David F. M. Brown • David W. Schoenfeld

BASICS

DESCRIPTION

- Dysfunction in either the reticular activating system in the upper brainstem or a large area of 1 or both cerebral hemispheres
- Definitions:
 - Confusion: A behavioral state of reduced mental clarity, coherence, comprehension, and reasoning
 - Drowsiness: The patient cannot be easily aroused by touch or noise and cannot maintain alertness for some time.
 - Lethargy: Depressed mental status in which the patient may appear wakeful but has depressed awareness of self and environment globally; cannot be aroused to full function
 - Stupor: The patient can be awakened only by vigorous stimuli, and an effort to avoid uncomfortable or aggravating stimulation is displayed.
 - Coma: The patient cannot be aroused by stimulation and no purposeful attempt is made to avoid painful stimuli.
 - Delirium: Acute onset of fluctuating cognition with impaired attention and consciousness, ranging from confusion to stupor.

ETIOLOGY

- Hypoxic:
 - Severe pulmonary disease
 - Anemia
 - Shock
 - Intracardiac shunting (especially in pediatrics)
- Metabolic:
 - Hypoglycemia; hyperglycemia
 - Diabetic ketoacidosis
 - Nonketotic hyperosmolar coma
 - Hyponatremia; hypernatremia
 - Hypocalcemia; hypercalcemia
 - Hypomagnesemia; hypermagnesemia
 - Hypophosphatemia
 - Acidosis; alkalosis
 - Dehydration
 - Deficiency: Thiamine, folic acid, B₁₂, niacin
 - Hyperammonemia (hepatic encephalopathy)

- Uremia (renal failure)
- CO₂ narcosis
- Toxicologic:
 - Toxic alcohols
 - Salicylates
 - Sedatives and narcotics
 - γ -hydroxybutyrate (GHB)
 - Anticonvulsants
 - Psychotropics
 - Isoniazid
 - Heavy metals
 - Carbon monoxide
 - Cyanide
 - Toxic plants (jimsonweed, mushrooms, etc.)
 - Sympathomimetics
 - Anticholinergic, cholinergic
 - Antiemetics
 - Antiparkinsonian medications
 - Withdrawal (especially alcohol, sedatives)
- Infectious:
 - UTI (especially in elderly)
 - Pneumonia
 - Sepsis; bacteremia
 - Meningitis, encephalitis, brain abscess
- Endocrine;:
 - Myxedema coma
 - Thyrotoxicosis
 - Hypothyroidism
 - Addison disease
 - Cushing disease
 - Pheochromocytoma
 - Hyperparathyroidism
- Environmental:
 - Hypothermia
 - Hyperthermia; heat stroke
 - High-altitude cerebral edema
 - Neuroleptic malignant syndrome
 - Malignant hyperthermia
- Vascular:
 - Hypertensive encephalopathy
 - Cerebral vasculitis
 - TTP, DIC, hyperviscosity

- MI
- Primary neurologic:
 - Seizures, nonconvulsive status epilepticus, and postictal state
 - Head trauma, concussion
 - Diffuse axonal injury
 - Structural brain lesions:
 - Hemorrhage (subdural, epidural, subarachnoid, intraparenchymal)
 - Infarction
 - Tumors
 - Demyelination disorders
 - Intracranial hypertension (pseudotumor)
 - HIV-related encephalopathy
 - Autoimmune/inflammatory encephalitis
 - Carcinoid meningitis
 - Primary neuronal or glial disorders:
 - Creutzfeldt–Jakob disease
 - Marchiafava–Bignami disease
 - Adrenoleukodystrophy
 - Gliomatosis cerebri
 - Progressive multifocal leukoencephalopathy
- Trauma; burns
- Porphyria
- Psychiatric
- Multifactorial (especially in elderly)

DIAGNOSIS

SIGNS AND SYMPTOMS

Confusion

- Difficulty in maintaining a coherent stream of thinking and mental performance:
 - Remember to consider level of education, language, and possible learning disabilities.
- Inattention
- Memory deficit:
 - Inability to recall any of the following:
 - The date, inclusive of month, day, year, and day of week
 - The precise place
 - Items of universally known information
 - Why the patient is in the hospital
 - Address, telephone number, or Social Security number
- Impaired mental performance:
 - Difficulty retaining 7 digits forward and 4 backward

- Difficulty naming ordinary objects
- Serial calculations: 3-from-30 subtraction test
- Disorganized and rambling language:
 - May be mistaken for aphasia
- Fever:
 - Infectious etiologies, drug toxicities, endocrine disorders, heat stroke
- Severe hypertension and bradycardia
 - Cushing reflex suggests intracranial lesion
- Hypotension:
 - Infectious, toxicologic etiologies, decreased cardiac output
- Eye movements:
 - Ocular bobbing:
 - Cyclical brisk conjugate caudal jerks of the globes followed by a slow return to midposition
 - Seen in bilateral pontine damage, metabolic derangement, and brainstem compression
- Ocular dipping:
 - Slow, cyclical, conjugate, downward movement of the eyes followed by a rapid return to midposition
 - Seen in diffuse cortical anoxic damage
- Pupil exam:
 - Nearly all toxic and metabolic causes of coma leave the pupillary reflexes sluggish but bilaterally intact.
- Focal findings (indicative of CNS process):
 - Hemiparesis
 - Hemianopsia
 - Aphasia
 - Myoclonus
 - Convulsions
 - Nuchal rigidity
- Asterixis:
 - Arrhythmic flapping tremor (almost always bilateral)
 - Seen in hepatic failure or severe renal failure

History

- Ask witnesses, family, pre-hospital personnel
- Baseline mental status
- Medical history (immunosuppressed, liver failure, depression, or chronic conditions)
- Recent events: Trauma, fever, illness
- Detailed medication list
- Substance abuse history

Physical-Exam

- Vital signs
- Head: Signs of trauma, pupils
- Fundoscopic exam: Hemorrhage, papilledema
- Neck: Rigidity, bruits, thyroid enlargement
- Heart and lungs
- Abdomen: Organomegaly, ascites
- Extremities: Cyanosis
- Skin: Diaphoretic/dry, rash, petechiae, ecchymoses, splinter hemorrhages, needle tracks
- Neurologic exam
- Mental status exam

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Dextrostix and glucose
- CBC
- Electrolytes (including Ca, Mg, P)
- BUN, creatinine
- Toxicologic screen (including toxic alcohols)
- ECG
- Urinalysis
- Blood and urine cultures (suspected infection)
- PT, PTT (anticoagulated, liver failure patients)
- Consider LFTs, thyroid function tests, ammonia, serum osmolarity, arterial blood gas
- Consider B₁₂, folic acid, RPR, urine porphobilinogen, heavy metal screening

Imaging

- Head CT scan:
 - Noncontrast only to rule out hemorrhage and mass effect
- Chest radiograph: To diagnose pneumonia
- MRI (if available):
 - Indicated when suspicious of ischemic stroke or other CNS abnormality
 - May be deferred when admitting the patient as part of the inpatient work-up

Diagnostic Procedures/Surgery

- Lumbar puncture (LP):
 - Indicated when the etiology remains unclear after lab and CT scan
 - Empiric antibiotics should be given before LP in patients with suspected

meningitis.

- EEG (inpatient): For suspected seizure, nonconvulsive status epilepticus
- Caloric stimulation of the vestibular apparatus to assess unresponsive patients:

DIFFERENTIAL DIAGNOSIS

- Locked-in syndrome:
 - Rare disorder caused by damage to the corticospinal, corticopontine, and corticobulbar tracts resulting in quadriplegia and mutism with preservation of consciousness.
 - Communication may be established through eye movements (maintain vertical eye movements).
- Psychogenic unresponsiveness:
 - Conversion reactions
 - Catatonia
 - Malingering
 - Akinetic mutism (abulic state)
- Dementia:
 - Multiple progressive cognitive deficits
 - Attention is preserved in the early stages.



TREATMENT

PRE HOSPITAL

- Airway management if loss of airway patency
- IV access, supplemental oxygen, cardiac monitor
- Spine immobilization if possibility of trauma
- “Coma cocktail”:
 - Dextrose
 - Naloxone
 - Thiamine
- Look for signs of an underlying cause:
 - Medications, medic alert bracelets
 - Document a basic neurologic exam, GCS, pupils, extremity movements
 - Gross signs of trauma
- CONTROVERSIES
 - Empirical dextrose should not be withheld or delayed if Dextrostix is not available
 - Glucose can be safely administered before thiamine.

INITIAL STABILIZATION/THERAPY

- IV D₅₀
- Naloxone

- Thiamine

ED TREATMENT/PROCEDURES

- Consider empiric use of antibiotics for altered mental status of undetermined etiology:
 - Broad spectrum with good CSF fluid penetration such as ceftriaxone and vancomycin
- Empiric treatment if a toxic ingestion is suspected:
- Correct body temperature:
- Specific therapy directed at underlying cause

MEDICATION

- Ceftriaxone: 2 g (peds: 50–75 mg/kg/d q12–24h) IV q12–24h
- Dextrose: 1–2 mL/kg of D₅₀W (peds: 2–4 mL/kg D₂₅W) IV
- Diazepam: 0.1–0.3 mg/kg slow IV (max 10 mg/dose) q10–q15min × 3 doses
- Lorazepam: 0.05–0.1 mg/kg IV (max. 4 mg/dose q10–q15min)
- Mannitol: 0.5–1 g/kg IV
- Naloxone: 0.01–0.1 mg/kg IV/IM/SC/ET
- Thiamine: 100 mg IM or 100 mg thiamine in 1,000 mL of IV fluid wide open
- Vancomycin: 1 g (peds: 10 mg/kg q8–12h) IV q12h



FOLLOW-UP

DISPOSITION

Admission Criteria

All patients with acute and persistent changes in mental status require admission.

Discharge Criteria

- Treated hypoglycemia related to insulin therapy with resolved symptoms
- Chronic altered mental status (e.g., dementia) without change from baseline
- Acute drug intoxication with return of patient's mental status to baseline with observation and drug has no potential for delayed toxicity

FOLLOW-UP RECOMMENDATIONS

Primary care follow-up to manage etiology which led to altered mental status (i.e., adjust medication dosing, drug abuse treatment referral)

PEARLS AND PITFALLS

- Consider reversible causes
 - Hypoglycemia (check glucose, give dextrose)

- Opiate overdose (trial of naloxone)
- Thiamine deficiency (trial of thiamine)
- Consider head CT for any patient with unclear etiology or neurologic abnormality
- Consider empiric antibiotics in patients with fever or unclear etiology

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CODES

ICD9

- 298.9 Unspecified psychosis
- 780.97 Altered mental status

ICD10

- R41.0 Disorientation, unspecified
- R41.82 Altered mental status, unspecified

AMEBIASIS

Ben Osborne • Joel C. Miller

BASICS

DESCRIPTION

- Invasive parasitic infection with both intestinal and extraintestinal manifestations
- Endemic worldwide, especially areas with poor sanitation
- Populations at risk:
 - _ Travelers to, citizens of, and immigrants from endemic areas
 - _ Institutionalized persons
 - _ Practitioners of oral–anal sexual activity
 - _ Men who have sex with men (MSM)
 - _ HIV infected individuals
- Risk factors for increased severity of disease and complications:
 - _ Immunocompromised: Corticosteroid use, HIV infection, malnutrition, malignancy
 - _ Pregnancy/postpartum state
 - _ Extremes of age

ETIOLOGY

- *Entamoeba histolytica*, an anaerobic, nonflagellated protozoa
- Fecal–oral transmission:
 - _ Humans are sole reservoir.
- Ingested organisms cause invasive colitis.
- Extraintestinal spread is hematogenous.

DIAGNOSIS

SIGNS AND SYMPTOMS

- Intestinal disease:
 - _ Onset 1 wk to 1 mo postexposure
 - _ Acute diarrhea (nondysenteric colitis):
 - 80% of cases
 - Afebrile
 - Occult blood in stool
 - Benign abdominal exam
 - _ Classic dysentery:
 - Bloody mucoid diarrhea
 - Abdominal pain/benign abdominal exam
 - Tenesmus

- Weight loss
- Fever (rare)
- Fulminant colitis:
 - Toxic-appearing patient
 - Rigid abdomen (25%)
 - Fever
 - Severe bloody diarrhea
 - Rapid progression to perforated bowel and frank peritonitis
 - >40% mortality
- Toxic megacolon:
 - Toxic-appearing patient
 - Profuse diarrhea (>10 stools per day)
 - Fever
 - Distended, tympanitic abdomen with signs of peritonitis
 - Associated with corticosteroid use
 - High mortality
- Ameboma:
 - Intraluminal granulated mass
 - Tender palpable mass on exam
- Amebic strictures:
 - Owing to chronic inflammation/scarring
 - Crampy abdominal pain
 - Nausea and vomiting (may be feculent)
 - Partial or complete bowel obstruction
- Chronic amebic colitis:
 - Mild recurrent episodes of bloody diarrhea, abdominal cramping, and tenesmus
 - Weight loss
 - May persist for years
- Extraintestinal disease:
 - Amebic liver abscess:
 - Most frequent extraintestinal manifestation (3–9% of cases)
 - Single abscess in right lobe (50–80%)
 - May develop months to years postexposure (median of 3 mo)
 - Fever
 - Right upper quadrant pain
 - Hepatomegaly with point tenderness
 - Rales at right lung base
 - Concurrent diarrhea unusual (20–33%)
 - Complication: Rupture into pleural cavity (10–20%), peritoneum, or pericardium (rare)
 - Increased risk of rupture if >5 cm in diameter or left lobe location
 - Extrahepatic amebic abscess:

- Brain
- Lung
- Perinephric
- Splenic
- Vaginal/cervical/uterine
- _ Cutaneous amebiasis:
 - Perineum and genitalia
 - Painful, irregularly shaped ulcers
 - Purulent exudate

Pediatric Considerations

Fulminant colitis is more likely

Pregnancy Considerations

Fulminant colitis is more likely

History

- Possible sources of exposure
- Membership in high-risk group

Physical-Exam

- Identify evidence of peritonitis, sepsis, or shock.
- Tender abdominal mass mandates workup for liver abscess or ameboma.
- Digital rectal exam shows gross or occult blood in >70% of patients.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC:
 - _ Leukocytosis in amebic liver abscess and peritonitis
- Alkaline phosphatase and ALT:
 - _ Elevated in amebic liver abscess
- Serum electrolytes, BUN/creatinine if prolonged diarrhea or evidence of dehydration
- Stool PCR is diagnostic gold standard:
 - _ 100% sensitive and specific
- Stool ELISA for *E. histolytica*-specific antigen:
 - _ 74–95% sensitive, 93–100% specific
- Serum for anti-*E. histolytica* antibodies:
 - _ Essential if suspecting liver abscess. These patients rarely shed parasites in stool
 - _ 90–100% sensitive in amebic liver abscess

- 70–90% sensitive in amebic colitis
- Stool microscopy is <60% sensitive and no longer the test of choice.
- Fecal leukocytes and culture:
 - Rule out infection of enteroinvasive bacteria;
 - Negative in amebiasis

Imaging

- Abdominal US:
 - 58–90% sensitive for liver abscess
 - Sensitivity influenced by size and location
 - Evaluate abscess for increased risk of rupture (>5 cm or located in left lobe)
- Abdominal CT or MRI:
 - Equivalent to US for delineating liver abscesses
 - Superior to US for detecting abscesses in other organs
- Head CT or MRI:
 - Suspect amebic brain abscess if patient with known amebiasis has altered mental status or focal neurologic findings.
 - Irregular nonenhancing lesions
- CXR:
 - Elevated right hemidiaphragm and/or right pleural effusion in liver abscess

Diagnostic Procedures/Surgery

- Colonoscopy with biopsy provides definitive diagnosis of amebic dysentery, colitis, ameboma, and amebic stricture.
- Percutaneous fine-needle aspiration of liver abscess to exclude bacterial abscess if nondiagnostic serology or antiamebic therapy fails
 - Not for primary treatment of liver abscesses

DIFFERENTIAL DIAGNOSIS

- Intestinal amebiasis:
 - Enteroinvasive bacterial infection (*Staphylococcus*, *E. coli*, *Shigella*, *Salmonella*, *Yersinia*, *Campylobacter*)
 - Inflammatory bowel disease
 - Ischemic colitis
 - Arteriovenous malformation
 - Abdominal aortic aneurysm
 - Perforated duodenal ulcer
 - Intussusception, diverticulitis
 - Pancreatitis
 - Colorectal carcinoma
- Amebic abscess:
 - Bacterial abscess

- Tuberculous cavity
- Echinococcal cyst
- Malignancy
- Cholecystitis
- Cutaneous amebiasis:
 - Carcinoma
 - STDs (condyloma acuminata, chancroid, syphilis)



TREATMENT

INITIAL STABILIZATION/THERAPY

- Airway, breathing, circulation (ABCs)
- IV 0.9% NS if signs of significant shock

ED TREATMENT/PROCEDURES

- Oral fluids if mild; IV if moderate/severe dehydration
- Avoid antidiarrheal agents.
- Correct serum electrolyte imbalances.
- Stool sample for *E. histolytica* PCR or ELISA, plus serology for anti-*E. histolytica* antibodies
- If stool or serum is positive for *E. histolytica*:
 - Metronidazole or tinidazole is 1st-line drug for systemic amebiasis (90% cure rate)
 - Chloroquine is an alternative systemic agent
 - Always follow systemic therapy with a luminal agent to eradicate intestinal colonization (erythromycin, iodoquinol, nitazoxanide, paromomycin, or tetracycline).
 - Do not use the luminal agents alone
- If stool or serum is negative for *E. histolytica*:
 - Refer to gastroenterologist for colonoscopy with biopsy.
 - Repeat serology in 7 days.
 - Consider empiric course of metronidazole if high suspicion for amebiasis and patient is critically ill.
- If evidence of peritonitis or sepsis:
 - Add IV antibiotic directed against anaerobic and gram-negative bacteria.
 - Surgery if toxic megacolon or perforation
- If liver abscess is suspected:
 - US or CT of hepatobiliary system with concurrent amebic serology
 - If imaging demonstrates an abscess but serology is negative, treat with amebicides and repeat serology in 7 days.
 - Consider abscess drainage by surgeon or interventional radiologist in conjunction with amebicidal therapy.

- If symptoms do not improve after 5–7 days of empiric amebicidal therapy, consider fine-needle aspiration to rule out bacterial abscess or hepatoma.

Pregnancy Considerations

- Use metronidazole with caution in 1st-trimester pregnancy, but do not withhold if patient has fulminant colitis or amebic abscess.
- Use erythromycin or nitazoxanide as intestinal amebicides along with metronidazole.
- Erythromycin or nitazoxanide may be used alone for mild dysentery in 1st-trimester pregnancy.
- Chloroquine, iodoquinol, paromomycin, tetracycline, and tinidazole are contraindicated.

MEDICATION

First Line

- Metronidazole: 500–750 mg (peds: 30–50 mg/kg/24 h) PO/IV q8h for 5–10 d
- Tinidazole: 2 g/d (peds: 50–60 mg/kg/d) PO for 3–6 d. For children older than 3 yr

Second Line

- Chloroquine: 1,000 mg/d PO for 2 d then 500 mg/d PO for 14 d; or 200 mg IM for 10–12 d
- Erythromycin: 250–500 mg (peds: 30–50 mg/kg/24 h) PO q6h for 10–14 d
- Iodoquinol: 650 mg PO q8h for 20 d
- Nitazoxanide: 500 mg PO q12. for 3 d (10 d if liver abscess) for children > 12 yr
- Paromomycin: 500 mg (peds: 25–30 mg/kg/24 h) PO q8h for 5–10 d
- Tetracycline: 250–500 mg (peds[> 12 yr]: 25–50 mg/kg/24 h) PO q6h for 10 d

Pediatric Considerations

- Chloroquine and iodoquinol are contraindicated.
- Tetracycline contraindicated in children < 8 yr

Pregnancy Considerations

Use erythromycin or nitazoxanide only.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Shock, sepsis, or peritonitis
- Hypotension or tachycardia unresponsive to IV fluids

- Children with > 10% dehydration
- Severe electrolyte imbalance
- Patients unable to maintain adequate oral hydration:
 - Extremes of age, cognitive impairment, significant comorbid illness
- Fulminant colitis or toxic megacolon
- Bowel obstruction
- Extraintestinal abscesses
- Failure of outpatient regimen

Discharge Criteria

- Nontoxic presentation of acute or chronic dysentery
- Able to maintain adequate oral hydration and medication compliance
- Dehydration responsive to IV fluids

Issues for Referral

Consult surgery if evidence of peritonitis, toxic megacolon, colonic perforation, or liver abscess.

FOLLOW-UP RECOMMENDATIONS

- Gastroenterology and infectious disease follow-up in 7 days for repeat serology and possible endoscopic evaluation.
- Physical exam in 14 days to assess for treatment effectiveness and for development of complications or extraintestinal disease.

PEARLS AND PITFALLS

- Avoid antidiarrheal medications
- Always give double therapy with both a systemic amebicidal (metronidazole, tinidazole, or chloroquine) plus an intestinal amebicidal (erythromycin, iodoquinol, nitazoxanide, paromomycin, or tetracycline) unless contraindicated.
- Always be vigilant for high-mortality complications such as fulminant colitis or extraintestinal disease.

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See Also (Topic, Algorithm, Electronic Media Element)

- Diarrhea
- Gastroenteritis



CODES

ICD9

- 006.0 Acute amebic dysentery without mention of abscess
- 006.1 Chronic intestinal amebiasis without mention of abscess
- 006.9 Amebiasis, unspecified

ICD10

- A06.0 Acute amebic dysentery
- A06.1 Chronic intestinal amebiasis
- A06.9 Amebiasis, unspecified

AMENORRHEA

Andrew J. French

BASICS

DESCRIPTION

- Absence of menstruation
- Primary amenorrhea:
 - No spontaneous uterine bleeding by age 16 yr or within 5 yr of breast development, which should occur by age 13.
- Secondary amenorrhea:
 - Absence of uterine bleeding for 3 mo in a woman with prior regular menses or for 9 mo in a woman with prior oligomenorrhea
 - More common than primary amenorrhea
 - Pregnancy is the most common cause.

ETIOLOGY

- Primary:
 - Gonadal failure
 - Hypothalamic-pituitary disorder
 - Chromosomal abnormalities
 - Imperforate hymen
 - Turner syndrome
- Secondary:
 - Pregnancy, breast-feeding, or postpartum
 - Asherman syndrome (intrauterine adhesions)
 - Dysfunction of the hypothalamic-pituitary-ovarian axis
 - Polycystic ovarian syndrome (PCOS)
 - Endocrinopathies
 - Obesity, starvation, anorexia nervosa, or intense exercise
 - Drugs:
 - Oral contraceptives
 - Antipsychotics
 - Antidepressants
 - Calcium channel blockers
 - Chemotherapeutic agents
 - Digitalis
 - Marijuana
 - Autoimmune disorders
 - Ovarian failure
 - Menopause

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Menarche and menstrual history
- Sexual activity
- Exercise, weight loss
- Chronic illness
- Medications
- Previous CNS radiation or chemotherapy
- Family history
- Infertility

Physical-Exam

- Low estrogen:
 - Atrophic vaginal mucosa
 - Mood swings, irritability
- High androgen:
 - Truncal obesity
 - Hirsutism
 - Acne
 - Male-pattern baldness
- Thyroid exam
- Pelvic/genital exam
- Tanner staging

ESSENTIAL WORKUP

Pregnancy test

DIAGNOSIS TESTS & INTERPRETATION

Lab

- If pregnancy test is negative, no further testing is needed emergently.
- May send TSH, prolactin, LH, FSH for follow-up by gynecology or primary care physician

Imaging

None needed emergently unless concern for ectopic pregnancy or other emergency as directed by patient's presentation

Diagnostic Procedures/Surgery

None needed emergently

DIFFERENTIAL DIAGNOSIS

Pregnancy



TREATMENT

PRE HOSPITAL

If amenorrhea is the result of pregnancy, stabilize patient as appropriate for pregnancy.

ED TREATMENT/PROCEDURES

Reassurance and referral for follow-up

MEDICATION

Defer for gynecology evaluation.



FOLLOW-UP

DISPOSITION

Admission Criteria

No need for admission unless concern for ectopic pregnancy

Discharge Criteria

Discharge with appropriate referral.

Issues for Referral

Referral to gynecology

FOLLOW-UP RECOMMENDATIONS

Gynecology follow-up is recommended.

PEARLS AND PITFALLS

- Pregnancy is the most relevant etiology of amenorrhea in the emergency department.
 - Urine pregnancy test (UPT) may give false negative with low urine specific gravity.
 - UPT sensitivity for β -HCG level may vary depending on type/manufacturer. High concern for amenorrhea due to pregnancy, specifically an ectopic, may warrant a qualitative serum pregnancy test
- Anorexia nervosa is an important consideration in patients with amenorrhea,

particularly in adolescents.

ADDITIONAL READING

- Heiman DL. Amenorrhea. *Prim Care Clin Office Pract.* 2009;36:1–17.
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- Rosenberg HK. Sonography of the pelvis in patients with primary amenorrhea. *Endocrinol Metab Clin N Am.* 2009;38:739–760.
- Santoro N. Update in hyper- and hypogonadotropic amenorrhea. *J Clin Endocrinol Metab.* 2011;96:3281–3288.



CODES

ICD9

- 256.8 Other ovarian dysfunction
- 626.0 Absence of menstruation

ICD10

- N91.0 Primary amenorrhea
- N91.1 Secondary amenorrhea
- N91.2 Amenorrhea, unspecified

AMPHETAMINE POISONING

James W. Rhee



BASICS

DESCRIPTION

- Increased release of norepinephrine, dopamine, and serotonin
- Decreased catecholamine reuptake
- Direct effect on α - and β -adrenergic receptors

ETIOLOGY

- Prescription drugs:
 - _ Amphetamine (Benzedrine)
 - _ Dextroamphetamine (Dexedrine)
 - _ Diethylpropion (Tenuate)
 - _ Fenfluramine (Pondimin)
 - _ Methamphetamine
 - _ Methylphenidate (Ritalin)
 - _ Phenmetrazine (Preludin)
 - _ Phentermine
- “Designer drugs”:
 - _ Variants of illegal parent drugs
 - _ Often synthesized in underground labs
 - _ “Crystal,” “Ice”:
 - Crystalline methamphetamine hydrochloride
 - Smoked, insufflated, or injected
 - Rapid onset; duration several hours
 - _ “Crank”
 - _ “Ecstasy” (3,4-methylenedioxymethamphetamine, MDMA, XTC, E):
 - Often used at dances and “rave” parties
 - Dehydration can lead to hyperthermia, hyponatremia, fatality
 - _ MDA (3,4-methylenedioxyamphetamine)
 - _ Methcathinone (“cat,” “Jeff,” “mulka”):
 - Derivative of cathinone, found in the evergreen tree *Catha edulis*
 - Frequently synthesized in home labs
 - Does not show up on urine toxicology screens
 - _ Mephedrone
 - May be contained in “bath salts”



DIAGNOSIS

SIGNS AND SYMPTOMS

- CNS:
 - Agitation
 - Delirium
 - Hyperactivity
 - Tremors
 - Dizziness
 - Mydriasis
 - Headache
 - Choreoathetoid movements
 - Hyperreflexia
 - Cerebrovascular accident
 - Seizures and status epilepticus
 - Coma
- Psychiatric:
 - Euphoria
 - Increased aggressiveness
 - Anxiety
 - Hallucinations (visual, tactile)
 - Compulsive repetitive actions
- Cardiovascular:
 - Palpitations
 - Hypertensive crisis
 - Tachycardia or (reflex) bradycardia
 - Dysrhythmias (usually tachydysrhythmias)
 - Cardiovascular collapse
- Other:
 - Rhabdomyolysis
 - Myoglobinuria
 - Acute renal failure
 - Anorexia
 - Diaphoresis
 - Disseminated intravascular coagulation (DIC)

History

- Determine the type, amount, timing, and route of amphetamine exposure
- Assess for possible coingestions
- Evaluate for symptoms of end organ injury:
 - Chest pain
 - Shortness of breath
 - Headache, confusion, and vomiting

Physical-Exam

- Common findings include:
 - Agitation
 - Tachycardia
 - Diaphoresis
 - Mydriasis
- Severe intoxication characterized by:
 - Tachycardia
 - HTN
 - Hyperthermia
 - Agitated delirium
 - Seizures
 - Diaphoresis
- Hypotension and respiratory distress may precede cardiovascular collapse
- Evaluate for associated conditions:
 - Cellulitis and soft tissue infections
 - Diastolic cardiac murmurs or unequal pulses
 - Examine carefully for trauma
 - Pneumothorax from inhalation injury
 - Focal neurological deficits

ESSENTIAL WORKUP

- Vital signs:
 - Temperature > 40°C:
 - Core temperature recording essential
 - Peripheral temperature may be cool
 - Indication for urgent cooling
 - Ominous prognostic sign
 - BP:
 - Severe hypertension can lead to cardiac and neurologic abnormalities.
 - Late in course, hypotension may supervene due to catecholamine depletion
- ECG:
 - Signs of cardiac ischemia
 - Ventricular tachydysrhythmias
 - Reflex bradycardia

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Urinalysis:
 - Blood
 - Myoglobin

- Electrolytes, BUN/creatinine, glucose:
 - Hypoglycemia may contribute to altered mental status.
 - Acidosis may accompany severe toxicity.
 - Rhabdomyolysis may cause renal failure.
 - Hyperkalemia—life-threatening consequence of acute renal failure
- Coagulation profile to monitor for potential DIC:
 - INR, PT, PTT, platelets
- Creatine phosphokinase (CPK):
 - Markedly elevated in rhabdomyolysis
- Urine toxicology screen:
 - For other toxins with similar effects (e.g., cocaine)
 - Some amphetamine-like substances (e.g., methcathinone) may not be detected.
- Aspirin and acetaminophen levels if suicide attempt is a possibility
- Arterial blood gas (ABG)

Imaging

- Chest radiograph:
 - Adult respiratory distress syndrome
 - Noncardiogenic pulmonary edema
- Head CT for:
 - Significant headache
 - Altered mental status
 - Focal neurologic signs
 - For subarachnoid hemorrhage, intracerebral bleed

Diagnostic Procedures/Surgery

Lumbar puncture for:

- Suspected meningitis (headache, altered mental status, hyperpyrexia)
- Suspected subarachnoid hemorrhage and CT normal

DIFFERENTIAL DIAGNOSIS

- Sepsis
- Thyroid storm
- Serotonin syndrome
- Neuroleptic malignant syndrome
- Pheochromocytoma
- Subarachnoid hemorrhage
- Drugs that cause delirium:
 - Anticholinergics:
 - Belladonna alkaloids
 - Antihistamines
 - Tricyclic antidepressants

- Cocaine
- Ethanol withdrawal
- Sedative/hypnotic withdrawal
- Hallucinogens
- Phencyclidine
- Drugs that cause HTN and tachycardia:
 - Sympathomimetics
 - Anticholinergics
 - Ethanol withdrawal
 - Phencyclidine
 - Caffeine
 - Phenylpropanolamine
 - Ephedrine
 - Monoamine oxidase inhibitors
 - Theophylline
 - Nicotine
- Drugs that cause seizures:
 - Carbon monoxide
 - Carbamazepine
 - Cyanide
 - Cocaine
 - Cholinergics (organophosphate insecticides)
 - Camphor
 - Chlorinated hydrocarbons
 - Ethanol withdrawal
 - Sedative/hypnotic withdrawal
 - Isoniazid
 - Theophylline
 - Hypoglycemics
 - Lead
 - Lithium
 - Local anesthetics
 - Anticholinergics
 - Phencyclidine
 - Phenothiazines
 - Phenytoin
 - Propoxyphene
 - Salicylates
 - Strychnine



TREATMENT

PRE HOSPITAL

- Patient may be uncooperative or violent.
- Secure IV access.
- Protect from self-induced trauma.

INITIAL STABILIZATION/THERAPY

- ABCs
- Establish IV 0.9% NS access.
- Cardiac monitor
- Naloxone, dextrose (or Accu-Chek), and thiamine if altered mental status

ED TREATMENT/PROCEDURES

- Decontamination:
 - _ Administration of activated charcoal
 - _ Whole-bowel irrigation with polyethylene glycol solution for body packers
- Hypertensive crisis:
 - _ Initially administer benzodiazepines if agitated.
 - _ α -blocker (phentolamine) as second-line agent
 - _ Nitroprusside for severe, unresponsive hypertension
 - _ Avoid β -blockers, which may exacerbate hypertension.
- Agitation, acute psychosis:
 - _ Administer benzodiazepines.
- Hyperthermia:
 - _ Benzodiazepines if agitated
 - _ Active cooling if temperature $> 40^{\circ}\text{C}$:
 - Tepid water mist
 - Evaporate with fan.
 - _ Paralysis:
 - Indicated if muscle rigidity and hyperactivity contributing to persistent hyperthermia
 - Nondepolarizing agent (e.g., vecuronium)
 - Avoid succinylcholine.
 - Intubation; mechanical ventilation
 - _ Apply cooling blankets.
- Rhabdomyolysis:
 - _ Administer benzodiazepines.
 - _ Hydrate with 0.9% NS.
 - _ Maintain urine output at 1–2 mL/min.
 - _ Hemodialysis (if acute renal failure and hyperkalemia occur)
- Seizures:
 - _ Maintain airway.
 - _ Administer benzodiazepines.
 - _ Phenobarbital if unresponsive to benzodiazepines

- Phenytoin contraindicated
- Hypotension:
 - May be late finding due to catecholamine depletion
 - Initially bolus with isotonic crystalloid solution
 - If no response, administer norepinephrine.
 - Dopamine may not be effective

MEDICATION

- Activated charcoal: 1–2 g/kg up to 100 g PO
- Dextrose: D₅₀W 1 amp: 50 mL or 25 g (peds: D₂₅W 2–4 mL/kg) IV
- Diazepam (benzodiazepine): 5–10 mg (peds: 0.2–0.5 mg/kg) IV
- Lorazepam (benzodiazepine): 2–6 mg (peds: 0.03–0.05 mg/kg) IV
- Nitroprusside: 1–8 µg/kg/min IV (titrated to BP)
- Phenobarbital: 15–20 mg/kg at 25–50 mg/min until cessation of seizure activity
- Phentolamine: 1–5 mg IV over 5 min (titrated to BP)
- Vecuronium: 0.1 mg/kg IVP



FOLLOW-UP

DISPOSITION

Admission Criteria

- Hyperthermia
- Persistent altered mental status
- Hypertensive crisis
- Seizures
- Rhabdomyolysis
- Persistent tachycardia

Discharge Criteria

- Asymptomatic after 6 hr observation
- Absence of the above admission criteria

FOLLOW-UP RECOMMENDATIONS

Patients may need referral for chemical dependency rehab and detoxification.

PEARLS AND PITFALLS

- Admit patients with severe or persistent symptoms.
- Monitor core temperature:
 - Hyperthermia > 40°C may be life threatening.
 - Treat with aggressive sedation and active cooling.

- Recognize rhabdomyolysis and hyperkalemia.
- Avoid physical restraints in agitated patients if possible.
- Consider associated emergency conditions:
 - Patients with chest pain should be evaluated for acute coronary syndromes and treated accordingly.
 - Consider infection in altered patients with fever and history of IV drug use.
 - Methamphetamine abuse frequently associated with traumatic injury
- Benzodiazepines are 1st-line therapy in symptomatic methamphetamine intoxication

ADDITIONAL READING

- Callaway CW, Clark RF. Hyperthermia in psychostimulant overdose. *Ann Emerg Med.* 1994;24:68–75.
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- Gray SD, Fatovich DM, McCoubrie DL, et al. Amphetamine-related presentations to and inner-city tertiary emergency department: A prospective evaluation. *Med J Aust.* 2007;186:336.
- Prosser JM, Nelson LS. The toxicology of bath salts: a review of synthetic cathinones. *J Med Toxicol.* 2012;8:33–42.
- Turnipseed SD, Richards JR, Kirk JD, et al. Frequency of acute coronary syndrome in patients presenting to the emergency department with chest pain after methamphetamine use. *J Emerg Med.* 2003;24(4):369–373.



See Also (Topic, Algorithm, Electronic Media Element)

- Sympathomimetic Poisoning
- Tricyclic Antidepressant Poisoning



CODES

ICD9

- 969.72 Poisoning by amphetamines
- 969.73 Poisoning by methylphenidate
- 969.79 Poisoning by other psychostimulants

ICD10

- T43.601A Poisoning by unsp psychostim, accidental, init
- T43.621A Poisoning by amphetamines, accidental (unintentional), init
- T43.631A Poisoning by methylphenidate, accidental, init

AMPUTATION TRAUMATIC/REPLANTATION

Charlotte A. Sadler

BASICS

DESCRIPTION

- Partial amputations have tissue connecting the distal and proximal parts and are treated by revascularization.
- Complete amputations have no connecting tissue and may be treated by replantation.
- Both of the above are treated the same from an emergency standpoint.

ETIOLOGY

Traumatic amputations may result from machinery, powered hand tools, household appliances, lawnmowers, getting caught between objects, motor vehicle collisions, crush injuries, blast injuries, gunshot wounds, knives, degloving injuries to digits (ring avulsions), and animal bites.

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Exact time of injury is critical, as ischemia time predicts success for replantation:
 - Irreversible muscle necrosis begins at 6 hr of ischemia.
 - The temperature and amount of muscle present in the tissue predicts the tolerable ischemia time.
 - Amputated digits have little muscle and can tolerate more ischemia time:
 - Warm ischemia time of 8–12 hr
 - Cool ischemia time of as much as 24 hr
 - Amputated limbs have more muscle mass and can tolerate less ischemia time:
 - Warm ischemia time of 4–6 hr
 - Cold ischemia time of 10–12 hr
- Mechanism of injury:
 - Clean-cut or “guillotine” amputations have better prognosis for replantation than crush or avulsion injuries.
- Comorbid illnesses that hinder successful replantation:
 - Diabetes, peripheral vascular disease, rheumatologic disease, smoking
- Handedness
- Social history, including occupation and major hobbies

Physical-Exam

- Assessment and documentation of injured extremity is crucial.
- Signs of neurologic compromise:
 - Loss of sensation and 2-point discrimination
 - Loss of active range of motion
- Signs of vascular compromise in partial amputations:
 - Distal part dusky or cyanotic
 - Delayed capillary refill (> 2 sec)
 - Diminished or absent pulses (Doppler or palpation)
 - Ribbon sign (twisting of the artery in the amputated digit or limb)
 - Use Allen test in hand injuries.
 - Pulse oximetry may be helpful.
- Soft tissue: Assess skin, muscle, tendon, and nail bed integrity.
- Identify exposed bone and fractures (gross deformity, tenderness, crepitus).

ESSENTIAL WORKUP

ED workup includes obtaining an accurate history and physical exam, stabilizing the patient and injured part, and consultation or transfer if replantation is an option.

DIAGNOSIS TESTS & INTERPRETATION

Lab

Preoperative lab studies, cultures from wounded area

Imaging

Radiographs of both amputated part and stump are important, but should not delay transport.

Diagnostic Procedures/Surgery

Determined by surgical consultant for replantation

DIFFERENTIAL DIAGNOSIS

- Involves neurologic, vascular, and soft tissue integrity and potential for replantation/revascularization
- Do not miss other major injuries with concurrent trauma.



TREATMENT

PRE HOSPITAL

- Collect all amputated body parts, including pieces of bone, tissue, and skin.
- See “Initial Stabilization” for care of amputated parts during transport.
- Transport patient and body parts to the nearest microvascular replantation center

unless other major injuries require transport to the nearest trauma center:

- Air transport from remote locations should be considered if ischemia time is of concern.

INITIAL STABILIZATION/THERAPY

- Consult surgical specialist as early as possible.
- Establish IV access.
- Limit blood loss:
 - Elevate injured limb.
 - Direct pressure using bulky pressure dressing or pressure points if ineffective.
 - Use tourniquet if above methods fail to give desired hemostasis (BP cuff 30 mm Hg > systolic BP [SBP]).
 - Partial amputations bleed more because of lack of both retraction and spasm of blood vessels.
- Avoid further damage to injured part:
 - Avoid vascular clamps, cautery, vessel ligation, or debridement.
 - Avoid repeated exams of the stump or amputated part.
- Care of amputated part (complete and partial):
 - Remove gross contamination/foreign material.
 - Gently irrigate with saline (avoid antiseptics).
 - Wrap in gauze moistened with saline.
 - Place in clean, dry plastic bag or specimen cup.
 - Place sealed bag/cup in ice water (half water, half ice) or refrigerate at 4°C.
 - Never place directly onto ice or into ice water.
 - Avoid dry ice to prevent freezing.
- Care of the stump:
 - Irrigate with saline and cover with saline dampened gauze.
 - Splint if necessary; keep partial amputations as near anatomic position as possible.
 - Keep any fragments of tissue (even if seemingly insignificant) because they may be used for skin, bone, or nerve grafting
 - If limb amputation, may cannulate proximal artery with 18G cannula and irrigate with tissue preservation formula, but this should be at the discretion of the surgeon
- Maintain normal blood volume with IV fluids or blood products if necessary.

ED TREATMENT/PROCEDURES

- Tetanus prophylaxis
- Adequate IV analgesia
- Patient NPO
- Prophylactic antibiotics if devitalized tissue, exposed bone, or contamination:
 - Cover *Streptococcus*, *Staphylococcus aureus*, and *Clostridium perfringens*

- All patients are candidates for surgical repair until a specialist deems otherwise.
- Limit ischemia time of the amputated part (i.e., early transfer if necessary).
- Patient considerations in decision to replant:
 - Age
 - Occupation/handedness
 - Degree of motivation
 - General physical condition and underlying diseases, particularly diabetes mellitus, peripheral vascular disease
- Indications for replantation (no absolute indications):
 - Thumb, any level (supplies 40% of hand function)
 - Multiple digits
 - Hand amputations through the palm and distal wrist
 - Individual digit distal to flexor digitorum superficialis tendon insertion and proximal to distal interphalangeal joint (DIP)
 - Some single-digit ring avulsion injuries
 - Arm proximal to midforearm (if sharp or moderately avulsed)
 - Virtually all pediatric amputations (younger patients have lower success rates but better functional outcomes)
- Contraindications to replantation:
 - Severely crushed or mangled parts
 - Injuries at multiple levels
 - Psychotic patients who willfully self-amputated the part
 - Single-digit amputations proximal to the flexor digitorum superficialis muscle insertion
 - Amputated parts with tendons avulsed from musculotendinous junctions
 - Lower extremities rarely attempted and usually in children
 - Unstable patients secondary to other serious injuries or diseases
 - Older patients or those with contraindications to general anesthesia
 - Inappropriately prolonged ischemia time
- Fingertip amputations: Most common type of upper extremity amputation:
 - Distal to DIP joint
 - Primary goals of treatment:
 - Maintenance of length
 - Good soft-tissue coverage
 - Painless fingertip with durable and sensate skin
 - Nail preservation
 - Better dorsal prognosis than ventral
 - No exposed phalanx:
 - Irrigate with saline, apply petrolatum-soaked gauze and allow to heal by secondary intention (best result in wounds < 1 cm²).
 - Small amount of exposed phalanx:
 - Shorten bone with rongeur below level of the tissue and close by

- primary intention or allow to heal by secondary intention.
 - Any bone left exposed requires additional operative procedures and consultation.
 - Replantation is an option for cosmetic reasons or for occupational consideration (e.g., musicians).
 - Considered open fractures if phalanx exposed, thus antibiotics are indicated.
 - Preserve nail bed and nail to optimize function and cosmesis.
 - Treat subungual hematomas.
 - Splint to prevent trauma to healing fingertip.
 - Consultation required if significant loss of bone or soft tissue for possible graft or flap
- Nonlimb amputations (penis, ear, nose): Amputated parts should be cared for similarly as above and emergently referred to a specialist for replantation:
 - Penile amputations: Most often secondary to self-mutilation and psychiatric illness
 - Successful replantation unlikely beyond 24 hr of cold ischemia or 6 hr of warm ischemia
 - Ear amputations: Should be considered for replantation by appropriate specialist
 - Nose amputations: Replantation has been successfully performed with variable results.

Pediatric Considerations

- All pediatric amputations considered for replantation
- Fingertip amputations often left to heal by secondary intention:
 - Spontaneous regeneration of fingertip occurs in children even with volar fingertip amputations.
 - Pediatric fingertip amputations distal to the lunula of the fingernail can be successfully replanted (unlike adults).

Geriatric Considerations

Advanced age not an absolute contraindication to replantation; however, underlying medical problems often make older patients poor surgical candidates.

MEDICATION

- First Line: Cefazolin: 0.5–1.5 g IV or IM q6–q8h (peds: 25–100 mg/kg/d divided q8h, max. 6 g/d)
- Second Line: Vancomycin 15–20 mg/kg IV q12h
- If concerned about clostridia, consider using Piperacilin/Tazobactam 80 mg/kg IV q8h



FOLLOW-UP

DISPOSITION

Admission Criteria

Hospitalization is required for all patients undergoing replantation or revascularization.

Discharge Criteria

- Mild fingertip amputations or mild degloving injuries with adequate repair and stable vasculature
- Close surgical or orthopedic follow-up is required.

Issues for Referral

- Know exact mechanism and time of injury
- Refer as early as possible
- Transfer imaging and amputated parts with patient, stored in appropriate medium

FOLLOW-UP RECOMMENDATIONS

Patients discharged but with significant skin loss should be considered for skin grafting and have close surgical follow-up.

PEARLS AND PITFALLS

- Every effort should be made to minimize ischemia time
- Expeditious consultation or transfer to appropriate surgeon and team is paramount.
- Avoid any direct contact of the amputated part with ice
- Perform thorough ATLS survey to avoid missing other less obvious, but potentially life threatening, injuries

ADDITIONAL READING

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- Lyn ET, Mailhot T. Hand, Runyon M. The Genitourinary System; McKay M, Mayersak R, Facial Trauma. In: Marx J, Hockberger RS, Walls RM, et al., eds. *Rosen's Emergency Medicine: Concepts and Clinical Practice*, 7th ed. Philadelphia, PA: Mosby/Elsevier; 2010.
- Maricevich M, Carlsen B, Mardini S, et al. Upper extremity and digital replantation. *Hand.* 2011;6:356–363.
- Sebastin SJ, Chung KC. A systematic review of the outcomes of replantation of distal digital amputation. *Plast Reconstr Surg.* 2011;128(3):723–737.



CODES

ICD9

- 885.0 Traumatic amputation of thumb (complete)(partial), without mention of complication
- 886.0 Traumatic amputation of other finger(s) (complete) (partial), without mention of complication
- 887.4 Traumatic amputation of arm and hand (complete) (partial), unilateral, level not specified, without mention of complication

ICD10

- S68.019A Complete traumatic metacarpo-phalangeal amputation of unspecified thumb, initial encounter
- S68.119A Complete traumatic metacarpo-phalangeal amputation of unspecified finger, initial encounter
- S68.129A Partial traumatic metacarpophalangeal amputation of unspecified finger, initial encounter

AMYOTROPHIC LATERAL SCLEROSIS

Richard S. Krause

BASICS

DESCRIPTION

- Progressive, incurable disease of adults
- Neurodegenerative disease of the motor system at all levels
- Some patients have associated dementia
- Manifestations:
 - Muscle weakness
 - Wasting
 - Fasciculations
 - Babinski sign
 - Hyperreflexia
- Variants with predominately upper or lower motor neuron manifestations also occur
- May begin with bulbar symptoms of dysphagia and dysarthria
- Also known as “Lou Gehrig Disease” after the famous baseball player who was affected
- Eventually leads to respiratory compromise secondary to weakness of diaphragm and other muscles of respiration
- 80% of cases begin between ages 40 and 70 yr
- Death (usually from respiratory paralysis) typically occurs within 3–5 yr of the diagnosis
- 50% die within 3 yr
- ~ 10% ALS patients live 10 yr or more
- Males > females

ETIOLOGY

- Etiology of amyotrophic lateral sclerosis (ALS) is unknown
- ~ 10% of affected patients have another affected family member
- Cigarette smoking and heavy metal exposure may be risk factors
- There is a disease cluster in the western Pacific
- Pathologically, there is loss of both upper and lower motor neuron cells
- Predilection for the motor system and sparing of other neurons

DIAGNOSIS

SIGNS AND SYMPTOMS

- Asymmetric limb weakness is the most common presentation of ALS (80%)

- May begin in either the arms (cervical onset) or the legs (lumbar onset):
 - Later all limbs are affected
- Bulbar ALS:
 - Usually presents with dysarthria or dysphagia
 - 2nd most common presentation
- Both lower motor neuron (weakness and wasting with fasciculation) and upper motor neuron signs (Babinski sign with hyperreflexia) occur
- Respiratory muscles and the vocal cords are affected late
- Muscle fasciculation is common, but may not be apparent to the patient
- Extraocular muscles, sphincters, cognition, and sensation are spared

History

- Most ED patients with ALS will present with an established diagnosis
- History should focus on clues regarding acute medical issues and functional decline
- When ALS is suspected due to a complaint of “weakness” consider that this occurs with many illnesses including:
 - Pulmonary disease
 - Cardiac disease
 - Anemia
 - Endocrine disorders
 - Toxidromes
 - Diseases of muscles or joints
 - Spinal cord abnormalities
 - Depression
- Differentiate true weakness from: Shortness of breath, chest pain, joint pain, fatigue, poor exercise tolerance, etc.
- True weakness often leads to complaints of inability to perform specific tasks:
 - Bulbar palsy:
 - Facial weakness
 - Weakness and fasciculation of tongue
 - Dysarthria
 - Cervical onset ALS:
 - Difficulty with washing hair, using comb
 - Impaired pincer grip
 - Lumbar onset ALS:
 - Frequent trips secondary to foot drop
 - Difficulty walking up stairs

Physical-Exam

- A detailed and thorough neurological exam is the key to diagnosis but is not typically performed in the ED
- Upper motor neuron disease causes slow uncoordinated movements and stiffness

- Lower motor neuron disease causes weakness accompanied by atrophy and muscle cramps are common
- Common findings:
 - _ Brisk reflexes
 - _ Fasciculations
 - _ Muscle wasting
- Exam should focus on excluding or confirming other conditions

ESSENTIAL WORKUP

- Previously undiagnosed ALS:
 - _ Diagnosis of ALS is clinical and rarely made in the ED:
 - Recognition of the possibility of this disease is sufficient and mandates referral for workup
 - _ If ALS is suspected, forced vital capacity (FVC) should be performed
- Known ALS patient:
 - _ Patients with known disease and progressive symptoms:
 - Evaluate potentially treatable complications with lab and imaging studies
 - _ FVC is a sensitive indicator of respiratory muscle weakness:
 - FVC < 50% of predicted is considered a sign of advanced disease
 - FVC < 50% usually requires ventilatory support
 - Start with noninvasive positive pressure ventilation when possible
 - Compare with the patient's previous baseline
 - _ CXR may reveal aspiration or pneumonia or comorbid conditions such as CHF
 - _ Pulse oximetry and blood gas analysis aid in the diagnosis of respiratory failure
 - _ Electrolytes and other blood chemistry tests may reveal a treatable cause of increasing weakness

DIAGNOSIS TESTS & INTERPRETATION

Lab

- In cases of undifferentiated weakness, consider CPK measurement along with blood chemistry:
 - _ Elevated CPK is associated with myopathy
- Electrolyte abnormalities such as hypokalemia, hypercalcemia, etc. may cause generalized weakness but this is typically in association with other signs and symptoms
- CBC, UA may be indicated to look for source of infection

Imaging

Cervical spine, other skeletal radiography, or head CT may be needed in case of falls

(common in ALS) or to rule out other conditions

Diagnostic Procedures/Surgery

- Check FVC
- Electromyography (EMG) may help confirm the diagnosis (not usually an ED test)

DIFFERENTIAL DIAGNOSIS

- Cervical cord compression:
 - Similar symptoms but usually acute onset with pain and sensory changes
 - Spinal MRI or myelography for diagnosis
- Thyrotoxicosis may mimic ALS:
 - Usually marked systemic symptoms
- Heavy metal poisoning (lead, mercury, arsenic)
- Syphilis and Lyme disease
- Lymphoma may have an associated lower motor neuron syndrome, which mimics ALS
- Bulbar ALS:
 - Esophageal cancer
 - Myasthenia gravis



TREATMENT

- There is no specific therapy for ALS
- The drug riluzole, a glutamate release inhibitor, has been shown to extend survival in ALS patients for an average of a few months
- Treatment issues in the ED revolve around symptomatic therapy and identification and treatment of complications

PRE HOSPITAL

Controversies:

- Many patients will have advanced directives:
 - Unless immediate intervention is essential, intubation should be avoided until directives have been ascertained
 - Noninvasive means of ventilatory support may be tried first

INITIAL STABILIZATION/THERAPY

- Respiratory insufficiency or failure:
 - Ascertain any advanced directives
 - Noninvasive ventilatory support
 - Intubation as indicated
- Weaning off the ventilator is very difficult:
 - Average survival after institution of ventilation is 19 mo

ED TREATMENT/PROCEDURES

- Sedation and pain control as indicated:
 - Joint pain may respond to NSAIDs
- Insomnia from pressure pain (owing to immobility) may respond to diphenhydramine or amitriptyline
 - Insomnia may also be treated with benzodiazepines
- Aspiration or drooling may be treated with amitriptyline, atropine, or hyoscyamine (dries secretions)
- Muscle cramps may respond to baclofen or tizanidine
- Constipation is related to immobility and diet:
 - Treated with laxatives, stool softeners, and dietary changes

MEDICATION

- Amitriptyline: 25–100 mg PO QHS
- Atropine: 0.4 mg PO q4–6h
- Baclofen: 10–25 mg PO TID
- Diphenhydramine: 25–50 mg PO nightly at bedtime
- Tizanidine 2–4 mg PO BID



FOLLOW-UP

DISPOSITION

Admission Criteria

- Need for respiratory support
- Dehydration
- Unable to be cared for at home owing to progression of illness
- Complications (e.g., infection) or other diagnosis that requires admission

Discharge Criteria

- *Suspected ALS*: Refer for outpatient evaluation if general condition permits and other serious conditions requiring admission are ruled out
- *Complication of known ALS*: Discharge if outpatient treatment available and stable respiratory status

FOLLOW-UP RECOMMENDATIONS

If considering diagnosis of ALS, prompt follow-up with a neurologist should be arranged

PEARLS AND PITFALLS

- ALS is a progressive neurodegenerative disease affecting all components of the motor system
- Many patients with ALS have advanced directives—*inquire prior to any aggressive*

intervention

- FVC <50% usually requires ventilatory support.

ADDITIONAL READING

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CODES

ICD9

335.20 Amyotrophic lateral sclerosis

ICD10

G12.21 Amyotrophic lateral sclerosis

ANAL FISSURE

Julia H. Sone

BASICS

DESCRIPTION

- Hard stool passes and “cuts” anoderm
- Linear tear extends from dentate line to anoderm:
 - Posterior midline 95%
 - Anterior midline 5%
 - Externally: Forms skin tag or sentinel pile
 - Internally: Forms hypertrophied anal papilla
 - Chronic fissure may reveal fibers of internal sphincter with sentinel pile.

ETIOLOGY

- Stress or an overly tight anal sphincter leads to local ischemia of posterior anoderm.
- Diarrhea or hard bowel movement tears anoderm.
- Local trauma from anal intercourse or sexual abuse may be the cause.
- Lateral fissures indicate underlying causative systemic disease:
 - Crohn's disease
 - Anal cancer
 - Leukemia
 - Syphilis
 - Previous anal surgery

DIAGNOSIS

SIGNS AND SYMPTOMS

- Bright red blood per rectum usually on toilet paper
- Sharp, cutting, throbbing or burning pain with bowel movement:
 - May last for hours
- Constipation; unable to pass stool owing to pain:
 - Hard, nondeformable stools

History

- Passage of hard stool or constipation
- Episode(s) of diarrhea
- Bright red blood on toilet paper

Physical-Exam

Anal exam:

- Gently retract buttocks and have patient bear down to visualize the fissure.
- Severe pain usually prevents a manual or digital exam:
 - Use lidocaine jelly or ELA-Max5, a topical lidocaine ointment, before attempting digital rectal exam.
 - Need to exclude abscess or tumor

Pediatric Considerations

A clear test tube may be used as an anoscope to visualize the anal canal/fissure.

ESSENTIAL WORKUP

Careful rectal exam

DIAGNOSIS TESTS & INTERPRETATION

Lab

Hematocrit if severe bleeding by history

Imaging

CT pelvis:

- To exclude anal rectal abscess/tumor if palpable mass on rectal exam

DIFFERENTIAL DIAGNOSIS

- Crohn's disease
- Chronic ulcerative colitis
- Anorectal carcinoma
- Perirectal abscess
- Thrombosed hemorrhoid
- Sexual abuse
- TB
- Syphilis
- Lymphoma
- Leukemia
- Previous anal surgery



TREATMENT

PRE HOSPITAL

Establish IV access for patients with significant rectal bleeding.

INITIAL STABILIZATION/THERAPY

Administer pain medications for patients with significant pain.

ED TREATMENT/PROCEDURES

- IV/IM/PO pain medications:
 - NSAIDs
 - Acetaminophen
 - Muscle relaxants to relieve sphincter spasm:
 - Cyclobenzaprine
 - Diazepam
 - Diltiazem 2% ointment
 - Nifedipine ointment 0.3%
- Topical anesthetics:
 - ELA-Max5
 - Lidocaine jelly 2%
- Sitz baths (with warm water) to relieve sphincter spasm

Diet

- High-fiber diet instruction:
 - Fiber/bran: 20 g/d
 - Psyllium seeds (Metamucil or Konsyl): 1–2 tsp (peds: 0.25–1 tsp/d) PO q24h
- Encourage consumption of 10–12 oz glasses of water per day.

MEDICATION

- Cyclobenzaprine (Flexeril): 10 mg (peds: Not indicated) PO TID
- Diazepam (Valium): 5 mg (peds: 0.12–0.8 mg/kg/d) PO TID PRN for spasm
- Diltiazem 2% ointment: Apply to fissure BID
- Docusate sodium (Colace): 50–200 mg (peds: younger than 3 yr, 10–40 mg/d; 3–6 yr, 20–60 mg/d; 6–12 yr, 40–150 mg/d) PO q12h
- ELA-Max5 (5% lidocaine anorectal cream): Apply to perianal area q4h PRN pain (pediatric dose: Not for those younger than 12 yr)
- Ibuprofen: 400–600 mg (peds: 40 mg/kg/d) PO q6h
- Nifedipine ointment 0.3%: Apply to fissure TID with Q-tip (peds: Not indicated)
- Nitroglycerin ointment 0.2%: Apply a small pea-sized dot to fissure BID—TID with cotton swab. (peds: Not indicated)



FOLLOW-UP

DISPOSITION

Admission Criteria

Severe abdominal pain/distention due to fecal impaction

Discharge Criteria

- Initial treatment is conservative therapy for acute anal fissures as an outpatient.

- Operative referral for chronic fissures

FOLLOW-UP RECOMMENDATIONS

Colorectal or GI follow-up for patients with symptomatic fissures

PEARLS AND PITFALLS

- Perform a careful physical exam of rectal area to delineate fissures and exclude other pathology.
- Provide combination of pain relief and muscle relaxants for patients with significant pain.
- Provide discharge medications/instructions to prevent constipation.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Hemorrhoid
- Perirectal Abscess



CODES

ICD9

565.0 Anal fissure

ICD10

- K60.0 Acute anal fissure
- K60.1 Chronic anal fissure
- K60.2 Anal fissure, unspecified

ANAPHYLAXIS

Sean-Xavier Neath



BASICS

DESCRIPTION

- An acute, widely distributed form of shock that occurs within minutes of exposure to antigen in a sensitized individual
- ~500–1,000 deaths are attributed annually in the US to anaphylaxis. There are ~10,000 ER visits per month for anaphylaxis in the US
- Involves release of bioactive molecules such as histamine, leukotrienes, and prostaglandins from inflammatory cells:
 - Mediator release results in increased vascular permeability, vasodilation, smooth-muscle contractions, and increased epithelial secretion.
 - Physiologically, this is manifested in a decrease in total peripheral resistance, venous return, and cardiac output, as well as intravascular volume depletion.

ETIOLOGY

- IgE mediated:
 - Antibiotics, particularly penicillin family
 - Venoms, especially bee and wasp
 - Latex
 - Vaccines
 - Foodstuffs (shellfish, soybeans, peanuts, tree nuts, wheat, milk, eggs, nitrates/nitrites)
 - Monoclonal antibody therapeutics
- Non-IgE mediated:
 - Iodine contrast media
 - Opiates
 - Vancomycin

Pediatric Considerations

In children, foods are an important trigger for IgE-mediated anaphylaxis:

- Milk, egg, wheat, and soy (MEWS) are the most common food allergens.
- Peanut allergies are increasingly common and can be more potent; children can develop anaphylaxis from residual peanut in a candy bar.



DIAGNOSIS

SIGNS AND SYMPTOMS

- Symptoms begin within seconds to minutes after contact with an offending antigen.
- Anaphylactic reactions almost always involve the skin or mucous membranes. > 90% of patients have some combination of urticaria, erythema, pruritus, or angioedema.
- Some patients may have an initial sensation of impending doom followed by more clearly definable symptomatology:
 - Respiratory: From sneezing and nasal congestion to frank bronchospasm and laryngeal edema
 - Cardiovascular: Hypotension, dysrhythmias, myocardial ischemia
 - Gastrointestinal: Nausea, vomiting, diarrhea
 - Ocular: Eye itching and tearing, conjunctival injection
 - Hematologic: Activation of intrinsic coagulation pathway sometimes leading to disseminated intravascular coagulation, thrombocytopenia
 - Neurologic: Seizures

History

- Anaphylaxis is a clinical diagnosis.
- A brief history should include previous history of allergy or anaphylaxis, as well as exposure to potential new triggers.

Physical-Exam

- Usually will include alterations in a combination of any 2 or more of the following systems: Cutaneous, respiratory, gastrointestinal, or cardiovascular system
- Hypotension or airway compromise is not always present at onset.

ESSENTIAL WORKUP

- Diagnosis is made based on clinical symptoms.
- It is important not to underestimate the potential severity of an allergic reaction in its early stages.
- ECG should be done in patients with previous cardiac history or ischemic symptoms; consider routinely in the elderly.

DIAGNOSIS TESTS & NTERPRETATION

Lab

The diagnosis of anaphylaxis is made on clinical grounds; laboratory tests are usually not useful in aiding diagnosis in the acute setting. Tryptase levels remain elevated after an attack and can be useful for later confirmation of a suspected anaphylactic episode.

Imaging

Hyperinflation can be seen on CXR.

DIFFERENTIAL DIAGNOSIS

- Pulmonary embolism
- Acute MI
- Airway obstruction
- Asthma
- Tension pneumothorax
- NSAID reaction
- Vasovagal collapse
- Hereditary angioedema
- Serum sickness
- Systemic mastocytosis
- Foreign body aspiration
- Hypoglycemia
- Pheochromocytoma
- Carcinoid syndrome



TREATMENT

PRE HOSPITAL

- IV access, O₂, cardiac and pulse oximetry monitoring
- Early intubation based on the initial progression of disease and response to treatment:
 - Laryngeal edema and spasm can progress rapidly.
 - Laryngeal edema can be managed with racemic epinephrine prior to intubation.
- IM epinephrine can be administered en route even prior to establishment of an IV.

ALERT

Note that current guidelines advocate the use of IM over SC epinephrine

INITIAL STABILIZATION/THERAPY

- ABCs
- Assure adequate ventilation
- Airway management:
 - Orotracheal intubation is the airway technique of choice.
 - If laryngeal edema, spasm, or soft tissue swelling present; consider using advanced airway adjuncts when available.
 - Consider blind nasotracheal intubation if soft tissue swelling prohibits an oral approach and there is absence of stridor.
 - Transtracheal jet insufflation or cricothyrotomy may be necessary to control the airway.
- Epinephrine IM/IV or endotracheal administration:

– Direct injection into the venous plexus at the base of the tongue is an option.

- Volume resuscitation with crystalloids or colloids

ED TREATMENT/PROCEDURES

- Continuous cardiac and vital sign monitoring until stable
- Persistent bronchospasm can be treated with β_2 -agonist bronchodilators.
- Hypotension should be treated with volume repletion. Vasopressors can provide additional support.
- Antihistamines (both H_1 and H_2 blockers) have been shown to be helpful in preventing histamine interactions with target tissues.
- Corticosteroids help prevent the progression or recurrence of anaphylaxis.
- Glucagon is particularly useful in epinephrine-resistant anaphylaxis from β -adrenergic blocking agents.

MEDICATION

ALERT

A patient's concomitant use of a β -blocker may antagonize the effects of epinephrine. For these patients consider glucagon as it increases intracellular cyclic adenosine monophosphate levels by a mechanism that does not depend upon β -receptors.

First Line

- Diphenhydramine: Adults—50 mg IV; (peds: 1–2 mg/kg, not to exceed 50 mg/dose) slow IVP
- Epinephrine: 0.3–0.5 mg; use 1:1,000 dilution for IM route and 1:10,000 for IV route (peds: epinephrine 0.01 mg/kg SC/IV)
- Hydrocortisone: Adults—500 mg IV (peds: 4–8 mg/kg/dose IV) OR
- Methylprednisolone: Adult—125 mg IV (peds: 1–2 mg/kg IV) OR
- Prednisone: Adult—60 mg PO (peds: 1 mg/kg PO)
- Albuterol: 0.5 mL of 0.5% solution in 2.5 mL of isotonic saline by nebulizer

Second Line

- Racemic epinephrine: 2.25% solution (0.5 mL placed in a nebulizer in 2.5 mL of normal saline)
- Glucagon: Adults—1–2 mg IV/IM/SC
- Ranitidine: Adult—50 mg IV (peds: 1 mg/kg) *or* cimetidine 300 mg IV



FOLLOW-UP

DISPOSITION

Admission Criteria

- Intubated patients or patients in respiratory distress should be admitted to an intensive care unit setting.
- A monitored inpatient bed may be necessary for the patient who has not had substantial response to initial therapy.
- Patients with significant generalized reactions and persistent symptoms should be admitted for observation for 24 hr. Biphasic or late phase reactions are known to occur.

Discharge Criteria

Patients with complete resolution of symptoms may be discharged after several hours of ED observation.

Issues for Referral

- Consultation with an allergist/immunologist is appropriate when desensitization to an antibiotic is being considered for the treatment of an infectious process.
- When a patient at high risk for contrast reaction needs a contrast study, consultation with the radiologist regarding pretreatment and choice of contrast agent is appropriate.

FOLLOW-UP RECOMMENDATIONS

- Patients with allergic reactions should have a follow-up within 48 hr of discharge to evaluate effectiveness of outpatient therapy.
- Refer patients who are treated and released from the ED after an episode of anaphylaxis, angioedema, or urticaria to an allergist for follow-up skin testing and consideration for desensitization.
- Patients should be advised to carry some type of treatment that can be self-administered in the event of future reactions such as the prefilled syringe EpiPen.
- Patients with a known trigger should be counseled on strict avoidance of that trigger.

PEARLS AND PITFALLS

- Failure to consider anaphylaxis early in presentation can lead to devastating hemodynamic compromise and airway collapse.
- Epinephrine given early is the most important intervention.
- Patients with a history of anaphylaxis should be educated about trigger avoidance and instructed in the correct use of epinephrine auto-injector pens.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Angioedema
- Urticaria



CODES

ICD9

- 989.5 Toxic effect of venom
- 995.0 Other anaphylactic reaction
- 995.60 Anaphylactic reaction due to unspecified food

ICD10

- T63.91XA Toxic effect of contact w unsp venomous animal, acc, init
- T78.00XA Anaphylactic reaction due to unspecified food, init encntr
- T78.2XXA Anaphylactic shock, unspecified, initial encounter

ANEMIA

Stevan A. Vuckovic • Paul J. Allegretti

BASICS

DESCRIPTION

- Reduction below normal in the mass of RBCs
- Measured by 1 or more of the major RBC components:
 - Hemoglobin (Hgb): Concentration of the major oxygen-carrying component in whole blood
 - Hematocrit (Hct): Percent volume of whole blood occupied by intact RBCs
 - RBC count: RBCs contained in a volume of whole blood
- Adult female: Hgb < 12 g/dL or Hct < 37%
- Adult male: Hgb < 14 g/dL or Hct < 42%
- Normal blood count values depend on age:
 - Birth: Hgb 16.5, Hct 51
 - 1 yr: Hgb 12, Hct 36
 - 6 yr: Hgb 12.5, Hct 37
 - Adult male: Hgb 14, Hct 42
 - Adult female: Hgb 12, Hct 37
- Hgb/Hct depend on oxygen pressure:
 - Increased in neonates and people living above 4,000 ft
- Hgb, Hct, and RBC count are concentrations:
 - Dependent on RBC mass and plasma volume
 - Values decrease if RBC mass decreases or plasma volume increases.
- Anemia is an indication of an underlying disorder or deficiency.

ETIOLOGY

- Never a normal variant:
 - May be the first manifestation of a systemic disorder
 - Always seek a cause.
- Excessive blood loss (most common cause):
 - Trauma
 - GI bleed
 - Menstruation
- Hemolysis (increased RBC destruction, RBC lifespan < 100 days):
 - Hypersplenism
 - Autoimmune hemolytic anemia
 - Mechanical trauma (prosthetic heart valves, vasculitis, thrombotic thrombocytopenic purpura [TTP], hemolytic uremic syndrome [HUS], or disseminated intravascular coagulation [DIC])

- Toxins
- Infections (malaria, babesiosis)
- Membrane abnormalities
- Intracellular RBC abnormalities (G6PD, sickle cell anemia, or thalassemia)
- Decreased RBC synthesis:
 - Classified by measurement of RBC size
 - Hypochromic/microcytic:
 - Iron deficiency
 - Thalassemia
 - Sideroblastic
 - Chronic disease
 - Normochromic/macrocytic:
 - Hypothyroidism
 - Folate deficiency
 - Vitamin B₁₂ deficiency
 - Liver disease
 - Myelodysplasia
 - Certain leukemias
 - Normochromic/normocytic:
 - Aplastic anemia
 - Chronic renal failure
 - Malignancy
 - Adrenal insufficiency
 - Hyperparathyroidism
 - Alcohol abuse
 - Acute blood loss

DIAGNOSIS

SIGNS AND SYMPTOMS

Depends on:

- Rapidity of onset:
 - Hypovolemia if acute
 - Asymptomatic if mild and chronic

History

- Underlying disease
- Severity and type of anemia
- Fatigue
- Decreased exercise intolerance
- Shortness of breath
- Dyspnea on exertion

- Chest pain/angina
- Syncope
- Blood in stool/tarry black stools
- Irregular or heavy menses
- Easy bruising or history of excessive bleeding

Physical-Exam

- Cardiovascular:
 - _ Tachycardia, cardiomegaly, or murmurs
 - _ Postural hypotension
- Dermatologic:
 - _ Skin:
 - Cool
 - Pallor
 - Jaundice
 - Purpura
 - Telangiectasia
 - Petechiae
 - Ecchymosis
 - _ Spoon-shaped nails (koilonychia)
- Neurologic:
 - _ Neuropathy
 - _ Altered mental status
- Bone (especially sternal) or joint pain (sickle cell disease)
- Hepatomegaly, splenomegaly
- Lymphadenopathy
- Findings reflect underlying disease

ESSENTIAL WORKUP

- CBC
- Vital signs/orthostatics
- Determine if:
 - _ Bleeding
 - _ Increased RBC destruction
 - _ Bone marrow suppression
 - _ Iron deficient

DIAGNOSIS TESTS & NTERPRETATION

Lab

- Type and crossmatch or type and screen
 - _ Depends on severity
- CBC

- RBC indices:
 - Mean corpuscular volume (MCV; normal: 80–100 μm^3)
 - Mean corpuscular hemoglobin (MCH; normal: 27–34 pg/cell)
 - Mean corpuscular hemoglobin concentration (MCHC; normal: 33–36%)
- Platelet count
 - Thrombocytosis suggests:
 - Iron deficiency
 - Myeloproliferative disorders
 - Inflammation
 - Infection
 - Neoplasm
 - Thrombocytopenia suggests:
 - Bone marrow malignancy
 - Hypersplenism
 - Sepsis
 - Vitamin B₁₂ or folate deficiency
 - Autoimmune disorders
- Reticulocyte (retic) count:
 - Normal 0.5–1.5% (retics/1,000 RBCs)
 - Increased retic count: Increased erythropoietic response to continued blood loss or hemolysis
 - Stable anemia with low retic count: Impaired RBC production
 - Active hemolysis or blood loss with low retic count: Concurrent disorder
 - Low retic count with pancytopenia: Aplastic anemia
 - Low retic count with normal WBC and platelets: Pure RBC aplasia
- Reticulocyte index (RI): $\text{Retic count (\%)} \times (\text{patient Hct}/\text{normal Hct})$:
 - RI < 2% implies inadequate RBC production.
 - RI > 2% implies increased RBC production with excessive RBC destruction or loss.
- WBC with differential and peripheral smear:
 - Leukopenia with anemia suggests bone marrow suppression, hypersplenism, or deficient vitamin B₁₂/folate
- Stool for occult blood
- Electrolytes, BUN, creatinine, glucose:
 - Chronic renal failure
- Urinalysis:
 - Hematuria
 - Hemoglobinuria in hemolytic anemia
- Workup strategy:
 - Hypochromic/microcytic anemias:
 - Iron

- Total iron-binding capacity
- Transferrin saturation
- Ferritin
- Macrocytic anemias:
 - Folate
 - Vitamin B₁₂
 - LFT
 - Thyroid function tests
- Hemolytic anemia:
 - Rapid fall in Hgb
 - Reticulocytosis
 - Fragmented RBCs
 - Increased LDH
 - Increased indirect bilirubin
 - Decreased serum haptoglobin
 - Coombs positive
- Special tests:
 - Peripheral smear:
 - Helmet cells/schistocytes—microangiopathic hemolysis
 - Teardrop cells—myelofibrosis
 - Spherocytes—autoimmune hemolysis
 - Leukoerythroblastic pattern—bone marrow replacement
 - Bite cells—oxidative hemolysis
 - RBC parasites—malaria or babesiosis
 - Target cells—liver disease
 - Burr cells—uremia
 - Sideroblasts—alcoholism or myelodysplasia
 - Howell–Jolly bodies—asplenia
 - Hgb electrophoresis for sickle cell/thalassemia
 - Iron, iron-binding capacity, transferrin saturation, ferritin:
 - Iron deficiency
 - Iron—decreased
 - Iron-binding capacity—increased
 - Transferrin saturation—decreased
 - Ferritin—decreased
 - Chronic disease:
 - Iron—decreased
 - Iron-binding capacity—decreased
 - Transferrin saturation—decreased/normal
 - Ferritin—normal/increased
 - Thalassemia:
 - Iron—normal

- Iron-binding capacity—normal
- Ferritin—normal
- Sideroblastic anemia:
 - Iron—increased
 - Iron-binding capacity—normal
 - Ferritin—increased

Diagnostic Procedures/Surgery

Bone marrow biopsy evaluates:

- Aplastic anemia
- Myelodysplasia
- Bone marrow malignancy
- Myeloproliferative disorders

DIFFERENTIAL DIAGNOSIS

- Acquired versus inherited anemia
- Anemia of chronic disease
- Blood loss
- CHF
- Dilutional anemia
- Hemolysis
- Malignancy
- Nutritional deficiency/malabsorption
- Toxic bone marrow suppression

Pediatric Considerations

- Hemolytic anemia of the newborn:
- Rh antibody crosses placenta when Rh-negative mother has Rh-positive child.

Pregnancy Considerations

- Physiologic or dilutional anemia in 3rd-trimester pregnancy:
- 25% increase of RBC mass and 50% increase in plasma volume

Geriatric Considerations

- Values for Hgb/Hct in healthy elderly are generally lower than in younger adults.
- This lower “normal” must be a diagnosis of exclusion.



TREATMENT

PRE HOSPITAL

Ongoing blood loss requires close assessment and rapid transport:

- Control bleeding to include wound packing and use of tourniquets if needed.

- Two large-bore IVs

INITIAL STABILIZATION/THERAPY

- Airway, breathing, circulation (ABCs)
- Oxygen
- IV fluid resuscitation with 0.9% NS if ongoing loss/hypotension

ED TREATMENT/PROCEDURES

- Depends on severity of anemia and acuteness of onset
- Transfusion for hemorrhage with unstable vital signs not responding to crystalloid resuscitation.
- Most anemias seen in ED are chronic and do not require immediate intervention.
- Therapy for specific anemia:
 - _ Iron deficiency:
 - FeSO₄: 300 mg PO TID
 - Investigate underlying cause.
 - Increased Hgb expected in 2–3 wk
 - _ Renal failure:
 - Endogenous erythropoietin is diminished.
 - Replace with recombinant erythropoietin
 - _ Autoimmune hemolytic anemia:
 - Corticosteroids (prednisone 60 mg/day until response)
 - Immunosuppressive agents
 - Plasmapheresis
 - Splenectomy if splenic sequestration
 - _ Drug-induced hemolytic anemia: Stop offending agent.
 - _ Anemia of chronic disease: Treat underlying disease.
 - _ Vitamin B₁₂ deficiency:
 - Vitamin B₁₂: 1,000 µg IM daily for 1 wk, then weekly for 1 mo, then monthly
 - Hematologic parameters normalize within 2 mo.
 - Neurologic symptoms present >6 mo may be permanent.
 - _ Folate deficiency:
 - Folic acid: 1 mg PO daily
 - _ Aplastic anemia
 - _ Antithymocyte globulin
 - _ Bone marrow transplantation:
 - Sickle cell anemia
 - Supportive care with oxygen, rehydration, analgesia
 - Treat precipitating cause.
 - _ Leukemia:
 - Bone marrow replacement

MEDICATION

- Iron supplements
- Erythropoietin for renal failure
- Corticosteroids for autoimmune
- Vitamin B₁₂
- Folic acid (B₉)



FOLLOW-UP

DISPOSITION

Admission Criteria

- Unstable vital signs
- Ongoing blood loss
- Symptomatic anemia—angina/dyspnea/syncope/neurologic symptoms
- Need for transfusion
- Need for aggressive evaluation
- Severe anemia
 - Initial, unexplained Hgb < 8 g/dL
 - Major difficulty in obtaining outpatient care for patients whose Hgb are significantly low or when comorbidity is present

Discharge Criteria

Discharge vast majority of stable patients for outpatient workup.

FOLLOW-UP RECOMMENDATIONS

Newly diagnosed anemic patients need to be worked up:

- If stable for discharge from the ED, provide follow-up options for workup

PEARLS AND PITFALLS

- Anemia is an indication of an underlying disorder or deficiency.
- Severe or life-threatening cases require immediate correction via blood transfusion.
- Most cases seen in the ED are chronic and do not require immediate intervention.

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See Also (Topic, Algorithm, Electronic Media Element)

- GI Bleeding
- Renal Failure
- Sickle Cell Disease



CODES

ICD9

- 280.0 Iron deficiency anemia secondary to blood loss (chronic)
- 285.1 Acute posthemorrhagic anemia
- 285.9 Anemia, unspecified

ICD10

- D50.0 Iron deficiency anemia secondary to blood loss (chronic)
- D62 Acute posthemorrhagic anemia
- D64.9 Anemia, unspecified

ANGIOEDEMA

Sean-Xavier Neath



BASICS

DESCRIPTION

- Nonpruritic, well-demarcated, nonpitting edema of the dermis
- Due to the release of inflammatory mediators that cause dilation and increased permeability of capillaries and venules:
 - Mast cell mediated
 - Kinin related from the generation of bradykinin and complement-derived mediators
- Similar in pathologic basis to urticaria except that affected tissue lies deeper:
 - Urticaria affects superficial tissue and causes irritation to mast cells and nerves in the epidermis leading to intense itching.
 - Angioedema occurs in deeper layers, which have fewer mast cells and nerves, therefore causing less itching.
 - Angioedema can affect tissues other than the skin, for example, the gut.
 - Angioedema may be seen in a mixed picture along with urticaria
- Hereditary and acquired etiologies are due to deficiencies in the quantity and/or function of C1-INH rather than classic hypersensitivity reactions
- Hereditary angioedema (HAE):
 - An autosomal dominant disorder caused by a deficiency of C1-INH
 - The prevalence of HAE is estimated to range from 1:10,000 to 1:150,000.
 - C1-INH has a regulatory role in the contact, fibrinolytic, and coagulation pathways.
 - Deficiency results in unregulated activity of the vasoactive mediators bradykinin, kallikrein, and plasmin.
 - More than 100 mutations of the C1-INH gene have been reported.
 - Type 1: Decreased expression of C1-INH
 - Type 2: Normal plasma levels of C1-INH but the protein is dysfunctional
 - Type 3: Mutation in coagulation factor XII resulting in increased kinin production:
 - Symptoms increased by estrogen-containing medications
 - Episodes occur in all 3 types when inflammation, trauma, or other factors lead to depletion of C1-INH.
- Acquired angioedema:
 - Normal quantities and function of C1-INH
 - Type 1 is associated with lymphoproliferative diseases and caused by consumption of the C1-INH protein by malignant cells.
 - Type 2 is autoimmune caused by circulating antibodies that inactivate C1-

INH.

- ACE inhibitor–induced angioedema:
 - Accounts for 2030% of all angioedema cases presenting to ER reported in 0.1–2.2% of patients taking ACE inhibitors
 - Usually occurs during the 1st mo of taking the medication, however the 1st event may occur spontaneously after many years.

ETIOLOGY

- Kinin-related etiologies:
 - HAE
 - Acquired angioedema:
 - Lymphoproliferative
 - Autoimmune
 - ACE inhibitor induced
- Mast cell–mediated etiologies:
 - Food allergies:
 - Additives
 - Nuts
 - Eggs/milk
 - Shellfish
 - Soy/wheat
 - Drug allergies:
 - Aspirin
 - NSAID
 - Antihypertensives
 - Narcotics
 - Oral contraceptives
 - Insect stings
 - Physically induced:
 - Cold/heat
 - Exercise/trauma/vibrations
 - Stress
 - UV light
- Hypereosinophilic syndromes such as Gleich syndrome
- Thyroid autoimmune disease
- Idiopathic recurrent AE



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- A family history or history of recurrent episodes can be useful in the diagnosis. Use of culprit foodstuffs or medications, especially ACEIs should increase index of suspicion
- Abdominal pain associated with nausea, vomiting, and diarrhea
- Attacks of HAE are not associated with hives or itching
- Emotional stress or physical trauma can trigger attacks.

Physical-Exam

- The lesions of angioedema are large, swollen, and nonpitting wheals.
- The eyelids and lips are frequently involved.
- Involvement of the pharynx and larynx may cause airway obstruction.
- Lesions are often asymmetric and do not typically involve gravitationally dependent areas

ESSENTIAL WORKUP

- Diagnosis is made of clinical grounds based on the presentation of large nonpitting, nonpruritic wheals.
- A family history need not be present: 25% of HAE patients have a new mutation and may not have a positive family history.

DIAGNOSIS TESTS & NTERPRETATION

Lab

- CBC with differential, ESR, ANA, rheumatoid factor, C4 and C3 levels.
- Skin biopsy if an urticarial lesion is accessible

Diagnostic Procedures/Surgery

Measurement of C1-INH levels (not routinely available in EDs):

- Patients affected with HAE type 1 have very low levels; carriers will have half-normal levels.
- C4 and C2 levels are low during attacks in both hereditary and acquired forms.

DIFFERENTIAL DIAGNOSIS

- Edema:
 - SVC syndrome
 - Right heart failure
 - Constrictive pericarditis
 - Renal failure
 - Nephrotic syndrome
- Allergic contact dermatitis
- Blepharochalasis
- Facial cellulitis
- Facial lymphedema

- Edema secondary to autoimmune disorders:
 - Dermatomyositis
 - Lupus
 - Polymyositis
 - Sjögren syndrome
- Hypothyroidism

Pediatric Considerations

Recurrent angioedema presenting around puberty should raise suspicion of HAE.



TREATMENT

PRE HOSPITAL

- Establish IV access
- Early intubation may be necessary due to the rapid progression of laryngeal swelling.
- Administration of H1 blocker when available
- Epinephrine to be considered for laryngeal edema especially if itching or other signs of a mast cell etiology with histamine release is suspected.

INITIAL STABILIZATION/THERAPY

- Active airway management and supportive measures are the primary goals of emergency treatment.
- Intubation may be necessary in severe cases:
 - Orotracheal intubation is the technique of choice but may be difficult because of laryngeal edema, spasm, or soft-tissue swelling.
 - Consider advanced airway adjuncts such as the gum elastic bougie to assist in securing endotracheal tube placement.
 - Blind nasotracheal intubation if soft tissue swelling prohibits an oral approach
 - Transtracheal jet insufflation or cricothyrotomy may be necessary to control the airway.
- Epinephrine, antihistamines, and steroids in obstructive airway swelling, although patient response can be variable.

ED TREATMENT/PROCEDURES

- Acute angioedema with features of a type 1 hypersensitivity reaction:
 - Treat similarly to an allergic reaction with H1 and H2 blockers along with corticosteroids.
 - Epinephrine should be used in refractory cases where the benefits outweigh the risks.
 - For abdominal attacks consider the addition of parenteral pain relief,

antiemetics, and IV fluid replacement.

- HAE and acquired angioedema:
 - C1-INH
 - Fresh frozen plasma (FFP) may be used as an alternative to C1-INH.
 - The kallikrein inhibitor ecallantide (Kalbitor) was approved in 2009 for the treatment of acute attacks of HAE.
 - Tranexamic acid (Cyklokapron), an antifibrinolytic agent, is not as effective for acute attacks and is used primarily in prevention.

ALERT

- C1 inhibition has been standard therapy in Europe for many years; however, the US FDA has only recently been approving these medications for use in the US, therefore clinician and pharmacist recognition of the utility of these drugs may be limited.
- Therapy with FFP (as a source of nonpurified C1-INH) is advised with caution as it may paradoxically worsen some attacks due to its high concentration of complement components.
- Attenuated androgens, such as the anabolic steroids and gonadotropin inhibitor danazol, are used in the long-term prophylactic treatment. They may not have any effect for 24–48 hr in the acute setting.
- Angioedema associated with ACE inhibitors occurs in 0.1–0.2% of cases and requires immediate withdrawal of the ACE inhibitor. ACE inhibitor–related angioedema usually occurs within a week after starting ACE inhibitor therapy, but may occur much later.

MEDICATION

General principles of pharmacologic treatment are based on suspected underlying cause:

- Suspected HAE:
 - C1-INH
 - Ecallantide
 - Icatibant
- Non-HAE:
 - H1 blockers
 - H2 blockers
 - Corticosteroids
- C1 esterase inhibitor replacement proteins (C1-INHP):
 - In the US currently only plasma-derived products:
 - Cinryze: 20 U/kg IV slow IV infusion
 - Berinert: 20 U/kg IV slow IV infusion with additional doses if no improvement in 2 h, sooner if worsening or if laryngeal symptoms
 - In the EU recombinant C1-INHP is also available (at this time not yet approved by US FDA)
 - Ruconest (not yet FDA approved) 50 U/kg, generally does not require

repeat dosing

- Cimetidine: 300 mg IV
- Danazol: 400–600 mg PO up to 1 g/d. *Contraindicated in children and pregnancy.*
- Diphenhydramine: Adult: 50 mg IV; peds: 1–2 mg/kg slow IVP
- Ecallantide (kallikrein inhibitor available US only): 30 mg SC given as 3 separate injections (about 1 mL each anatomically distant from AE affected area)
- Epinephrine: 0.3–0.5 mg (use 1:1,000 dilution for SC route, and 1:10,000 for IV route); peds: 0.01 mg/kg SC/IV
- Racemic epinephrine: 2.25% solution (0.5 mL placed in a nebulizer in 2.5 mL of NS)
- FFP (if C1-INH is unavailable): Adult: 2 U
- Hydrocortisone: Adult: 500 mg IV; peds: 4–8 mg/kg/dose IV.
- Icatibant (bradykinin B2 receptor antagonist): 30 mg SC once
- Methylprednisolone: Adult: 125 mg IV; peds: 1–2 mg/kg IV
- Prednisone: Adult: 60 mg PO; peds: 1 mg/kg PO
- Ranitidine: Adult: 50 mg IV
- Stanazolol: 2 mg PO up to 16 mg/d:
 - Discontinued in the US
 - Contraindicated in children and in pregnancy
- Tranexamic acid: 1 g PO q3–4h for up to 48 h if necessary

Pediatric Considerations

Safety and efficacy of newer HAE treatment agents (such as C1-INH Cinryze and Berinert) have not been established in children as of this writing. Dosing for adolescents is suggested to be weight based at 20 U/kg.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients with systemic symptoms that do not resolve completely will need to be hospitalized for observation.
- A monitored bed is recommended for those with airway involvement.

Discharge Criteria

- Patients presenting with minor symptoms of angioedema without progression after 4–6 hr of observation may be safely discharged home on a short course of steroids and antihistamines.
- Patients should be provided with an EpiPen and instructions on its use.

Issues for Referral

Patients should be evaluated by an allergist/immunologist after the initial presentation, especially if there is a family history of angioedema, or if the angioedema is accompanied by abdominal pain, or triggered by trauma.

FOLLOW-UP RECOMMENDATIONS

Patients without systemic symptoms who are stable for discharge should be seen in outpatient follow-up in a few days.

PEARLS AND PITFALLS

- Early measures should be employed to maintain the patient's airway.
- Consider use of newer agents in HAE patients (e.g., C1-INH and Kallikrein inhibition).

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- Wahn V, Aberer W, Eberl W, et al. Hereditary angioedema (HAE) in children and adolescents—a consensus on therapeutic strategies. *Eur J Pediatr*. 2012;171(9):1339–1348.
- Zuraw B. Hereditary angioedema. *N Engl J Med*. 2008;359:1027–1036.



See Also (Topic, Algorithm, Electronic Media Element)

- Anaphylaxis
- Urticaria



CODES

ICD9

- 277.6 Other deficiencies of circulating enzymes
- 995.1 Angioneurotic edema, not elsewhere classified

ICD10

- D84.1 Defects in the complement system
- T78.3XXA Angioneurotic edema, initial encounter

ANKLE FRACTURE/DISLOCATION

Sarah V. Espinoza • Leslie C. Oyama



BASICS

DESCRIPTION

Common mechanisms and injury patterns of the ankle:

- Mechanism of injury:
 - Inversion injury: Lateral ankle distraction and medial ankle compression
 - Avulsion fracture of the lateral malleolus
 - Oblique fracture of the medial malleolus
 - Eversion injury: Medial ankle distraction and lateral ankle compression
 - Avulsion fracture of medial malleolus
 - Oblique fracture of the fibula
 - External rotation injury:
 - Disruption of the tibiofibular syndesmosis, or a fibular fracture above the plafond
 - Anterior or posterior tibial fracture with separation of the distal tibia and fibula (unstable fracture)
 - Inversion and external rotation (Maisonneuve fracture):
 - Medial malleolus avulsion fracture or deltoid ligament tear
 - Disruption of the tibiofibular syndesmosis
 - Oblique fracture of the proximal fibula
 - Inversion and dorsiflexion (snowboarders' fracture):
 - Fracture of the lateral process of the talus
- Epidemiology
 - Most ankle fractures are malleolar
 - Common in young male and 50–70 yr old female
 - Associated with cigarette use and high BMI

Pediatric Considerations

- Ankle fractures in children often involve the physis (growth plate):
 - May result in angular deformity from growth plate injury
 - Associated with sports requiring sudden changes in direction and obese children
 - In children < 10 yr old, growth plate is weaker than epiphysis
- *Tillaux fracture*: Salter–Harris type III injury of the anterolateral tibial epiphysis external rotation of the foot
- *Triplane fracture*: Uncommon fracture of distal tibia with fracture lines in 3 distinct planes (coronal, transverse, sagittal)

SIGNS AND SYMPTOMS

- History of trauma
- Local ankle pain, swelling, deformity
- Inability to bear weight
- Soft tissue injury, swelling, ecchymosis, skin tenting, skin blanching
- Neurovascular compromise:
 - _ Diminished capillary refill
 - _ Diminished posterior tibialis (PT) or dorsalis pedis (DP) pulses
- Limited range of motion

History

- Discover the position of the ankle at the time of injury and area of most significant pain
- Determine if patient was able to bear weight immediately or if he or she needed assistance to walk afterward
- Ask if the patient heard audible “pop” or “snap,” as this may indicate partial or full tendon rupture

Physical-Exam

- *Ottawa Ankle Rules* (OAR), 100% sensitive: Decision tool for ordering radiographs in patients with suspected injury to the ankle and midfoot:
 - _ Malleolar zone (if any finding is present, then *ankle* radiographs are indicated):
 - Bony tenderness at the posterior edge or distal 6 cm of either malleoli (points A and B)
 - Inability to bear weight for 4 consecutive steps both immediately after the injury and in ED
 - _ Midfoot zone (if either finding is present, then *foot* radiographs are indicated):
 - Bony tenderness at the base of the 5th metatarsal (point C)
 - Bony tenderness of the navicular (point D)
 - Inability to bear weight for 4 consecutive steps both immediately after the injury and in ED
 - _ Considered a reliable tool in children > 5 yr
- Assess the skin for swelling, ecchymosis, skin tenting, disruption, or ischemia
- Careful evaluation of distal neurovascular status:
 - _ Capillary refill
 - _ Palpation or Doppler of DP and PT pulses
- Palpate proximal fibula for tenderness, especially when medial malleolus or deltoid ligament tenderness is present:

- Peroneal nerve is at risk for injury with a Maisonneuve fracture:
 - Wraps around the fibular head
 - Test anterior tibialis and extensor hallucis longus
 - Assess sensation in the 1st web space

DIAGNOSIS TESTS & INTERPRETATION

Imaging

- Radiography:
 - Evaluate the mortise view for widening: Distance between talus to the medial and lateral malleoli should be uniform
- Unstable ankle fractures or dislocations require post reduction radiographs in all 3 planes after splinting
 - Anteroposterior (AP), lateral, and mortise (AP with a 20° lateral angle)
- AP and lateral radiographs of the tibia and fibula are indicated if a Maisonneuve fracture is suspected clinically
- Stress testing of the ligaments in a painful ankle is unnecessary in the ED if the patient will be re-examined in 3–7 days
- Stress radiographs of the ankle are usually unnecessary acutely
- CT scan or MRI:
 - Assess the degree of injury to the tibial plafond and associated ligamentous injury

Diagnostic Procedures/Surgery

N/A

DIFFERENTIAL DIAGNOSIS

- Ankle sprain
- Achilles tendon injury
- Os trigonum fracture
- 5th metatarsal fracture (Jones fracture)
- Peroneal tendon dislocation or injury
- Talar fractures
- Talar dome fracture/lesion
- Subtalar dislocations
- Calcaneal fractures
- Foot fractures
- Ankle diastasis
- Rattlesnake envenomation

Pediatric Considerations

- Injury to the growth plates may not be apparent on plain radiographs
- Consider splint immobilization, nonweight-bearing status, and orthopedic referral

- if clinical suspicion warrants, even in the setting of negative radiographs
- CT scan or MRI may be warranted to delineate the extent of the injury
- Inform parents of the possibility of growth abnormalities in patients with injury to the physis



TREATMENT

PRE HOSPITAL

- Immobilize with soft splint to reduce pain, bleeding, and further injury
- Cautions:
 - _ Traction devices are usually unnecessary:
 - Contraindicated with open injuries
 - _ Protruding bone should not be reduced; the wound should be covered with a clean dressing

INITIAL STABILIZATION/THERAPY

- Nonweight bearing
- Ice
- Compression
- Elevation

ED TREATMENT/PROCEDURES

- Ankle fracture:
 - _ All ankle fractures or dislocations require orthopedic referral
 - _ Open ankle fractures:
 - Remove contaminants
 - Apply moist sterile dressing
 - Assess tetanus immunity
 - Antibiotics
 - Emergent orthopedic consultation
 - _ Closed ankle fractures:
 - Dislocations should be reduced promptly to prevent complications
 - Apply *posterior splint* to immobilize foot in 90° angle with the application of bulky dressings and covered by a volar posterior and coaptation (U-shaped stirrup) splint
 - Sugar tong (coaptation) can be added for mediolateral support
 - _ *Stable injury*: (one-sided nondisplaced malleolar fracture without ligamentous injury)
 - Isolated injury to the lateral malleolus without medial involvement is virtually always stable
 - Apply posterior splint
 - _ *Unstable injury*: (both sides of the ankle are injured i.e., bi- or trimalleolar

fractures)

- Urgent orthopedic consultation
- Posterior splint as in stable injuries
- May require open reduction and internal fixation (ORIF) emergently before significant swelling develops
- *Neurovascular injury* requires emergent orthopedic consultation
- Ankle dislocations:
 - Closed reduction should be performed as rapidly as possible to minimize ischemia to the skin and reduce the risk of avascular necrosis of the talus
 - Skin tenting and evidence of neurovascular compromise are indications for immediate reduction, even prior to radiographs
 - Most ankle dislocations require ORIF
 - After reduction, place a posterior splint

MEDICATION

- Closed fractures:
 - Primarily analgesics (opioids)
- Dislocations or displaced fractures requiring closed reduction consider:
 - Short-acting benzodiazepine (midazolam 0.05–0.1 mg/kg IV) or barbiturate (methohexital 1–1.5 mg/kg IV) with opioid analgesic
- Open fractures:
 - Cefazolin: 2 g loading dose (peds: 50 mg/kg) IV
 - Gentamicin: 5–7 mg/kg q24h (peds: 2.5 mg/kg q8h) IV
 - Vancomycin: 1 g loading dose (10 mg/kg in children) if penicillin allergic
 - Tetanus toxoid if indicated



FOLLOW-UP

DISPOSITION

Admission Criteria

- Unstable ankle fractures require urgent orthopedic consultation and may require admission
- Open ankle fractures and dislocations should be admitted for debridement, irrigation, and IV antibiotics
- Ankle dislocations that are treated with either open or closed reduction
- Concern for compartment syndrome or neurovascular injury

Discharge Criteria

Simple nondisplaced stable ankle fractures without neurovascular compromise may be splinted for immobilization and discharged

FOLLOW-UP RECOMMENDATIONS

- Splinting
- Elevation of affected lower extremity
- Fitted for crutches and shown how to use them
- Placed on nonweight-bearing status of affected joint, until seen by orthopedist

PEARLS AND PITFALLS

- To reduce a dislocated ankle, partial flexion of knee of affected limb will decrease tension on Achilles tendon and ankle
- Differentiate between ankle fracture and subtalar fracture on physical exam: While the latter is rare, it is also rarely reducible
- Remember to look for other injuries including lumbar spine, hip, tibia, fibula, especially the proximal fibular neck, and foot

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Ottawa Ankle Rules Figure



CODES

ICD9

- 824.0 Fracture of medial malleolus, closed
- 824.8 Unspecified fracture of ankle, closed
- 824.9 Unspecified fracture of ankle, open

ICD10

- S82.56XA Nondisp fx of medial malleolus of unsp tibia, init
- S82.66XA Nondisp fx of lateral malleolus of unsp fibula, init

- S82.899A Oth fracture of unsp lower leg, init for clos fx

ANKLE SPRAIN

Taylor Y. Cardall



BASICS

DESCRIPTION

- Injuries to ligamentous supports of the ankle
- Ankle joint is a hinge joint composed of the tibia, fibula, and talus.
- Injuries may range from stretching with microscopic damage (grade I) to partial disruption (grade II) to complete disruption (grade III).

ETIOLOGY

- Forced inversion or eversion of the ankle
- Forceful collisions
- 85–90% of ankle sprains involve lateral ligaments:
 - Anterior talofibular (ATFL)
 - Posterior talofibular (PTFL)
 - Calcaneofibular (CFL)
 - Usually the result of an inversion injury
 - The ATFL is the most commonly injured.
 - If the ankle is injured in a neutral position, the CFL is often injured.
 - The PTFL is rarely injured alone.
- Injury to the deltoid ligament (connecting the medial malleolus to the talus and navicular bones) is usually the result of an eversion injury:
 - Often associated with avulsion at the medial malleolus or talar insertion
 - Rarely found as an isolated injury
 - Suspect associated lateral malleolus fracture or fracture of the proximal fibula (Maisonneuve fracture).
- Syndesmosis sprains (injury to the tibiofibular ligaments or the interosseous ligament of the leg):
 - Occur most commonly in collision sports
 - Syndesmosis injuries (“high ankle sprains”) have a higher morbidity and potential for long-term complications.

Pediatric Considerations

- Children <10 yr with traumatic ankle pain and no radiologic evidence of fracture most likely have a Salter–Harris I fracture.
- The ligaments are actually stronger than the open epiphysis.



DIAGNOSIS

SIGNS AND SYMPTOMS

History

History may predict the type of injury found and should include:

- Time of injury
- Mechanism
- The presence of a “pop” or “crack”
- History of previous trauma
- Relevant medical conditions (e.g., bone or joint disease)
- Treatments attempted prior to arrival
- Ability to bear weight subsequent to the injury at scene and ED

Physical-Exam

- Aimed at detecting joint instability and any associated injuries:
 - Note the presence or absence of bony tenderness at posterior edge of medial and lateral malleoli as well as at the base of the 5th metatarsal.
- Document neurovascular status distal to the injury.
- Assess range of motion and compare it with the uninjured side.
- Stress testing in the ED is often limited by pain and may impair detection of ligament injury.
- The squeeze test helps identify syndesmosis injuries:
 - Squeeze tibia and fibula together at the midcalf; pain felt in the ankle indicates a positive test.

ESSENTIAL WORKUP

- The Ottawa Ankle Rules, a selective strategy for obtaining ankle radiographs in adults, suggest that foot or ankle radiographs are unnecessary except when any of the following are present:
 - Bony tenderness at the posterior edge of the distal 6 cm or tip of either malleolus
 - Bony tenderness along the base of the 5th metatarsal or navicular bone
 - Inability to take 4 unassisted steps both immediately after the injury and in the ED
- The rules have been prospectively validated by the original authors as well as independently by groups in the US, the UK, France, and other countries.

DIAGNOSIS TESTS & INTERPRETATION

Imaging

- Ankle injuries should be radiographed if there is concern for fracture.
- Stress radiographs are rarely useful in the ED and should not be routinely ordered unless requested by a consultant.

DIFFERENTIAL DIAGNOSIS

- Ankle fracture (lateral, medial, or posterior malleolus) or dislocation
- Achilles tendon injury
- Maisonneuve fracture
- Os trigonum fracture
- 5th metatarsal fracture (Jones fracture)
- Transchondral talar dome fracture
- Peroneal tendon dislocation or injury



TREATMENT

PRE HOSPITAL

Immobilize ankle as necessary.

INITIAL STABILIZATION/THERAPY

- Prevent further injury; avoid weight bearing if painful.
- RICE (rest, ice, compression, elevation)

ED TREATMENT/PROCEDURES

- The goal of treatment is reduction of pain and return to normal activity without long-term pain or joint laxity.
- Existing evidence supports early mobilization and functional treatment:
 - Unstable ankles (i.e., grade III) or those with severe pain may benefit from brief immobilization followed by early return to functional treatment.
- Grade I or II sprains can be treated with functional support (elastic bandage, air splint, gel splint, etc.):
 - Recent evidence suggests an elastic bandage dressing coupled with an air stirrup splint is superior to other forms of immobilization.
- Grade III sprains can be treated by immobilization (sugar tong with posterior splint or elastic bandage dressing coupled with air stirrup splint) and early orthopedic consultation or referral.
- Crutches may be needed initially for comfort, but encourage weight bearing as tolerated for grades I and II.
- Once acute pain and swelling have resolved, strengthening exercises and proprioceptive training (e.g., balance board, small circle walking) improve ankle strength and function and prevent reinjury.
- Full sports activities may be resumed only when running and turning are pain free.
- Ankle taping, air splints, or gel splints reduce the risk of recurrent injury in high-risk sports such as basketball, volleyball, soccer, and running.

MEDICATION

- NSAIDs are useful in treating acute pain:

- Ibuprofen: 800 mg (peds: 5–10 mg/kg) PO TID
- Topical NSAIDs have been shown to control pain and shorten healing time with acute ankle sprain:
 - Diclofenac sodium 1% gel: Apply 4g to affected area QID
- Narcotic analgesics may be required for severe pain.



FOLLOW-UP

DISPOSITION

Admission Criteria

An isolated ankle sprain should not require admission.

Discharge Criteria

An isolated ankle sprain may be safely discharged from the ED with appropriate treatment, prescriptions, aftercare instructions, and referrals.

Issues for Referral

Patient copies of any radiographs obtained may facilitate early follow-up.

FOLLOW-UP RECOMMENDATIONS

- Patients with grade I and II sprains should be instructed to follow up with the primary care physician in 1–2 wk.
- Patients with grade III sprains and syndesmosis injuries should be referred to an orthopedic surgeon or sports medicine specialist within 7–10 days.

PEARLS AND PITFALLS

- The Ottawa Ankle Rules may decrease the need for radiographs.
- Immobilization with an elastic bandage dressing coupled with an air stirrup splint followed by early functional therapy may shorten healing time.

ADDITIONAL READING

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CODES

ICD9

- 845.00 Sprain of ankle, unspecified site
- 845.02 Sprain of calcaneofibular (ligament) of ankle
- 845.09 Other sprains and strains of ankle

ICD10

- S93.409A Sprain of unsp ligament of unspecified ankle, init encntr
- S93.419A Sprain of calcaneofibular ligament of unsp ankle, init
- S93.499A Sprain of other ligament of unspecified ankle, init encntr

ANKYLOSING SPONDYLITIS

Daniel R. Lasoff • Brian K. Snyder

BASICS

DESCRIPTION

- Chronic inflammatory disease, primarily affects the axial skeleton with predilection toward the spine and sacroiliac (SI) joints:
 - SI joints 100%
 - Cervical spine 75%
 - Thoracic spine 70%
 - LS spine 50%
 - Hip joints 30%
 - Shoulder joints 30%
- *Spondylitis* (inflammation of vertebrae) of ankylosing spondylitis (AS) begins at the insertions of the outer fibers of the annulus fibrosus (enthesitis) of the vertebrae:
 - Ossification (syndesmophyte formation) may lead to complete fusion, *ankylosis*, of the vertebrae.
 - Extensive spinal involvement causes the radiographic appearance of the brittle “bamboo spine.”
- Onset 15–35 yr of age
- Male to female ratio is between 2:1 and 3:1.

ALERT

AS patients are at 4 times the risk for fracture and paralysis compared to the general population. They are 11 times more likely to have spinal cord injuries.

RISK FACTORS

Genetics

Strong genetic component. HLA-B27 is present in 80–90% of patients with AS.

ETIOLOGY

Disease is likely triggered by environmental factors such as infection in genetically predisposed individuals.

DIAGNOSIS

SIGNS AND SYMPTOMS

- **Spinal:** Low back pain with sacroiliitis is the most common presentation:
 - Inflammatory back pain, improving with movement and exercise.

- Higher risk for serious injury from milder traumatic mechanisms.
- Extraspinal inflammatory conditions (which may precede spinal symptoms):
 - Ocular (the most common):
 - Uveitis (25–40% occurrence). Usually acute and unilateral in onset. Can alternate eyes.
 - Cardiac:
 - Slight increased risk of CAD
 - Increased risk for valvular incompetence with prolonged course of AS.
 - Pulmonary:
 - Progressive restrictive lung disease due to limited expansion and fibrosis
 - GI:
 - 5–10% of patients with inflammatory bowel disease.
 - GU:
 - Risk for IgA nephropathy or amyloidosis. Also increased risk for NSAID nephropathy from anti-inflammatory use.
 - Enthesitis (inflammation at tendon or ligament insertion):
 - Often Achilles tendonitis or plantar fasciitis

History

- Patients <40 yr of age with insidious onset of low back pain >3 mo, radiating into gluteal areas from SI region, and progressing to involve the entire spinal region:
 - Worse with rest and improved with mild activity. Pain in 2nd half of night waking patient from sleep
 - Women may have more cervical and extraspinal manifestations than men.
- Possible prior history of uveitis, restrictive pulmonary disease, inflammatory bowel disease, enthesitis, or migrating or polyarthritis.

Physical-Exam

- Tenderness over SI joints elicited with direct pressure over both of patient's ASIS simultaneously.
- Dactylitis or enthesitis.
- Flattening of the normal lumbar lordosis
- Exaggeration of thoracic kyphosis
- Limitation of spinal movement
- Reduction in chest expansion

Pediatric Considerations

- Patients with juvenile ankylosing spondylitis (JAS) may commonly be misdiagnosed as recurrent sprains
- Onset of JAS is late childhood or adolescence (between 8 and 12 yr, before age

20); primarily boys.

- JAS has a much greater predilection for extraspinal joints and entheses of the lower extremities; in addition to SI tenderness, examine for:
 - Asymmetrical pauciarthritides of the joints of the lower extremities
 - Enthesitis of the ankle, knee, or tarsal bones. Plantar fasciitis and Achilles tendonitis are often common findings.

ESSENTIAL WORKUP

- Exclude fracture and neurologic injury in any patient with suspected AS for any new spinal pain (even without trauma).
- Exclude sepsis or septic joint if clinically indicated.
- Evaluate for sacroiliitis with pelvic rock test (compression) or Patrick test (downward pressure on the knee of a flexed and externally rotated leg and the contralateral ASIS causing sacral distraction).

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC may show mild leukocytosis with slight to moderate anemia and thrombocytosis.
- BUN, creatinine, and electrolytes may be useful to assess renal involvement.
- Erythrocyte sedimentation rate (ESR) or C-reactive protein (CRP) may be elevated, but are of limited use in the ED.
- HLA-B27 testing can be performed by a specialist. A negative result does not rule out AS.

Imaging

- Pelvic radiograph: Should be done in any adult patient suspected of undiagnosed ankylosing spondylitis:
 - Sacroiliitis is essential to the diagnosis of AS; this is seen initially as subchondral bony erosions on the iliac side of the SI joint, which later manifest as bony proliferation and sclerosis.
 - If plain films are negative for sacroiliitis, MRI should be considered.
- Lumbar, thoracic, and cervical spine radiographs to exclude fracture for complaint of new pain to these areas with or without trauma
- CT should be performed to further evaluate possible fractures on plain radiographs.
- MRI should be performed emergently on any patient with neurologic deficit.
- Chest radiograph may show patchy inflammatory infiltrates or apical fibrosis.

Diagnostic Procedures/Surgery

- Electrocardiogram indications:
 - Symptoms of acute coronary syndrome (slightly increased risk compared to

general population for CAD)

- Symptomatic arrhythmia:
 - o AV block
- Echocardiogram indications:
 - New murmur: Increased predilection for aortic insufficiency with AS
 - Evidence of new heart failure.

DIFFERENTIAL DIAGNOSIS

- **JAS:**
 - Onset before age 20
 - More enthesitis and extraspinal joint involvement.
- **Reactive arthritis (formerly Reiter syndrome):**
 - Arthritis, urethritis, and conjunctivitis beginning about 1 mo after an episode of urethritis or enteritis.
- **Enteropathic arthritis:**
 - Crohn's disease or ulcerative colitis
 - Primarily involves knee, elbow, ankle, or wrist, and usually exacerbated by flares of the bowel disease
- **Psoriatic arthritis:**
 - Psoriasis rash
 - Much greater predilection for the hands and feet with higher incidence of dactylitis.
- **Septic arthritis:**
 - Exclude with arthrocentesis if clinically suspected in single joint involvement.
- **Mechanical low back pain:**
 - Improved with rest and exacerbated by exercise without signs of systemic inflammatory process.
- **Spinal epidural abscess:**
 - More constant, unremitting, and typically associated with fever and history of IVDA or immunosuppression.
- **Neoplastic low back pain:**
 - Typically in patients older than 40, more constant and unremitting, and more characteristically at night.



TREATMENT

PRE HOSPITAL

ALERT

- High risk of spinal injury from minor trauma.
- Spinal immobilization must avoid creating further injury:

- Cushion stabilization and scoop board in position of comfort may be a better approach than cervical collar and/or backboard.
- Intubation difficulty
 - Cervical and TMJ restriction may limit success in all but fiberoptic techniques.
 - Consider alternative airway approaches such as LMA or bag valve mask with oral airway until definitive airway can be achieved safely (usually fiberoptic).
- Ventilation difficulty
 - Chest wall restriction from deformity and pulmonary fibrosis
- CPR may carry a higher likelihood of rib fractures

ED TREATMENT/PROCEDURES

- Exclude cord compression if clinically suspected (MRI is the study of choice).
- Exclude spinal fracture for any new spinal pain (CT may be necessary).
- Exclude infection if clinically suspected with laboratory analysis and arthrocentesis.
- Control pain and inflammation with NSAIDs

MEDICATION

- Nonselective NSAIDs:
 - Ibuprofen: 35 mg/kg/d divided QID, max. 50 mg/kg/d (adult: 300–800 mg PO TID or QID)
 - Indomethacin: 1–2 mg/kg/d divided BID or QID, max. 4 mg/kg/d (adult: 25 mg PO BID or TID)
 - Not well tolerated, especially at higher doses because of GI and CNS effects
 - Naproxen: 10 mg/kg/d divided BID, max. 1,000 mg/d (adult: 250–500 mg PO BID)
- COX 2 inhibitors:
 - Celecoxib (adult: 100 mg–200 mg PO BID)
- TNF- α inhibitors:
 - Adalimumab (adult: 40mg SubQ q2wk)
 - Etanercept (adult: 50mg SubQ qwk)

Pregnancy Considerations

- NSAIDs should be avoided in pregnancy.
 - Acetaminophen is 1st line
 - Opioids are 2nd line

Geriatric Considerations

NSAID use may increase risk in the elderly for cardiovascular disease, GI bleeding, renal function, and hypertension. Although effective in select patients, close follow-up is

prudent.

ALERT

- NSAIDs:
 - _ GI bleeding risks
 - Elderly, history of PUD, concurrent use of glucocorticoids, anticoagulants, aspirin, smoking, alcohol.
 - Consider celecoxib or adding an H2 blocker or PPI if patient is at higher risk for GI bleeding.

Second Line

Consider if NSAIDs or acetaminophen are ineffective at appropriate doses:

- Opioid analgesics, muscle relaxants, or low-dose steroids.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Acute neurologic impairment
- Intractable pain
- Sepsis or septic joint cannot be excluded.

Discharge Criteria

- No serious injuries or neurologic deficit
- Pain is manageable to the patient

Issues for Referral

- The patient should be encouraged to obtain a medical alert bracelet.
- Rheumatology:
 - _ Patients with evidence of a new diagnosis of AS should be considered for early referral to a specialist in rheumatology for immunomodulative therapy.
- Physical medicine and rehabilitation:
 - _ Resting splints for inflamed joints
 - _ Orthoses for enthesitis (such as heel cushion inserts to rest Achilles tendon attachment)

FOLLOW-UP RECOMMENDATIONS

- Routine primary care re-evaluation within 1–2 wk to assess response to treatment.
- Referral to a rheumatologist for immunomodulating medications.
- Earlier follow-up in any patient with higher risk for adverse response to NSAIDs:

- Elderly, hypertensive patients, and patients with higher GI bleeding risks.

PEARLS AND PITFALLS

- Intubation is likely to be difficult and should avoid neck repositioning due to risk of C1 subluxation.
 - Consider airway adjuncts (such as LMA) until a definitive airway (usually fiberoptic) can be safely assured.
- Immobilization must avoid creating additional injury
 - Consider cushion/tape stabilization in position of comfort rather than standard cervical collar and backboard
- Minor traumatic injuries in AS can result in spinal fracture and possible cord injury. Maintain a high clinical suspicion.

ADDITIONAL READING

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- Chakravarty SD, Paget SA. Ankylosing spondylitis: Pathogenesis, diagnosis, and therapy. *Rheumatology*. 2012;40:39--43.
- Sieper J, Braun J. *Ankylosing Spondylitis: In Clinical Practice*. Britain: Springer, 2010.



See Also (Topic, Algorithm, Electronic Media Element)

<http://www.spondylitis.org>



CODES

ICD9

720.0 Ankylosing spondylitis

ICD10

- M45.2 Ankylosing spondylitis of cervical region
- M45.8 Ankylosing spondylitis sacral and sacrococcygeal region
- M45.9 Ankylosing spondylitis of unspecified sites in spine

ANTICHOLINERGIC POISONING

Patrick M. Whiteley



BASICS

DESCRIPTION

- Central and peripheral cholinergic blockade
- Depending on the drug involved, antagonism occurs at muscarinic (most common), nicotinic, or both receptors.
- Onset of activity: 15–30 min after ingestion
- Duration of effect: 2–24 hr

ETIOLOGY

- Many drugs contain anticholinergic properties:
 - Mild at therapeutic doses
 - Life threatening in overdose
- Anticholinergic substances:
 - Antihistamines
 - Belladonna alkaloids and synthetic congeners
 - Antiparkinsonian drugs
 - Cyclic antidepressants
 - Antipsychotics (neuroleptics)
 - Mydriatics
 - Skeletal muscle relaxants (orphenadrine, cyclobenzaprine)
 - Antispasmodics
 - Mushrooms—*Amanita muscaria*, *Amanita pantherina*
 - Plants—deadly nightshade, mandrake, henbane
 - Jimson weed—smoked or ingested



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Onset and duration of symptoms
- Type and extent of ingestion/exposure

Physical-Exam

- Classic toxidrome:
 - “**Mad as a hatter**”—altered mental status
 - “**Hot as a hare**”—hyperthermia

- “*Red as a beet*”—flushed skin
- “*Dry as a bone*”—dry skin and mucous membranes
- “*Blind as a bat*”—blurred vision secondary to mydriasis
- General:
 - Hyperthermia
 - Altered mental status
- Ocular:
 - Unreactive mydriasis
 - Inability to accommodate
- Cardiovascular:
 - Sinus tachycardia
 - Dysrhythmias (rare except in massive ingestions)
 - Hypotension/HTN
 - Cardiogenic pulmonary edema
- Pulmonary:
 - Tachypnea
 - Respiratory failure
- GI:
 - Decreased/absent bowel sounds
 - Dysphagia
 - Decreased GI motility
 - Decreased salivation
- Genitourinary (GU):
 - Urinary retention
- Integument:
 - Decreased sweating
 - Flushed skin
 - Dry skin and mucous membranes
- CNS:
 - Altered mental status
 - Auditory or visual hallucinations
 - Coma
 - Seizures

ESSENTIAL WORKUP

Diagnosis based on clinical presentation and an accurate history

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Urine toxicologic screen if clinically indicated
- Electrolytes, BUN, creatinine, and glucose
- CBC

- Creatine phosphokinase (CPK) if suspected rhabdomyolysis
- Urinalysis
- Acetaminophen and salicylate levels:
 - _ Detects occult ingestion (e.g., Tylenol PM)

Imaging

ECG:

- Sinus tachycardia most common
- QRS prolongation
- AV blockade
- Bundle branch block pattern
- Dysrhythmias

DIFFERENTIAL DIAGNOSIS

- Sympathomimetic intoxication
- Withdrawal syndrome
- Acute psychiatric disorders
- Sepsis
- Thyroid disorder



TREATMENT

PRE HOSPITAL

Transport all pills/pill bottles involved in overdose for identification in ED.

INITIAL STABILIZATION/THERAPY

- Airway, breathing, and circulation (ABCs):
 - _ Airway control essential
 - _ Administer supplemental oxygen.
 - _ IV access
 - _ Cardiac monitor and pulse oximetry
- Naloxone, thiamine, D₅₀ (or Accu-Chek) if altered mental status

ED TREATMENT/PROCEDURES

- Supportive care:
 - _ IV rehydration with 0.9% NS
 - _ Standard aggressive cooling measures for hyperthermia
 - _ Use benzodiazepines for treatment of agitation:
 - Avoid phenothiazines owing to anticholinergic effects.
 - _ Treat seizures with benzodiazepines and barbiturates.
 - _ Dysrhythmias:
 - Use standard antidysrhythmics.

- Avoid class Ia antidysrhythmic owing to the quinidine-like effect of many anticholinergic drugs.
- Sodium bicarbonate boluses may reverse the quinidine-like effects.
- Decontamination:
 - Administer activated charcoal for oral ingestions if within 1 hr.
 - Ocular lavage for eyedrop exposure
- Physostigmine (Antilirium):
 - Reversible acetylcholinesterase inhibitor that crosses the blood–brain barrier
 - Short-term reversal of both central and peripheral anticholinergic effects
 - Indicated in the presence of peripheral anticholinergic signs and the following:
 - Seizures unresponsive to conventional therapy
 - Uncontrollable agitation
 - Use with caution if prolonged QRS is present on ECG owing to risk of dysrhythmias (especially asystole), seizures, and cholinergic crises:
 - Place on cardiac monitor.
 - Observe for cholinergic symptoms.
 - Contraindications:
 - Cyclic antidepressant overdose (potentiates toxicity)
 - Cardiovascular disease
 - Asthma/bronchospasm
 - Intestinal obstruction
 - Heart block
 - Peripheral vascular disease
 - Bladder obstruction

MEDICATION

- Activated charcoal: 1 g/kg PO
- Dextrose: 50–100 mL D₅₀ (peds: 2 mL/kg of D₂₅ over 1 min) IV; repeat if necessary
- Diazepam: 5–10 mg (peds: 0.2–0.5 mg/kg) IV every 10–15 min
- Dopamine: 2–20 µg/kg/min IV with titration to effect
- Lorazepam: 2–4 mg (peds: 0.03–0.05 mg/kg) IV every 10–15 min
- Physostigmine: 0.5–2.0 mg (peds: 0.02 mg/kg) IV over 5 min; repeat if necessary in 30–60 min
- Phenobarbital: 10–20 mg/kg IV (loading dose); monitor for respiratory depression
- Thiamine (vitamin B₁): 100 mg (peds: 50 mg) IV or IM

First Line

Lorazepam or Diazepam

Second Line

Physostigmine (use with caution and consult with medical toxicologist)



FOLLOW-UP

DISPOSITION

Admission Criteria

- ICU admission for moderate to severe anticholinergic symptoms (agitation control, temperature control, and observation for seizures or dysrhythmias)
- Any patient receiving physostigmine

Discharge Criteria

Mild and improving symptoms of anticholinergic toxicity after 6–8 hr of ED observation

Issues for Referral

- Substance abuse referral for patients with recreational anticholinergic abuse
- Patients with unintentional (accidental) poisoning require poison prevention counseling.
- Patients with intentional (e.g., suicide) poisoning require psychiatric evaluation.

FOLLOW-UP RECOMMENDATIONS

Appropriate psychiatric referral for intentional ingestions

PEARLS AND PITFALLS

- Aggressively treat hyperthermia.
- Antipyretic medications are not effective in toxic hyperthermia.
- Use physostigmine cautiously and consult with medical toxicologist when available.

ADDITIONAL READING

- Burns MJ, Linden CH, Graudins A, et al. A comparison of physostigmine and benzodiazepines for the treatment of anticholinergic poisoning. *Ann Emerg Med.* 2000;35:374–381.
- Ceha LJ, Presperin C, Young E, et al. Anticholinergic toxicity from nightshade berry poisoning responsive to physostigmine. *J Emerg Med.* 1997;15:65–69.
- Delaney KA. Anticholinergics and antihistamines (H1 antagonists). In: Ford MD, Delaney KA, Ling LJ, et al., eds. *Clinical Toxicology*. Philadelphia, PA: WB Saunders; 2001;472–477.
- Hidalgo HA, Mowers RM. Anticholinergic drug abuse. *Ann Pharmacother.* 1990;24:40.
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- Reilly KM, Chan L, Mehta NJ, et al. Systemic toxicity from ocular homatropine.



CODES

ICD9

971.1 Poisoning by parasympatholytics (anticholinergics and antimuscarinics) and spasmolytics

ICD10

T44.3X1A Poisoning by oth parasympath and spasmolytics, acc, init

ANTIDEPRESSANT POISONING

Patrick M. Lank



BASICS

DESCRIPTION

- Antidepressants are the most commonly prescribed psychiatric medications in US.
- Patients who overdose on antidepressants may be on various antidepressants, divided into SSRIs, SNRIs, and atypical. Concomitant usage of atypical antipsychotics and mood stabilizing medications, some of which are FDA approved for the treatment of depressive disorders, is common.
- Antidepressants may be prescribed for multiple other indications, including chronic pain syndromes, anxiety, eating disorders, substance abuse, and sleep disorders.
- Tricyclic antidepressants (TCAs) are covered in a separate chapter

ETIOLOGY

Mechanism

- SSRIs:
 - Increase serotonin at the synapse by preventing the reuptake of serotonin by the presynaptic neuron.
 - SSRIs include paroxetine, fluoxetine, sertraline, citalopram, and escitalopram
- SNRIs
 - Similar to SSRIs, but also inhibit reuptake of norepinephrine.
 - Developed because said to have fewer side effects than SSRIs at therapeutic dose, although not true for toxicity.
 - SNRIs include venlafaxine, desvenlafaxine, and duloxetine.
- Atypical antidepressants:
 - Have variable effects on serotonin, norepinephrine, and dopamine.
 - Include mirtazapine, trazodone, and bupropion
- Atypical antipsychotics:
 - Most antipsychotics have activity at dopamine receptors, although variable agonism/antagonism depending on medication and dopamine receptor.
 - Additional activity at serotonin, α -adrenergic, histamine, and muscarinic receptors.
- Psychiatric medications also have variable potassium and sodium channel blockade, leading to cardiotoxicity (QT and QRS prolongation, respectively).



DIAGNOSIS

SIGNS AND SYMPTOMS

- SSRIs:
 - Traditional SSRIs (fluoxetine, paroxetine, sertraline):
 - Sedation
 - Serotonin syndrome
 - In single substance overdose, rarely will cause severe medical effects
 - Citalopram/Escitalopram:
 - Somnolence, vomiting, tachycardia
 - QTc prolongation
 - Seizures (more common in citalopram)
 - Exhibit *delayed* toxicity (up to 12 hr after ingestion)
- SNRIs:
 - Duloxetine:
 - Somnolence, vomiting, tachycardia
 - Seizures rare
 - Venlafaxine/Desvenlafaxine:
 - Seizures
 - QTc prolongation
- Atypical antidepressants:
 - Bupropion
 - Sedation
 - Seizures
 - QRS & QTc prolongation
 - Trazodone:
 - Sedation
 - QTc prolongation
 - Hypotension
 - Priapism
 - Mirtazapine:
 - Sedation
 - QTc prolongation
 - Possible neutropenia in chronic dosing
- Atypical antipsychotics:
 - Variable dopamine receptor activity
 - Developed for fewer extrapyramidal symptoms (EPS), particularly tardive dyskinesia, than typical antipsychotics
 - Most common symptoms in overdose include sedation, tachycardia, and miosis
 - Clozapine:
 - Agranulocytosis (in up to 1% taking chronically)
 - Sialorrhea
 - Cardiomyopathy
 - Anticholinergic delirium

- Olanzapine:
 - Anticholinergic delirium
 - QTc prolongation
- Quetiapine:
 - Hypotension from significant α_1 -antagonism
 - QTc prolongation
 - Anticholinergic delirium
- Ziprasidone:
 - Sedation
 - QTc prolongation → increased risk of torsade
- Aripiprazole:
 - No QTc prolongation
 - Hypotension
 - Prolonged CNS dysfunction

ESSENTIAL WORKUP

- Determine agents ingested, dose, and time of ingestion:
 - Investigate for coingested drugs.
- Rapid bedside glucose if altered mental status

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Specific drug levels rarely available and do not guide emergent management
- ECG:
 - For evaluation of QTc and QRS width
- Urine pregnancy:
 - In females of childbearing age
- Electrolytes, BUN, creatinine, glucose
- Urine drug of abuse screening:
 - Rarely changes clinical management
- Salicylate and acetaminophen concentrations
 - Very common coingestants in suicidal patients
- Serum ethanol:
 - To evaluate ethanol intoxication as contributing to altered mental status

Imaging

- CT of brain to evaluate for other causes of depressed mental status
- CXR if intubated or hypoxic

DIFFERENTIAL DIAGNOSIS

- TCA overdose
- Ethanol overdose

- Isoniazid overdose
- Hypoglycemia
- Hypoxemia
- Hyponatremia
- Hypocalcemia
- Withdrawal syndromes
- Serotonin syndrome
- Head trauma
- Opioid intoxication
- Sedative–hypnotic overdose
- Mood stabilizer/antiepileptic overdose
- DKA



TREATMENT

PRE HOSPITAL

- In cases of suspected overdose, bring all medication bottles to hospital with patient.
- ABCs
- 0.9% NS IV fluids as needed for hypotension
- Benzodiazepines as needed for seizures

INITIAL STABILIZATION/THERAPY

- ABCs:
 - Administer oxygen.
 - Place on cardiac monitor and measure pulse oximetry.
 - Establish IV access
 - Intubate as needed for airway protection or respiratory status.
- Rapid bedside glucose measurement
- Naloxone or D₅₀W as indicated for altered mental status and rapid clinical evaluation
 - Flumazenil is not recommended for mixed-overdose patients, patients with underlying seizure disorder, or patients chronically on benzodiazepines.
- May give diphenhydramine 25–50 mg IM/IV or Cogentin 1 mg IV for EPS

ED TREATMENT/PROCEDURES

- GI decontamination:
 - Do not attempt decontamination in a patient who cannot protect their airway.
 - Intubation solely for decontamination purposes, however, is not recommended.
 - Activated charcoal may be beneficial in early presenting overdoses.

- For QRS widening, administer sodium bicarbonate IV bolus.
 - Sodium bicarbonate infusion (i.e., “bicarb drip”) is NOT appropriate for use with QRS widening, as it is ineffective and potentially limits ability to provide sodium bicarbonate boluses.
- Treat hypotension unresponsive to IV fluids with norepinephrine rather than dopamine owing to α_1 receptor antagonism.
- Treat seizures with:
 - Initial therapy: Benzodiazepines
 - For refractory seizures: Barbiturates
- Treat symptoms of serotonin syndrome (fever, AMS, tachycardia, rigidity, hyperreflexia) with benzodiazepines and active cooling

MEDICATION

- Activated charcoal: 50–75 g PO initial dose; better to give 10g charcoal per 1g ingested xenobiotic as tolerated up to 100g PO
- Benzotropine 1 mg PO/IV
- Diazepam: 5–10 mg IV bolus (peds: 0.1 mg/kg IV bolus or 0.5 mg/kg rectal)
- Diphenhydramine 25–50 mg IM/IV (peds 1 mg/kg)
- Lorazepam: 2–4 mg (peds: 0.1 mg/kg) IV bolus
- Naloxone: 0.4–2 mg (peds: 0.1 mg/kg) initial bolus; may repeat up to a total of 10 mg
- Norepinephrine: 0.5–2 μ g/kg IV infusion
- Phenobarbital: 15–20 mg/kg IV max. dose is 2 g; caution: Likely to develop respiratory depression with IV loading doses
- Sodium bicarbonate: 1 mEq/kg IV bolus (adult 8.4%; peds: < 50 kg, 4.2%)



FOLLOW-UP

DISPOSITION

Admission Criteria

- 24 h telemetry admission for ingestions of the following: Citalopram, escitalopram, venlafaxine, desvenlafaxine, bupropion
 - Asymptomatic patients 6 hr after ingestion of other antidepressant medications do not require medical admission
- Coma
- Altered mental status
- Symptoms of NMS
- Hemodynamic compromise
- ECG changes
- Suicidal patients should be on a 1:1 observation

Discharge Criteria

- Asymptomatic patients of less toxic antidepressants > 6 hr after ingestion may be medically cleared for psychiatric admission.
- Discharge only asymptomatic patients who are not suicidal.

FOLLOW-UP RECOMMENDATIONS

Psychiatry referral for patients with intentional overdose

PEARLS AND PITFALLS

- For QRS widening, administer sodium bicarbonate IV bolus.
- Overdose with citalopram, venlafaxine, and bupropion have the possibility of being more severe than overdoses with other SSRIs and SNRIs and should prompt medical observation prior to clearance for psychiatric hospitalization.
- For any overdose, call your regional poison center at 1-800-222-1222

ADDITIONAL READING

- Boyer EW, Shannon M. The serotonin syndrome. *N Engl J Med*. 2005;352(11):1112–1120.
- Cooke MJ, Waring WS. Citalopram and cardiac toxicity. *Eur J Clin Pharmacol*. 2013;69(4):755–760.
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See Also (Topic, Algorithm, Electronic Media Element)

Tricyclic Antidepressant Poisoning



CODES

ICD9

- 969.00 Poisoning by antidepressant, unspecified
- 969.02 Poisoning by selective serotonin and norepinephrine reuptake inhibitors
- 969.05 Poisoning by tricyclic antidepressants

ICD10

- T43.201A Poisoning by unsp antidepressants, accidental, init
- T43.211A Poison by slctv serotonin/norepineph reup inhibtr, acc, init
- T43.221A Poison by selective serotonin reuptake inhibtr, acc, init

AORTIC DISSECTION, THORACIC

Jeffrey I. Schneider • Jonathan S. Olshaker



BASICS

DESCRIPTION

- Aortic dissection begins when there is an intimal tear.
- Blood then dissects through the media under aortic systolic pressure.
- It is thought that hypertension is a major factor in the dissection process.
- Dissections can start proximally at the root and dissect distally to involve any or all branches of the aorta, such as the carotid and subclavian arteries.
- The dissection process can also proceed proximally to involve the aortic root, the coronary ostia, and the pericardium.
- Dissection that progresses proximally may lead to occlusion of the coronary ostia, aortic valve incompetence, or cardiac tamponade.
- Classification related to portion of aorta involved:
 - Stanford classification:
 - Type A: Ascending aorta
 - Type B: Distal to ascending aorta
 - DeBakey classification:
 - DeBakey I: Intimal tear in aortic arch or root
 - DeBakey II: Ascending aorta
 - DeBakey III: Distal to takeoff of left subclavian artery
- Peak age for occurrence:
 - Proximal dissection: 50–55 yr
 - Distal dissection: 60–70 yr

Pregnancy Considerations

Risk of dissection increases in the presence of pregnancy:

- In women < 40 yr of age, 50% of dissections occur during pregnancy.

ETIOLOGY

Any process that affects the mechanical properties of the aortic wall can lead to dissection:

- Hypertension (72% of patients in the Registry of Acute Aortic Dissection)
- Congenital heart disease (bicuspid aortic valve, coarctation)
- Aortic wall connective tissue abnormalities (cystic medial necrosis)
- Connective tissue disease (Marfan disease, Ehlers–Danlos syndrome)
- Pregnancy
- Infectious/inflammatory conditions that can cause vasculitis (lupus, syphilis, endocarditis, giant cell arteritis, rheumatoid arthritis, Takayasu arteritis)

- Previous cardiac surgery including CABG, aortic valve repair
- Tobacco use

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Chest pain:
 - May be absent in as many as 15% of patients
 - Substernal if type A dissection
 - Intrascapular if descending thoracic dissection
 - Lumbar if abdominal aorta involved
 - Starts abruptly
 - Usually described as sharp
 - Most severe at onset
- Back pain:
 - Commonly interscapular or lumbar
- Combination of chest, back, and abdominal pain
- Neurologic complaints:
 - Visual changes
 - Stroke symptoms
- Aortic dissection may present with atypical symptoms that can result in a delay of diagnosis
 - Abdominal pain
 - Chest pressure
 - Leg pain
 - Syncope
 - Fever
 - Nausea, vomiting

Geriatric Considerations

Elderly are less likely to undergo surgery and have a higher mortality rate

- Elderly are less likely to describe their pain as abrupt in onset, have a pulse deficit, or have aortic insufficiency

Physical-Exam

- HTN:
 - 35–40% may be normotensive.
- Pulse deficits:
 - Discrepancies in BP between limbs
 - Usually in upper extremities

- Neurologic/spinal cord deficits
- Murmur of aortic regurgitation:
 - Occurs in up to 31% of patients
 - Musical, vibrating quality with variable intensity
 - Heard best along right sternal border
- Shock
 - If pericardial rupture or myocardial infarction (MI) from dissection into a coronary artery
- Atypical presentations
 - Ischemic lower extremity
 - Altered mental status
 - Congestive heart failure

ESSENTIAL WORKUP

ECG:

- Useful in ruling in or out ST-elevation MI or ischemia
- Dissection may involve coronary ostia and cause MI:
 - Inferior MI (right coronary artery lesion) is more common than left coronary artery territory.
- Useful for evaluating the presence of left ventricular hypertrophy
- A normal ECG in the presence of severe, acute-onset chest/back pain should heighten one's suspicion of an aortic dissection.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Leukocytosis
- Hematuria
- Elevated BUN and creatinine
- Elevated amylase secondary to bowel ischemia
- Elevated cardiac enzymes due to myocardial ischemia
- D-dimer < 500 ng/mL makes the diagnosis of dissection unlikely

Imaging

- CXR:
 - Useful in excluding other etiologies such as pneumothorax and pneumonia
 - In dissection, there may be a widened mediastinum or abnormal aortic contour.
 - An enlarged heart secondary to pericardial fluid (blood) may be present.
 - May be completely normal in as many as 12–18% of cases
- Echo—transthoracic or transesophageal:
 - Transthoracic:
 - Not very helpful in the diagnosis of aortic dissection

- May be used to evaluate for complications of a known dissection such as tamponade, valvular incompetence, or MI (from ostial occlusion)
- Transesophageal:
 - May be performed in the ED
 - Patients may require intubation.
 - Provides information regarding extent of dissection and complications
- CT:
 - Very useful in defining extent of dissection
 - May also be used in diagnosing clinical entities such as pulmonary embolism
 - Has a high sensitivity for the diagnosis of aortic dissection and is the diagnostic modality of choice in many centers
- MRI:
 - Highly sensitive and specific
 - Requires patient transport out of ED for extended period of time
 - Lack of immediate availability may be a problem
 - Study of choice in those with renal insufficiency or dye allergy
- Aortography:
 - High sensitivity and specificity
 - Useful for preoperative planning
 - Difficult to obtain in many centers
- Cardiac catheterization:
 - Due of overlap of symptomatology with cardiac ischemia, some patients may have diagnosis made by cardiac catheterization when an intimal flap is visualized.

DIFFERENTIAL DIAGNOSIS

- MI/ischemia
- Unstable angina
- Pneumothorax
- Esophageal rupture
- Pulmonary embolism
- Pericarditis
- Pneumonia
- Musculoskeletal pain



TREATMENT

PRE HOSPITAL

- Monitor
- IV access
- Oxygen

INITIAL STABILIZATION/THERAPY

- 2 large-bore IV lines
- Continuous cardiac monitoring
- Pulse oximetry
- Oxygen
- Type and cross

ED TREATMENT/PROCEDURES

- BP reduction to reduce shearing forces on aortic wall and slow down the dissection process
- Medications: IV β -blockade and nitroprusside
 - Medications are used to control HTN and cardiac contractility and decrease shearing forces.
 - Esmolol (IV) or labetalol (IV):
 - Contraindications: Bradycardia, COPD, hypotension
 - Nitroprusside (commonly used in conjunction with IV β -blocker)
 - Caution when using the above together: To prevent an initial increase in shear forces, β -blocker therapy should be started prior to the addition of nitroprusside therapy
- Emergent surgery:
 - Treatment of choice for type A dissection
 - Treatment for type B dissections in those who have failed medical therapy
- Medical management:
 - Treatment of choice for stable type B dissections

ALERT

Symptoms of aortic dissection may be similar to those of cardiac ischemia/infarction and pulmonary embolus. Treatment with thrombolytics and anticoagulants may be harmful and potentially fatal if aortic dissection is present.

MEDICATION

- Esmolol: 500 μ g/kg IV bolus, then 25--50 μ g/kg/min drip
- Labetalol: 10–20 mg IV over 2 min q10–15min. Then 2–4 mg/min IV drip. Total dose not to exceed 300 mg.
- Nitroprusside: 0.5 μ k/kg/min IV and titrate upward to desired effect. Dose should be based on IBW.



FOLLOW-UP

DISPOSITION

Admission Criteria

- All patients with acute aortic dissection should be admitted to the intensive care

unit.

- Emergency cardiothoracic surgery consultation should be obtained, especially in cases of type A dissection.

Discharge Criteria

None

FOLLOW-UP RECOMMENDATIONS

Close follow-up with cardiology and/or cardiothoracic surgery is of paramount importance.

PEARLS AND PITFALLS

- Untreated, nearly 75% of patients with ascending aortic dissection can be expected to die within 2 wk, with a mortality of 1–3%/hr in the 1st 48 hr.
- Majority of patients present with pain (90%) of severe intensity (90%) that occurred suddenly (84%).
- Although some recent literature has suggested a role for D-dimer testing, there is insufficient evidence to support its use as the sole screening test for aortic dissection.
- Should consider the diagnosis in patients with chest pain in whom conventional therapy (nitrates, β -blockers) are ineffective, and in those who have chest pain in addition to another complaint (extremity weakness, back pain, paresthesias, abdominal pain).
- Identification of risk factors is critical. These include:
 - HTN
 - Male gender
 - Cocaine use
 - Advanced age
 - Pregnancy
 - Connective tissue disorders, such as Marfan syndrome or cystic medial necrosis
 - Bicuspid aortic valve
 - Turner syndrome
 - Family history
 - Previous cardiac or valvular surgery

ADDITIONAL READING

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- Khan IA, Nair CK. Clinical, diagnostic, and management perspectives of aortic

dissection. *Chest*. 2002;122(1):311–328.

- Klompas M. Does this patient have an acute thoracic aortic dissection? *JAMA*. 2002;287:2262–2272.
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CODES

ICD9

441.01 Dissection of aorta, thoracic

ICD10

I71.01 Dissection of thoracic aorta

AORTIC RUPTURE, TRAUMATIC (TAI)

Stephen R. Hayden

BASICS

DESCRIPTION

- Traumatic aortic rupture (also referred to as traumatic aortic injury or TAI) is the cause of death in an estimated 20% of lethal motor vehicle collisions.
- An estimated 85% of patients with TAI die before reaching the hospital.
- Patients surviving to the ED usually have a contained rupture as aortic blood is tamponaded by the adventitia.
- Without proper treatment, of the 15% that survive the initial event, 49% will die within the 1st 24 hr, and 90% within 4 mo.
- Mean age of patients sustaining aortic rupture is 33 yr, and 70% are male.
- Most tears are transverse, not longitudinal.
- Tears may be partially or completely circumferential.

ETIOLOGY

- Most commonly results from motor vehicle collisions > 30 mph
- Unrestrained passengers, driver seat occupants (injuries from steering column and instruments), and ejected occupants.
- Other mechanisms: Auto versus pedestrian, airplane crashes, falls from height > 10 ft, crush and blast injuries, direct blow to chest
- Proposed mechanisms of aortic injury:
 - Shear forces arising from unequal rates of deceleration of the relatively fixed descending aorta and the more mobile arch
 - “Bending” stress at the aortic isthmus may cause flexion of the aortic arch on the left mainstem bronchus and pulmonary artery.
 - Twisting of the arch forces it superiorly and causes it to stretch.
 - Osseous structures (e.g., medial clavicles, manubrium, 1st rib) cause pinching of the trapped aorta as they strike the vertebral column.
 - “Waterhammer” fluid wave causes explosive rupture of aorta just distal to the aortic valve.

DIAGNOSIS

SIGNS AND SYMPTOMS

ALERT

Despite the severe nature of the injury, clinical manifestations are often deceptively subtle or nonexistent as patients frequently present with multiple coexisting injuries.

1/3–1/2 of these patients do not have external signs of chest trauma.

History

- Substernal chest pain is the most common symptom, but only present in ~25% of cases.
- Dyspnea, hoarseness, and stridor (tracheal compression from expanding hematoma) are less common.

Physical-Exam

- Neither sensitive nor specific for aortic injury
- Generalized HTN may occur from stimulation of sympathetic afferent nerves located near aortic isthmus.
- Harsh precordial or midscapular systolic murmur (1/3 of patients)
- Ischemic pain in lower extremities, oliguria/anuria, paraplegia from decreased aortic blood flow distal to aortic arch
- Swelling of base of neck (extravasation of blood)
- Acute coarctation syndrome (1/3 of patients): Upper extremity HTN with decreased pressures in low extremities, caused by periaortic hematoma compressing aortic lumen

ESSENTIAL WORKUP

Plain CXR is the primary screening tool with ~90% sensitivity, but low specificity.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC
- Chemistry
- Prothrombin time/partial thromboplastin time
- Type and cross-match (6–8 units PRBC)

Imaging

- Plain CXR:
 - Findings suggestive of mediastinal hemorrhage, hematoma, or associated injuries:
 - Widening of the superior mediastinum at the level of aortic arch (defined as >8 cm on a supine film, >6 cm in an upright PA film, or >0.25 mediastinum-width to chest-width ratio) is the most sensitive sign.
 - Obscuration of the aortic knob is also a sensitive sign.
 - More specific, but less sensitive, signs include opacification of the aortopulmonary window, rightward displacement of nasogastric tube, widened paratracheal stripe, and widened right paraspinal interface.

- 7–10% false-negative rate with normal mediastinum on x-ray; consider use of helical chest CT with high-speed deceleration mechanisms.
- In pediatric patients: The most common findings are a left apical cap, pulmonary contusion, aortic obscuration, and mediastinal widening.
- Helical chest CT angiography:
 - Preferred confirmatory study in stable patients
 - Nearly 100% sensitivity and specificity for detecting aortic rupture with improved CT technology
 - Has largely eliminated need for aortography
 - Advantages over aortography include noninvasive, provides information on other thoracic structures, more rapid
- Aortography:
 - Still considered by some to be the gold standard for diagnosis of TAI
 - Provides precise anatomic localization of aortic tears, useful for aorta injured at > 1 site (15–20% of cases)
 - Risk of further damage to aorta from catheter
 - Need for this modality is declining given advances in CT imaging quality.
- Transesophageal Echo (TEE):
 - Can be done rapidly in the ED
 - Can detect associated cardiac injuries (contusion, effusion, etc.)
 - Reported 87–100% sensitivity and 98–100% specificity
 - Contraindicated in patients with cervical, maxillofacial, or esophageal injuries
- MRI:
 - High accuracy
 - Lengthy study time and difficulty monitoring patients limit use
- Intravascular US:
 - Newer modality, availability is limited
 - Preliminary data suggest high sensitivity and specificity.

Pediatric Considerations

Presence of large thymus may make diagnosis of widened mediastinum difficult.

DIFFERENTIAL DIAGNOSIS

- Supine CXR can lead to false positive for widened mediastinum; obtain upright PA if possible.
- Mediastinal hematoma owing to other causes
- Mediastinal lymphadenopathy or tumor
- Redundant aorta resulting from HTN



TREATMENT

PRE HOSPITAL

Important information to retrieve at scene of injury:

- Vehicular speed
- Patient in driver or passenger seat
- Damage to steering column if driver is patient
- Ejection or use of seat belt

INITIAL STABILIZATION/THERAPY

- Follow advanced trauma life support protocols.
- Life-threatening intracranial, peritoneal, and retroperitoneal injuries take precedence.

ED TREATMENT/PROCEDURES

- Immediate trauma surgery consultation
- Immediate cardiothoracic or vascular surgery consultation (institution dependent)
- Avoid maneuvers that may result in a Valsalva-like response (e.g., gagging, straining)
- Aggressive pharmacologic treatment of BP and heart rate, as emerging data suggest delaying surgical repair may lead to improved outcomes
- Goal of medical therapy is to target heart rate 60 ± 5 bpm, systolic BP 100–120 mm Hg, and mean arterial BP 70–80 mm Hg to decrease risk of sudden free rupture and exsanguination:
 - β -blockers such as esmolol and labetalol are 1st-line agents
 - Calcium-channel blockers in patients with contraindications to β -blockade (CHF, COPD, 2nd- or 3rd-degree atrioventricular block)
 - Add vasodilator (nitroprusside) if needed to reach target BP and heart rate goals.
 - Antihypertensives are relatively contraindicated in acute coarctation syndrome.
- For significant hypotension, initiate rapid volume expansion, including blood.
- Vasopressors for refractory hypotension; norepinephrine and phenylephrine are preferred
- Central venous and arterial catheters

ALERT

Only administer vasodilator after initiating negative inotrope (β -blocker or calcium-channel blocker), as vasodilator alone can cause an increase in shearing forces on the intact aortic adventitia.

MEDICATION

- Esmolol: 500 μ g/kg bolus IV (peds: 100–500 μ g/kg bolus), then 50–150 μ g/kg/min IV infusion (peds: 25–100 μ g/kg/min IV infusion)
- Labetalol: 20 mg IV, followed by additional doses of 40 mg and 80 mg (peds: 0.2–

- 10 mg/kg per dose, max. 20 mg per dose) IV q10–15min, to 300 mg IV total; start infusion at 2 mg/min and titrate up to 10 mg/min (peds: 0.4–3 mg/kg/h infusion)
- Diltiazem: 20 mg (0.25 mg/kg) IV over 2 min; 2nd bolus 25 mg (0.35 mg/kg) in 15 min if needed; infusion 5–15 mg/h
- Norepinephrine: Start with 0.5–1 µg/min and titrate to desired response; 8–30 µg/min is usual dose (peds: Start 0.05–0.1 µg/kg/min, max. 2 µg/kg/min)
- Phenylephrine: 0.1–0.5 mg IV boluses q10–15min, initial dose not to exceed 0.5 mg (peds: 5–20 µg/kg/dose q10–15min); 100–180 µg/min or 0.5 µg/kg/min titrated to desired effect (peds: 0.1–0.5 µg/kg/min, titrated to desired effect)



FOLLOW-UP

DISPOSITION

Admission Criteria

All patients with aortic injuries must be admitted to the ICU if not taken directly to the OR.

FOLLOW-UP RECOMMENDATIONS

All patients with TAI are admitted to the hospital.

PEARLS AND PITFALLS

- Maintain a high degree of suspicion for TAI in patients with injuries from significant deceleration mechanisms.
- Clinical signs and symptoms may be subtle or nonexistent, necessitating some reliance on radiologic imaging for diagnosis.
- Special attention should be given to assessment of the mediastinum on CXR in trauma patients.
- Early pharmacologic control of BP and heart rate is of utmost importance when diagnosis is confirmed.

ADDITIONAL READING

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- Weidenhagen R, Bombien R, Meimarakis G, et al. Management of thoracic aortic lesions—the future is endovascular. *Vasa*. 2012;41(3):163–176.



CODES

ICD9

- 901.0 Injury to thoracic aorta
- 902.0 Injury to abdominal aorta

ICD10

- S25.01XA Minor laceration of thoracic aorta, initial encounter
- S25.02XA Major laceration of thoracic aorta, initial encounter
- S25.09XA Other specified injury of thoracic aorta, initial encounter

APHTHOUS ULCERS

Matthew R. Berkman

BASICS

DESCRIPTION

Painful ovoid or round ulcerations on the mucous membranes of the mouth, tongue or genitals:

- Commonly referred to as “canker sores”

ETIOLOGY

- Unknown
- Etiology likely multifactorial with some correlation with:
 - Immunologic dysfunction; alteration of cell-mediated immune system
 - Infection
 - Food hypersensitivities (i.e., gluten)
 - Vitamin deficiency
 - Pregnancy
 - Menstruation
 - Trauma
 - Stress
 - Ethnicity
 - Immunodeficiency
 - Medications: β -blockers, anti-inflammatory
- Epidemiology: Usually occurs in children and young adults (Peak age of onset: Between 10 to 19 yr old)
 - Most common inflammatory ulcerative condition of the oral cavity (20-40% of general population)
 - More common in women
 - May be familial

DIAGNOSIS

SIGNS AND SYMPTOMS

- Minor aphthous ulcers:
 - 70–90% of all aphthae
 - <5 mm in diameter; up to 5 appear at a time
 - Painful, shallow ulcers with necrotic centers
 - Raised, circumscribed margins and erythematous halos
 - Gray-white pseudomembrane
 - Affect nonkeratinized mucosa of anterior oral cavity

- Labial and buccal mucosa
- Floor of mouth
- Ventral surface of tongue
- Rarely found on dorsum of tongue, hard palate, or gingiva
- Last for 10–14 days; do not scar
- Fever/constitutional symptoms rarely associated
- Major aphthous ulcers or Sutton disease:
 - 10–15% of all aphthae
 - Similar in appearance but more painful than minor form
 - >5 mm in diameter; 1–10 ulcers at a time
 - Deeper than minor form
 - Involve all areas of oropharynx including pharynx, soft/hard palate, lips
 - Last for weeks to months, may scar
 - Onset after puberty
 - Often associated with underlying disease
 - Fever is rarely associated
- Herpetiform aphthous ulcers:
 - 7–10% of all aphthae
 - Multiple small clusters
 - <5 mm in diameter, 10–100 at any time, may coalesce into plaques
 - Herpetiform in nature, but herpes simplex virus cannot be cultured from lesions.
 - Predisposition for women
 - Last for 7–30 days; scarring can occur

History

- Prodrome of burning or pricking sensation of oral mucosa 1–2 days prior to appearance of ulcers
- Inquire about patient or family history of:
 - Systemic lupus erythematosus (SLE)
 - Inflammatory bowel disease (IBD)
 - Behçet disease
 - Reiter disease
 - Gluten sensitivity
 - Cancer
 - HIV
- Inquire about patient sexual history of syphilis or herpes virus
- Inquire about current medications:
 - NSAIDs
 - β -blockers

Physical-Exam

- See “Signs and Symptoms.”
- Look for signs of dehydration:
 - Vital signs should be within normal limits.
 - Evaluate mucus membranes.
- Evaluate for signs of secondary infection.
- Evaluate for signs of systemic causes of ulcers (see “History”).

ESSENTIAL WORKUP

- Diagnosis is made by history and clinical presentation.
- Rule out oral manifestation of systemic disease:
 - More likely if persists > 3 wk or associated with constitutional symptoms
- Focus on symptoms of eyes, mouth, genitalia, skin, GI tract, allergy, diet history and physical exam

DIAGNOSIS TESTS & INTERPRETATION

Lab

Routine lab testing not indicated:

- Needed only when systemic etiologies causing ulcers are suspected
- Biopsy should be considered for ulcers lasting more than 3 wk
- Should be guided by history and physical exam:
 - CBC series
 - Rapid plasma reagin (RPR) (syphilis)
 - Fluorescent treponemal antibody-absorption test
 - Antinuclear antibody test
 - Tzanck stain: Inclusion giant cells (herpes virus)
 - Biopsy: Multinucleated giant cells (cytomegalovirus)
 - Fungal cultures

Diagnostic Procedures/Surgery

An outpatient biopsy should be considered for any ulcer > 3 wk

DIFFERENTIAL DIAGNOSIS

- Trauma:
 - Biting
 - Dentures
 - Braces
- Drug exposure:
 - NSAIDs
 - Nicorandil
 - β -blockers
- Infection:
 - Herpes virus:

- Vesicular lesions
- Ulcers on attached mucosa
- Cytomegalovirus:
 - Immunocompromised patient
- Varicella virus:
 - Characteristic skin lesions
- Coxsackievirus:
 - Ulcers preceded by vesicles
 - Hand, foot, and buttock lesions
- Syphilis:
 - Other skin or genital lesions
- Erythema multiforme:
 - Lip crusting
 - Lesions on attached and unattached mucosa skin lesions
- *Cryptosporidium* infection, mucormycosis, histoplasmosis
- Necrotizing gingivitis
- Underlying disease:
 - Behçet syndrome:
 - Genital ulceration
 - Uveitis
 - Retinitis
 - Reactive arthritis (Reiter syndrome):
 - Uveitis
 - Urethritis
 - HLA-B27-associated arthritis
 - Sweet syndrome:
 - Fever
 - Erythematous skin plaques/nodules
 - In conjunction with malignancy
 - IBD:
 - Bloody or mucous diarrhea
 - GI ulcerations
 - Weight loss
 - Gluten-sensitive enteropathy:
 - Weight loss
 - SLE:
 - Malar rash
 - ANA positive
 - Bullous pemphigoid/pemphigus vulgaris:
 - Vesiculobullous lesions on attached and unattached mucosa
 - Diffuse skin involvement
 - Cyclic neutropenia:
 - Periodic fever

- Squamous cell carcinoma:
 - o Chronic
 - o Head/neck adenopathy
- Immunocompromised patient:
 - HIV
 - Agranulocytosis
 - Malignancy



TREATMENT

ED TREATMENT/PROCEDURES

- Treatment guided by severity and duration of symptoms
- Goal is for symptomatic pain relief and reduction of inflammation.

MEDICATION

- Mild to moderate disease:
 - Avoid oral trauma/acidic foods
 - Topical anesthetic
 - o Magnesium hydroxide/diphenhydramine hydrochloride 5 mg/5 mL in 1/1 mix swish and spit
 - o Viscous lidocaine 2–5%: Applied to ulcer QID after meals until healed
 - Protective bioadhesives
 - o Topical OTC preparations (Orabase, Anbesol): Applied to ulcer QID after meals until healed
 - Topical anti-inflammatory
 - o Amlexanox 5% paste (Aphthasol): applied to ulcer QID after meals until healed
 - Antimicrobial mouthwash
 - o Chlorhexidine gluconate aqueous mouthwash 0.12% (Peridex): Mouth rinse QID after meals until healed
- Severe disease:
 - Prednisone tablets: 30–60 mg PO per day × 7 d
 - Thalidomide: 50–200 mg PO per day × 4 wk



FOLLOW-UP

DISPOSITION

Admission Criteria

- Unable to eat or drink after appropriate analgesia
- Abnormal vital signs or evidence of dehydration

Discharge Criteria

- Tolerating fluids
- Adequate analgesia
- Normal vital signs

Issues for Referral

Follow up with primary care physician if lesions have not resolved within 2 wk.

FOLLOW-UP RECOMMENDATIONS

- Avoid oral trauma (hard foods) or acidic foods.
- Referral to a specialist if underlying disease suspected

PEARLS AND PITFALLS

- The vast majority of aphthous ulcers are benign, self-limited, and treated symptomatically
- ED physicians must consider underlying systemic cause of ulcers.

ADDITIONAL READING

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- Wanda C, Chi AC, Neville BW. Common oral lesion: Part I. Superficial mucosal lesions. *Am Fam Physician.* 2007;75:501–507.



CODES

ICD9

- 528.2 Oral aphthae
- 608.89 Other specified disorders of male genital organs
- 616.50 Ulceration of vulva, unspecified

ICD10

- K12.0 Recurrent oral aphthae

- N50.8 Other specified disorders of male genital organs
- N76.6 Ulceration of vulva

APNEA, PEDIATRIC

Sarah M. Halstead



BASICS

DESCRIPTION

- Absence of respiratory airflow for a period of 20 sec, with or without decreased heart rate:
 - Central apnea:
 - Disruption in the generation or propagation of respiratory signals in the brainstem and descending neuromuscular pathways
 - Obstructive apnea:
 - Respiratory effort is present, but there is no airflow
 - Structural airway obstruction, often with paradoxical chest wall movement
 - Functional obstruction from airway collapse
 - Mixed
- Apparent life-threatening event (ALTE):
 - Episode that is associated with a combination of apnea, color change, change in tone, choking, or gagging
 - A clinical presentation, not a diagnosis

ETIOLOGY

- Infection:
 - Sepsis
 - Meningitis or encephalitis
 - Pneumonia
 - Pertussis/chlamydia
 - RSV and other viral respiratory infections
- Respiratory:
 - Obstructive airway lesions
 - Enlarged tonsils and adenoids
 - Vocal cord dysfunction
 - Laryngotracheomalacia
 - Vascular ring
 - Foreign body
 - Craniofacial abnormality
 - Choanal atresia or stenosis
 - Functional obstruction from airway collapse
 - Infection
 - Immaturity/prematurity

- Abnormal ventilatory response to hypoxia/hypercarbia
- Neurologic:
 - Seizure
 - Intracranial hemorrhage
 - Increased intracranial pressure
 - Tumor
 - Arnold–Chiari or other CNS malformation
 - Ingestion
 - Toxin
 - Carbon monoxide
 - Hypoxic injury
 - Neuromuscular disorder
 - Central hypoventilation syndrome
- Cardiac:
 - Dysrhythmia
 - Congenital heart disease
 - CHF
 - Myocarditis
 - Cardiomyopathy
- GI:
 - GERD
 - Volvulus
 - Intussusception
- Child abuse
- Endocrine/metabolic:
 - Hypoglycemia
 - Electrolyte disorders
 - Inborn errors of metabolism
- Other:
 - Transient choking episode
 - Laryngospasm
 - Periodic breathing
 - Breath-holding spell



DIAGNOSIS

ALERT

If the patient is apneic, treatment must commence at once.

SIGNS AND SYMPTOMS

Apnea may be current, historical, or impending.

History

- Duration of apnea
- State:
 - Asleep, awake, crying
 - Relationship to feeds and position (supine, prone)
- Respiratory effort:
 - None, shallow breathing, increased work of breathing, struggling to breathe, choking
- Presence and location of any color change
- Position of eyes
- Description of movements and muscle tone
- Interventions done by the caregiver
- Antecedent symptoms such as fever or cough
- Antecedent trauma
- Past medical history, including prematurity, cardiopulmonary, GI, or neurologic conditions
- Any past history of ALTEs in this patient or family members

Physical-Exam

- Vital signs with temperature
- Growth parameters:
 - Weight pattern
 - OFC (head circumference) pattern
- Pulse oximetry
- Exam of airway and lungs:
 - Assess impending apnea
 - Stridor or other evidence of upper airway obstruction
 - Fast or slow respirations
 - Use of accessory muscles
 - Adventitial lung sounds
- Exam of heart:
 - Irregular rhythm
 - Murmur
 - Evidence of CHF
- Neurologic exam:
 - Assess mental status
 - Assess for trauma, seizure, or toxidrome
 - Muscle tone and reflexes
 - Funduscopic exam

ESSENTIAL WORKUP

- Complete history and physical exam
- The historical factors and exam will direct the diagnostic evaluation and treatment

- Check/clear out upper airway as appropriate.
- Remove or suction any obstruction as appropriate
- Ensure proper head positioning with special consideration for occult trauma

DIAGNOSIS TESTS & INTERPRETATION

Lab

Perform as appropriate for presentation:

- Dextrostix
- CBC
- Urinalysis
- CSF studies
- Blood, urine, and CSF cultures
- Electrolytes (including calcium)
- BUN, creatinine
- Blood gas
- RSV and respiratory viral studies
- Pertussis and chlamydia tests
- Consider toxicologic screen (including toxic alcohols and acetaminophen)
- Consider LFTs and ammonia

Imaging

Perform as appropriate for presentation:

- CXR
- Head CT or MRI
- ECG
- UGI or swallowing study
- Polysomnography in follow-up in patient with suspected central or obstructive sleep apnea
- EEG in follow-up
- 0 Bone survey and other studies as indicated

DIFFERENTIAL DIAGNOSIS

- Multiple etiologies as previously noted
- Special considerations:
 - _ Breath-holding spells:
 - Reflexive cessation of respiratory effort during expiration
 - Cyanotic and pallid types
 - Paroxysmal event occurring in 0.1–5% of healthy children 6 mo–6 yr of age
 - _ Periodic breathing may be seen in neonates:
 - 3 or more respiratory pauses lasting > 3 sec with < 20 sec of respiration between pauses

- May be normal event

ALERT

In a neonate, strongly consider occult sepsis.



TREATMENT

PRE HOSPITAL

- Respiratory support as indicated
 - _ High-flow oxygen if breathing resumes
 - _ Check/clear out upper airway
 - _ Bag-mask ventilation
 - _ Endotracheal intubation if continued apnea
- IV access, cardiac monitoring
- Look for signs of an underlying cause:
 - _ Medications
 - _ Document a basic neurologic exam:
 - GCS
 - Pupils
 - Extremity movements
 - _ Gross signs of trauma
 - _ Talk with family/pre-hospital personnel for information

INITIAL STABILIZATION/THERAPY

- Establish unresponsiveness
- Check/clear out upper airway
- Remove or suction any obstruction
- Ensure proper head positioning

ED TREATMENT/PROCEDURES

- If currently apneic, ventilate with the bag-valve-mask device and high-flow oxygen
- Endotracheal intubation is required if apnea persists
- Resuscitation medications and antibiotics as indicated
- Support and counseling if breath holding suspected

MEDICATION

- Antibiotic doses in ED
 - _ Ceftriaxone: 50 mg/kg IV
 - _ Vancomycin: 15 mg/kg IV
 - _ Neonates:
 - Ampicillin: 50 mg/kg IV
 - Gentamicin: 2.5 mg/kg IV
- Dextrose: 2–4 mL/kg D₂₅W IV or 5-10 mL/kg D₁₀W IV

- Neonates: 1 mo 2–4 mL/kg D₁₀W IV
- Naloxone: 0.01–0.1 mg/kg IV/IM/SC/ET
 - Caution: May precipitate withdrawal symptoms in patients with chronic opiate use



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients who were or may become apneic should be admitted to an inpatient unit for appropriate monitoring. Those with persistent abnormal vital signs need intensive care monitoring.
- Variables that identify most children requiring admission include those with an obvious need for admission including abnormal vital signs or a medical history, or >1 apparent ALTE event in 24 hr.
- Recommend referral for pediatric evaluation and follow-up as indicated. Interventions may include further studies (i.e., EEG), antireflux medications or caffeine, and home monitoring.

Discharge Criteria

In patients without true apnea who are low risk and have no abnormalities noted during the period of observation and evaluation, discharge may be considered, assuming that parents are compliant and comfortable with their child and follow-up and support are definitively established.

Issues for Referral

Primary care physician and subspecialist, reflecting suspected etiology

PEARLS AND PITFALLS

- Consider occult sepsis, especially in a neonate
- Consider occult trauma

ADDITIONAL READING

- Brand AD, Altman RL, Purtill K, et al. Yield of diagnostic testing in infants who have had an apparent life-threatening event. *Pediatrics*. 2005;115:885–893.
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See Also (Topic, Algorithm, Electronic Media Element)

- Sudden Infant Death Syndrome
- Neonatal Sepsis



CODES

ICD9

- 327.23 Obstructive sleep apnea (adult)(pediatric)
- 770.81 Primary apnea of newborn
- 786.03 Apnea

ICD10

- G47.33 Obstructive sleep apnea (adult) (pediatric)
- P28.4 Other apnea of newborn

APPENDICITIS

Colleen N. Hickey • Jennifer L. Kolodchak



BASICS

DESCRIPTION

- Most common abdominal emergency
- Acute obstruction of appendiceal lumen results in distension followed by organ ischemia, bacterial overgrowth, and eventual perforation of the viscus
- Pain migration:
 - Periumbilical pain: Appendiceal distension stimulates stretch receptors, which relay pain via *visceral* afferent pain fibers to 10th thoracic ganglion.
 - RLQ pain: As inflammation extends to surrounding tissues, pain occurs owing to stimulation of *parietal* nerve fibers and localizes to position of appendix.

Pediatric Considerations

- 28–57% misdiagnosis in patients <12 yr (nearly 100% in patients <2 yr)
- 70–90% perforation rate in children <4 yr
- Perforation correlates strongly with delayed diagnosis.

Geriatric Considerations

- Decreased inflammatory response
- 3 times more likely to have perforation owing to anatomic changes
- Diagnosis often delayed owing to atypical presentations

Pregnancy Considerations

- Slightly higher rate in 2nd trimester compared to 1st/3rd/postpartum periods
- Increased perforation rate (25–40%), highest in 3rd trimester
- RLQ pain remains the most common symptom
- 7–10% fetal loss, up to 24% in perforated appendicitis

ETIOLOGY

- Luminal obstruction of appendix
- Appendiceal lumen becomes distended, inhibiting lymphatic and venous drainage.
- Bacterial invasion of wall, with edema and blockage of arterial blood flow
- Perforation and spillage of contents into peritoneal cavity, causing peritonitis (usually 24–36 hr from onset)
- May wall off and form abscess
- Gram-negative rods and anaerobic organisms predominate

SIGNS AND SYMPTOMS

History

- **Abdominal pain: Primary symptom:**
 - Normal location:
 - RLQ pain
 - 35% of patients have appendix located within 5 cm of “normal” location.
 - Retrocecal appendix (28–68%):
 - Back pain
 - Flank pain
 - Testicular pain
 - Pelvic appendix (27–53%):
 - Suprapubic pain
 - Urinary or rectal symptoms
 - Long appendix (< 0.2%):
 - Inflamed tip may cause pain in RUQ or LLQ.
 - Anorexia
 - Vomiting
- **Change in bowel habits: Diarrhea (33%), constipation (9–33%)**
- **Classic presentation (< 75% adults):**
 - Initially periumbilical pain
 - Followed by anorexia (1st symptom in 95%) and nausea
 - Localizes to RLQ (1–12 hr after onset)
 - Finally, vomiting with fever

Pediatric Considerations

- **Presentations often nonspecific and difficult to localize (< 50% have classic presentation)**
- **Anorexia, vomiting, and diarrhea more common (half-eaten meal hours before complaints of pain may more accurately indicate duration of symptoms)**
- **Observe child before exam for subtle indicators of local inflammation:**
 - Limping gait
 - Hesitation to move or climb
 - Flexed right hip

Physical-Exam

- **Vital signs:**
 - Often normal
 - Fever: Normal to mild elevation (< 1°F) initially, increases with perforation

- Abdominal exam:
 - Tenderness at McBurney point (1/3 of distance from right anterior iliac spine to umbilicus)
 - Guarding:
 - Voluntary guarding early owing to muscular resistance to palpation
 - Involuntary guarding (rigidity) later as inflammation progresses and perforation occurs
 - Rebound:
 - Pain with any rapid movement of peritoneum (e.g., bumping stretcher)
 - Specific signs (less useful in pediatrics):
 - *Rovsing sign*: Pain in RLQ when palpating LLQ
 - *Psoas sign*: Increased pain on extension of right hip with patient lying on her or his left side, owing to inflamed appendix touching iliopsoas muscle.
 - *Obturator sign*: Pain with passive internal rotation and flexion of right hip
- Rectal exam:
 - Limited value: May localize tenderness/mass
- Pelvic exam:
 - Important to differentiate gynecologic disease
 - Vaginal discharge and/or adnexal tenderness or mass suggests gynecologic disease.
 - Cervical motion tenderness when present suggests PID, but can be seen in up to 25% of women with appendicitis
- Patient position:
 - Supine or decubitus with legs (particularly the right) drawn up
 - Prefer not to move
- Shuffling gait—known as “appy walk”

Pediatric Considerations

Almost all children have generalized abdominal tenderness with some rigidity.

Pregnancy Considerations

- Enlarging uterus displaces appendix upward and laterally.
- Hyperemesis gravidarum and other nonsurgical causes of vomiting should not cause abdominal tenderness.

Geriatric Considerations

Typical signs of peritonitis may be absent in elderly.

ESSENTIAL WORKUP

- Suggestive history and physical exam sufficient to establish preoperative diagnosis and warrant surgical consultation
- Tests listed below may be used to assist in diagnosis
- Atypical cases: Repeat serial exams in conjunction with some of the tests listed below is effective, with decreased rates of negative appendectomies and no increase in rates of perforation

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC:
 - WBC > 10,000, with left shift (80%)
 - Normal WBC does *not* exclude diagnosis
- C-reactive protein:
 - Overall sensitivity 62%, specificity 66%
 - May not be elevated early (< 12 hr)
 - Increased sensitivity with serial measurements
- Urinalysis:
 - Generally normal
 - Mild pyuria, bacteriuria, or hematuria (25–30%)
 - Pyuria present if inflamed appendix lies near ureter or bladder
- Pregnancy test for females of child-bearing age

Imaging

- Unnecessary when diagnosis is clear
- Most helpful in female patients of child-bearing age where diagnosis is often unclear
- Abdominal radiographs—not recommended
- US: Sensitivity 86–90%; specificity 92–95%:
 - Noncompressible appendix 6 mm anteroposterior (AP) diameter
 - Presence of appendicolith
 - Periappendiceal fluid/mass
 - Limited by obesity, bowel gas, retrocecal appendix, and operator
 - Negative study of limited use
- CT: Sensitivity 91–100%; specificity 94–97%:
 - Highest yield using oral and rectal contrast with focused appendiceal technique (5 mm cuts from 3 cm above cecum extending distally 12–15 cm)
 - Fat stranding (100%)
 - Appendix 6 mm in diameter (93%)
 - Focal cecal apical thickening
 - Defines appendiceal masses (phlegmon vs. abscess)
 - Best study for finding alternative diagnoses
 - Nonvisualized appendix does not rule out appendicitis

- MRI: Sensitivity 97–100%, specificity 92–94%:
 - Appendix 7 mm in diameter
 - Periappendiceal fat stranding
 - Advantages: Lack of ionizing radiation, excellent safety profile of gadolinium contrast agents
 - Disadvantages: High cost, limited availability, lengthy exam, lack of radiologist familiarity in appendicitis
 - No gadolinium in early pregnancy (class C drug)

Pediatric Considerations

American College of Radiology recommends US followed by CT as needed for suspected appendicitis

Diagnostic Procedures/Surgery

- Laparoscopy:
 - Diagnostic and therapeutic use
 - Gross pathology may be absent with positive microscopic findings
- Open appendectomy
- Percutaneous drainage

DIFFERENTIAL DIAGNOSIS

- Gastroenteritis
- Meckel diverticulum
- Epiploic appendicitis
- Crohn's disease
- Diverticulitis
- Volvulus
- Abdominal aortic aneurysm
- Intestinal obstruction
- UTI
- Pyelonephritis
- PID
- Ectopic pregnancy
- Ovarian cyst/torsion
- Tubo-ovarian abscess
- Endometriosis
- Renal stone
- Testicular torsion
- Mesenteric adenitis
- Henoch–Schönlein purpura
- Diabetic ketoacidosis
- Streptococcal pharyngitis (children)
- Biliary disease



TREATMENT

INITIAL STABILIZATION/THERAPY

- Airway, breathing, and circulation management (ABCs)
- Fluid resuscitation with LR or 0.9% NS

ED TREATMENT/PROCEDURES

- IV fluids, correct electrolyte abnormalities
- Immediate surgical consult for convincing history and physical exam:
 - Laparoscopic versus open technique
 - Negative appendectomy rate of 10% in males and 20% in females
 - Percutaneous drainage, IV antibiotics, bowel rest and possible interval appendectomy in 6–8 wk in appendiceal abscesses
- Perioperative antibiotics
- NPO
- Order CT if palpable mass is present in RLQ to define phlegmon versus abscess
- If diagnosis is uncertain, send serial labs, observe, and repeat exams (6–10% negative appendectomy rate with observation protocols)
- Analgesics:
 - Administration of analgesics, including narcotics, does not adversely affect abdominal exam or mask pathology

MEDICATION

- Ampicillin/sulbactam: 3 g (peds: 100–200 mg ampicillin/kg/24 h) IV q6h
- Cefoxitin: 2 g (peds: 80–100 mg/kg/24 h) IV q6h
- Ceftriaxone: 1 g (peds: 50–100 mg/kg) IV q24h
- Ciprofloxacin: 400 mg (peds: 20–40 mg/kg) IV q12h
- Ertapenem: 1 g IM/IV q24h
- Metronidazole: 500 mg (peds: 30–50 mg/kg/24 h) IV q8–12h
- Morphine sulfate: 3–5 mg (peds: 0.1–0.2 mg/kg per dose q2–q4h) IV, every 15 min titrated to effect
- Piperacillin/tazobactam: 3.375 g (peds: 150–300 mg/kg/d if < 6 mo; 240–400 mg/kg/d if > 6 mo) IV q6h



FOLLOW-UP

DISPOSITION

Admission Criteria

- Surgical intervention of acute appendicitis
- Observation or further diagnostic workup if diagnosis is uncertain

Discharge Criteria

Patients with abdominal pain thought not to be appendicitis may be discharged if they meet the following criteria:

- Resolved or resolving symptoms
- Minimal or no abdominal tenderness
- No lab/radiologic abnormalities
- Able to tolerate PO intake
- Adequate social support and able to return if symptoms worsen

FOLLOW-UP RECOMMENDATIONS

24–48 hr recheck for patients discharged from the ED with abdominal pain of unclear etiology

PEARLS AND PITFALLS

- Pediatric and geriatric patients present atypically and have increased perforation rates
- Imaging is not required in a classic presentation of acute appendicitis
- Appendicitis cannot be ruled out on any imaging modality if the appendix is not visualized

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Abdominal Pain
- Vomiting, Adult; Vomiting, Pediatric



CODES

ICD9

- 540.1 Acute appendicitis with peritoneal abscess
- 540.9 Acute appendicitis without mention of peritonitis
- 541 Appendicitis, unqualified

ICD10

- K35.3 Acute appendicitis with localized peritonitis
- K35.80 Unspecified acute appendicitis
- K37 Unspecified appendicitis

ARSENIC POISONING

Vinodinee L. Dissanayake



BASICS

DESCRIPTION

- Acute toxicity:
 - Caused by intentional ingestion, malicious poisoning, or medication error
- Minimal lethal ingested dose ~ 2 mg/kg
- Chronic toxicity:
 - Resulting from occupational exposures, water or food contamination, or use of folk remedies containing arsenic
- Ingestion is the primary route of exposure
- Inhalational toxicity is possible from arsine gas exposure

ETIOLOGY

- Most cases seen in the ED result from intentional ingestion or malicious poisoning
- Sodium arsenate, found in ant killer, is the most common acute exposure in the US
- Contaminated food and water supplies are the most common cause worldwide
- Inorganic arsenic trioxide has been recently approved as a chemotherapeutic agent for acute myelogenous leukemia (AML)
- Melarsoprol, an organic arsenical, has been used to treat trypanosomiasis since 1949
- Found in pesticides, certain folk remedies (herbal balls), industrial wood preservatives
- May be released as arsine gas from combustion of zinc- and arsenic-containing compounds

Mechanism

- Arsenic exists in several forms—gas (arsine, or lewisite), organic, elemental, and inorganic
- Inorganic forms (pentavalent and trivalent arsenic) are most frequently involved in toxic exposures:
 - Pentavalent arsenic uncouples oxidative phosphorylation
 - Most pentavalent arsenic is converted to the more toxic trivalent arsenic in the body
 - Trivalent arsenic binds sulfhydryl groups and interferes in hemoglobin production
 - Some trivalent arsenic may be methylated into species of varying toxicity
 - The more reactive species are DNA damaging and genotoxic

DIAGNOSIS

SIGNS AND SYMPTOMS

- CNS:
 - Altered mental status/encephalopathy
 - Neurodevelopmental deficits in children
 - Peripheral neuropathy
 - Acute: Sensory neuropathy
 - Subacute: Sensorimotor neuropathy
 - Peripheral dysesthesias
 - Headache
 - Seizures
- Cardiovascular:
 - Prolonged QTc interval
 - Hypotension (acute) or hypertension (chronic)
 - Dysrhythmias, primarily ventricular
 - Nonspecific ST segment changes
 - Noncardiogenic pulmonary edema
- Pulmonary:
 - Inhalational exposure increases lung cancer risk and respiratory mortality
 - Large acute ingestion (8 mg/kg) may lead to severe respiratory distress
 - Pulmonary edema, hemorrhagic bronchitis, and bronchopneumonia
- GI:
 - Nausea, vomiting after ingestion and possibly inhalation
 - Protracted and may be refractory to antiemetics at usual doses
 - Can have hemorrhagic gastroenteritis; corrosive to GI tract
 - Rice water diarrhea
 - Abdominal pain
 - Garlic odor to breath, vomit, stools
 - Causes acute hepatitis; chronically, can cause portal HTN
 - A possible association with diabetes mellitus in chronic exposure
- Miscellaneous (usually associated with chronic exposure)
 - Acute rhabdomyolysis
 - Blackfoot disease in Taiwan: Gangrene from loss of circulation to extremities
 - Dermatitis, such as toxic erythroderma and hyperkeratotic, hyperpigmented lesions
 - Hemolytic anemia (more pronounced with arsine gas exposure)
 - Hypothyroidism (antagonizes thyroid hormone)
 - Increased risk of carcinoma (liver/basal cell/squamous cell of skin/bronchogenic)
 - Leukopenia (after several days)

- Mees lines (white bands across the nails owing to growth arrest caused by arsenic)
- Patchy alopecia
- Raynaud phenomenon and vasospasticity

ESSENTIAL WORKUP

- Spot urine arsenic level
- CBC

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Spot urine arsenic level $> 1,000 \mu\text{g/L}$ may confirm diagnostic suspicion:
 - Peaks 10–50 hr postingestion
- Definitive test is 24 hr urine collection with speciation into organic and inorganic types of arsenic.
- Blood levels not routinely helpful owing to short half-life in serum (~ 2 hr)
- CBC to evaluate for anemia, leukopenia, basophilic stippling
- Electrolytes, BUN/creatinine, and glucose
- Urinalysis to look for evidence of hemolysis/rhabdomyolysis
- Liver function tests
- Total creatine phosphokinase (CPK) for rhabdomyolysis
- Hair and nail arsenic levels:
 - Do not help in acute setting
 - May help determine chronicity of exposure in select populations

Imaging

- Plain abdominal radiographs to look for radiopaque foreign body
- Cranial CT/other studies as indicated by patient's condition

DIFFERENTIAL DIAGNOSIS

- Acute toxicity:
 - Acute appendicitis/colitis/gastroenteritis
 - Celiac disease
 - Cholera
 - Distributive shock
 - Encephalopathy
 - Toxic ingestions
 - *Amanita* mushroom poisoning
 - Cyclic antidepressants or other seizure-inducing toxins
 - Organophosphates
- Chronic toxicity:
 - Addison disease

- Guillain–Barré syndrome or other neuropathy
- Raynaud phenomenon
- Thromboangiitis obliterans, or other vasculitides
- Vitamin deficiency (B₃, B₆, or B₁₂)
- Wernicke–Korsakoff syndrome



TREATMENT

PRE HOSPITAL

ALERT

- If possible to do so safely, bring containers in suspected overdose/poisoning.
- Decontaminate skin.
- Support airway/breathing/circulation.
- Cardiac monitoring

INITIAL STABILIZATION/THERAPY

- ABCs:
 - Cardiac monitor
 - Isotonic crystalloids as needed for hypotension
- Naloxone, thiamine, and dextrose (D50W) as indicated for altered mental status
- Cardiovascular:
 - Vasopressors if refractory hypotension is present
 - Central venous pressure monitoring to prevent pulmonary/cerebral edema
 - Avoid type IA, IC and III antidysrhythmic agents, which worsen QTc prolongation
 - Continuous cardiac monitoring for QTc prolongation
- Neurologic:
 - Treat seizures with benzodiazepines
 - Assist ventilation for respiratory failure from neuromuscular weakness
- Renal:
 - Hemodialysis for renal failure
- Alimentary:
 - Dextrose, enteral or parenteral feeding may be beneficial

ED TREATMENT/PROCEDURES

- Decontamination:
 - Orogastric lavage or aspiration may be helpful within the 1st hr of ingestion
 - Activated charcoal does not bind arsenic
 - If opacities are seen on abdominal film, administer whole bowel irrigation (polyethylene glycol) at 1–2 L/hr until repeat radiographs are clear
 - If dermal exposure, decontaminate skin as 1st step in management

- Ensure that no one else is contaminated and environment is evaluated
- Ensure that electrolytes such as calcium, magnesium, and potassium are replaced
- Evaluate need for chelation therapy, based on levels, acuity of exposure, clinical symptoms:
 - Consult with medical toxicologist/poison center
 - Agents
 - Dimercaprol (British anti-Lewisite)
 - DMSA (succimer)
- Elimination:
 - Hemodialysis not routinely effective
 - Consider for patient with renal failure or other hemodialysis indications
 - Continue chelation throughout hemodialysis sessions

MEDICATION

- Dimercaprol (British anti-Lewisite): 3 mg/kg deep IM q4h for 24 h, then q6h for the next 24 h, then q12h until able to tolerate PO
 - Caution: Contraindicated in patients with peanut allergies
- Dextrose 50%: 25 g (50 mL) (peds: 0.5 g/kg D₂₅W) IV for hypoglycemia
- DMSA (succimer): 10 mg/kg PO q8h for 5 d, then q12h for 14 d
- Sodium bicarbonate: 1 mEq/kg IV bolus, followed by infusion of 150 mEq in 1 L of D₅W at 150 mL/h
 - Used to treat rhabdomyolysis
 - Ensure that potassium and other electrolytes are monitored and replaced during infusion
- Naloxone: 0.4–2.0 mg (peds: 0.1 mg/kg) IV, may repeat up to 10 mg for suspected opioid intoxication
- Thiamine: 100 mg IM or IV (peds: 1 mg/kg)
- Vasopressors after sufficient fluids
 - Dopamine 5 µg/kg/min, increase by 5–10 µg/kg/min (q10–30min) Max.: 20 µg/kg/min
 - Norepinephrine 0.01–3 µg/kg/min, start at 2 µg/min, titrate to MAP 65–90 mm Hg
- Max.: 20 µg/min



FOLLOW-UP

DISPOSITION

Admission Criteria

Symptomatic arsenic exposures should be admitted to an intensive care setting.

Discharge Criteria

- Asymptomatic patients with a spot urinary arsenic level < 50 µg/L may be discharged
- Suspected chronic exposures who do not require admission should be referred for outpatient evaluation and 24 hr urine collection
- Ensure that home environment is safe for patient prior to discharge

FOLLOW-UP RECOMMENDATIONS

- Psychiatric follow-up for intentional overdoses
- Primary care follow-up for cancer screening and monitoring

PEARLS AND PITFALLS

- Arsenic poisoning results in a myriad of signs and symptoms
 - Suspect acute arsenic poisoning when patients present with gastrointestinal distress and neurologic findings.
 - Suspect chronic arsenic poisoning in patients who present with neurologic deficits, nonspecific wasting, and hyperkeratotic skin lesions.
- Consult a medical toxicologist/poison center regarding the need for chelation therapy.

A special thanks goes to Dr. Gerald Maloney Jr, who contributed to the previous edition.

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CODES

ICD9

985.1 Toxic effect of arsenic and its compounds

ICD10

- T57.0X1A Toxic effect of arsenic and its compounds, accidental (unintentional), initial encounter
- T57.0X2A Toxic effect of arsenic and its compounds, intentional self-harm, initial encounter
- T57.0X3A Toxic effect of arsenic and its compounds, assault, initial encounter

ARTERIAL GAS EMBOLISM (AGE)

Nicole L. Lunceford • Catherine M. Visintainer • Peter J. Park

BASICS

DESCRIPTION

- Results when air bubbles enter the pulmonary venous return from ruptured alveoli, then propagate through the systemic vasculature:
 - Clinical manifestations depend on location of air bubbles in systemic vasculature system.
- Also known as dysbaric air embolism or cerebral air embolism
- Caused by overpressurization of lung tissue, causing pleural tear with air entering the vascular circulation:
 - Trapped air (in lungs with closed glottis) expands on diver ascent.
 - Boyle law: At a constant temperature, pressure (P) is inversely related to volume (V):
 - $PV = K$ (constant) or $P_1V_1 = P_2V_2$
 - As pressure increases/decreases, volume decreases/increases.

ETIOLOGY

- Pulmonary atrioventricular (AV) shunts, or as paradoxical embolism via a patent foramen ovale
- Breath holding during ascent:
 - Symptoms attributable to a shower of bubbles and multiple blood vessel involvement
- Iatrogenically during placement of central venous pressure (CVP) lines, cardiothoracic surgery, or hemodialysis
- Penetrating injuries to heart, with emergent repair of cardiac wound

DIAGNOSIS

SIGNS AND SYMPTOMS

- Cerebral:
 - Rapid onset:
 - Almost all cases of AGE present within 1st 5 min of surfacing, although most often symptoms are evident in 1st 2 min
 - Dive-related stroke
 - 2 main presentations:
 - Apnea and full cardiopulmonary arrest
 - Any combination of neurologic deficits
 - Presentation depends on arterial distribution of gas embolism:

- Stupor or confusion (24%)
- Coma without seizure (22%)
- Coma with seizures (18%)
- Unilateral motor deficits (14%)
- Visual disturbances (9%)
- Vertigo (8%)
- Unilateral sensory deficits (8%)
- Bilateral motor deficits (8%)
- Collapse (4%)
- Spontaneous improvement minutes after initial deficits may occur:
 - High incidence of relapse
 - Improvement may be transiently related to postural changes that affect distribution of bubbles flowing to brain.
- Pulmonary:
 - Dyspnea
 - Hemoptysis, pleuritic chest pain
 - Subcutaneous air
- Cardiac:
 - MI owing to air in coronary vessels
 - Reduced cardiac output owing to air trapped in ventricle
 - Hamman sign: Crepitus on auscultation of heart
- Renal:
 - Renal infarction owing to air embolism

History

Elicit time of symptom onset in relation to dive surfacing (almost all symptoms occur within the 1st 10 min).

Physical-Exam

Careful neurologic exam owing to the wide variety of neurologic manifestations

ESSENTIAL WORKUP

- Clinical diagnosis: Recognize risk factors and various clinical presentations.
- Inquire as to unusual circumstances during ascent:
 - Breath holding
 - Panic/out-of-air situation
- Thorough neurologic exam must carefully document the extent of the deficits to the motor, sensory, cerebellar, and cranial nerves.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Serum creatinine kinase activity:

- Marker of the severity of cerebral AGE
- CBC
- Electrolytes, BUN, creatinine, glucose
- ABG when respiratory symptoms are present

Imaging

- CXR:
 - For evidence of pneumothorax or mediastinal emphysema (both rare)
- Chest CT
 - For evidence of local lung injury or hemorrhage
- Ventilation Perfusion Scan
 - For evidence consistent with pulmonary emboli
- EKG
- Echo:
 - Looking for evidence of patent foramen ovale
- CT head:
 - For altered mental status
 - Do not delay recompression for CT when AGE almost certain clinically.

DIFFERENTIAL DIAGNOSIS

- Cerebrovascular accident (CVA) from causes unrelated to gas embolism
- Neurologic deficits owing to decompression sickness



TREATMENT

PRE HOSPITAL

- Cautions:
 - Patients who experience sudden neurologic recovery can relapse quickly as bubble positions change.
 - Recognize AGE as a potential diagnosis.
- Altered mental status within 10 min of surfacing from compressed air dive
- Sudden neurologic decompensation following placement of central line
- Controversies:
 - Trendelenburg positioning patients with suspected AGE is not effective:
 - Hypothesized that elevation of legs could cause air bubbles to migrate away from cerebral circulation and that increased hydrostatic pressure in brain will shrink bubbles
 - Trendelenburg positioning may in fact increase injury by increasing intracerebral pressure.

INITIAL STABILIZATION/THERAPY

ABCs:

- 100% oxygen by tight-fitting mask
- Intubation for ventilation/protection of airway required
- IV access with volume augmentation

ED TREATMENT/PROCEDURES

- Hyperbaric oxygen recompression therapy (see “Hyperbaric Oxygen Therapy”):
 - _ For all AGE
 - _ Arrange transportation to nearest hyperbaric facility.
 - _ Aircraft capable of cabin pressurization below 1,000 feet barometric pressure best suited for transfers
 - _ Prophylactic chest tube for simple pneumothorax to prevent conversion to tension pneumothorax during recompression
 - _ Fill endotracheal and Foley catheter balloons with water or saline to avoid shrinkage/damage during recompression.
- Divers alert network (DAN):
 - _ Based at Duke University Medical Center
 - _ Provides 24 hr emergency hotline for medical consultation on treatment of dive-related injuries and for referrals to hyperbaric chambers (telephone: [919] 684-8111)



FOLLOW-UP

DISPOSITION

Admission Criteria

Admit all following initial hyperbaric therapy for observation and re-exam.

Discharge Criteria

No AGE patients should be discharged from the ED.

FOLLOW-UP RECOMMENDATIONS

Hyperbaric oxygen referral for patients with arterial gas embolisms

PEARLS AND PITFALLS

- Symptoms occur during ascent or within 10 min of reaching the surface.
- Patients who experience sudden neurologic recovery can relapse quickly as bubble positions change.
- Fill endotracheal and Foley catheter balloons with water or saline to avoid shrinkage/damage during recompression.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Barotrauma
- Decompression Sickness
- Hyperbaric Oxygen Therapy



CODES

ICD9

958.0 Air embolism

ICD10

T79.0XXA Air embolism (traumatic), initial encounter

ARTERIAL OCCLUSION

Richard E. Wolfe

BASICS

DESCRIPTION

Immediate and severe compromise of the blood supply to a limb, threatening its viability, secondary to the sudden blockage of a peripheral artery

- Arterial embolization
 - Thrombus or plaque
 - Originates from aneurysms or atherosclerotic lesions
 - Emboli typically lodge where there is an acute narrowing of the artery
 - 75% of emboli involve an axial limb vasculature
 - Femoral 28%
 - Arm 20%
 - Aortoiliac 18%
 - Popliteal 17%
 - Visceral and other 9%
- Thrombosis
- Arterial dissection
- Trauma
 - Crush injuries
 - Compression
 - Arterial contusion and thrombosis
 - Arterial transection
- Limb ischemia >6 hr usually results in functional impairment or limb loss.
 - If acute on chronic, collateral circulation may preserve tissue beyond 6 hr.

ETIOLOGY

- Embolus:
 - Atrial fibrillation
 - Myocardial infarction
 - Valvular disease
 - Endocarditis
 - Atrial myxoma
 - Aneurysm
 - Atherosclerotic plaques
 - Paradoxical embolus
 - Patent foramen ovale
- Thrombosis
 - Vascular grafts

- Atherosclerosis
- Thrombosis of an aneurysm
- Entrapment syndrome
- Blood clotting disorders
- Low flow state
- Heparin-induced thrombosis
- Arterial dissection
- Arterial injury:
 - Intimal flap
 - Dissection
 - Pseudoaneurysms
 - Iatrogenic
 - Catheterization
 - Arteriography
 - Balloon angioplasty
 - Complication of arterial puncture
 - Penetrating trauma
 - Gunshot, stab wounds, shotgun, shrapnel
 - IV drug use
 - Blunt trauma
 - Joint displacement
 - Fracture
 - Compartment syndrome

DIAGNOSIS

SIGNS AND SYMPTOMS

- Sudden onset of a cold, painful leg
- The 6 Ps:
 - Pain
 - Gradual, initially increasing in severity then decreasing with progressive sensory loss
 - Distal progressing proximally
 - Sudden onset with embolization
 - Pallor
 - Paresthesias
 - Paralysis
 - Pulseless (late finding)
 - Polar (Cold)
- Progressive peripheral nerve dysfunction
 - Early loss of proprioception and light touch
 - Loss of sensation and weakness follows

- Blue toe syndrome:
 - Development of blue or violaceous discoloration in one or more toes
 - The affected digits are often painful.
 - The cyanosis initially blanches with pressure or leg elevation.
- Signs of severe obstruction and poor prognosis
 - Absent capillary flow
 - Skin marbling
 - Loss of distal pulses
 - Paralysis

History

- Time of onset
- History of claudication or cramps
 - Reproducible discomfort of a defined group of muscles that is induced by exercise and relieved with rest
- Past medical history to identify risk factors for thrombosis or embolus

Physical-Exam

- Sensory loss
- Muscle weakness
- Skin color changes
- Loss of pulse
- Signs of chronic arterial insufficiency:
 - Hair loss
 - Atrophic skin
- Ankle-brachial pressure index measurement
 - Measure arm systolic pressure with the Doppler flowmeter for accuracy
 - Record pressure in both arms and both tibial arteries at the ankle.
 - Ratio of systolic BP in the lower legs to the brachial pressure in the arm:
 - Place cuff above malleoli to measure pressure in lower legs
 - Use Doppler at posterior tibial or dorsalis pedis artery
 - Chronic PVD < 0.9
 - Acute arterial occlusion < 0.5
- Demarcation of the warm part of the extremity to the cold part to estimate level of the obstruction

ESSENTIAL WORKUP

ALERT

Elevation, cool compress or ice, or warm compress to the affected extremity is contraindicated.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Electrolytes/anion gap
- BUN
- Creatinine
- CBC
- Creatine phosphokinase

Imaging

- The utility of imaging in the ED is limited as most of the decision making is based on the clinical presentation
- Duplex US
 - _ Provides a “roadmap” of stenosis of the arteries of the lower extremities
- CT angiography
 - _ With multidetectors, performance is similar to angiography
 - _ Like angiography it requires IV contrast bolus and exposure to radiation
- MRI
 - _ Viable alternative to angiography.
 - _ Noninvasive
 - _ Does not required contrast material
- Angiography

Classification

- Class 1: Viable
 - _ Pain but no paralysis or sensory loss
 - _ Needs attention, not in immediate danger
- Class 2: Threatened but salvageable
 - _ 2A: Some sensory loss, no paralysis: No immediate threat.
 - _ 2B: Sensory and motor loss: Needs immediate treatment
- Class 3: Irreversible/nonviable:
 - _ Sensory loss, paralysis, absent capillary flow, skin marbling, absent arterial Doppler flow
 - _ Will require amputation

DIFFERENTIAL DIAGNOSIS

- Lumbar spine disorders
- Back pain, mechanical
- Decreased cardiac output owing to advanced atherosclerotic disease
- Frostbite
- Peripheral neuropathy
- Aneurysm, abdominal
- Ankle injury, soft tissue
- Deep venous thrombosis
- Septic thrombophlebitis

- Superficial thrombophlebitis
- Trauma, peripheral vascular injuries



TREATMENT

PRE HOSPITAL

- Early recognition and rapid transport to an emergency department
- Place the limb in a dependent position
- Keeping the limb warm
- Oxygen by nasal cannula
- Aspirin

ED TREATMENT/PROCEDURES

- Prompt consultation with vascular surgeon
- Heparin bolus followed by an infusion
- Class 1: Viable
 - _ Most often due to thrombosis
 - _ Intra-arterial thrombolytic agents versus surgical revascularization or endovascular repair depending on viability of limb
- Class 2: Threatened but salvageable
 - _ Immediate surgical revascularization
 - _ Embolectomy if indicated
 - _ Angiography and oral anticoagulation post op
- Class 3: Nonviable
 - _ Prompt amputation
 - _ Clinical assessment, imaging usually not required
- Pain control

MEDICATION

- Heparin: Weight-based protocol anticoagulation with typical 80 U/kg loading bolus; 18 U/kg/h IV



FOLLOW-UP

DISPOSITION

Admission Criteria

- All patients with clinical diagnosis of acute arterial occlusion or (ABI < 0.5) should be admitted after an emergency consultation with a vascular surgeon.

Discharge Criteria

- Patients with chronic occlusive disease, resolved pain, and stable ABI

measurements

- No other acute medical issues (e.g., new atrial fibrillation)
- Vascular surgical follow-up can be ensured.
- Patients should be instructed to return for any recurrent or progressive symptoms.

Issues for Referral

- PVD patients in which illness is not severe or acute as to require inpatient treatment may be discharged with appropriate follow-up with a vascular surgeon.
- Potential effects of various activities and medications on the course of their illness should be discussed.
- Education on smoking cessation, temperature extremes, and vasoconstricting medications should be considered.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Peripheral Vascular Disease



CODES

ICD9

- 444.9 Embolism and thrombosis of unspecified artery
- 444.21 Arterial embolism and thrombosis of upper extremity
- 444.22 Arterial embolism and thrombosis of lower extremity

ICD10

- I74.2 Embolism and thrombosis of arteries of the upper extremities
- I74.3 Embolism and thrombosis of arteries of the lower extremities
- I74.9 Embolism and thrombosis of unspecified artery

ARTHRITIS, DEGENERATIVE

Patrick H. Sweet

BASICS

DESCRIPTION

- Degenerative arthritis or osteoarthritis (OA) is the most common progressive joint disease, with 20–30 million cases in the US
- Found almost exclusively in the elderly

ETIOLOGY

- Mechanism
- Repetitive stress to synovial joints associated with age
- May be seen in younger patients secondary to joint trauma
- Articular cartilage destruction:
 - Reactive changes in joint margin bone and subchondral sclerosis
- Risk factors include age, obesity, trauma, genetics, sex, and environment.

DIAGNOSIS

SIGNS AND SYMPTOMS

- Chronic progressive joint pain:
 - Worse with weight bearing, improved with rest
- Asymmetric joint involvement:
 - Involves hand, foot, knee, hip, and spine joints
- Morning joint stiffness usually < 30 min
- Joint deformity late in presentation with limited range of motion
- Heberden nodes at the distal interphalangeal joints
- Bouchard nodes at the proximal interphalangeal joints
- Absence of systemic symptoms
- Crepitus common

ESSENTIAL WORKUP

- Thorough joint exam with assessment of range of motion and functional ability
- Radiographic exam: Typical findings in OA are decreased joint space, irregular bone at the joint margin, and osteophytes.
- Synovial fluid analysis in the setting of effusion may be therapeutic and diagnostic (see below), but is absolutely necessary if presents with warmth and erythema so as to rule out a septic joint or gout.
- ESR, CRP, and CBC if infection is in the differential as arthrocentesis may be indicated if a more superficial infection cannot be ruled out (e.g., septic bursitis,

cellulitis, etc.).

DIAGNOSIS TESTS & INTERPRETATION

Lab

Synovial fluid exam typically reveals the following:

- Clear
- Elevated leukocyte cell count, but $< 4,000/\text{mm}^3$
- $< 25\%$ polymorphonuclear leukocytes
- Glucose level similar to blood levels (95–100%)

Imaging

- Radiographs
- Joint space narrowing
- Osteophyte formation
- Marginal bone erosion
- Subchondral sclerosis

DIFFERENTIAL DIAGNOSIS

- Gout or pseudogout
- Septic arthritis
- Rheumatoid arthritis
- Charcot joint
- Hemarthrosis
- Overlying bursitis or soft tissue infection



TREATMENT

The general goal of treatment is to provide relief from symptoms. A patient may have significant radiographic evidence of disease but have very few symptoms. Therefore the treatment regimen is tailored to the patient's symptomatology.

PRE HOSPITAL

Immobilization of affected joint may be indicated until fracture is excluded.

INITIAL STABILIZATION/THERAPY

- Pain management acutely
- Begin a daily medication that can be managed on follow-up with primary care physician.
- Instructions for gentle strengthening exercises
- Avoidance of unnecessary joint immobilization

ED TREATMENT/PROCEDURES

Intra-articular (IA) arthrocentesis and injection:

- Ultrasound (US) guidance is recommended when expertise and instrument is available
- Shown to be an effective low-risk intervention for OA with or without effusion
- Though relatively rare in larger joints, dry tap is a possible finding due to anatomic features of joint and periarticular soft tissue (e.g., fat pad).
 - US very useful in this case
- Careful attention must be given to aseptic technique while joint is in proper position to reduce muscle tension, exposing joint space.
- Vapor coolant or lidocaine 1% or 2% can be used for local anesthesia.
- Usually 1.5 in or greater 22G or 18G hypodermic needle should be used with 1 syringe for arthrocentesis and another for IA corticosteroid injection.
- If septic joint cannot be ruled out, corticosteroids should not be administered after arthrocentesis.

Corticosteroid dosing equivalents:

- Small joints—wrist and foot:
 - Methylprednisolone 10–20 mg, triamcinolone 10 mg, betamethasone 0.75–1.5 mg
- Medium-sized joints—elbow and ankle:
 - Methylprednisolone 40–80 mg, triamcinolone 20 mg, betamethasone 3–6 mg
- Large joints—knee and shoulder:
 - Methylprednisolone 80–120 mg, triamcinolone 40 mg; betamethasone 6–9 mg
- Some studies show triamcinolone to be more efficacious than other corticosteroids; the author recommends this, if available.

MEDICATION

General guidelines:

- Acetaminophen is drug of choice initially as it has a safer medication profile compared with NSAIDs and has been shown to be as efficacious in some patients.
- If one class fails, consider another class (e.g., salicylates vs. COX-2 inhibitors).
- The 2 alternative medications below have been shown to have a small but positive effect by meta-analysis of recent studies and can be considered adjuncts.
- Postprandial administration is recommended for all these. Patients at increased risk for GI bleeding (e.g., history of peptic ulcer disease, etc.) should be placed on COX-2 inhibitors or alternatively a proton pump inhibitor can be given with a nonselective COX inhibitor.
- NSAIDs:
 - Celecoxib (reversible COX-2-selective) 400 mg PO q24:
 - Note: Contraindicated in sulfonamide allergy
 - Ibuprofen (reversible nonselective COX): 400–600 mg PO q6h
 - Naprosyn (reversible nonselective COX): 500 mg PO q12h

- Meloxicam (reversible nonselective COX): 7.5 mg PO q12h or 7.5–15 mg PO q24h
- Analgesics:
 - Acetaminophen: 500 mg (peds: 10–15 mg/kg, do not exceed 5 doses/24 h) PO q4–6h, do not exceed 4 g/24 h
 - Tramadol: 50 mg PO q4–6h:
 - Note: Use cautiously in elderly, patients with seizure disorders, concurrently using antidepressants, or in hepatic or renal dysfunction.
 - Other opioid narcotics rarely used
- Alternative therapies (separate or in combination):
 - Glucosamine: 500 mg PO q8h
 - Chondroitin: 1200 mg PO q24h
- Lifestyle modification:
 - Weight loss for the obese
 - Strengthening exercises



FOLLOW-UP

DISPOSITION

Admission Criteria

Rarely indicated in the absence of fracture

Discharge Criteria

- Ambulatory and capable of activities of daily living
- Improvement in symptoms (i.e., pain)

Sports Medicine Follow-up

- Consider referral to sports medicine clinic for definitive management

ADDITIONAL READING

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- Vangsness CT Jr, Spiker W, Erickson J. A review of evidence-based medicine for glucosamine and chondroitin sulfate use in knee osteoarthritis. *Arthroscopy*. 2009;25(1):86–94.



CODES

ICD9

- 715.90 Osteoarthrosis, unspecified whether generalized or localized, involving unspecified site
- 715.94 Osteoarthrosis, unspecified whether generalized or localized, hand
- 715.97 Osteoarthrosis, unspecified whether generalized or localized, ankle and foot

ICD10

- M19.049 Primary osteoarthritis, unspecified hand
- M19.079 Primary osteoarthritis, unspecified ankle and foot
- M19.90 Unspecified osteoarthritis, unspecified site

ARTHRITIS, JUVENILE IDIOPATHIC

Kathleen A. Kerrigan • Kenneth G. Christian



BASICS

DESCRIPTION

- Previously called JRA
- JIA comprises persistent, unexplained arthritis lasting >6 wk, occurring <17 yr of age, and affecting a heterogeneous group of children.
- Prevalence up to 1 in 1,000 children
- Girls > boys for most forms
- Classified into 7 subgroups: Systemic onset, polyarticular RF⁺, polyarticular RF⁻, pauciarticular, psoriatic, enthesitis, other or unclassified.
- Subtypes are based on number, type, and symmetry of joints involved; presence of systemic symptoms; skin involvement; family history; and lab values.
- Up to 20% of JIA patients remain unclassified or are classified in multiple categories.
- Natural course of the disease depends on the subtype (pauciarticular with overall best prognosis), but full resolution occurs in <50% of JIA patients.
- Many will have a fluctuating course and ongoing disease through adulthood.

ETIOLOGY

Believed to be an autoimmune disease triggered by an unknown environmental trigger in a genetically susceptible host



DIAGNOSIS

Subtypes

- Systemic onset:
 - 10% of cases, girls = boys
 - Associated fever and arthritis:
 - Fever: Diurnal (>39°C) of >2 wk duration, child looks ill during temperature spike
 - Arthritis: May involve any number of joints and may appear only weeks to months after onset of fever
 - In addition there must be 1 of the following:
 - Maculopapular, salmon-colored rash on trunk and axillae
 - Lymphadenopathy
 - Hepatosplenomegaly
 - Serositis
 - Most acute, ill appearing of subtypes

ALERT

- Systemic onset JIA patients are at risk for macrophage activation syndrome (MAS).
- MAS is a proliferation of macrophages causing a DIC-like picture with resultant fever, mucosal bleeding, neurologic changes and multisystem failure.
- ESR may be normal in active MAS
- MAS mortality is 8–22%.
- Pauciarticular:
 - 50% of cases of IJA
 - 80% girls, peak incidence 2–4 yr olds
 - Insidious onset and child appears healthy
 - ≤ 4 joints involved at 6 mo:
 - Involves larger joints, (89% knee) *hip rarely affected*
 - Joints swollen, mildly tender, with decreased range of motion (ROM), possible leg-length discrepancy
 - Uveitis in about 20%; no other systemic signs
 - Subset termed *extended* pauciarticular progresses to greater joint involvement after 6 mo and has worse prognosis
- Polyarticular:
 - 10–30% of cases, girls > boys, bimodal peaks: 2–5 and 10–14 yr
 - > 4 joints involved at 6 mo:
 - Arthritis often symmetrical, small or large joints—commonly knees, wrists, and ankles
 - Decreased ROM of cervical and lumbar spine and temporomandibular joint (TMJ)
 - Systemic involvement rare except for fatigue and anemia
 - Older girls with RF⁺ often go on to develop typical adult rheumatoid arthritis (RA) and are placed in a separate subtype.
- Psoriatic:
 - Arthritis; asymmetric large joints of lower extremities and back
 - Psoriatic rash in patient or 2 of the following: Dactylitis, nail pitting, psoriatic rash in 1st-degree relative
 - Enthesitis-related (*enthesitis* means pain at the insertion of a muscle or tendon)
 - Arthritis; asymmetric large joints of lower extremities
 - Boys > girls, age usually > 6 yr
 - Sacroiliac (SI) joint pain
 - Limited flexion of lumbar spine
 - Uveitis
 - Often FH
- Otherwise unclassified:

– Arthritis not fitting into any distinct category

History

- Findings based on specific subtype
- Classic presentation is insidious arthritis, worse in AM and with periods of immobility, improved with ROM.
- New-onset systemic subtype most likely to use ED because they appear acutely ill, whereas other subtypes have a more insidious onset.

ALERT

- Child with severe pain and red-hot joint probably does not have new-onset JIA.
- Rapid onset of polyarticular joint involvement is atypical for JIA; infectious or reactive cause of arthritis should be ruled out.
- Beware of occult infection in patients on immunosuppressants.

Physical-Exam

- Determine if child is systemically ill: Search for fever, rash, or other nonarthritic involvement.
- Do careful joint evaluation, documenting the number of joints involved and noting whether they are red, warm, and swollen or have limited ROM.

ESSENTIAL WORKUP

- Rule out septic joint and malignant bone tumor.
- Rule out other identifiable causes of joint inflammation.
- Rule out complications from long-term drug therapy.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC, ESR; if ill appearance, add blood cultures
- Other labs if suspicious of specific subtype: rheumatoid factor (RF), antinuclear antibodies (ANA), HLA-B27, LFTs:
 - Systemic—ESR often elevated, leukocytosis, thrombocytosis, anemia, minor AST/ALT elevations, positive RF or ANA rarely seen, MAS may be associated with elevated LFTs and abnormal clotting factors but will often have normal ESR
 - Pauciarticular—common to have positive ANA in young girls; other labs usually normal; if anemic or elevated ESR, are probably misclassified or pauciarticular extended subtype
 - Polyarticular—may be anemic; if positive RF more likely to go on to adult RA, ESR may be elevated
 - Enthesitis—more likely positive HLA; presence of positive RF or positive ANA specifically excludes enthesitis subtype

- Psoriatic arthritis—usually seronegative RF
- Unfortunately, RF and ESR may also be elevated in acute infection unrelated to JIA.

ALERT

Consider adding Lyme titer if new joint swelling in endemic area.

Imaging

- Joint radiograph:
 - Early presentation: Soft tissue swelling, joint effusion
 - Late presentation: Osteoporosis, joint destruction, early growth plate closure
- Ultrasound:
 - Evaluate for small effusion, especially if tap considered.

Diagnostic Procedures/Surgery

Arthrocentesis if concern for septic arthritis: 5,000–8,000 WBC/mm³ with negative Gram stain and culture typical for JIA

DIFFERENTIAL DIAGNOSIS

- Trauma
- Infection:
 - Septic arthritis, viral infection (especially parvovirus), Lyme disease, rheumatic fever, tuberculosis, subacute endocarditis, malaria, *Neisseria gonorrhoeae* infection
- Other rheumatic/connective tissue diseases:
 - Systemic lupus erythematosus, polyarteritis nodosa, Henoch–Schönlein purpura, sarcoid
 - Legg–Calvé–Perthes disease/slipped capital femoral epiphysis
- Neoplasm:
 - Be suspicious of neoplasm in a severely uncomfortable child with midshaft bone pain.
- Hematologic disease:
 - Sickle cell disease, hemophilia
- Drug reactions



TREATMENT

INITIAL STABILIZATION/THERAPY

Toxic-appearing children: IV access, O₂

ED TREATMENT/PROCEDURES

- ED treatment is directed toward ruling out a septic joint and other causes of acute arthritis
- If the diagnosis of JIA is already established and the child presents with an acute flare, a treatment plan/medication adjustment should be coordinated with the child's rheumatologist.

MEDICATION

- Medication in children with JIA is geared toward eliminating clinical signs of active disease, maximizing joint function, and preserving growth. (Chronic inflammation can make affected limb slightly longer.)
- Efficacy depends on JIA subtype and disease severity.
- Most pauciarticular JIA responds to NSAIDs and joint injections; polyarticular and systemic JIA usually require disease-modifying antirheumatic drug (DMARD) therapy and/or biological agents.
- Antibiotics are indicated only if the joint is infected.

ALERT

As early aggressive therapy may prevent some of the long-term complications of JIA, it is now common for children to be placed on DMARDs and biological agents early in the disease course. These medications have serious potential side effects, including:

- Immunosuppression
- Decreased vaccine response (live vaccinations are contraindicated)
- Increased potential for malignancy
- NSAIDs:
 - _ Responsiveness differs within NSAID subtype
 - _ Used alone in mild JIA subtype or with other medications
 - _ Ibuprofen: 30–45 mg/kg divided TID–QID
 - _ Naproxen: 10–20 mg/kg divided BID; maximum daily dose 1,000 mg
 - _ Side effects: Gastritis, hepatitis, renal, headache, dermatitis
 - _ Intra-articular steroids: Triamcinolone hexacetonide: 1 mL/mg of 20 mg/mL solution
 - _ Often provide long-term (6–18 mo) relief
- DMARDs:
 - _ Include corticosteroids, methotrexate, sulfasalazine
 - _ Corticosteroids
 - _ Use judiciously because of long-term complications, but high-dose pulse therapy may be needed in acute attack.
 - _ Prednisone: 0.5–2 mg/kg PO
 - _ Methylprednisolone: 30 mg/kg daily IV up to 1 g for 1–5 days for high-dose pulse steroids
 - _ Side effects: Gastritis, adrenal suppression, osteopenia, Cushing syndrome, infection
 - _ Methotrexate: 5–15 mg/m² PO/SC or IM per week

- Considered 1st-line DMARD, as most will respond
- Side effects: GI, nausea, liver toxicity, teratogenic
- Sulfasalazine: 30 mg/kg/d divided QID
- Poorly tolerated in up to 30%
- Side effects: GI, rash, anorexia
- Biological agents—engineered to target specific key cytokines, very expensive
- Tissue necrosis factor binders
- Etanercept: 0.8 mg/kg SC once a week
 - Adalimumab: < 30 kg: 20 mg, > 30 kg: 40 mg given SC administered every other week
 - Side effects: Infection, injection-site reactions, inhibit T-cell activation
 - Abatacept: 10 mg/kg infusion q4wk
 - Side effects: Infusion reaction, HA, cough, nausea, infection
- Non-FDA–approved therapies:
- Remicade, rituximab, anakira, leflunomide IL-1 and IL-6 blockers
- Stem cell transplants are used rarely for severe cases unresponsive to medical treatment:
 - Treatment for MAS is nonstandardized but may include high-dose steroids, cyclosporine, cyclophosphamide, or intravenous immunoglobulin



FOLLOW-UP

DISPOSITION

Admission Criteria

Unclear diagnosis in ill-appearing child or if concern of secondary joint infection

Discharge Criteria

- No evidence of septic joint, systemic infection, or organ failure from drug therapy
- Patient appears comfortable.
- Appropriate follow-up has been arranged.

Issues for Referral

- Orthopedics if septic joint suspected
- Rheumatologist if meds need adjustment

FOLLOW-UP RECOMMENDATIONS

- Children need long-term consults with a rheumatologist.
- Children with JIA need frequent eye exams to rule out uveitis (which is often asymptomatic until permanent damage has occurred).

PEARLS AND PITFALLS

- Rule out acute joint infection (always consider Lyme disease in the appropriate geographic context).
- Consider systemic onset JIA in child with prolonged diurnal febrile illness that is unresponsive to antibiotics.
- Consider MAS in systemic onset JIA patients who appear septic.
- Review patient's medications to identify potential side effects or immunosuppression.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Septic Joint
- Lyme Disease



CODES

ICD9

- 714.30 Polyarticular juvenile rheumatoid arthritis, chronic or unspecified
- 714.31 Polyarticular juvenile rheumatoid arthritis, acute
- 714.32 Pauciarticular juvenile rheumatoid arthritis

ICD10

- M08.00 Unsp juvenile rheumatoid arthritis of unspecified site
- M08.3 Juvenile rheumatoid polyarthritis (seronegative)
- M08.90 Juvenile arthritis, unspecified, unspecified site

ARTHRITIS, MONOARTICULAR

Paul Blackburn



BASICS

DESCRIPTION

- Localized to 1 joint, not migratory
- 1 etiology does not exclude another
- *Infectious (septic) arthritis*: Rapidly destructive process causes significant disability
 - Contiguous extension (cellulitis, osteomyelitis), hematogenous spread, direct inoculation
 - Predisposing factors:
 - Local pathology (inflammatory arthritis, trauma, prosthetic joint)
 - Immunosuppression
 - IV drug use
- *Crystalline*:
 - *Gout*: Uric acid overproduction or underexcretion, deposited within and around joints.
 - *Pseudogout*: Calcium pyrophosphate
- *Noninflammatory conditions*
 - Osteoarthritis (DJD), trauma (fractures, hemarthrosis), autoimmune disorders
 - Progressive joint destruction; mechanical dysfunction
 - Bone reactive changes (spurring)
 - Subchondral bony erosions

ETIOLOGY

- *Infectious (septic)*
 - Most common organisms nongonococcal
 - Gram-positives: *Streptococcus*, *Staphylococcus* (80%)
 - Some associations:
 - *Staphylococcus aureus*: (trauma, IV drug use)
 - *Neisseria gonorrhoea* (STD)
 - Salmonella (sickle cell) but most common causes in sickle cell same (*Staphylococcus*, *Streptococcus*)
 - Less common: Fungal (chronic), spirochete (Lyme), viral (polyarticular), mycobacteria (TB)
- *Crystalline*:
 - *Gout*: Uric acid overproduction, underexcretion within, around joints
 - *Tophi*: Crystal deposits near recurrent flare sites. Progressive enlargement, may ulcerate “spit out” (discharge) crystals

- Negatively birefringent crystals
- *Pseudogout*: Calcium pyrophosphate
- Positively birefringent crystal
- Bariatric surgery: Postoperative gout flares common, frequent, significant. Prophylactic treatment effective, recommended
- *Inflammatory*
 - Diligent search for underlying cause, resultant conditions: arthritides (rheumatoid, psoriatic), inflammatory bowel disease, Reiter syndrome
- *Noninflammatory conditions*
 - Osteoarthritis or degenerative joint disease (DJD), overuse, overload (obesity)
 - Trauma (fractures, hemarthrosis)
 - Hemorrhagic disorders
 - Neuropathic disorders (Charcot joint)

Pediatric Considerations

- *Infectious (septic) arthritis*
 - Low incidence, high morbidity, sepsis (8%)
 - Most common: *S. aureus*, hip > knee, 50% coexisting osteomyelitis
 - Present like adults: Joint swollen, painful, worsened with weight bearing, movement; constitutionally ill (fever, lassitude)
 - Immediate aspiration, empiric treatment, admission mandatory
- *Inflammatory*
 - A diagnosis only after septic joint excluded; then considerations same as adults
- *Noninflammatory:*
- Orthopedic considerations to not overlook:
 - Salter–Harris epiphyseal plate fractures
 - Congenital hip dysplasia
 - Slipped capital femoral epiphysis (SCFE)
 - Overweight adolescents
 - Legg–Calve–Perthes:
 - Osteonecrosis femoral head
 - Age 4–9
 - Bleeding disorders, hemorrhage



DIAGNOSIS

Early accurate diagnosis allows directed therapy, earlier resumption of function, activities of daily living (ADL); longer-term morbidity lessened

SIGNS AND SYMPTOMS

- Isolated to 1 joint, not migratory

- Acute pain, swelling, redness, warmth
- Decreased range of motion, nonweight bearing (effusion, pain, osteomyelitis)
- *Infectious (septic) arthritis*:
 - Constitutionally ill, fever, chills
 - Larger joints swollen, painful range motion
 - Knee > hip = shoulder > ankle > wrist
 - *N. gonorrhoea*: Urethral discharge painful, purulent (males)
 - Lyme disease:
 - Spirochete *Borrelia burgdorferi*
 - Deer tick (*Ixodes dammini*)
 - Circular expanding, centrally clearing, eruption (*erythema chronicum migrans*)
 - Knees, shoulders most common
- *Crystalline*:
 - Sudden, severe pain, swelling, erythema
 - Recurrent, self-limited flares
 - *Gout*: Great toe joint (“podagra”) > ankle > tarsal joints > knee
 - *Tophi*: Crystal granulomas overlying affected joints; ulcerate, drain crystals
 - *Pseudogout*: Knee > wrist > ankle = elbow
- *Inflammatory*:
 - Protean manifestations, findings related to systemic conditions
 - Individual, multiple, combination organ system involvement. Example: Reiter syndrome: Iritis, urethritis, arthritis
- *Noninflammatory conditions*
 - Osteoarthritis (DJD):
 - Stiffness AM (inactivity), after activity (synovial gelling), relieved with rest
 - Trauma: Acute or distant, gradual swelling episodes, pain pattern same as DJD
 - Neuropathic: Charcot joint (“bag of bones”), little or no pain—chronic neuropathy
 - Hemarthrosis, hemorrhagic disorders

History

- See “Description,” “Etiology,” “Pediatric Considerations,” and “Signs and Symptoms.”
- Complete, meticulous history: Joint issues or involvement (recent, remote), systemic conditions (direct, local, remote manifestations), immune status (HIV, medications, disease process), STD (history, exposure, treatment type and duration), IV drug use.

Physical-Exam

See “Description” and “Signs and Symptoms.”

ESSENTIAL WORKUP

- Meticulous history and physical exam
- Condition—related diagnostic studies
- Arthrocentesis for synovial fluid analysis is the definitive diagnostic procedure.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Blood testing never the key diagnostic studies of choice for monoarticular arthritis; provides ancillary, corroborative, exclusionary information.
- Arthrocentesis for synovial fluid aspiration and analysis the definitive diagnostic procedure and studies.
- Synovial fluid culture the definitive synovial study regarding infection, but results not immediate (inherent nature of the test)
- Fluid appearance: Clear versus turbid, serous versus bloody, viscosity (“string sign”), volume removed, associated pain (levels, trends)
- Synovial fluid white blood cells (WBC), polymorphonuclear (PMN) predominance suggests septic etiology
 - WBC $> 50,000/\text{mm}^3$ increases likelihood of septic arthritis
- Synovial glucose: Most useful compared to concurrent blood glucose levels
 - Synovial glucose less than half blood value indicates likely septic process
- *Gram stain (positive stain)*: Directs initial antibiotic selection, administration
 - Gram + cocci: Vancomycin
 - Gram – cocci: Ceftriaxone
 - Gram – rods: Ceftazidime
- *Gram stain (negative stain)*: Clinical suspicion for septic joint: Empiric vancomycin + ceftazidime or aminoglycoside
- Viscosity: “String sign”
 - Slowly drip fluid off needle or syringe, noting the length of the “stringing.”
 - Noninflammatory fluid has longer strings
 - Inflammatory fluid will drip like water
- Crystal analysis: Polarized light microscopy for birefringent crystals: Gout (negative), pseudogout (positive)
- Rheumatologic “screening panel” for suspected disease: Uric acid, C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), rheumatoid factor (RF), anticyclic citrullinated peptide (ACCP)
- Lyme testing (anti-*Borrelia* titers or “Lyme titers”) for monoarticular arthritis presenting in endemic areas
- Fat globules in bloody aspirate: Suspect fracture with marrow (fat globules), synovial space communication

Imaging

- *Plain films* reasonable cost-effective choice:
 - Joint surfaces: Chondral, subchondral erosions, joint margin destruction or reactive bone formation (osteophytes, “spurring”), loose bodies, fractures
 - *Infectious*: As above plus soft-tissue swelling, joint capsule distortion
 - *Crystalline*: As above plus soft-tissue calcification, tophi usually located on or near frequent, repetitive joint flares
- *Ultrasound*: Detects joint fluid, tissue and vascular perfusion, periarticular structures, foreign bodies (especially if small, superficial organic composition of tissue density)
 - Guides aspiration attempts
- *MRI* detects bone necrosis, subtle fractures
- Bone or gallium scans do not distinguish Infectious versus inflammatory, especially chronic

Diagnostic Procedures/Surgery

See “Diagnostic Tests & Interpretation” above.

- Arthrocentesis for synovial fluid aspiration, analysis the definitive diagnostic procedure and laboratory studies.

DIFFERENTIAL DIAGNOSIS

See “Etiology” and “Signs and Symptoms” “Pediatric Considerations” above.



TREATMENT

PRE HOSPITAL

Alluding to subsequent headings containing all pertinent consideration.

INITIAL STABILIZATION/THERAPY

- Joint immobilization, position of comfort
- Vascular access for rapid, titratable, predictable medication effects
- Symptom control: Pain, nausea, vomiting, fluid replenishment
- Joint aspiration as soon as practicable; analysis directs therapy, disposition

ED TREATMENT/PROCEDURES

- See “Diagnostic Procedures” above
- *Septic arthritis*:
 - Urgent empiric IV bactericidal antibiotics: Ceftriaxone, vancomycin for *Staphylococcus*, *Streptococcus*, gonococcal arthritis
 - Subsequent outpatient antibiotics (PICC line, oral) therapy duration variable, multifactorial: Joint involved, organism, underlying health, patient compliance, medical costs

- Surgical irrigation considerations (“washouts”): Joint involved, open versus closed, single versus multiple sequential, comorbid conditions, patient compliance, medical costs
- *Crystalline*:
 - Treatment goals: (1) Quell the acute flare: NSAIDs (indomethacin, naproxen), colchicine, steroids; (2) only after acute exacerbation quelled:
 - Prophylaxis with flare prevention medications (colchicine, naproxen), begin urate-lowering therapy (allopurinol, febuxostat)
 - Febuxostat as effective or greater than allopurinol for gout flares, tophus area, uric acid levels.
 - Probenecid: Efficacious long-term uricosuric, alone or in conjunction with allopurinol
- *Noninflammatory*:
 - NSAIDs, analgesics
 - Physical therapy, rehabilitation
 - Orthopedic trauma: Immobilize, pain control, ensure neurovascular status intact
 - Hemorrhagic causes: Correction of factor levels, component replacement



FOLLOW-UP

DISPOSITION

Admission Criteria

- Unable to perform ADL
- Evidence systemic illness, metabolic derangement (sepsis, DKA)
- Any joint requiring surgical intervention (including serial washouts)
- Intractable pain
- *All septic arthritis*
 - General medical/surgical bed
 - Intensive care unit if generalized sepsis, metabolic derangement
- *Crystalline*:
 - Intractable nausea, vomiting, diarrhea
 - Septic joint superimposed on other arthritis

Discharge Criteria

- Symptoms (including pain) controlled, comorbid conditions stable, managed appropriately
- Medication compliance: Can obtain medications (economically, logistically), understands dosages, time intervals.
- Timely follow-up possible

Issues for Referral

Immediate consultation, admission for infectious etiologies, intractable pain, poorly controlled comorbid illnesses, interference with ADL

FOLLOW-UP RECOMMENDATIONS

- As soon as practicable, with health care providers best suited, capable of treating the condition in question.
- If unable to acquire the appropriate care in a timely manner, return to ED (safety net).

PEARLS AND PITFALLS

- Joint aspiration with Gram stain of fluid is the most important aspect of securing a diagnosis, directing initial management
- Suspect septic arthritis in the presence of Intra-articular corticosteroid administration, diabetes, drug abuse, trauma, injections through cellulitis and extra-articular infection

ADDITIONAL READING

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- Genes N, Chisolm-Straker M. Monoarticular arthritis update: Current evidence for diagnosis and treatment in the emergency department. *Emerg Med Pract.* 2012;14(5):1–19.
- Smith BG, Cruz AI Jr, Milewski MD, et al. Lyme disease and the orthopaedic implications of Lyme arthritis. *J Am Acad Orthop Surg.* 2011;19(2):91–100.



CODES

ICD9

- 274.00 Gouty arthropathy, unspecified
- 711.90 Unspecified infective arthritis, site unspecified
- 716.90 Arthropathy, unspecified, site unspecified

ICD10

- M00.9 Pyogenic arthritis, unspecified
- M10.00 Idiopathic gout, unspecified site
- M19.90 Unspecified osteoarthritis, unspecified site

ARTHRITIS, RHEUMATOID

Charles F. Lanzillo • Stephen R. Hayden



BASICS

DESCRIPTION

- Chronic systemic inflammatory disorder that attacks the joints:
 - Nonsuppurative, proliferative synovitis
 - Destruction of the articular cartilage
 - Ankylosis of the joint
- Involvement of knee is common.
- Baker cysts may be seen in chronic disease.
- Involvement of spine is limited to cervical region:
 - May cause atlantoaxial subluxation
 - Rarely results in cord compression

Pediatric Considerations

Juvenile rheumatoid arthritis (JRA) is a distinct entity (see “Arthritis, Juvenile Idiopathic”).

- Genetics:
 - Genetic predisposition related to HLA-DR4
 - Female-to-male ratio is 3:1.
 - Typical age of onset is between 30 and 50.

ETIOLOGY

- Etiology is unknown.
- Possible triggers include infection and autoimmune response.
- Prevalence is about 1% of both US and world population.



DIAGNOSIS

SIGNS AND SYMPTOMS

- Malaise, fatigue
- Generalized musculoskeletal pain
- After weeks to months, patients develop swollen, warm, painful joints.
- Often worse in morning
- Joint involvement usually symmetric and polyarticular
- Starting in small joints of hands and feet:
 - Later wrists, elbow, and knees
- Distal interphalangeal (DIP) joints of hand generally not involved:
 - Presence of swelling in these joints should suggest another type of arthritis.

- Synovitis is typically gradual.
- Classic joint findings in long-standing disease:
 - Metacarpophalangeal (MCP) swelling with ulnar deviation
 - Swan neck and boutonniere deformities
- Extra-articular complications:
 - SC nodules
 - Vasculitis
 - Pericarditis or myocarditis
 - Pulmonary fibrosis
 - Pneumonitis
 - Sjögren syndrome
 - Mononeuritis multiplex
- Evidence of mild pericarditis on echocardiogram is found in up to 1/3 of patients.
- Consider ECG evaluation in these patients
- Patients usually present to ED owing to exacerbations of the disease or complication in other organ systems:
 - Airway obstruction from cricoarytenoid arthritis or laryngeal nodules
 - Heart block, constrictive pericarditis, pericardial effusion with possible tamponade or myocarditis
 - Pulmonary fibrosis, pleuritis, intrapulmonary nodules, or pneumonitis
 - Hepatitis
- Neurologic findings may result from cervical spine subluxation or ocular manifestations such as scleritis and episcleritis.
 - Can also have retinal vasculitis in periphery, and recurrent iritis—consider in patients with photophobia, red eye, and decreased vision. These patients need ophthalmologic evaluation
- Complications of chronic steroid use:
 - Infections
 - Steroid-induced osteopenia and fractures
 - Insulin resistance
 - Glaucoma or IOP elevation, accelerated cataracts
- Patients may present with side effects related to chronic salicylate or NSAID use such as GI bleeding.
- Drugs such as methotrexate, gold, or d-penicillamine also have toxic side effects, most commonly GI but also neuropathic.

ESSENTIAL WORKUP

- Primary diagnosis of rheumatoid arthritis (RA) is rarely made in the ED.
- Synovitis should be present for at least 6 wk; a minimum of 4 of the following 7 criteria as established by the American Rheumatism Association must be met to make the diagnosis:
 - Stiffness of the involved joints in the morning for at least 1 hr
 - Arthritis in 3 or more joints with effusion or soft tissue swelling

- Arthritis of joint in hand (wrist, MCP, or proximal interphalangeal [PIP] joint)
- Symmetric arthritis
- Rheumatoid nodules on extensor surfaces or juxta-articular surfaces
- Significantly elevated rheumatoid factor
- Characteristic radiographic changes include erosions and decalcification (not attributable to osteoarthritis).
- Other pertinent history: Malaise, weakness, weight loss, myalgias, bursitis, tendonitis, fever of unknown cause
- Initial workup should focus on demonstrating that other causes of arthritis are not present, especially septic arthritis, reactive arthritis, or gout.
- Arthrocentesis of a joint effusion may be required.

DIAGNOSIS TESTS & INTERPRETATION

ECG, chest radiograph, C-spine or extremity radiograph, and hemoglobin testing are helpful if patient presents with complications of RA.

Lab

- CBC: Mild anemia with leukocytosis and thrombocytosis
- Erythrocyte sedimentation rate (ESR): Often > 30 . Guide for elevation is $\text{age}/2$ in men, $(\text{age} + 10)/2$ in women. Consider GCA in patients with elevated markers and RA with vision loss that is acute.
- C-reactive protein correlates with erosive disease
- Antinuclear antibodies (ANA) 30–40% positive screening tool
- Rheumatoid factor: Elevated in $\sim 70\%$ of cases
- Joint fluid analysis:
 - Typically between 4,000 and 50,000 white cells
 - Neutrophil predominance
 - Microscopic Gram stain of fluid should show no organisms and no crystals.
- ECG: Conduction defects are rare, but heart block may be seen. May see evidence of pericarditis.

Imaging

- Joint radiograph:
 - Joint effusion
 - Juxta-articular erosions and decalcification
 - Narrowing of joint space
 - Loss of cartilage
- MRI of joints can detect early inflammation before plain radiograph
- CXR reveal pulmonary fibrosis, pleural changes, nodular lung disease, or pneumonitis:
 - Cardiac silhouette may show changes related to myocarditis.
- Cervical spine radiograph:

- Atlantoaxial joint subluxation may occur.

DIFFERENTIAL DIAGNOSIS

- Osteoarthritis
- Septic arthritis
- Reactive arthritis
- Gonococcal arthritis
- Lyme disease
- Gout
- Connective tissue disorders
- Systemic lupus erythematosus (SLE), dermatomyositis, polymyositis, vasculitis, Reiter syndrome, and sarcoid
- Rheumatic fever
- Malignancy



TREATMENT

PRE HOSPITAL

Cervical spine immobilization and airway support as indicated

INITIAL STABILIZATION/THERAPY

- ABCs:
 - Manage airway with attention to C-spine immobilization during intubation.
- Treat complications of RA as appropriate.

ED TREATMENT/PROCEDURES

- Salicylates or NSAIDs are 1st-line treatment for RA:
 - If 1 NSAID fails, another NSAID from a different chemical class may work better.
- Early treatment of RA is important as joint changes may be most progressive during the 1st 18 mo.

MEDICATION

- Glucocorticoids, methotrexate, and other 2nd-line therapies should be initiated by a rheumatologist.
- Aspirin (ECASA): Adult: 900 mg PO QID (2.6–5.4 g/d); peds: 60–90 mg/kg/d QID up to 3.6 g

Note: Enteric coated aspirin has delayed absorption and its analgesic effects will be delayed compared to regular aspirin. Doses of aspirin needed for anti-inflammatory effect approach toxic doses. Patients should be closely monitored and dose carefully titrated to avoid toxicity.

- Auranofin: 3–9 mg/d (peds: 0.15 mg/kg/d up to 9 mg) divided BID
- Celecoxib (Celebrex): 100–200 mg PO BID; peds: N/A

- Hydroxychloroquine: Adult: 200–600 mg/d divided BID
- Ibuprofen (Ibuprofen, Advil, Motrin): 200–800 mg (peds: 10 mg/kg) PO q6h
- Leflunomide: 100 mg PO daily for 3 d, then maintenance dose of 10–20 mg PO daily; peds: N/A
- Methotrexate: 7.5 mg once/wk
- Prednisone: Maintenance: 5–10 mg PO daily; acute exacerbations: 20–50 mg PO daily; peds: Maintenance: 0.1 mg/kg/d PO, acute exacerbations: 2–5 mg/kg/d PO
- Sulfasalazine: Adult: 500–1,000 mg PO BID; peds: 30–60 mg/kg/d BID. up to 2 g
 - Not recommended in children <6 yr
- NSAIDs and Tramadol for breakthrough pain.
- Newer DMARDs and monoclonals need to be dosed by a rheumatologist and should likely not be prescribed in the ED: Abatacept, Adalimumab, Anakinra, Etanercept, Infliximab, Rituximab, Tocilizumab.

ALERT

Recent studies have shown possibly increased risk of cardiovascular event with NSAID medications, particularly with COX-2 inhibitors.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients with severe or life-threatening presentations of RA and its complications should be admitted to hospital.
- Admission is warranted when diagnosis is unclear and serious illnesses such as septic joint or systemic vasculitis may be present or cannot be ruled out.
- Admission may be required for pain control.
- Admission may be required if patient has inadequate social support and is unable to maintain activities of daily living.
- Pediatric patients with fever and arthritis should be strongly considered for admission.

Discharge Criteria

Patients without serious complications may be managed as outpatients with appropriate medications and follow-up.

Issues for Referral

All patients should have primary physician for further therapy and care as well as appropriate specialty care referral such as rheumatologists, cardiologists, and orthopedics.

PEARLS AND PITFALLS

- Recognize that symmetric arthritis is more consistent with RA.
- Even patients with RA can get septic arthritis.
- Consult rheumatologist rather than initiate steroids or TNF antagonists from ED.

ADDITIONAL READING

- Imboden JB. The immunopathogenesis of rheumatoid arthritis. *Annu Rev Pathol.* 2009;4:417–434.
- Sanders S, Harisdangkul V. Leflunomide for the treatment of rheumatoid arthritis and autoimmunity. *Am J Med Sci.* 2002;323(4):190–193.
- Smedslund G, Byfuglien MG, Olsen SU, et al. Effectiveness and safety of dietary interventions for rheumatoid arthritis: A systematic review of randomized controlled trials. *J Am Diet Assoc.* 2010;110(5):727–735.
- Smith JB, Haynes MK. Rheumatoid arthritis: A molecular understanding. *Ann Intern Med.* 2002;136(12):908–922.
- The American College of Rheumatology Subcommittee on Rheumatoid Arthritis Guidelines. Guidelines for the management of rheumatoid arthritis: 2002 update. *Arthritis Rheum.* 2002;46:328–346.



CODES

ICD9

714.0 Rheumatoid arthritis

ICD10

- M06.9 Rheumatoid arthritis, unspecified
- M06.049 Rheumatoid arthritis without rheumatoid factor, unsp hand
- M06.079 Rheumatoid arthritis w/o rheumatoid factor, unsp ank/ft

ARTHRITIS, SEPTIC

Amin Antoine Kazzi • Elie R. Zaghrini



BASICS

DESCRIPTION

- Bacteria can be introduced into a joint by:
 - Hematogenous spread (most common)
 - Invasive procedures
 - Contiguous infection (e.g., osteomyelitis, cellulitis)
 - Direct inoculation such as plant thorns or nails
- Acute inflammatory process results in migration of WBCs into joint.
- Synovial hyperplasia, cartilage damage, and formation of a purulent effusion
- Irreversible loss of function in up to 50%
- Mortality rate reported as high as 11%

Pediatric Considerations

- Hip infections are most common:
 - Often in patients with otitis media, upper respiratory tract infections or history of femoral venipuncture
 - Complications of septic arthritis (SA) of hip in children: Avascular necrosis, epiphyseal separation, pathologic dislocation, and arthritis
- 50% occur in children < 3 yr old.
- Infants present with irritability, fever, and loss of appetite.
- Older children present with fever, and a limp or refusal to bear weight or use joint.

ETIOLOGY

- Risk factors:
 - Old age, infancy
 - Rheumatoid arthritis and degenerative joint disease
 - Intravenous drug user (IVDU), endocarditis
 - Females (gonococcal [GC] infection)
 - Immunosuppression (AIDS, diabetes, chemotherapy, steroid therapy)
 - Repeated joint injections, pre-existing joint diseases, trauma, or prosthesis
 - Skin infection, cutaneous ulcers
- No bacterial pathogen is identified in 10–20%.
- Most common organisms:
 - *Staphylococcus aureus* in adults, hip infections (80%), and patients with rheumatoid arthritis or diabetes
 - *Multidrug-resistant S. aureus (MRSA)* has been noted in some studies to be the most common organism in community-onset adult SA.

- *Neisseria gonorrhoeae* most common in young, healthy, sexually active patients (incidence has decreased over the past decades due to a decrease in the incidence of mucosal GC infections)
- Other pathogens: Group A β -hemolytic and group B, C, and G streptococci:
 - Gram-negative rods (e.g., *Pseudomonas aeruginosa*, *Escherichia coli*) in 10% of cases
 - *Neisseria meningitidis* (12% of patients with meningococcal meningitis)
- Common in old age, infancy, immunosuppression, and IVDU (*Pseudomonas*)
- Anaerobes: Diabetes, prosthetic joints
- Mycobacterial and fungal causes: Atypical (e.g. in advanced HIV); more indolent course

DIAGNOSIS

SIGNS AND SYMPTOMS

- Presents abruptly as a single painful, swollen, warm, tender joint
- Common findings include:
 - Fever
 - A separate source of infection (e.g., skin)
 - Extremely painful joint motion in all planes
 - A joint effusion (less evident in sacroiliac, hip, and shoulder)
- Any joint can be involved:
 - Typically a single joint is involved.
 - Most commonly knee, then hip, shoulder, and ankle
- Commonly seen in IVDUs: Sacroiliac costochondral and sternoclavicular joints:
 - Vertebral involvement such as lumbar facets possible
- Human and animal bites, plant thorns, local steroid therapy, and trauma may lead to infection in atypical locations.
- Polyarticular involvement in 10–20%:
 - Mostly with rheumatoid arthritis; delay in diagnosis from low suspicion and more subtle presentations (fever in only 50%)
 - Patients with sepsis
- GC SA features:
 - Develops in 1–3% of untreated gonorrhea and in 42–85% of disseminated GC infection:
- Typically monoarticular but commonly polyarticular
- Migratory polyarthralgia, tenosynovitis (present in 20% of patients with arthritis), and dermatitis:
 - Involves small joints (e.g., fingers, wrist, elbow, ankle)
- Signs of urethral or vaginal GC infection may be present.
- Painless maculopapular lesions on trunk, arms, legs, and around affected joint

ESSENTIAL WORKUP

Arthrocentesis

- Perform joint aspiration in any suspected case.
- Send fluid for protein and glucose, cell count, Gram stain, and culture.
- Typical SA findings:
 - A turbid, purulent, or serosanguineous fluid
 - A leukocytosis (50,000–150,000/mm³) with a polymorphonuclear predominance (>75%)
 - Often a decreased glucose and elevated protein level
- Appearance of crystals does not rule out SA.
- Use special stain or culture media when indicated (e.g., GC, anaerobes, fungus, mycobacterium)
- Intra-articular lidocaine reduces the sensitivity of subsequent cultures; immediate emptying of aspirated sample into a blood culture flask increases the yield.
- In non-GC SA, Gram stain and culture are positive in 50% and 90% of cases, respectively:
 - Drops to nearly 10% and 50% in GC SA, respectively
- Real-time PCR can detect bacterial pathogen DNA in many culture-negative aspirates.
- Fluoroscopic, sonographic, or CT guidance can be used in technically difficult aspirations.
- CT scan and MRI may aid in the diagnosis for joints such as the sacroiliac joint.
- Arthrocentesis is contraindicated whenever there is an underlying joint prosthesis or an overlying skin infection:
 - If cellulitis present, use an alternate approach through normal skin.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Nonspecific serum leukocytosis (more common in children), left shift, and C-reactive protein (CRP) and ESR elevation are usually present.
- Procalcitonin can be a helpful aid to rule in rather than rule out SA
- UA and culture can reveal a urologic source for the pathogen.
- Blood cultures may be useful: Positive in 50–70% of non-GC SA.
- Culture any potential focus of infection (pharynx, urine, cervix, or anus), particularly when suspecting GC.

Imaging

- Plain radiographs to identify:
 - Effusion
 - Baseline status of the joint
 - Contiguous osteomyelitis
 - Concurrent rheumatologic diseases

- Fractures or foreign body
- Joint loosening (a late nonspecific sign)
- US, CT, and MRI are more sensitive:
 - US may be used to guide aspiration of some joints (e.g., hip) and to detect joint effusions.
- Scintigraphic techniques are sensitive and specific in diagnosis of SA. However, they are often not available through ED.
- Other tests:
 - Bacterial DNA amplification techniques in rapid detection and identification of organisms

DIFFERENTIAL DIAGNOSIS

- Viral arthritis
- Rheumatoid arthritis
- Gout or pseudogout
- HIV-associated arthritis
- Reactive arthritis
- Lyme disease
- Osteomyelitis
- Endocarditis
- Septic bursitis
- Trauma
- In children:
 - Juvenile idiopathic arthritis
 - Slipped capital femoral epiphysis
 - Legg–Calvé–Perthes disease
 - Metaphyseal osteomyelitis
 - Transient synovitis

Pediatric Considerations

- Because of vaccine, *Haemophilus influenzae* is no longer the most common agent.
- *S. aureus* is most common.
- Group B streptococcus, enterobacteria, and gram-negative rods in the newborn



TREATMENT

PRE HOSPITAL

No specific considerations

INITIAL STABILIZATION/THERAPY

- Patient may be septic and require resuscitation.
- If patient is toxic, do not delay antibiotics for aspiration results.

ED TREATMENT/PROCEDURES

- Promptly aspirate joint fluid.
- Obtain cultures.
- Start empiric antibiotics based on Gram stain (if available) and age group or risk factors—consider staphylococcal, streptococcal, and gram-negative coverage; and MRSA in the appropriate setting. Recommended duration of treatment is 2–4 wk. Intra-articular antibiotics are contraindicated.
- No risk factors for atypical organisms:
 - Use Flucloxacillin or equivalent 2 g QDS IV. Local policy may be to add gentamicin IV.
 - If penicillin allergic, clindamycin 450–600 mg QDS IV or 2nd or 3rd generation cephalosporin IV.
- High risk of gram-negative sepsis (elderly, frail, recurrent UTI, and recent abdominal surgery):
 - 2nd or 3rd generation cephalosporin for example, cefuroxime 1.5 g TDS IV. Local policy may be to add flucloxacillin IV to 3rd generation cephalosporin.
 - Gram stain may influence antibiotic choice.
- MRSA risk (known MRSA, recent inpatient, nursing home resident, leg ulcers or catheters, or other risk factors determined locally):
 - Vancomycin IV + 2nd or 3rd generation cephalosporin IV
- Suspected gonococcus or meningococcus:
 - Ceftriaxone IV or similar
 - Dependent on local policy or resistance
- IVDUs: Discuss with microbiologist
- ICU patients, known colonization of other organs (e.g., cystic fibrosis): Discuss with microbiologist
- Early orthopedic consultation to evaluate eligibility for surgical drainage
- Pain control: Narcotics and moderately flexed splinting
- Immunologic therapies are experimental.
- Prosthesis: Some may try to preserve the limb unless it is loose on plain films.
- Patients should be at rest with joint maintained in optimal position to prevent damage.

MEDICATION

- Cefazolin: 1–2 g IV q6h
- Ceftazidime: 1–2 g IV q8h
- Cefotaxime: 2 g IV q8h; peds: 50 mg/kg q12h
- Ceftriaxone: 2 g IV QD; peds: 50 mg/kg
- Ciprofloxacin: 400 mg IV q12h
- Flucloxacillin: 2 g QD IV
- Gentamicin: 2–5 mg/kg IV load
- Nafcillin: 2 g IV q4h; peds: 25 mg/kg q6h

- Tobramycin: 1 mg/kg IV q8h; peds: 2.5 mg/kg q8h
- Vancomycin: 1 g IV q12h; peds: 10 mg/kg q6h

Pediatric Considerations

- Open surgical drainage is the method of choice in pediatric hip SA.
- Cover *H. influenzae* type B if prior immunization cannot be established.



FOLLOW-UP

DISPOSITION

Admission Criteria

- All patients with suspected SA should be admitted until SA is ruled out.
- May undergo drainage of joint, as indicated, by serial aspirations, arthroscopy, or arthrotomy

Discharge Criteria

Cases where suspected SA has been adequately ruled out

PEARLS AND PITFALLS

- CRP and ESR can be used to follow up response to treatment
- It can be difficult to distinguish SA from toxic synovitis or crystal arthropathy; have a low threshold for arthrocentesis.

ADDITIONAL READING

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CODES

ICD9

- 711.00 Pyogenic arthritis, site unspecified
- 711.05 Pyogenic arthritis, pelvic region and thigh
- 711.45 Arthropathy associated with other bacterial diseases, pelvic region and thigh

ICD10

- M00.9 Pyogenic arthritis, unspecified
- M00.052 Staphylococcal arthritis, left hip
- M00.059 Staphylococcal arthritis, unspecified hip

ASCITES

Paul J. Allegretti • Keri Robertson



BASICS

DESCRIPTION

- Pathologic accumulation of serous fluid in the peritoneal cavity
- Portal hypertension (> 12 mm Hg) starts fluid retention.
- Avid sodium retention state
- Retained sodium and water increases plasma volume.
- Water excretion becomes impaired.
- Increased release of antidiuretic hormone (ADH)
- Urinary sodium retention, increased total body sodium, and dilutional hyponatremia
- Degree of hyponatremia correlates with disease severity; prognostic factor.
- Decreased plasma oncotic pressure from hypoalbuminemia
- Peritoneal irritation owing to infection, inflammation, or malignancy

ETIOLOGY

- Parenchymal liver disease:
 - Cirrhosis and alcoholic hepatitis:
 - 80% of adult patients
 - Fulminant hepatic failure
- Hepatic congestion:
 - CHF
 - Constrictive pericarditis
 - Veno-occlusive disease and Budd–Chiari syndrome
- Malignancies:
 - Peritoneal carcinomatosis
 - Hepatocellular carcinoma or metastatic disease
- Infections:
 - TB, fungal, or bacterial peritonitis
- Hypoalbuminemic states:
 - Nephrotic syndrome
 - Malnutrition; albumin < 2.0 g/dL
- Other conditions:
 - Pancreatic ascites
 - Biliary ascites
 - Nephrogenous ascites
 - Ovarian tumors
 - Chylous ascites from lymphatic leak

- Connective tissue disease
- Myxedema
- Granulomatous peritonitis

Pediatric Considerations

Most pediatric cases owing to:

- Malignancy (Burkitt lymphoma, rhabdomyosarcoma)
- Nephrotic syndrome
- Malnutrition



DIAGNOSIS

SIGNS AND SYMPTOMS

- Abdominal distention, discomfort
- Weight gain; sometimes weight loss
- Dyspnea
- Orthopnea
- Edema
- Abdominal hernias
- Muscle wasting
- Shifting dullness, flank fullness, fluid wave, puddle sign
- Signs and symptoms of underlying disease
- Stigmata of chronic liver disease

History

- Risk factors for liver disease
- Description of onset of symptoms:
 - Distinguishes ascites from obesity
 - Patients less tolerant of rapid accumulation of ascitic fluid
- New-onset ascites in known cirrhotic signifies 1 of the following:
 - Progressive liver disease
 - Superimposed acute liver injury (alcohol, viral hepatitis)
 - Hepatocellular carcinoma

Physical-Exam

- Detection difficult in obese patients
- Flank dullness is a prominent physical finding:
 - 500 mL for flank dullness
 - Fluid wave
 - Shifting dullness

ESSENTIAL WORKUP

- Search for liver disease, CHF, TB, malignancy, and other systemic disorders.
- Abdominal paracentesis:
 - Necessary for:
 - New ascites
 - Worsening encephalopathy
 - Fever
 - Abdominal pain/tenderness
- Determine if fluid infected or presence of portal hypertension
- Test ascitic fluid for:
 - Cell count and differential:
 - Most helpful to determine infection quickly
 - Order on every specimen
 - Albumin
 - Protein
 - Gram stain
 - Culture twice in blood culture bottles with 10 mL of fluid
 - Lactate dehydrogenase (LDH)
 - Glucose
 - TB culture
 - Amylase
 - Triglyceride
 - Cytology
 - Bilirubin
 - Carcinoembryonic antigen
- Spontaneous bacterial peritonitis (SBP):
 - Ascitic fluid infection without an intra-abdominal surgically treatable source
 - Fever, abdominal pain/tenderness, altered mentation
 - Polymorphonuclear neutrophils (PMNs) > 250 cells/mm³
 - Ascitic fluid protein < 1 g/dL
 - Low concentration of opsonins
- Secondary bacterial peritonitis:
 - Bacterial peritonitis from a surgically treatable intra-abdominal source
 - Gut perforation or intra-abdominal abscess (i.e., perinephric abscess)
 - PMNs > 250 cells/mm³ with multiple micro-organisms on Gram stain + 2 of the following found with secondary bacterial peritonitis:
 - Total protein > 1 g/dL
 - Glucose < 50 mg/dL
 - LDH greater than the upper limit of normal for serum

DIAGNOSIS TESTS & INTERPRETATION

- CBC
- Basic chemistry
- LFTs
- PT, PTT, INR
- Arterial blood gas (ABG) or pulse oximeter
- Urinalysis
- Urine sodium
- Hepatitis panel
- Amylase/lipase
- α -fetoprotein
- TSH

Imaging

- US:
 - _ Confirm ascites, especially if < 500 mL
 - _ Evaluate liver, pancreas, spleen, and ovaries
 - _ Guides paracentesis
- Doppler study: Evaluate hepatic blood flow
- CT scan
- CXR: CHF, effusions, cavitory, or mass lesion
- ECG

Diagnostic Procedures/Surgery

- Peritoneoscopy: Ascites of unknown cause; especially TB
- Paracentesis:
 - _ Clinical diagnosis of SBP without paracentesis is inadequate.
 - _ Safety of paracentesis:
 - 70% of ascitic patients have coagulopathy.
 - Benefits of a diagnostic paracentesis outweigh the risks.
 - Paracentesis is still indicated unless disseminated intravascular coagulation (DIC) is present.
 - Transfusion of plasma or platelets prior to paracentesis is not supported.

DIFFERENTIAL DIAGNOSIS

- 1 of the 5 “F” causes of abdominal swelling:
 - _ Fluid (including cysts)
 - _ Fat
 - _ Flatus
 - _ Fetus
 - _ Feces
 - _ Other: Organomegaly
- Serum-ascites albumin gradient (SAAG) = serum albumin – ascitic albumin:

- Replaced ascitic fluid total protein in the differential diagnosis of ascites
- SAAG ≥ 1.1 g/dL:
 - 97% accurate in predicting portal hypertension
 - Cirrhosis
 - Alcoholic hepatitis
 - Cardiac
 - Liver metastases
 - Fulminant hepatic failure
 - Portal vein thrombosis
 - Veno-occlusive disease
 - Myxedema
 - Budd—Chiari
 - Fatty liver of pregnancy
 - SBP
- SAAG < 1.1 g/dL:
 - Peritoneal carcinomatosis
 - TB
 - Pancreatic ascites
 - Nephrotic syndrome
 - Bowel obstruction or infarction
 - Vasculitis
 - Postoperative lymphatic leak



TREATMENT

PRE HOSPITAL

Symptomatic hypotension:

- Airway, breathing, circulation (ABCs), IV 0.9 NS

INITIAL STABILIZATION/THERAPY

Sudden increase in abdominal girth, pain, or fever requires urgent evaluation for possible complicating factor such as:

- Infection
- Hepatoma
- Obstruction of hepatic outflow
- Decompensated liver function

ED TREATMENT/PROCEDURES

- Successful treatment depends on accurate diagnosis of underlying cause.
- Treat underlying cause.
- Minimize ascitic fluid and peripheral edema without causing intravascular volume depletion.

- Early detection of complications is necessary:
 - SBP:
 - High degree of suspicion
 - Low threshold for paracentesis
 - Prompt therapy
 - Tense ascites and hydrothorax:
 - Supplemental oxygen
 - Therapeutic paracentesis or thoracentesis for respiratory distress
 - Abdominal hernias:
 - Watch for incarceration, ulceration, or rupture.
 - Therapeutic paracentesis
 - Surgical consultation
 - Persistent leak at paracentesis site:
 - Remove more fluid.
 - Stomal barrier device
 - Meralgia paresthetica:
 - Owing to pressure on the lateral femoral cutaneous nerve
 - Relieve the pressure by paracentesis or diuresis.
- Large-volume paracentesis:
 - 5–10 L (100 mL/kg)
 - Performed safely in the ED with stable hemodynamics
 - Consider replacement with IV albumin (5–10 g/L fluid removed) if >5 L removed.
 - Monitor the patient for 8 hr prior to discharge.
- Nonparacentesis reduction of ascites:
 - Strict sodium restriction:
 - <2 g/day
 - Restrict water if serum sodium <120–125 mEq/L
 - Spironolactone:
 - Works best for cirrhotic ascites
 - Alternatives: Amiloride or triamterene
 - Furosemide:
 - Works best for other causes of ascites
 - Add to spironolactone in cirrhotics at spironolactone/furosemide ratio of 100 mg/40 mg.
 - Add metolazone for less responsive cases.
 - Diuretic principles:
 - Administer diuretics as single morning dose.
 - Obtain spot-urine sodium to evaluate response.
 - Patients with urinary Na >10 mEq/L are more responsive to diuretics.
 - Diuretic-induced weight loss should not exceed 2 lb/day in patients without edema and 5 lb/day in patients with edema.

- Monitor electrolytes and renal function.
- Avoid hypokalemia since hypokalemia enhances renal ammonia production, precipitating hepatic encephalopathy.
- Refractory ascites:
 - Accounts for 10% of patients
 - Ensure compliance with diet and medications.
 - Treated with peritoneovenous shunt—transjugular intrahepatic portosystemic shunt
 - Liver transplantation
- Avoid NSAIDs:
 - Diminish response to diuretics
 - Decrease renal plasma flow and GFR.
 - Cause sodium retention/reduces urinary Na excretion
- Treat underlying cause of ascites owing to conditions other than cirrhosis:
 - TB, CHF

MEDICATION

First Line

- Albumin: 5–10 g/L of fluid removed if >5 L removed
- Cefotaxime: 2 g IV q8h
- Spironolactone: 100–400 mg/d (peds: 1–6 mg/kg) PO in 2 divided doses per day
- Furosemide: 40–160 mg/d (peds: 1–3 mg/kg) PO

Second Line

- Amiloride: 5–20 mg/d PO
- Metolazone: 5 mg/d
- Triamterene: 100–300 mg/d PO in 2 divided doses per day



FOLLOW-UP

DISPOSITION

Admission Criteria

- Fulminant liver failure
- Hepatic encephalopathy
- SBP
- Hepatorenal syndrome
- GI bleeding
- Tense ascites not responding to ED treatment

Discharge Criteria

Patients responding to ED management

FOLLOW-UP RECOMMENDATIONS

- GI for all new cases
- Primary doctor or GI for previously established cases

PEARLS AND PITFALLS

- New cases need full workup and GI consultation for management.
- SBP symptoms are frequently vague.
- Must have a high suspicion and low threshold for paracentesis when considering SBP
- Benefits of confirming SBP outweigh risks of bleeding in a coagulopathic patient undergoing paracentesis.
- US guidance is helpful when performing paracentesis in lower-volume ascites.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Cirrhosis



CODES

ICD9

- 789.5 Ascites
- 789.51 Malignant ascites
- 789.59 Other ascites

ICD10

- R18 Ascites
- R18.0 Malignant ascites
- R18.8 Other ascites

ASTHMA, ADULT

Melissa H. White • Carolyn Maher Overman



BASICS

DESCRIPTION

- Increased expiratory resistance:
 - Airway inflammation
 - Bronchospasm
 - Mucosal edema
 - Mucous plugging
 - Smooth muscle hypertrophy
- Consequences:
 - Air trapping
 - Airway remodeling
 - Increased dead space
 - Hyperinflation
- Status asthmaticus refers to disease that does not respond to therapy within 30–60 min
- Risk factors for life-threatening disease:
 - Prior intubations
 - Intensive care unit admissions
 - Chronic steroid use
 - Hospital admission for asthma during the past year
 - Inadequate medical management
 - Increasing age
 - Ethnicity (African Americans)
 - Lack of access to medical care
 - Multiple comorbidities

ETIOLOGY

- Inflammatory process of the airways evidenced by episodic and reversible airflow obstruction and hyper-responsiveness with many cells and cellular elements contributing to the disease:
 - Neutrophils
 - Mast cells
 - Eosinophils
 - Macrophages
 - T lymphocytes
 - Epithelial cells
 - Cytokines

- Triggers:
 - Pollen
 - Dust mites
 - Molds
 - Animal dander
 - Other environmental allergens
 - Viral upper respiratory infections
 - Occupational chemicals
 - Tobacco smoke
 - Environmental change
 - Cold air
 - Exercise induced
 - Emotional factors
 - Menstrual associated
 - Drugs:
 - Aspirin
 - NSAIDs
 - β -blockers



DIAGNOSIS

SIGNS AND SYMPTOMS

- Wheezing
- Dyspnea
- Chest tightness
- Cough
- Tachypnea
- Tachycardia
- Respiratory distress:
 - Posture sitting upright or leaning forward
 - Use of accessory muscles
 - Inability to speak in full sentences
 - Diaphoresis
 - Poor air movement
- Impending failure:
 - Altered mental status
 - Worsening fatigue
- Pulsus paradoxus > 18 mm Hg

ESSENTIAL WORKUP

- Primarily a clinical diagnosis
- Measure and follow severity with peak expiratory flow rate (PEFR)
- Assess for underlying disease

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Arterial blood gas:
 - _ Not helpful during the initial evaluation
 - _ The decision to intubate should be based on clinical criteria.
 - _ Mild-moderate asthma: Respiratory alkalosis
 - _ Severe airflow obstruction and fatigue: Respiratory acidosis and $\text{PaCO}_2 > 42$
- Pulse oximetry:
 - _ $< 90\%$ is indicative of severe respiratory distress.
 - _ Patients with impending respiratory compromise may still maintain saturation above 90% until sudden collapse.
- WBC:
 - _ Leukocytosis is nonspecific
 - _ Pneumonia
 - _ Chronic steroid use
 - _ Stress of an asthma exacerbation
 - _ Demargination occurs after administration of epinephrine and steroids.

Diagnostic Procedures/Surgery

- PEFR:
 - _ Estimates the degree of airflow obstruction:
 - Normal peak flow (adult) is 400–600.
 - 100–300 indicates moderate airway obstruction.
 - < 100 is indicative of severe airway obstruction.
 - Use serially as an objective measure of the response to therapy
- Forced expiratory volume (FEV):
 - _ More reliable measure of lung function than PEFR
 - _ Difficult to use as a screening tool
 - _ Often unavailable in the ED
 - _ Severe airway obstruction: $\text{FEV}_1 < 30\text{--}50\%$
- CXR:
 - _ Indications:
 - Fever
 - Suspicion of pneumonia
 - Suspicion of pneumothorax or pneumomediastinum
 - Foreign body aspiration
 - 1st episode of asthma
 - Comorbid illness: For example: Diabetes, renal failure, CHF, AIDS, cancer
 - Not responding to treatment
 - _ Typical findings:

- Hyperinflation
- Scattered atelectasis
- ECG:
 - _ Indicated in patients at risk for cardiac disease:
 - Dysrhythmias
 - Myocardial ischemia
 - _ Transient changes in severe asthma:
 - Right axis deviation
 - Right bundle branch block
 - Abnormal P-waves
 - Nonspecific ST–T-wave changes

DIFFERENTIAL DIAGNOSIS

- Allergic reaction
- Angioedema
- Bronchiolitis
- Bronchitis
- Carcinoid tumors
- Chemical pneumonitis
- Chronic cor pulmonale
- Chronic obstructive pulmonary disease
- CHF
- Croup
- Foreign body aspiration
- Immersion injury
- Myocardial ischemia
- Pneumonia
- Pulmonary embolus
- Smoke inhalation
- Upper airway obstruction
- Venous air embolus



TREATMENT

PRE HOSPITAL

- Recognize the “quiet chest” as respiratory distress.
- Supplemental oxygen
- Continuous nebulized β -agonist
- Administration of IM/SC epinephrine

INITIAL STABILIZATION/THERAPY

- Immediate initiation of inhaled β -agonist treatment

- Intubate for fatigue and respiratory distress.
- Steroids

ED TREATMENT/PROCEDURES

- Oxygen:
 - _ Maintain an oxygen saturation > 90%
- β -adrenergic agonist:
 - _ Selective β_2 -agonists (albuterol)
 - Mild–moderate asthmatic: Administer every 20 min
 - Severe asthmatic: Continuous nebulized treatment
 - _ SC β -agonist (terbutaline and epinephrine):
 - Severe exacerbations
 - Limited inhalation of aerosolized medicine
 - More side effects because of systemic absorption
 - Terbutaline—longer acting β -2 agonist with bronchodilating effects equivalent to epinephrine in acute asthma.
 - Relative contraindication: Age > 40 yr and coronary disease
- Corticosteroids:
 - _ Reduce airway wall inflammation
 - _ Administered early
 - _ Onset of action may take 4–6 hr
 - _ Administer IV or PO
 - _ IV Solu-Medrol in the treatment of severe asthma exacerbation
 - _ Mild–moderate exacerbations may be treated with oral prednisone burst or Depo-Medrol IM
 - _ Inhaled corticosteroids are currently not recommended as initial therapy.
- Anticholinergic agents:
 - _ If minimal response to initial β -agonist treatment
 - _ Severe airflow obstruction
 - _ Inhaled anticholinergic agents should be used in conjunction with β -agonists.
- Magnesium sulfate:
 - _ No benefit in mild–moderate asthma
 - _ May have a benefit in severe asthma
- Aminophylline:
 - _ Rare utility in acute management
- Leukotriene inhibitors:
 - _ Not currently recommended for acute exacerbation
- Heliox:
 - _ Mixture of helium and oxygen (80:20, 70:30, 60:40)
 - _ Less dense than air
 - _ Decrease airway resistance.
 - _ Decrease in respiratory exhaustion

- Not currently recommended for routine use
- Consider in severe asthma
- Noninvasive positive pressure ventilation:
 - CPAP and BiPAP
 - May improve oxygenation and decrease respiratory fatigue
 - Can only be used in an alert patient
 - Should not replace intubation
 - Not currently recommended for routine use
 - Consider in severe asthma
- Ketamine:
 - Bronchodilator and an anesthetic agent
 - Useful as an induction agent during intubation
 - Contraindications:
 - HTN
 - Coronary disease
 - Preeclampsia
 - Increased intracranial pressure
- Halothane:
 - Inhalation anesthetics are potent bronchodilators.
 - Refractory asthma in intubated patients
- Intubation of the asthmatic patient:
 - Rapid sequence intubation
 - Lidocaine to attenuate airway reflexes
 - Etomidate or ketamine as an induction agent
 - Succinylcholine should be administered to achieve paralysis.
 - A large endotracheal tube >7 mm should be used to facilitate ventilation.
 - May need to mechanically exhale for the patient
 - Permissive hypercapnia

MEDICATION

- β -agonists
 - Albuterol: 2.5 mg in 2.5 mL NS q20min inhaled (peds: 0.1–0.15 mg/kg/dose q20min [min. dose 1.25 mg])
 - Epinephrine: Adult: 0.3 mg (1:1,000) SC q0.5h–q4h \times 3 doses (peds: 0.01 mg/kg up to 0.3 mg SC)
 - Terbutaline: 0.25 mg SC q0.5h \times 2 doses (peds: 0.01 mg/kg up to 0.3 mg SC)
- Corticosteroids:
 - Methylprednisolone: 60–125 mg IV (peds: 1–2 mg/kg/dose IV or PO q6h \times 24 h)
 - Prednisone: 40–60 mg PO (peds: 1–2 mg/kg/d in single or divided doses)
 - Depo-Medrol 160 mg IM
- Anticholinergics

- Ipratropium bromide: 0.5 mg in 3 mL NS q1h × 3 doses
- Magnesium: 2 g IV over 20 min (peds: 25–75 mg/kg)
- Aminophylline: 0.6 mg/kg/h IV infusion
- Rapid sequence intubation:
 - Etomidate: 0.3 mg/kg IV, *or* ketamine: 1–1.5 mg/kg IV
 - Lidocaine: 1–1.5 mg/kg IV
 - Succinylcholine: 1.5 mg/kg IV



FOLLOW-UP

DISPOSITION

Admission Criteria

Medical Wards

- PEFR < 40% and minimal air movement
- Persistent respiratory distress:
 - Factors that should favor admission:
 - Prior intubation
 - Recent ED visit
 - Multiple ED visits or hospitalizations
 - Symptoms for more than 1 wk
 - Failure of outpatient therapy
 - Use of steroids
 - Inadequate follow-up mechanisms
 - Psychiatric illness

Observation Unit

- PEFR > 40% but < 70% of predicted
- Patients without subjective improvement
- Patients with continued wheeze and diminished air movement
- Patients with moderate response to therapy and no respiratory distress

Discharge Criteria

- PEFR > 70% should be > 300
- Patient reports subjective improvement
- Clear lungs with good air movement
- Adequate follow-up within 48–72 hr

FOLLOW-UP RECOMMENDATIONS

Encourage patients to contact their PMD or pulmonologist for asthma related problems over the next 3–5 days.

PEARLS AND PITFALLS

- Altered mental status in asthma equals ventilatory failure.
- Patients should be able to demonstrate the correct use of their inhaler or nebulizer:
 - Discharge with a peak flow meter
- If no signs or symptoms of dehydration, no evidence that IVF will clear airway secretions.
- Antibiotics should generally be reserved for patients with purulent sputum, fever, pneumonia, or evidence of bacterial sinusitis.

ADDITIONAL READING

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- National Asthma Education and Prevention Program Expert Panel Report 3. Guidelines for diagnosis and management of asthma. U.S. Dept of Health and Human Services, October 2007.



CODES

ICD9

- 493.90 Asthma, unspecified type, without mention of status asthmaticus
- 493.91 Asthma, unspecified type, with status asthmaticus
- 493.92 Asthma, unspecified type, with (acute) exacerbation

ICD10

- J45.901 Unspecified asthma with (acute) exacerbation
- J45.902 Unspecified asthma with status asthmaticus
- J45.909 Unspecified asthma, uncomplicated

ASTHMA, PEDIATRIC

Nathan Shapiro



BASICS

DESCRIPTION

- 2.7 million children (<18 yr) affected in US
- 850,000 ED visits per year in US
- Inflammatory events, usually viral, lead to bronchoconstriction:
 - Compounded by hyper-reactivity of airways
 - Mediators of the inflammatory cascade exacerbate symptoms
- Airway obstruction produces increased airway resistance and gas trapping:
 - Mucosal edema
 - Bronchospasm
 - Mucous plugging
- Infants more vulnerable to respiratory failure:
 - Increased peripheral resistance
 - Decreased elastic recoil with early airway closure
 - Unstable rib cage
 - Mechanically disadvantaged diaphragm
- Family history of allergy
- Medical history of early injury to airway (bronchopulmonary dysplasia, pneumonia, intubation, croup, reflux, passive exposure to smoking), reactions to foods and drugs, other allergic manifestations
- Environmental exposures such as pets, smoke, carpets, or dust may trigger or exacerbate

ETIOLOGY

Precipitating/Aggravating Factors

- Infection:
 - Viral
 - Bacterial
- Allergic/irritant:
 - Environment: Pollens, grasses, mold, house dust mites, and animal dander
 - Occupational chemicals: Chlorine, ammonia—food and additives
 - Irritants: Smoke, pollutants, gases, and aerosols
 - Exercise
 - Cold weather
 - Emotional: Stress, phobia
 - Intoxication: β -blockers, aspirin, NSAIDs

SIGNS AND SYMPTOMS

General

- Fatigue, somnolence
- Diaphoresis, agitation
- Hypoxia, cyanosis
- Tachycardia
- Dehydration
- Pulsus paradoxus

Respiratory

- Wheezing, rales, rhonchi
- Cough, acute or chronic
- Tachypnea
- “Tight chest”
- Dyspnea, shortness of breath with prolonged expiratory phase
- Retractions, accessory muscle use, nasal flaring
- Hyperinflation
- Often a history of recurrent episodes and chronic restrictions
- Complications:
 - Recurrent pneumonia, bronchitis
 - Atelectasis
 - Pneumothorax, pneumomediastinum
 - Respiratory distress/failure/death

History

- Precipitating events or known triggers
- Chronicity of symptoms
- Comorbid illnesses
- History of disease:
 - Previous hospitalizations for asthma
 - Previous intubations and intensive care
 - Regular and sporadic medications

Physical-Exam

- Vital signs, including oximetry and respiratory status
- Wheezing: Absence of wheezing may be associated with markedly impaired air movement and decreased breath sounds
- Signs of hypoxia
- Skin and nail bed color bluish

- Signs of respiratory fatigue, distress, or failure:
 - Use of accessory muscles of respirations or retractions
 - Lethargy or confusion

ESSENTIAL WORKUP

- Clinical diagnosis based primarily on physical exam and history; assess ventilation by observation for retractions and use of accessory muscles as well as auscultating for air exchange.
- Follow response to bronchodilator therapy with present illness and past episodes.
- Exclude other differential considerations.
- Pulse oximetry:
 - Initial SaO₂ < 91% (sea level) associated with significant illness: Admission, relapse, prolonged course
- Peak flow meters in cooperative patients (usually > 5 yr old)
 - < 50–70% predicts moderate to severe obstruction.
 - > 70–90% associated with mild to moderate obstruction
 - > 90% considered normal

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Arterial blood gas (ABG) may be an adjunct to pulse oximetry to measure oxygenation and clinical exam to assess ventilation; not mandatory or routinely done.
- CBC as a nonspecific marker of infection
- Theophylline level: Only for patients on theophylline (not recommended)

Imaging

Chest radiograph considered in the following patients, esp. focusing on the presence of infiltrates, bronchial wall thickening, or hyperexpansion.

- < 1 yr of age to exclude foreign body or atelectasis
- First episode of significant wheezing (suggested to assess chronicity of illness and assist in excluding other conditions)
- Increasing respiratory distress or minimal response to therapy
- Respiratory distress/failure
- Shortness of breath in the absence of wheezing

Diagnostic Procedures/Surgery

Peak flow measurement (see above)

DIFFERENTIAL DIAGNOSIS

- Infection/inflammation:
 - Bronchiolitis: Clinically difficult to differentiate except by age and clinical

- history.
- Pneumonia: Viral, bacterial, chemical, or hypersensitivity
- Aspiration
- Retropharyngeal/mediastinal abscess/mass
- Anaphylactic reaction
- Anatomic:
 - Pneumothorax
 - Foreign body
- Vascular disorder:
 - Compression of trachea by vascular anomaly
 - Pulmonary embolism
 - CHF
- Congenital disease:
 - Cystic fibrosis
 - Tracheoesophageal fistula
 - Bronchogenic cyst
 - Congenital heart disease
- Intoxication: Metabolic acidosis
- Neoplasm
- Vocal cord dysfunction (VCD)
- Pulmonary edema—cardiogenic or noncardiogenic
- Gastroesophageal reflux



TREATMENT

PRE HOSPITAL

- Oxygen and oxygen saturation monitoring
- Nebulized β -adrenergic agonist: Albuterol
- Intubate for respiratory failure or severe fatigue.
- IV fluids if evidence of dehydration
- Rapid transport and good communication with ED

INITIAL STABILIZATION/THERAPY

- Maintain $\text{SaO}_2 > 90\text{--}95\%$.
- β -adrenergic nebulizer(s): Albuterol
- Intubate for respiratory failure.
- 20 mL/kg 0.9% NS bolus if evidence of dehydration.

ED TREATMENT/PROCEDURES

- Assess patient for signs of potential respiratory failure:
 - Cyanosis
 - Severe anxiety or irritability

- Lethargy, somnolence, fatigue
- Persistent tachypnea
- Poor air entry, ventilation
- Severe retractions
- Monitor oxygenation; titrate oxygen saturation to $\text{SaO}_2 > 95\%$ (sea level).
- β -adrenergic nebulizer: Albuterol:
 - Frequent or continuous for severe asthma
 - Levalbuterol may require less frequent dosing and may be associated with less side effects.
- Ipratropium bromide may be added as adjunct to β -adrenergic agonists. Most effective when combined with 1st 3 doses of β -adrenergic agent in moderate to severely ill children
- Steroid therapy:
 - Oral for moderate exacerbations in those able to take oral meds
 - IV for severe exacerbations or in those unable to take oral meds
 - 1 dose of dexamethasone may be equivalent to traditional steroids
- SC epinephrine or terbutaline for severe or refractory asthma (rarely used)
- Magnesium sulfate may be useful in severe disease following standard therapy.
- Intubate for respiratory failure:
 - Ketamine is a useful induction agent.
- 20 mL/kg of 0.9% NS bolus if evidence of dehydration
- Heliox (oxygen and helium) may be useful but studies are inconclusive

MEDICATION

- Albuterol (0.5% solution or 5 mg/mL):
 - Nebulizer: 0.15 mg/kg per dose, up to 5 mg per dose, q15–30min PRN
 - Metered-dose inhaler (MDI) (with spacer) (90 μg /puff): 2 puffs q5–10min, max. 10 puffs
 - Also available for nebulizer as 0.083% solution or 2.5 mg/3 mL
- Dexamethasone 0.3 mg/kg/dose (max.: 16 mg)
- Epinephrine (1:1,000) (1 mg/mL): 0.01 mg/kg SC, up to 0.35 mL per dose, q20min for 3 doses
- Ipratropium bromide: Nebulizer (0.02% inhaled sol 500 μg /2.5 mL), 250–500 μg per dose q6h
- Ketamine (for intubation): 1–2 mg/kg IV as induction agent
- Levalbuterol (0.63 and 1.25 mg vials): q6–8h by nebulizer
- Magnesium sulfate: 25 mg/kg per dose IV over 20 min; max. 1.2–2 g per dose
- Methylprednisolone: 1–2 mg/kg per dose IV q6h; max. 125 mg per dose
- Prednisolone: 1–2 mg/kg per dose PO q12h (available as 15 mg/5 mL)
- Prednisone: 1–2 mg/kg per dose PO q6–12h; max. 80 mg per dose
- Terbutaline/ (available as 1 mg/1 mL) (0.01%): 0.01 mL/kg SC q15–20min up to 0.25 mL per dose, q20min for 2 doses

First Line

- Albuterol
- Steroids
- Ipratropium

Second Line

- Epinephrine or terbutaline
- Magnesium sulfate



FOLLOW-UP

DISPOSITION

Admission Criteria

- Need to individualize based upon subjective and objective assessment
- Persistent respiratory difficulty:
 - Persistent wheezing
 - Increased respiratory rate/tachypnea
 - Retraction and use of accessory muscles
- $\text{SaO}_2 < 93\%$ (sea level) on room air
- Peak expiratory flow rate (PEFR) $< 50\text{--}70\%$ predicted levels
- Inability to tolerate oral medicines or liquids
- Prior ED visit in last 24 hr
- Comorbidity:
 - Congenital heart disease
 - Bronchopulmonary dysplasia
 - CF
 - Neuromuscular disease
- Concomitant illness:
 - Pneumonia or severe viral infection

Intensive Care Unit Criteria

- Severe respiratory distress
- $\text{SaO}_2 < 90\%$ or $\text{PaO}_2 < 60$ mm Hg on 40% oxygen
- $\text{PaCO}_2 > 40$ mm Hg
- Significant complications:
 - Pneumothorax
 - Dysrhythmia

Discharge Criteria

- Good response to therapy. Observe in ED 60 min after last treatment before

discharging:

- PEFR > 70% predicted based on age/height
- SaO₂ > 93% on room air (sea level)
- Respiratory rate normal
- No retractions
- Clear or minimal wheezing
- No or minimal dyspnea
- Good follow-up and compliance. Reduce exposure to irritants (smoking) or allergens
- Discharge treatment:
 - Intensive β -adrenergic regimen for 3–5 days
 - Short course (3–5 days) of steroids (2 mg/kg/day) for those presenting with moderate symptoms with consideration of ongoing therapy using nebulized or MDI routes. Patients with moderate or severe exacerbations should have arrangements made for inhaled steroids over a 1–2 mo period such as fluticasone, budesonide, or beclomethasone
 - Follow-up appointment 24–72 hr
 - Instructions to return for shortness of breath refractory to home regimen
 - Long-term therapy should be considered for children with recurrent episodes, persistent symptoms, or activity limitations.

FOLLOW-UP RECOMMENDATIONS

Primary care physician for maintenance therapy, often including nebulized or MDI steroid therapy and education about acute rescue management.

PEARLS AND PITFALLS

- Rapid treatment with continuous re-evaluation to detect any progression of disease is essential.
- When admitting patients, assure that β -adrenergic agent therapy is not interrupted.

ADDITIONAL READING

- Krebs SE, Flood RG, Peter JR, et al. Evaluation of a high dose continuous albuterol protocol for treatment of pediatric asthma in the emergency department. *Pediatr Emerg Care*. 2013;29:191–196.
- National Heart, Blood and Lung Institute; National Asthma Education and Prevention Program. *Guidelines for the diagnosis and management of asthma*. Bethesda, MD: NIH; 2007.
- Robinson PD, Van Asperen P. Asthma in childhood. *Pediatr Clin North Am*. 2009;56(1):191–226.
- Scarfone RJ, Friedlaender E. Corticosteroids in acute asthma: Past, present, and

future. *Pediatr Emerg Care*. 2003;19(5):355–361.



See Also (Topic, Algorithm, Electronic Media Element)

- Bronchiolitis, Pediatric
- Pneumonia, Pediatric



CODES

ICD9

- 493.00 Extrinsic asthma, unspecified
- 493.02 Extrinsic asthma with (acute) exacerbation
- 493.90 Asthma, unspecified type, without mention of status asthmaticus

ICD10

- J45.901 Unspecified asthma with (acute) exacerbation
- J45.902 Unspecified asthma with status asthmaticus
- J45.909 Unspecified asthma, uncomplicated

ASYSTOLE

David F. M. Brown • Calvin A. Lee

BASICS

DESCRIPTION

Absence of ventricular electrical activity

ETIOLOGY

- An end-stage rhythm, sometimes degrading from:
 - Prolonged bradycardia
 - Prolonged ventricular fibrillation (VF)
 - Prolonged pulseless electrical activity
- Patient is extremely unlikely to survive when asystole occurs outside the hospital:
 - ~40% will have return of spontaneous circulation and survive to hospital admission, but <15% survive to hospital discharge.
- Prognosis is similarly poor for those patients who develop asystole after countershock for ventricular tachycardia/VF; <10% survive to hospital discharge.
- Potentially reversible causes include:
 - Hypoxia
 - Hypovolemia (blood loss)
 - Acidosis
 - Hyperkalemia
 - Hypokalemia
 - Drug overdose
 - Hypothermia
 - Pulmonary embolism
 - Myocardial infarction
 - Tension pneumothorax
 - Cardiac tamponade

DIAGNOSIS

SIGNS AND SYMPTOMS

- Unresponsive patient
- Pulseless
- Agonal or absence of spontaneous respirations

ESSENTIAL WORKUP

- Confirm asystole in 2 limb leads to exclude VF.
- Confirm lead and cable connections.

- Confirm monitor power is on.
- Confirm monitor gain is up.
- Identify reversible causes (see above)

DIAGNOSIS TESTS & INTERPRETATION

Lab

Arterial blood gas (potassium and hemoglobin)

Imaging

Cardiac US to exclude pericardial tamponade

DIFFERENTIAL DIAGNOSIS

“Fine” VF (which may be mistaken for asystole)



TREATMENT

PRE HOSPITAL

- No intervention should be made for a patient with a valid Do Not Resuscitate document.
- No intervention if patient can be verified as dead:
 - Rigor mortis
 - Dependent livedo
 - Injury incompatible with life (e.g., decapitation)

INITIAL STABILIZATION/THERAPY

- Initiate CPR, with emphasis on minimally interrupted, high-quality chest compressions
- Confirm asystole with cardiac monitor
- Place airway device (ETT preferred, but BVM acceptable), confirm placement, and provide 100% inspired oxygen and a slow ventilation rate (6–12 breaths/minute). Minimize interruption in chest compressions during airway placement
- Establish IV or IO access.
- Apply continuous waveform capnography to optimize quality of chest compressions (PETCO₂ correlates with cardiac output and myocardial blood flow during CPR)
- Epinephrine every 3–5 min.
- Consider and treat potentially reversible causes (see above)
- Sodium bicarbonate if hyperkalemia or drug overdose suspected
- No proven benefit to an empiric single countershock
- No proven benefit to electrical pacing
- Provide defibrillation without delay, IF the patient develops VF or VT

ED TREATMENT/PROCEDURES

- Initiate induced hypothermia in comatose patients with return of spontaneous circulation
- Consider termination of resuscitation efforts if the following conditions are met:
 - High-quality chest compressions performed for a period of time
 - Tracheal intubation to ensure normal oxygenation
 - Fine VF excluded
 - Reversible causes corrected or excluded
 - Bedside US without pericardial effusion
 - No tension pneumothorax clinically

MEDICATION

- Epinephrine: 1 mg (peds: 0.01 mg/kg) IV q3–5min
- Sodium bicarbonate: 1 mEq/kg IV only if:
 - Pre-existing acidosis
 - Hyperkalemia
 - Tricyclic antidepressant overdose is suspected.



FOLLOW-UP

DISPOSITION

Admission Criteria

All patients with return of spontaneous circulation

Discharge Criteria

None—all patients with return of spontaneous circulation need admission to an ICU for post-arrest care

FOLLOW-UP RECOMMENDATIONS

A permanent pacemaker may be considered only if asystole is found to be due to primary heart block

Patient Monitoring

ICU for cardiac monitoring and induced hypothermia as appropriate

PEARLS AND PITFALLS

- Emphasis on high-quality, minimally uninterrupted chest compressions while considering reversible causes of asystole
- Resuscitation is likely to be successful only if reversible causes are found and corrected immediately

ADDITIONAL READING

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- Bernard SA, Gray TW, Buist MD, et al. Treatment of comatose survivors of out-of-hospital cardiac arrest with induced hypothermia. *N Engl J Med*. 2002;346(8):557–563.
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- Silvestri S, Ralls GA, Krauss B, et al. The effectiveness of out-of-hospital use of continuous end-tidal carbon dioxide monitoring on the rate of unrecognized misplaced intubation within a regional emergency medical services system. *Ann Emerg Med*. 2005;45:497–503.



CODES

ICD9

427.5 Cardiac arrest

ICD10

- I46.2 Cardiac arrest due to underlying cardiac condition
- I46.8 Cardiac arrest due to other underlying condition
- I46.9 Cardiac arrest, cause unspecified

ATAXIA

Lara K. Kulchycki

BASICS

DESCRIPTION

- Inability to perform coordinated movements
- Caused by a disorder of the cerebellum or its connections:
 - Ipsilateral signs with lateral cerebellar lesions
 - Truncal ataxia with midline lesions

ETIOLOGY

Usually cerebellar in origin, but may occur with sensory, motor, or vestibular dysfunction:

- Trauma
- Mass lesions
- Vascular disorders
- Infections or postinfectious processes
- Toxins/drugs
- Metabolic/endocrine derangements
- Demyelinating diseases
- Congenital malformations
- Hereditary disorders:
 - Inborn errors of metabolism
 - Progressive degenerative ataxias
- Nutritional deficiencies

DIAGNOSIS

SIGNS AND SYMPTOMS

- Gait disturbance:
 - Ataxia often presents with unsteady gait
 - Initial sense of insecurity while walking
 - Problems with special skills (bicycling, skiing, climbing)
 - Sense of imbalance
 - Wide base stance and staggering gait
 - Test tandem gait to identify subtle ataxia
- Limb ataxia:
 - Incoordination
 - Intention tremors
 - Clumsiness with writing, picking up objects, buttoning

- Dysmetria: Under- or overshooting on finger-to-nose and heel-to-shin testing
- Dysdiadochokinesis: Difficulty with rapid alternating movements
- Truncal ataxia:
 - Head tremors
 - Truncal instability
 - Titubation: Swaying of the head/trunk while at rest
- Dysarthria and bulbar symptoms:
 - Slurred speech
 - Staccato, scanning speech
 - Choking from incoordination of swallowing
- Visual abnormalities:
 - Blurry vision
 - Vertigo:
 - Distinguish central from peripheral vertigo
 - Peripheral vertigo is often severe, triggered by movement, and may be accompanied by ear pain, hearing loss, or tinnitus
 - Nystagmus:
 - Gaze-evoked nystagmus: Repetitive drifts to the midline followed by fast phase to the eccentric side
 - Rebound nystagmus
- Abnormalities of muscle tone and strength:
 - Isometrataxia: Difficulty sustaining constant force during hand use:
 - Ask patient to hold slight, steady pinching pressure against examiner's finger (examiner will feel irregular pressure)
 - True muscle weakness or hypotonia uncommon in cerebellar disease
- Sensory ataxia:
 - Paresthesias
 - Numbness
 - Cautious, steppage gait
 - Marked worsening of coordination with eyes closed:
 - A positive Romberg sign is the classic finding in sensory ataxia
 - Loss of position/vibration sense
 - Difficulty with fine motor skills

History

- A careful history is essential since gait changes may be caused by pain, weakness, lightheadedness, vertigo, or incoordination
- Onset:
 - Hours–days: Acute
 - Weeks–months: Subacute
 - Months–years: Chronic
- Symmetric or focal symptoms
- Presence of fever, mental status changes, weakness, sensory loss, or urinary

incontinence

- Recent viral illness or immunizations
- History of trauma or toxic ingestion
- Family history of movement disorder

Physical-Exam

- Perform a complete physical exam, including neurological and gait testing
- Assess for signs or symptoms of acute, life-threatening disorders such as hemorrhage, stroke, or CNS infection:
 - Altered mental status
 - Headache
 - Focal neurological deficits
 - Elevated intracranial pressure:
 - Bradycardia, HTN, abnormal respiratory pattern
 - Papilledema
 - Bulging fontanelles
 - Fever
 - Meningismus
 - Nystagmus
 - Nausea/vomiting
 - Examine ears and perform provocative testing for nystagmus (Dix–Hallpike)
- Note the presence of intoxication or toxidromes in patients with suspected ingestion

ESSENTIAL WORKUP

A detailed history and physical exam will help determine which tests are necessary

DIAGNOSIS TESTS & NTERPRETATION

Lab

- Blood glucose level
- Serum electrolytes
- Toxicology screen:
 - Standard panels may not include the drugs of interest in the ataxic patient
- Thyroid function testing
- Target additional testing to likely exposures, such as anticonvulsants

Imaging

- CT:
 - Head CT can identify mass, hemorrhage, subacute infarct, or hydrocephalus
 - Consider CT with and without IV contrast if mass suspected
 - CT angiography can be performed to evaluate for vascular disease
- MRI:

- Excellent study to evaluate for acute ischemia, mass, demyelinating lesions, and vascular abnormalities
- Superior for imaging the posterior fossa
- MR angiography of head/neck may be indicated if vascular abnormality is suspected
- EKG:
 - Not indicated as part of ataxia workup, but may be useful in evaluation of nonspecific dizziness

Diagnostic Procedures/Surgery

- Lumbar puncture:
 - Indicated if infection or Guillain–Barré suspected

DIFFERENTIAL DIAGNOSIS

- Acute symmetric ataxia:
 - Head trauma
 - Drug use/toxic ingestion:
 - Alcohol
 - Lithium
 - Phenytoin
 - Barbiturates
 - Carbamazepine
 - Phenobarbital
 - Valproic acid
 - Benzodiazepines
 - Diphenhydramine
 - Dextromethorphan
 - Acute viral cerebellitis
 - Meningitis/encephalitis
 - Hydrocephalus
 - Postinfectious syndrome
 - Hypoglycemia
 - Hyponatremia
 - Severe heat stroke
- Acute focal ataxia:
 - Posterior circulation infarction
 - Anterior cerebral artery syndrome
 - Vertebrobasilar insufficiency (VBI)
 - Cerebellar hemorrhage
 - Subdural hematoma
 - Cerebellar abscess
 - Acute disseminated encephalomyelitis
 - Complicated migraine

- Atypical seizure
- Subacute symmetric ataxia:
 - Drug use/toxic ingestion:
 - Mercury
 - Lead
 - Hydrocarbons
 - Glue sniffing
 - Cytotoxic chemotherapy
 - Organophosphates
 - Vitamin B₁ or B₁₂ deficiency
 - Paraneoplastic syndromes:
 - Breast/ovarian cancer
 - Hodgkin lymphoma
 - Neuroblastoma
 - Lyme disease
 - Toxoplasmosis
 - Creutzfeldt–Jakob disease
- Subacute focal ataxia:
 - Cerebellar glioma
 - Metastatic tumors
 - Lymphoma
 - Multiple sclerosis
 - Guillain–Barré syndrome
 - AIDS-related progressive multifocal leukoencephalopathy
 - Syringomyelia
 - Cervical spondylosis
- Chronic ataxia:
 - Alcohol-related cerebellar degeneration
 - Stable gliosis
 - Inherited disorders:
 - Spinocerebellar ataxias
 - Friedreich ataxia
 - Ataxia telangiectasia
 - Niemann–Pick disease
 - Hypothyroidism
 - Vitamin E deficiency
 - Tabes dorsalis
 - Congenital malformation:
 - Arnold—Chiari
 - Dandy–Walker
- Disease states that cause peripheral vertigo can mimic the gait findings in ataxia:
 - Benign paroxysmal positional vertigo

- Acute labyrinthitis
- Ménière disease

Pediatric Considerations

- May present with a refusal to walk
- Acute ataxia in children is usually a benign, self-limited process:
 - 60% of cases caused by acute cerebellar ataxia or drug ingestion
- Acute cerebellar ataxia:
 - Postinfectious cerebellar demyelination
 - Usually occurs in children 2–5 yr old
 - Onset 1–3 wk after triggering illness
 - Over 1/4 of cases occur after varicella, but linked to many viral infections and immunizations
 - Normal mental status
 - No fever, focal deficits, or seizures
 - Mild cases may be managed at home, but require injury prevention counseling
 - Most children recover completely within 3 mo without intervention
- Drug/toxic ingestions:
 - Expect mental status changes
 - Assess access to medications and order appropriate toxicological testing
- Guillain–Barré syndrome:
 - 15% present with sensory ataxia
 - Miller-Fisher variant: Clinical triad of ataxia, areflexia, and ophthalmoplegia
- Neoplasm:
 - More than 50% of childhood brain tumors occur in the brainstem or cerebellum
 - Opsoclonus-myoclonus-ataxia syndrome:
 - Paraneoplastic autoimmune syndrome affecting cerebellum
 - Over 50% due to neuroblastoma
- Stroke:
 - Rare in children, but can occur in patients with sickle cell disease or hypercoagulable states

Geriatric Considerations

- Gait disorders in the elderly are often multifactorial
- Underlying cognitive deficits may make it difficult to distinguish presyncope, weakness, vertigo, and incoordination
- Posterior circulation cerebrovascular syndromes, like VBI and stroke, are more common in the elderly and may present with vague symptoms, like dizziness
- Evaluate for signs of orthostasis or extrapyramidal disorders, like Parkinsonism



TREATMENT

PRE HOSPITAL

ALERT

- Acute onset of ataxia may be due to stroke or hemorrhage
- Deterioration in mental status may warrant field endotracheal intubation

INITIAL STABILIZATION/THERAPY

- ABCs
- IV access
- Supplemental oxygen
- Cardiac monitor
- Fingertick blood glucose:
 - _ Administer dextrose if hypoglycemic
 - _ Consider thiamine in alcoholics and malnourished patients

ED TREATMENT/PROCEDURES

- Institute fall precautions
- Treatment must be tailored to the patient's presentation and underlying pathology
- Cerebellar infarction can lead to significant edema with mass effect and herniation:
 - _ Neurosurgery consultation may be needed for decompressive craniectomy

MEDICATION

- Dextrose: D₅₀W 1 amp (50 mL or 25 g) (peds: D₂₅W 2–4 mL/kg) IV
- Thiamine (vitamin B₁): 100 mg IV



FOLLOW-UP

DISPOSITION

Admission Criteria

- Acute and subacute ataxia, particularly if a benign etiology cannot be established
- Patients who cannot ambulate safely
- Admit patients with cerebellar hemorrhage or mass effect to the ICU

Discharge Criteria

- Reversible or mild symptoms
- Normal mental status
- Able to ambulate safely

FOLLOW-UP RECOMMENDATIONS

Follow up with primary care or neurology depending on likely etiology of symptoms

PEARLS AND PITFALLS

- Failure to distinguish true ataxia from other causes of gait instability
- Failure to note trauma in intoxicated patients
- Failure to realize the limitations of CT scan in evaluating the posterior fossa
- Failure to recognize the risk of herniation in cerebellar lesions, including stroke

ADDITIONAL READING

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CODES

ICD9

- 334.2 Primary cerebellar degeneration
- 334.3 Other cerebellar ataxia
- 781.3 Lack of coordination

ICD10

- G11.1 Early-onset cerebellar ataxia
- G11.9 Hereditary ataxia, unspecified
- R27.0 Ataxia, unspecified

ATRIAL FIBRILLATION

Edward Ullman • Terrance T. Lee



BASICS

DESCRIPTION

- Dysrhythmia characterized by seemingly disorganized atrial depolarizations without effective atrial contraction
- Caused by multiple re-entrant waveforms within the atria
- Atrial rate ranges from 350–600 beats per minute (bpm).
- Results in loss of organized atrial contractions and rapid ventricular rate:
 - Decrease in cardiac output
 - Prone to embolus formation
- Most common clinical arrhythmia:
 - Prevalence increasing with age
 - Men are at higher risk

ETIOLOGY

- Systemic disease:
 - HTN
 - Hyperthyroidism
 - Chronic pulmonary disease
 - Infection
 - Pulmonary embolus
 - Hypoxia
 - Drugs (e.g., sympathomimetics)
 - Acute alcohol ingestion (holiday heart syndrome)
 - Obesity
 - Electrolyte disturbance
 - Thyroid disease
- Underlying cardiac disease:
 - Cardiomyopathy
 - CAD
 - Valvular disease, especially mitral
 - Pericarditis
 - Sick sinus syndrome
 - Myocardial contusion
 - CHF
 - Congenital heart disease
- Idiopathic:
 - Absence of any known etiologic factor

- No clinical or echocardiographic evidence of heart disease



DIAGNOSIS

SIGNS AND SYMPTOMS

- Palpitations
- Decreased cardiac output:
 - Weakness
 - Light headedness
 - Syncope
 - Hypotension
 - Angina
 - Pulmonary edema
 - Altered mental status
 - Lower extremity edema
 - Hepatojugular reflex
- Embolus formation:
 - Acute neurologic injury
 - Mesenteric ischemia

History

- Onset of symptoms
- Duration
- Inciting factors
- Prior episodes of fibrillation
- Prior heart disease

Physical-Exam

- Palpitations
- Irregularly irregular pulse
- Absence of A- waves in the jugular venous pulse
- Pulse deficit with more rapid ventricular rates:
 - The auscultated or palpated apical rate is faster than the rate palpated at the wrist

ESSENTIAL WORKUP

- History and physical exam:
 - Assess for instability and need for immediate cardioversion
 - Duration of symptoms > 48 hr or < 48 hr
 - Evidence of systemic disease or underlying cardiac disease
- ECG: Signs of congestive heart failure
 - Absent P-waves replaced by fibrillatory (f) waves, 350–600 bpm

- F-waves vary in amplitude, morphology, and intervals
- R-R intervals are irregularly irregular
- Absence of an isoelectric baseline
- Ventricular rate ranges from 80–150 bpm:
 - If rate > 200 associated with wide-irregular QRS, consider bypass tract
- Slower rate suggests abnormal AV node or presence of AV nodal blocking medication
- Usually narrow QRS complexes unless:
 - Functional aberration
 - Pre-existing bundle branch
 - Pre-excitation with an accessory pathway

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC
- Electrolytes
- Cardiac enzymes—if ischemia is a concern
- Thyroid function
- Digoxin level—if patient is taking
- Anticoagulation parameters
- Urine drug screen

Imaging

- CXR
- ECG

DIFFERENTIAL DIAGNOSIS

- Atrial flutter with variable AV block
- Multifocal atrial tachycardia
- Sinus rhythm with frequent premature atrial contractions
- Atrial tachycardia with variable AV block



TREATMENT

PRE HOSPITAL

- IV access
- Monitor
- Oxygen
- Cardioversion:
 - In settings where patient is unstable

INITIAL STABILIZATION/THERAPY

- IV
- Oxygen
- Monitor
- Immediate synchronized electrical cardioversion starting at 200 J if the patient is unstable

ED TREATMENT/PROCEDURES

- Hemodynamically unstable and life threatening:
 - _ Myocardial infarction, pulmonary edema, heart failure that does not respond promptly to pharmacological measures
 - _ Synchronized electrical cardioversion
 - Biphasic: Start at 100 J, higher success rate
 - Monophasic: Start at 200 J
 - Sx duration < 48 hr: Consider IV heparin bolus prior.
 - Sx duration > 48 hr: IV heparin, transesophageal echo to exclude atrial clot, cardioversion. Anticoagulate for 4 wk. Do not delay echo if life-threatening arrhythmia.
 - Consider pretreatment with antiarrhythmic drugs and use anterior-posterior pad placement to increase likelihood of success
 - _ Chemical cardioversion:
 - Choice of drug depends on history of CHF, high BP, LV hypertrophy, and CAD
 - Medications may be proarrhythmic and should be used with caution
 - As with electrical cardioversion, appropriate anticoagulation will be necessary depending on the duration and presence/absence of clot
 - Ibutilide
 - Procainamide
 - Flecainide
 - Propafenone
 - Sotalol
- Hemodynamically stable, mildly symptomatic:
 - _ Treat underlying cause if 1 is identified.
 - _ Identify if symptoms are < 48 hr. If so consider synchronized cardioversion.
 - _ > 48 hr: Rhythm control does not offer mortality benefit over rate control
 - _ Use procainamide to treat stable patients with a suspected bypass tract
 - _ Rate control:
 - Not necessary if rate < 100 bpm or if rhythm spontaneously converts to sinus
 - AV nodal blockers (calcium channel blockers, β -blockers, and digoxin) contraindicated if bypass tract suspected such as WPW
 - Calcium channel blockers: Consider in patient with pulmonary disease. Use cautiously in patient with uncompensated CHF and 2nd-

- or 3rd-degree heart block
 - β -blockers: Consider in patient with coronary artery disease (CAD). Use cautiously in patient with uncompensated CHF, 2nd- or 3rd-degree heart block, and pulmonary disease
 - Digoxin: Consider in patient with pre-existing CHF.
 - Amiodarone: Consider in refractory atrial fibrillation
- Rhythm control and prophylaxis:
 - Includes procainamide, sotalol, amiodarone, dofetilide
 - Amiodarone: Only agent with strong data to support initiation for outpatient treatment
- Elective cardioversion:
 - Oral anticoagulation with therapeutic levels for 3 wk prior to and 4 wk after
- Stable patients with atrial fibrillation and WPW can be treated with procainamide or ibutilide, although cardioversion may be preferred
- Anticoagulation determined by CHADS2 scoring:
 - 1 point for each of the following:
 - History of cardiac failure
 - History of HTN
 - Age ≥ 75 yr
 - Diabetes
 - 2 points for a history of stroke or TIA
 - Score of 0:
 - 81–325 mg/day of aspirin
 - Score of 1:
 - Either 81–325 mg/day of aspirin or adjusted-dose warfarin with a target INR of 2.5
 - Score > 1 :
 - Adjusted-dose warfarin with a target INR of 2.5 (range 2–3)
 - Adjusted annual stroke rate increases from 1.9% for a CHADS2 score of 0 to 18.2% for a CHADS2 score of 6
 - Aspirin:
 - Patients with contraindications to anticoagulation and unreliable individuals
 - Patients with low stroke risk

MEDICATION

- Metoprolol:
 - 5–10 mg slow IV push at 5 min intervals to total of 15 mg
 - 25 mg–100 mg oral BID
- Diltiazem:
 - 0.25 mg/kg IV over 2 min; if unsuccessful, repeat in 15 min as 0.35 mg/kg IV over 2 min; maintenance infusion of 5 mg/h usually started to maintain

- rate control.
 - 120–300 mg oral daily
- Digoxin:
 - 0.5 mg IV initially, then 0.25 mg IV q4h until desired effect
- Esmolol:
 - 0.5 mg/kg over 1 min; maintenance infusion at 0.05 mg/kg/min over 4 min
- Propranolol:
 - 0.1 mg/kg IV divided into equal doses at 2–3 min intervals
- Verapamil:
 - 2.5–5 mg IV bolus over 2 min; may repeat with 5–10 mg q15–30min to max. of 20 mg
 - 120–300 mg PO daily
- Amiodarone:
 - 5–7 mg/kg over 30–60 min, then 1.2–1.8 g/d continuous infusion or in divided PO doses until 10 g total
 - 600–800 mg/d divided dose until 10 g total, then 200–400 mg/d maintenance
- Procainamide: 15–18 mg/kg loading dose administered as a slow infusion over 30 min. Max.: 1 g. Then 2–6 mg/min infusion.
- Quinidine gluconate: 324–648 mg PO q8–12h: (extended release tabs)
- Ibutilide: 1 mg IV for patients >60 kg; 0.01 mg/kg IV for patients <60 kg infused over 10 min; can be repeated once if sinus rhythm not restored within 10 min. Requires normal QTc, no history of torsades, no hypokalemia. Patients must be monitored for 4 h for QT prolongation, Torsades de Pointes, and ventricular tachycardia.
- Flecainide: 2 mg/kg IV at 10 mg/min PO. Do not give in patients with structural heart disease.
- Propafenone: 1–2 mg/kg IV at 10 mg/min
- Sotalol: 75 mg infused IV over 5 h BID if CrCl > 60 mL/min. Give QD if CrCl 40–60 mL/min
- Heparin: Load 80 U/kg IV; infusion at 18 U/kg/h. Dosage adjustment required in obese patients
- Low-molecular-weight heparin: 1 mg/kg SQ BID
- Warfarin sodium: 2.5–5 mg/d PO, dosage adjustments based on INR
- Aspirin: 50–325 mg/d

ALERT

IV form for flecainide, propafenone, and sotalol not approved for use in US; *must be infused slowly.*



FOLLOW-UP

DISPOSITION

Admission Criteria

- Unstable AF:
 - Inability to control rate
- High risk for stroke:
 - Prior cardiovascular accident
 - CHF
- Associated medical problems contributing to the AF that require inpatient management

Discharge Criteria

- Conversion to sinus rhythm if symptoms < 48 hr
- Chronic AF with appropriate ventricular rate control and anticoagulation
- New-onset AF with rate control and anticoagulation

Issues for Referral

- Cardiology or an electrophysiologist
- Evaluation for outpatient cardioversion

FOLLOW-UP RECOMMENDATIONS

- INR check if placed on warfarin
- The patient should return to the ED if feeling faint, dizzy, numbness or weakness of the face or limbs, or trouble seeing or speaking

PEARLS AND PITFALLS

- If hemodynamically unstable and life threatening, synchronized cardioversion is warranted
- Rate or rhythm control is an individualized option for stable atrial fibrillation using β -blockers, calcium channel blockers, or antiarrhythmics
- Do not mistake F-waves or U-waves as P-waves. Can misdiagnose AF as a sinus rhythm.
- Do not use channel blockers, β -blockers, or digoxin in AF with a wide complex AF in a patient with an underlying bypass tract

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CODES

ICD9

427.31 Atrial fibrillation

ICD10

- I48.0 Paroxysmal atrial fibrillation
- I48.1 Persistent atrial fibrillation
- I48.91 Unspecified atrial fibrillation

ATRIAL FLUTTER

Liesl A. Curtis



BASICS

DESCRIPTION

- Atrial dysrhythmia
- 200,000 new cases each year
- A macroreentrant circuit in the right atrium is thought to be the underlying mechanism.
- Most sensitive rhythm to cardioversion
- Seldom occurs in the absence of organic heart disease
- Less common than supraventricular tachycardia (SVT) or atrial fibrillation
- Typically paroxysmal, lasting seconds to hours
- Occurs in ~ 25–35% of patients with atrial fibrillation
- Untreated, may promote cardiomyopathy

ETIOLOGY

- Alcoholism
- Cardiomyopathies and myocarditis
- CHF
- Electrolyte abnormalities
- Ischemic heart disease
- Pulmonary embolus and other pulm diseases
- Valvular heart diseases
- Post op following cardiac surgery (often in 1st postoperative week)
- Thyrotoxicosis

Pediatric Considerations

- Occurs in children but is often asymptomatic
- Associated mortality is highest in the neonatal period.
- Associated with:
 - Congenital heart disease
 - Infectious etiologies, such as rheumatic fever or myocarditis
- Be sure to consider potential toxic ingestions in pediatric patients with new AV block



DIAGNOSIS

SIGNS AND SYMPTOMS

- Palpitations

- Syncope/presyncope
- Chest pain
- Fatigue
- Dyspnea
- Poor exercise capacity
- Tachycardia—HR > 150 bpm:
- Hypotension
- Heart failure

Pediatric Considerations

- Infants do not tolerate atrial flutter well.
- The aortic valve (AV) node is capable of very rapid conduction.
- Extremely rapid ventricular rates can lead to shock or CHF.
- Atrial flutter can occur in the fetus and young infants without associated cardiac defects:
 - _ Often does not recur beyond neonatal period
- Most older children have an underlying cardiac abnormality
 - _ More likely to recur and difficult to control

ESSENTIAL WORKUP

- EKG
- Labs
- CXR

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Electrolytes and mineral panel
- Cardiac enzymes
- Digoxin level
- PT/PTT

Imaging

- CXR:
 - _ May identify cardiomyopathy or CHF
- Echo:
 - _ May identify regional wall motion abnormalities or valvular dysfunction

DIFFERENTIAL DIAGNOSIS

- SVT
- Sinus tachycardia
- Atrial fibrillation
- Multifocal atrial tachycardia

- Ventricular tachycardia (VT)



TREATMENT

PRE HOSPITAL

- Oxygen, monitor, IV access
- Unstable patients should be cardioverted in the field:
 - _ Immediate synchronized cardioversion
 - _ Start with 100 J

INITIAL STABILIZATION/THERAPY

- Oxygen, monitor, IV access
- Immediate synchronized cardioversion if unstable
 - _ Current guidelines recommend starting at 150–200 J min to improve initial success and to limit cumulative energy doses.

ED TREATMENT/PROCEDURES

- Rate control:
 - _ Rate control should be instituted prior to giving an antidysrhythmic to avoid risk of a 1:1 AV conduction ratio and hemodynamic collapse.
 - _ May be difficult to achieve
- Anticoagulation:
 - _ Same guidelines as for atrial fibrillation:
 - INR 2–3 for 3 wk prior to cardioversion if > 48 hr or unknown duration
 - Recommended even if negative transesophageal echo
 - Risk of thromboembolism ranges from 1.7–7%.
 - _ CHADS₂ score: Used for decision regarding anticoagulation
 - CHF history (1 point)
 - Hypertension history (1 point)
 - Age ≥ 75 (1 point)
 - DM history (1 point)
 - Stroke symptoms or TIA history (2 points):
 - Score 0: Aspirin is sufficient prophylaxis
 - Score 1: Oral anticoagulants preferred
 - Score 2 or more: Oral anticoagulants strongly recommended
 - _ Patients at higher thromboembolism risk:
 - Valvular heart disease
 - Fluctuating a fib/flutter rhythms
 - Left ventricular (LV) dysfunction
 - Prior stroke or thromboembolism
 - Longer symptom duration (> 48 hr)

- Antiarrhythmic drugs:
 - Adenosine:
 - Unlikely to break atrial flutter
 - May aid in the diagnosis of atrial flutter by unmasking the flutter waves
 - Amiodarone:
 - Rate control in patients with pre-excited atrial arrhythmias (i.e., WPW)
 - Preferable antiarrhythmic agent for patients with severely impaired heart function
 - Major adverse effects are hypotension and bradycardia, slower infusions can prevent this.
 - Calcium channel blockers:
 - Rate control
 - Verapamil has higher incidence of symptomatic hypotension than diltiazem.
 - Verapamil should only be used in narrow-complex arrhythmias
 - β -blockers:
 - Rate control
 - Added benefit of cardioprotective effects for patients with ACS
 - Magnesium sulfate:
 - Rate control
 - Low-level evidence
 - Digoxin:
 - Rate control
 - 3rd-line drug
 - Has inotropic properties so may be useful in patients with ventricular dysfunction
 - Longer onset to therapeutic effect
 - Procainamide:
 - Rhythm control
 - Drug of choice for patients with known pre-excitation syndromes (i.e., WPW) and preserved ventricular function
 - Caution if patient has QT prolongation
 - Sotalol:
 - Rhythm control
 - Not a 1st-line drug
 - For use in WPW and preserved ventricular function if duration of arrhythmia is ≤ 48 hr
 - Ibutilide:
 - Rhythm control
 - For acute pharmacologic rhythm conversion in patients with preserved ventricular function (EF > 30%) if duration of arrhythmia

- is ≤ 48 hr
 - Correct potassium and magnesium before use
 - Contraindicated if QTc > 440 msec or in patients with severe structural heart disease
 - Efficacy rate of 38–76%
 - Mean time to conversion is 30 min.
 - Incidence of sustained polymorphic VT 1.2–1.7%
 - Observe for 4–6 hr after administration for QT prolongation or VT.
- Cardioversion:
 - 100–360 J
 - Sedation when possible
 - Safest and most effective means of restoring sinus rhythm
- Maintenance of sinus rhythm after cardioversion:
 - High recurrence rate: $\sim 50\%$ at 1 yr; however, difficult to determine rate because data combines atrial fibrillation with atrial flutter
 - Amiodarone most effective
- Percutaneous catheter ablation:
 - Acute success rates exceed 95%.
 - 5–10% recurrence in 1–2 yr of follow-up
 - Low complication rate
 - Candidates include:
 - Recurrent episodes of drug-resistant atrial flutter
 - Patients who are drug intolerant
 - Patients who do not desire long-term drug therapy

Pediatric Considerations

- Verapamil is not recommended in infants and young children as it is associated with a low cardiac output and serious cardiovascular compromise.
- Digoxin is the 1st-line drug therapy for pediatric atrial flutter.
- Consider cardioversion as 1st-line therapy in neonates.

MEDICATION

- Amiodarone: 150 mg IV over 10 min, then continuous infusion at 1 mg/min for 6 hr, then 0.5 mg/min infusion over 18 hr; supplemental 150 mg infusions can be dosed PRN to a max. daily dose of 2.2 g (peds: 5 mg/kg IV loading dose over 20–60 min, may repeat to max. of 15 mg/kg/d IV)
- Adenosine: 6 mg IV $\times 1$. May give 12 mg IV q1–2min $\times 2$ if no conversion. Give all doses IV push
- Atenolol: 5 mg IV over 5 min, may repeat in 10 min if tolerated, then 50 mg PO q12h
- Digoxin: Loading dose 8–12 U μ g/kg lean body weight, half of which is administered initially over 5 min, and remaining portion at 25% fractions at 4–8 hr intervals (peds: 8–12 μ g/kg)

- Diltiazem: 0.25 mg/kg IV over 2 min followed in 15 min by 0.35 mg/kg IV over 2 min, maintenance infusion of 10–15 mg/h titrated to heart rate
- Dofetilide: CrCl > 60 mL/min and QTc 440 msec or less) initial dose 500 µg ORALLY twice daily; determine QTc 2–3 h after 1st dose; if QTc increases by more than 15% OR is > 500 msec (550 msec in patients with ventricular conduction abnormalities), reduce dose to 250 µg ORALLY twice daily; MAX. dose 500 µg ORALLY twice daily
- Esmolol: 0.5 mg/kg over 1 min; maintenance infusion at 0.05 mg/kg/min; can repeat loading dose and increase in increments of 0.05 mg/kg/min q4min up to 0.3 mg/kg/min
- Flecainide: A single dose of flecainide 300 mg (body weight 70 kg or greater), and flecainide 200 mg (body weight < 70 kg) [3]; prior to antiarrhythmic initiation, a β-blocker or nondihydropyridine calcium channel antagonist should be administered to prevent rapid AV conduction if atrial flutter occurs
- Ibutilide: 1 mg IV over 10 min for patients > 60 kg; 0.01 mg/kg IV for patients < 60 kg infused over 10 min; dose can be repeated once if normal sinus rhythm not restored within 10 min after infusion
- Magnesium sulfate: 1–2 g diluted in D5W over 5–60 min; slower rate preferable if patient is stable.
- Metoprolol: 5 mg IV push over 5 min at 5 min intervals to total of 15 mg, then 50 mg PO BID
- Procainamide: 20 mg/min until arrhythmia suppressed, hypotension, QRS prolongation of 50%, or total of 17 mg/kg; may be given at rate up to 50 mg/min (peds: 15 mg/kg IV over 30 min, then 20–80 µg/kg/min continuous infusion)
- Propranolol: 0.5–1 mg over 1 min, repeated after 2 min up to a total dose of 0.1 mg/kg (peds: 0.01–0.15 mg/kg/dose slow IV push over 5 min, max. 1 mg/dose)
- Sotalol: 1–1.5 mg/kg over 5 min (US packaging recommends infusion over 5 h)
- Verapamil: 2.5–5.0 mg IV bolus over 2 min; may repeat with 5–10 mg q15–30min to a max. of 20–30 mg.



FOLLOW-UP

DISPOSITION

Admission Criteria

- New-onset atrial flutter requiring antidysrhythmics, rate control
- Symptomatic (i.e., chest pain that warrants a rule out or cardioversion)
- CHF

Discharge Criteria

- New-onset atrial flutter who meet these criteria:
 - _ Rate or rhythm has been controlled

- Underlying cause has been investigated and addressed
- Anticoagulation has been initiated
- Appropriate follow-up is arranged
- Chronic atrial flutter with good rate control and appropriate anticoagulation

FOLLOW-UP RECOMMENDATIONS

Cardiologist: Radiofrequency ablation of atrial flutter emerging as treatment of choice for patients with symptomatic atrial flutter without identifiable reversible cause

PEARLS AND PITFALLS

- Be aware of WPW:
 - Do not use adenosine, β -blockers, calcium channel blockers, and digoxin (Class III can be harmful).
 - Can cause increased ventricular response, which can deteriorate to ventricular fibrillation
- Do not delay cardioversion in an unstable patient for IV placement.
- Use β -blockers with caution in patients with pulmonary disease or CHF.
- 4 major treatment issues:
 - Rate control
 - Prevention of systemic embolization
 - Reversion to sinus rhythm
 - Maintenance of sinus rhythm

ADDITIONAL READING

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ICD9

427.32 Atrial flutter

ICD10

- I48.3 Typical atrial flutter
- I48.4 Atypical atrial flutter
- I48.92 Unspecified atrial flutter

ATRIOVENTRICULAR BLOCKS

Colleen N. Roche

BASICS

DESCRIPTION

- Impaired conduction between the atrium and the ventricle through the AV node or His–Purkinje system
- 1st-degree AV block:
 - Prolonged conduction through the AV node
 - Ventricular impulses are not lost.
 - Generally benign, and occurs in 1.6% healthy adults.
- 2nd-degree AV block:
 - Marked by a failure of some atrial impulses to reach ventricles
 - Mobitz Type I (Wenckebach):
 - Usually secondary to conduction deficit in AV node.
 - Progressive prolongation of the pulse-rate (PR) interval until a nonconducted P-wave and a dropped QRS complex occur
 - Generally benign, but may be a complication of an inferior wall MI
 - Mobitz Type II:
 - Conduction deficit is usually below the level of the AV node.
 - PR intervals are constant until single or multiple beats are abruptly dropped.
 - High likelihood of progression to complete heart block
 - Worse prognosis if associated with an acute MI
 - Less common than Type I
- 3rd-degree AV block:
 - Also known as complete heart block
 - All atrial impulses are unable to reach the ventricular conducting system; a ventricular escape pacemaker then takes over, resulting in AV dissociation.
 - Constant PP and RR intervals with variable PR intervals because PP and RR intervals are independent of each other.
 - More severe symptoms occur when the block is lower in the conducting system.
 - If secondary to toxicologic agents, often resolves upon omission of offending toxin
 - Never a benign condition

ETIOLOGY

- Essentially due to:
 - A structural lesion

- Increase in inherent refractory period
- Marked shortening of the supraventricular cycle
- MI:
 - 1st-degree block and Type I 2nd-degree AV block may be associated with an inferior wall MI:
 - These blocks are transient.
 - AV conduction usually returns to normal with no increased morbidity or mortality.
 - Type II 2nd-degree AV block may be associated with an anterior wall MI:
 - 5% anterior wall MIs are associated with AV blocks.
 - Increased mortality secondary to ventricular arrhythmias and left-heart failure
- Coronary artery disease:
 - Chronic ischemic injury can lead to fibrosis around the AV node
- Toxicologic:
 - Digoxin
 - β -blockers
 - Calcium-channel blockers
 - Amiodarone
 - Procainamide
 - Class 1C agents: Propafenone, encainide, flecainide
 - Clonidine
- Congenital
- Valvular heart disease
- Surgical trauma:
 - S/P coronary artery bypass graft or valvular replacement
- Increased vagal tone
- Infectious:
 - Syphilis
 - Diphtheria
 - Chagas disease
 - TB
 - Toxoplasmosis
 - Lyme disease
 - Myocarditis
 - Endocarditis
 - Rheumatic fever
 - Abscess formation in interventricular septum
- Collagen vascular diseases
- Infiltrative diseases:
 - Sarcoidosis
 - Amyloidosis
 - Hemochromatosis

- Cardiomyopathy
- Electrolyte disturbances:
 - Hyperkalemia
- Myxedema
- Hypothermia

Pediatric Considerations

- Occurs in children, but is often asymptomatic
- Associated mortality is highest in the neonatal period.
- Associated with:
 - Congenitally acquired maternal antibodies
 - Congenital heart disease
 - Infectious etiologies, such as rheumatic fever or myocarditis
- Be sure to consider potential toxic ingestions in pediatric patients with new AV block



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- 1st-degree AV block:
 - Asymptomatic
- Type I 2nd-degree AV block:
 - Pulse irregularities
- Type II 2nd-degree AV block and 3rd-degree block:
 - Exercise intolerance
 - Palpitations
 - Chest pain
 - Presyncope/syncope
 - Altered mental status
 - Dyspnea, orthopnea

Physical-Exam

- 1st-degree AV block:
 - No discrete physical exam findings
- Type I 2nd-degree AV block:
 - Regularly irregular pulse
- Type II 2nd-degree AV block and 3rd-degree block:
 - Irregular pulse
 - Hypotension
 - Mental status changes

- Signs of heart failure:
 - o Rales
 - o Cyanosis
 - o Jugular venous distention

ESSENTIAL WORKUP

- A 12-lead EKG to determine the type of block and identify evidence of infarction
- 1st-degree AV block:
 - PR interval > 0.20 sec
- 2nd-degree AV block:
 - Type I: Progressive prolongation of PR interval until there is a nonconducted P-wave and a dropped QRS complex; occurs in repeated cycles; QRS is usually narrow.
 - Type II: PR interval remains constant; atrial impulses are not conducted intermittently, giving the appearance of an occasionally dropped ventricular beat; QRS may be prolonged depending on the level of the lesion.
- 3rd-degree AV block:
 - P-waves occur at consistent intervals.
 - QRS complexes occur independently from P-waves but also at consistent intervals.
 - QRS complexes are usually narrow unless there is an infranodal conduction disturbance or a ventricular escape rhythm.

DIAGNOSIS TESTS & INTERPRETATION

Additional studies aid in confirming the etiology of the identified AV block.

Lab

- Electrolytes
- Calcium, magnesium
- Cardiac enzymes:
 - Especially for Type II 2nd-degree and 3rd-degree blocks
- Digoxin level, if patient has been exposed to this medication

Imaging

- CXR:
 - May identify cardiomyopathy or CHF
- ECG:
 - May identify regional wall motion abnormalities or valvular dysfunction

DIFFERENTIAL DIAGNOSIS

- Accelerated junctional rhythm
- Idioventricular rhythm

- Sinus bradycardia
- SA block



TREATMENT

PRE HOSPITAL

- Transcutaneous pacing for unstable Type II 2nd- or 3rd-degree block
- Atropine:
 - _ Avoid with Type II 2nd-degree block because it may precipitate complete heart block
 - _ Contraindicated in 3rd-degree heart block with a widened QRS complex
- Attempts should be made to prevent increases in vagal tone.

INITIAL STABILIZATION/THERAPY

- Transcutaneous pacemaker:
 - _ Necessary for the unstable patient with signs of hypoperfusion:
 - Hypotension
 - Chest pain
 - Dyspnea
 - Mental status changes
- Atropine:
 - _ Can be administered in:
 - Complete heart block with a narrow QRS
 - Symptomatic sinus bradycardia

ED TREATMENT/PROCEDURES

- 1st-degree AV block:
 - _ No treatment required
 - _ Avoid AV nodal blocking agents
 - _ Evaluate for associated MI, electrolyte abnormalities, medication excess in the appropriate clinical scenarios
- Type I 2nd-degree AV block:
 - _ Usually no treatment needed
 - _ If symptomatic, atropine will enhance AV conduction
- Type II 2nd-degree AV block:
 - _ Temporary transcutaneous or transvenous pacemaker
 - _ Atropine is not effective and should be avoided
- 3rd-degree AV block:
 - _ 1st line of treatment: Emergent pacemaker
 - _ May transiently respond to atropine with narrow QRS complexes
 - _ If block is identified to be toxin-mediated, specific treatments include:
 - Digoxin-specific antibodies (digoxin overdose)

- Glucagon and calcium (β -blocker or calcium-channel blocker overdose)

MEDICATION

- Atropine: 0.5–1.0 mg (peds: 0.01–0.03 mg/kg) IV q5min as necessary
- Digoxin-specific antibodies: 10 vials (380 mg) is an appropriate loading dose if digoxin toxicity is strongly suspected:
 - _ Serum level \times weight (kg) = number of vials to be administered
- Glucagon: 5–10 mg (peds: 50 μ g/kg) IV over 5 min
- Calcium chloride: 250–500 mg (peds: 20 mg/kg) IV



FOLLOW-UP

DISPOSITION

Admission Criteria

Monitored bed:

- Type II 2nd-degree block
- 3rd-degree block

Discharge Criteria

Asymptomatic 1st-degree and Type I 2nd-degree blocks: Ensure follow-up for further outpatient workup.

FOLLOW-UP RECOMMENDATIONS

Asymptomatic 1st-degree and Type I 2nd-degree blocks can follow-up with a cardiologist on a routine outpatient basis.

PEARLS AND PITFALLS

- Obtaining an EKG rapidly in symptomatic patients is paramount.
- Once a high-degree AV block has been diagnosed, initiate transcutaneous pacing immediately.
- Obtain a complete history from all available resources; it may help you identify an offending toxin rapidly.
- Common pitfalls:
 - _ Failure to interpret EKG properly
 - _ Failure to diagnose AV block appropriately
 - _ Failure to initiate transcutaneous pacing in a timely fashion
 - _ Failure to consult cardiology for permanent pacemaker in a timely fashion

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Bradyarrhythmias
- Cardiac Pacemakers



CODES

ICD9

- 426.10 Atrioventricular block, unspecified
- 426.11 First degree atrioventricular block
- 426.13 Other second degree atrioventricular block

ICD10

- I44.0 Atrioventricular block, first degree
- I44.1 Atrioventricular block, second degree
- I44.30 Unspecified atrioventricular block

BABESIOSIS

Philip D. Anderson



BASICS

DESCRIPTION

- Tick-borne, infectious disease caused by intraerythrocytic protozoa of the genus *Babesia*, infects wide array of vertebrate animals, causes lysis of host RBCs
- Asymptomatic to severe, life-threatening infection depending on the species of *Babesia* and the immune status of the patient
 - Asymptomatic infection:
 - 50% of children and 25% of adults with infection have no symptoms
 - Mild–moderate disease:
 - Usually immune competent patients
 - Infections typically self-limited or resolve with antibiotic therapy
 - Mortality usually <5%
 - Severe disease:
 - Defined as hospitalization >2 wk, ICU stay >2 days, or ending in death
 - Typically associated with immune compromise: Splenectomy; cancer; HIV; hemoglobinopathy; chronic heart, lung, or liver disease
 - Other groups at higher risk for severe disease: Neonates, >50 yr old, on immune-suppressive drugs (e.g., rituximab or anticytokine therapy [e.g., etanercept, infliximab])
 - Mortality can be as high as 21% among immune-suppressed patients
- Complications develop in approximately one-half of the hospitalized patients:
 - ARDS, DIC most common
 - Can also see CHF, coma, liver failure, renal failure, splenic rupture
- Co-occurrence with other tick-borne diseases should be considered in endemic regions under the following conditions:
 - Lyme disease (*Borrelia burgdorferi*) – associated rash
 - Human granulocytic anaplasmosis (*Anaplasma phagocytophilum*) – protracted symptoms with leukopenia

ETIOLOGY

- *Babesia*:
 - Species causing human disease:
 - *Babesia microti* – Northern and Midwestern US (most common cause of disease in US)
 - *Babesia divergens* – Europe
 - *Babesia duncani* – Northern US Pacific coast

- Case reports of Babesiosis in Asia, Africa, Australia, and South America
- Animal reservoirs:
 - *B. microti* – white-footed mouse, white-tailed deer
 - *B. divergens* – cattle, rats
- Transmission via *Ixodes* tick vector:
 - Most common vector for transmission of babesiosis to humans
 - *Ixodes* requires blood meal from a vertebrate host to pass through each stage of life cycle (larva, nymph, adult)
 - Most cases result from nymphal tick bites in late spring through summer, adult ticks can also transmit disease
- Pathogen life cycle, pathogenesis:
 - Protozoa pass from tick salivary glands to mammalian bloodstream where they penetrate erythrocytes, mature and divide.
 - Mature protozoa exit from RBC resulting in membrane damage, lysis, hemolytic anemia, and hemoglobinuria.
 - Damaged RBCs become less deformable, enhancing removal by spleen; however, asplenic patients less able to clear infected RBCs, leading to more severe disease.
 - Damaged RBCs may result in microvascular stasis with secondary ischemic organ injury to liver, spleen, heart, kidney, or brain.
- Transmission via transfusion of RBCs, platelets:
 - > 150 cases since 1979, 75% of these since 2000
 - *B. microti* is the most common pathogen
 - Low-level parasitemia may not be visible on donor blood smears, yet can still transmit disease
 - Often results in severe cases as recipients of blood products often immune compromised or have significant comorbidities

Pediatric Considerations

Transmission can occur in utero and during delivery; youngest reported case was a 4-wk-old infant.

DIAGNOSIS

SIGNS AND SYMPTOMS

Gradual onset of malaise and fatigue, associated with fever as high as 105°F (40.6°C), 1–4 wk after tick bite, or 1–9 wk after transfusion with contaminated blood products

- Common symptoms include chills and sweats, headache, anorexia, nonproductive cough, arthralgia, and nausea
- Less common symptoms include vomiting, sore throat, abdominal pain, conjunctival injection, photophobia, weight loss, emotional lability, depression, and hyperesthesia

History

- Febrile, flu-like illness in patients who
 - live in, or traveled to an endemic area within past 2 mo (especially during spring, summer)
 - have had blood product transfusions within past 6 mo
- Shock or sepsis presentation in patients with above history, especially in presence of risk factors for severe disease (see above)

Physical-Exam

- Fever (most common finding)
- Hepatosplenomegaly
- Pharyngeal erythema
- Jaundice
- Retinopathy with splinter hemorrhages
- Retinal infarcts
- Rash may be seen:
 - Petechiae, ecchymosis
 - Erythema chronicum migrans (suggests concurrent Lyme disease)
- Severe disease:
 - Tachypnea
 - Hypoxia
 - Hypotension
 - Altered mental status

ESSENTIAL WORKUP

- Microscopy of thin blood smear with Giemsa or Wright staining to demonstrate *Babesia* organisms
- When smears are negative, polymerase chain reaction (PCR) assay can be used
- Indirect immunofluorescent antibody testing can be used to recognize babesial antigens when microscopy and PCR assays are negative

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Microscopy:
 - Intraerythrocytic parasites can be round, oval, or pear shaped.
 - Parasites in budding tetrad formation (Maltese cross) are pathognomonic for babesiosis, but not commonly seen.
 - Most common finding is intraerythrocytic round or oval (pyriform) rings with pale blue cytoplasm and red-staining nucleus.
 - Extracellular parasites may be seen with high levels of parasitemia.
 - Parasitemia levels are generally between 1% and 10%, but can be as high as 80%; may be <1% in early stages of disease.

- Ring forms may appear similar to *Plasmodium falciparum* (malaria); in babesiosis there are no pigment deposits (hemozoin) that are usually seen with malaria.
- PCR:
 - Amplification of babesial 18s rRNA gene is more sensitive than microscopy
 - Results can be available within 24 hr
 - Useful in cases with low levels of parasitemia
- Serology:
 - Indirect immunofluorescent antibody testing may be useful when microscopy and PCR testing are negative.
 - IgM antibody usually detectable 2 wk after onset of illness
 - IgG titers $\geq 1:256$ suggest active or recent infections; IgM titers $\geq 1:64$ suggest acute infection.
- Nonspecific lab abnormalities that may be seen in babesiosis:
 - Mild-to-moderate hemolytic anemia (low hematocrit/hemoglobin, low haptoglobin, elevated reticulocyte count, elevated lactate dehydrogenase, elevated total bilirubin)
 - Thrombocytopenia is common
 - LFTs (elevated alkaline phosphatase, transaminases, lactate dehydrogenase, bilirubin)
 - Urinalysis (hemoglobinuria, proteinuria)
 - Elevated BUN, creatinine suggests renal insufficiency
 - Hyperkalemia may result from massive hemolysis

DIFFERENTIAL DIAGNOSIS

- Malaria
- Other tick-borne diseases:
 - Lyme disease
 - Human granulocytic anaplasmosis
 - Ehrlichiosis
 - Rocky Mountain spotted fever
 - Colorado tick fever
 - Q fever
 - Tularemia
 - Relapsing fever
- Typhoid fever
- Acute hemolytic anemia



TREATMENT

PRE HOSPITAL

- Ensure a patent airway in patients with respiratory distress.

- Provide supplemental oxygen and ventilatory assistance as needed.
- If patient presents in shock, establish IV access and administer a fluid bolus of 0.9% NS 500 mL (peds: 20 mL/kg) IV.

INITIAL STABILIZATION/THERAPY

- Airway management, ventilatory support for patients with acute respiratory distress
- IV access should be established in patients with evidence or risk factors for severe disease.
- IV fluids, pressor support for patients in shock
- Cardiac monitor: Patients with severe disease may develop cardiac ischemia, arrhythmias.

ED TREATMENT/PROCEDURES

- Antipyretics for fever
- Start antibiotic therapy in symptomatic patients after confirming diagnosis on microscopy or PCR
- Mild–moderate disease:
 - Oral atovaquone + azithromycin for 7–10 days is the regimen of choice.
 - Clindamycin + quinine is an effective alternative, but associated with significant side effects (tinnitus, vertigo, gastroenteritis) that may require reduced dosing or stopping medication in up to one-third of patients.
- Severe disease:
 - IV clindamycin + oral quinine for 7–10 days is regimen of choice (IV quinine may be used, but may cause ventricular arrhythmias and requires QT monitoring)
 - RBC exchange transfusion is indicated in patients with parasitemia > 10%; hemoglobin < 10 g/dL; or pulmonary, renal, or hepatic complications.
 - Persistent or relapsing disease may be seen in immunocompromised patients; these patients should receive antibiotic therapy for at least 6 wk, continuing for 2 wk after the last positive blood smear; can use standard combinations (see above).
- Asymptomatic infection:
 - Antibiotics are not indicated unless parasitemia on blood smears persists > 3 mo.

MEDICATION

- Acetaminophen: 500 mg (peds: 10–15 mg/kg, do not exceed 5 doses/24 h) PO q4–6h, do not exceed 4 g/24 h
- Atovaquone: 750 mg (peds: 20 mg/kg; max. 750 mg/dose) PO BID for 7 days
- Azithromycin: 500 mg (peds: 10 mg/kg; max. 500 mg) PO on day 1, followed by 250 mg (peds: 5 mg/kg; max. 250 mg) PO per day: 6 days
- Clindamycin: 300–600 mg (peds: 7–10 mg/kg q6–8h) IV q6h; 600 mg (peds: 7–10

- mg/kg q6–8h) PO q8h for 7–10 days
- Ibuprofen: 400 mg (peds: 20–40 mg/kg/d) PO q6–8h PRN
- Quinine: 650 mg (peds: 25 mg/kg/d) PO q8h for 7–10 days



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients with parasitemia > 4%, severe anemia (hemoglobin < 10 g/dL), significant symptoms or complications, or need for exchange transfusion require admission:
 - Respiratory distress
 - Hypotension or shock
 - New renal insufficiency or hepatic failure
 - Altered mental status
 - Severe hemolysis (jaundice, hematuria)
- Admission should also be considered in patients without any of the above, but who have risk factors for developing severe disease (see above):
 - Elevated alkaline phosphatase, elevated WBC counts, and male gender were predictive of more severe outcomes

Discharge Criteria

- Patients with asymptomatic, mild, or moderate disease
- Parasitemia < 4%
- Intact spleen, immune competent
- Able to tolerate oral medications

Issues for Referral

Immunodeficient patients are more likely to have persistent or relapsing disease following initial treatment and should be referred for infectious disease consultation.

FOLLOW-UP RECOMMENDATIONS

Patients diagnosed with babesiosis should follow up with their primary care physician or infectious disease specialist for monitoring of parasitemia levels following completion of antibiotic course in symptomatic patients and at 3 mo in asymptomatic patients.

PEARLS AND PITFALLS

- Babesiosis can be a life-threatening disease in asplenic patients.
- Consider babesiosis as a potential cause of respiratory distress/shock in patients with a travel history to an endemic area.

- Microscopy findings may not be present in early stages of disease when parasitemia levels are low.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Lyme Disease



CODES

ICD9

088.82 Babesiosis

ICD10

B60.0 Babesiosis

BACK PAIN

James Willis • Eric Legome



BASICS

DESCRIPTION

- Low back pain (LBP):
 - Refers to pain in the area between the lower rib cage and the gluteal folds, often with radiation into the thighs
- Sciatica:
 - Pain in the distribution of the lower lumbar spinal roots
 - May be accompanied by neurosensory and motor deficits
- Pain classification:
 - Acute: < 6 wk
 - Subacute: 6–12 wk
 - Chronic: > 12 wk

ETIOLOGY

- Nonspecific musculoligamentous source (great majority) (e.g., muscle, ligament, fascia)
- Herniation of the nucleus pulposus
- Degenerative joints or discs
- Spinal stenosis
- Anatomic abnormalities—especially spondylolisthesis
- Fractures from trauma and osteoporosis
- Underlying systemic diseases (minority):
 - Neoplasm
 - Infections
 - Vascular (dissection, aneurysm, and thrombosis)
 - Renal
 - GI
 - Pelvic organ pathology



DIAGNOSIS

SIGNS AND SYMPTOMS

- Musculoligamentous:
 - Poorly localized and dull back/gluteal pain without radiation past the knee.
 - Usually no objective neurologic signs.
 - Back spasm is a variable and poorly reproducible finding.
- Sciatica:

- Sharp, shooting, well-localized pain
- Leg complaints often greater than back
- May present with
 - asymmetric deep tendon reflexes
 - decreased sensation in a dermatomal distribution
 - objective weakness
- Massive central disc herniation (cauda equina):
 - Decreased perineal sensation
 - Urinary retention with overflow incontinence
 - Fecal incontinence
- Infectious processes:
 - Fever
 - Localized percussion tenderness of the vertebral bodies
- Bony lesion:
 - Continuous pain that does not change with rest
 - Constitutional symptoms
- Vascular etiology:
 - Severe, often “ripping or tearing” pain
 - May be associated with cold or insensate extremities

History

- Can assist with focusing and narrowing differential diagnosis. Helps rule out concerning pathology for pain:
 - Intensity
 - Quality
 - Location and radiation
 - Onset
 - Exacerbating or remitting factors
 - Social or psychological factors
 - Response to previous therapy
- Risk factors for serious disease:
 - Fever
 - Constitutional symptoms
 - Trauma
 - Age >60 yr
 - History of cancer:
 - Especially those that metastasize to bone
 - Chronic steroid use
 - IV drug use
 - Recent instrumentation or bacteremia
 - Night pain

Physical-Exam

- Fever
- Spasm or soft tissue tenderness is a poorly reproducible finding:
 - Vertebral tenderness sensitive but nonspecific for infection
- Straight leg raise—elevating the leg while supine reproduces sciatic symptoms:
 - Ipsilateral raise highly sensitive but not specific
 - Crossed leg raise highly nonspecific but insensitive
- Ankle and great toe dorsiflexion and ankle plantar flexion (L5, S1 nerve roots)
- Ankle deep tendon reflexes (S1)
- Dermatomal sensory exam:
 - Assess for saddle anesthesia
- Rectal sphincter tone

ESSENTIAL WORKUP

- Thorough history and physical exam, including detailed neurologic and vascular exam
- No specific tests are needed for uncomplicated musculoligamentous or sciatic pain
- Rapid diagnostic testing and vascular consultation concerning aortic etiology

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Urinalysis for suspected:
 - UTI/pyelonephritis
 - Prostatitis
- ESR:
 - Highly sensitive, though nonspecific for infectious or inflammatory etiologies
 - Used for screening to help rule out disease

Imaging

- Lumbosacral radiograph:
 - Significant trauma
 - Age > 50–60 yr
 - History or signs/symptoms of cancer
 - Fever
 - IV drug user
 - Pain at rest
 - Suspicion of inflammatory etiology
 - Pain that does not improve after 4 wk
- Bedside US:
 - Full bladder suggests urinary retention
 - Abdominal aortic aneurysm (AAA)

- Abdominal CT if patient stable
- MRI:
 - Suspicion of abscess:
 - Fever, immunocompromised, IVDA, history of bacteremia
 - Suspicion of metastatic tumor:
 - Systemic cancer, weight loss
 - Suspicion of hematoma:
 - Anticoagulation, recent spinal anesthesia
 - Rapidly progressing neurologic symptoms
 - Urinary retention or fecal incontinence associated with back pain
- CT:
 - Secondary modality for diagnosis of abscess, cancer, or massive disc when MRI unavailable
 - Test of choice in imaging potential unstable fractures
 - Excellent sensitivity to evaluate vascular etiology in stable patient

DIFFERENTIAL DIAGNOSIS

- Spinal origins—in the majority of patients no precise anatomic site is discovered:
 - Musculoligamentous (majority)
 - Discogenic
 - Fracture
 - Spondylolisthesis
 - Ankylosing spondylitis
 - Osteomyelitis
 - Epidural abscess/hematoma
 - Neoplasm
- Nonspinal causes:
 - AAA
 - Prostatitis
 - Upper UTI
 - Abdominal neoplasm
 - Renal colic
 - Aortic dissection



TREATMENT

PRE HOSPITAL

- Immobilization is not generally recommended for nontraumatic pain.
- Rapid transport for vascular concerns

ED TREATMENT/PROCEDURES

- NSAIDs:

- Musculoligamentous pain
- Renal colic
- Similar benefits as APAP but less optimal side-effect profile
- APAP: Considered 1st-line therapy for mild-to-moderate pain
 - Moderate but conflicting evidence for benefit of NSAID and acetaminophen combination over each individually in postoperative pain
 - APAP and NSAIDs not effective for sciatica pain
- Muscle relaxants:
 - Cyclobenzaprine, methocarbamol, carisoprodol, or tizanidine
 - Benefits must be balanced by side effects, mostly sedation, dizziness, and dry mouth
- Benzodiazepines:
 - No clear difference from skeletal muscle relaxants
 - Likely higher risk profile for addiction
- Narcotics:
 - A reasonable (3–5 days) course may be given for severe pain not relieved by anti-inflammatory or APAP. Effective for neuropathic pain
 - Risk benefit profile should be considered and discussed with patient
- Corticosteroids:
 - No benefit in radicular or nonradicular back pain
- Spinal manipulation:
 - A short course (< 2 wk) may be helpful in acute LBP without sciatica
- Physical therapy/exercise:
 - No clear consensus for indications
 - May be helpful in symptomatic relief, preventing further episodes and teaching patients
- Acupuncture:
 - Controversial, probable benefit for chronic musculoskeletal pain
 - No clear benefit over other modalities
 - Trigger point therapy with minimal to no evidence of benefit for chronic LBP not studied for acute
- Massage:
 - May be beneficial when combined with exercises and education
- Heat/cold therapy:
 - Limited evidence to support that heat wrap therapy may help reduce pain and disability for patients with back pain < 3 mo. Improved as adjunct to exercise.
- Bed rest:
 - Unhelpful to speed recovery and may impede improvement. If patient requires bed rest acutely or is symptomatically improved, 1 or 2 days may be recommended.
- Back exercises:
 - Unlikely to be useful in acute phase; may assist with prevention of future

episodes

- Expected recovery to pain-free state:
 - Conflicting data, mostly in non-ED setting
 - ~33% within 1 wk
 - ~90% within 6–8 wk
 - Low SES, female sex, baseline disability and chronic LBP significant for worse functional outcome at 1 and 3 wk
 - Newer ED data suggests functional limitation in 50% of patients with pain at 3 mo.
- Recurrence is common: ~40%

MEDICATION

First Line

- Acetaminophen: 500 mg (peds: 10–15 mg/kg, do not exceed 5 doses/24h) PO q4–6h, do not exceed 4 g/24h
- Hydrocodone/acetaminophen: 5/500 mg PO q4–6h
- Ibuprofen: 600–800 mg PO q6–8h (peds: 10 mg/kg q6h)
- Naproxen: 250–500 mg PO q12h
- Oxycodone/acetaminophen: 5/500 mg PO q4–6h

Second Line

- Cyclobenzaprine: 5–10 mg PO TID. Caution patient regarding drowsiness.
- Methocarbamol: 500–1,500 PO q6h. Caution patient regarding drowsiness.
- Valium: 5–10 mg PO q8h
- You may combine 1st- and 2nd-line therapies but side-effect profile will increase



FOLLOW-UP

DISPOSITION

Admission Criteria

- Severe pain with inability to ambulate
- Pain unresponsive to ED management
- Progressive neurologic deficits
- Signs of cauda equina syndrome
- Infectious, vascular, or neoplastic etiologies

Discharge Criteria

Uncomplicated presentation with ability to control pain and ambulate

Geriatric Considerations

- Maintain a high suspicion for serious disease including vascular etiology, neoplasm, or infection.
- Have a low threshold for imaging or diagnostic testing.
- Follow up patients on NSAIDs or opioids more carefully for complications or adverse events related to therapy.

Pediatric Considerations

- Back pain is unusual in the pediatric patient; a high suspicion for an infectious etiology must be maintained.
- For musculoligamentous pain, a single trial found that Ibuprofen provides good pain control with a low side-effect profile.

Pregnancy Considerations

Limited evidence suggests that strengthening and pelvic tilt exercises combined with routine prenatal care may have benefit in treating back pain; unclear if they prevent pain

Issues for Referral

Urgent neurosurgical or orthopedic consultation for definite diagnosis or high suspicion for abscess or lesion (disc, neoplasm, or other) with rapidly progressive objective neurologic findings

FOLLOW-UP RECOMMENDATIONS

- Uncomplicated back pain: PCP in 1–2 wk
- New sciatica without neurologic findings: PCP or specialist in 7–10 days
- Complicated with sensory findings only or minimal motor symptoms: 24–48 hr
- Marked or rapidly progressive motor symptoms, or bowel/bladder findings warrant specialist consultation in the ED or transfer if unavailable.

PEARLS AND PITFALLS

- Consider MRI for history of IVDA to rule out epidural abscess or if concerns of nonbony spinal metastases.
- Elderly with minimal trauma may sustain fractures.
- Consider vascular etiology in elderly patients with 1st-time presentation of back pain.
- Advise patients that this is often a prolonged course and they should not expect rapid resolution.
- Opioids should be limited to a short course from the ED.

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CODES

ICD9

- 724.2 Lumbago
- 724.3 Sciatica
- 724.5 Backache, unspecified

ICD10

- M54.5 Low back pain
- M54.9 Dorsalgia, unspecified
- M54.30 Sciatica, unspecified side

BACTERIAL TRACHEITIS

Noah K. Rosenberg • Gary Bubly

BASICS

DESCRIPTION

- A tracheal infection potentially causing acute airway obstruction. Also known as bacterial croup and laryngotracheobronchitis. Exudative tracheitis can refer to a less severe form of disease
- Usually secondary bacterial infection of trachea, complicating antecedent viral infection, or less commonly, instrumentation
- Fatal in 0–20%
- Tracheal membrane formation, purulent discharge, subglottic edema, erosions, with normal epiglottis
- Classically presents with prodrome similar to croup followed by rapid deterioration and loss of airway patency
- Mean age 5 yr; rarely occurs in adults
- More common in children than epiglottitis, presumably due to success of *Haemophilus influenzae* immunization
- More frequent August–December

ALERT

Patients may present with a fairly benign course, followed by rapid deterioration, with respiratory distress, toxic appearance, and acute airway obstruction.

ETIOLOGY

- *Staphylococcus aureus* (with occ. methicillin-resistant *S. aureus* [MRSA])
- *Moraxella catarrhalis*
- *Streptococcus pneumoniae*
- Group A streptococcal species
- *Pseudomonas aeruginosa*
- *H. influenzae* type B
- *Escherichia coli*
- Anaerobes
- *Klebsiella pneumoniae*
- *Nocardia*
- Associated with influenza A (including H1N1) and B, parainfluenza, adenovirus, and RSV viral infections
- *Aspergillus*, HSV in immunocompromised hosts (HIV)

DIAGNOSIS

SIGNS AND SYMPTOMS

History

Usually preceding viral infection with acute deterioration in course of illness

Physical-Exam

- Fever
- Cough
- Retractions
- Inspiratory/expiratory stridor
- Toxic appearance
- Hoarseness
- Cyanosis
- Nasal flaring
- Sore throat/neck pain
- Dysphonia (drooling uncommon)
- Complications:
 - Respiratory:
 - Airway obstruction
 - Subglottic stenosis
 - Pulmonary edema
 - Pneumothorax
 - ARDS
 - Endotracheal tube (ETT) plugging
 - Infection:
 - Septic shock
 - Toxic shock syndrome (TSS)
 - Pneumonia
 - Retropharyngeal cellulitis
 - Cardiopulmonary arrest
 - Renal failure

ESSENTIAL WORKUP

- Clinical assessment and management of airway takes priority over diagnostic workup; secure airway, optimally in operating room under controlled conditions.
- Ensure adequate oxygenation before proceeding:
 - Pulse oximetry

DIAGNOSIS TESTS & INTERPRETATION

Lab

- WBC variably elevated
- Blood cultures usually negative

- Request tracheal cultures from endoscopist/surgeon.

Imaging

Radiographs of neck soft tissue:

- If done, perform in ED; accompany and monitor at all times.
- Tracheal margin irregularities
- Subglottic narrowing
- Clouding of tracheal air column
- Irregular intratracheal densities
- Normal epiglottis

Diagnostic Procedures/Surgery

- Flexible fiberoptic laryngoscopy:
 - Permits direct visualization of epiglottis
 - Mucosal edema
 - Subglottic edema, secretions, membrane
- Bronchoscopy:
 - Direct visualization of trachea
 - Laryngotracheal inflammation and erosions
 - Mucopurulent secretions
 - Membranes
 - Therapeutic stripping of membranes
 - Enables direct culture of material

DIFFERENTIAL DIAGNOSIS

- Infection:
 - Croup (failure to respond to treatment, older age, rapid deterioration or toxic appearance should raise suspicion for bacterial tracheitis rather than croup.)
 - Epiglottitis
 - Peritonsillar abscess
 - Retropharyngeal abscess
 - Uvulitis
 - Laryngeal diphtheria
- Angioedema
- Intraluminal obstruction:
 - Foreign body aspiration
- Caustic ingestion
- Trauma



TREATMENT

PRE HOSPITAL

- Assess airway/breathing:
 - Supplemental oxygen
 - Racemic epinephrine aerosol if easily tolerated
 - Reassurance; avoid agitating child
- Bag-valve-mask (BVM) ventilation if in respiratory failure
- Intubate if unable to maintain airway with BVM and other measures.
- Immediate transport
- Notify receiving ED of airway status.

INITIAL STABILIZATION/THERAPY

Airway management:

- Anticipate difficult airway
- Intubation required in ~75% (40–100%) of patients. More frequently required in younger patients. Active airway management ensures stable airway and facilitates suctioning.
- Intubation should ideally be performed in the operating room with surgical airway backup.
- Select an ETT 1–2 sizes smaller than usual for age/size.
- Meticulous ETT care and suctioning
- If BVM ventilation needed, use appropriately sized mask with 2-hand seal.
- Supplemental humidified oxygen

ED TREATMENT/PROCEDURES

- Continue monitoring of ventilation and oxygenation.
- IV fluids, bolus, as necessary
- Bronchoscopy if not rapidly deteriorating:
 - Assess need for intubation
 - Therapeutic stripping of membranes
- IV antibiotics to cover typical pathogens:
 - Ceftriaxone and nafcillin or vancomycin
 - Vancomycin or clindamycin for penicillin-allergic patients
 - Consider corticosteroid therapy

MEDICATION

- Ceftriaxone: 50 mg/kg IV, max. 2 g
- Nafcillin: 50 mg/kg IV; max. 2 g
- Ampicillin/sulbactam: 50 mg/kg IV; max. 3 g
- Vancomycin: 15 mg/kg IV; max. 1 g
- Clindamycin: 10 mg/kg IV; max. 1 g
- Racemic epinephrine: 2.25% solution diluted 1:8 with water in doses of 2–4 mL via aerosol
- Dexamethasone: 0.6 mg/kg IV

First Line

Ceftriaxone plus nafcillin

Second Line

Vancomycin or clindamycin:

- Consider if penicillin allergic, and in areas of high prevalence of MRSA



FOLLOW-UP

DISPOSITION

Admission Criteria

All patients with suspected or documented bacterial tracheitis:

- Admit to PICU.
- PICU length of stay varies from 3–9 days.

Discharge Criteria

None

Issues for Referral

Critical care, otolaryngologist, or pulmonologist should be consulted.

FOLLOW-UP RECOMMENDATIONS

Few long-term complications

PEARLS AND PITFALLS

- Consider in patients with croup-like illness who rapidly deteriorate.
- May be more severe in younger patients due to narrower tracheal diameters.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Epiglottitis, pediatric
- Epiglottitis, adult
- Croup



CODES

ICD9

- 464.4 Croup
- 464.11 Acute tracheitis with obstruction
- 464.21 Acute laryngotracheitis with obstruction

ICD10

- J04.11 Acute tracheitis with obstruction
- J05.0 Acute obstructive laryngitis [croup]

BARBITURATES POISONING

Shaun D. Carstairs • David A. Tanen



BASICS

DESCRIPTION

- Class of sedative–hypnotic agents
- Derivatives of barbituric acid
- Mechanism:
 - Enhances activity of γ -aminobutyric acid (GABA)
 - At high levels, directly opens GABA-A associated chloride channel
 - Leads to inhibition of vascular smooth muscle tone
 - May lead to direct myocardial depression

ETIOLOGY

Overdose of barbiturates:

- Intentional or nonintentional



DIAGNOSIS

SIGNS AND SYMPTOMS

- CNS:
 - Lethargy
 - Slurred speech
 - Incoordination
 - Ataxia
 - Coma (can mimic brain death)
 - Loss of reflexes
- Cardiovascular:
 - Hypotension
 - Bradycardia
- Ophthalmologic:
 - Miosis (generally associated with deep coma)
 - Nystagmus
 - Dysconjugate gaze
- Other:
 - Respiratory depression
 - Hypothermia
 - Bullae or “barb blisters”

History

- Determine if there was an intentional overdose:
 - _ Pill bottles at the scene
 - _ History of depression or suicidal ideation
- Determine if there was a medication error:
 - _ What other medications was the patient taking?
 - _ Were there any recent changes in dose?
- Estimate how long the patient may have been unresponsive.

Physical-Exam

- CNS abnormalities:
 - _ Ataxia to coma
- Respiratory depression
- Cardiovascular:
 - _ Bradycardia and hypotension
- Ophthalmologic:
 - _ Miosis
 - _ Nystagmus
 - _ Dysconjugate gaze
- Hypothermia
- Bullae or “barb blisters”

ESSENTIAL WORKUP

- Fingerstick glucose
- Oxygen saturation monitor
- Monitor BP

ALERT

Barbiturate poisoning can mimic brain death:

- Cannot pronounce a patient brain dead until barbiturate poisoning has been ruled out

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Electrolytes, BUN/creatinine, glucose:
 - _ Calculate anion gap
 - _ Assess for renal failure
- Urinalysis:
 - _ For myoglobin
 - _ For crystalluria (Primidone)
- Creatine phosphokinase for evidence of rhabdomyolysis
- Urine toxicology screen
- Obtain serum phenobarbital level (if suspected)

- Acetaminophen and salicylate levels if suspected suicide attempt
- Thyroid function tests

Imaging

- CT scan of head for altered mental status
- CXR for evidence of aspiration

Diagnostic Procedures/Surgery

- Noncontrast head CT
- Lumbar puncture

DIFFERENTIAL DIAGNOSIS

- Sedative–hypnotic poisoning (including γ -hydroxybutyrate [GHB] and its precursors)
- Carbon monoxide poisoning
- CNS infections
- Space-occupying lesions of the head
- Hypoglycemia
- Uremia
- Electrolyte imbalance (i.e., hypermagnesemia)
- Postictal state following seizure
- Hypothyroidism
- Liver failure
- Psychiatric illness



TREATMENT

PRE HOSPITAL

- Moderate to severe poisonings require paramedic transport.
- Intubation is often necessary because of respiratory depression or loss of gag reflex.
- IV access and supplemental oxygen:
 - IV fluid bolus for hypotension

INITIAL STABILIZATION/THERAPY

- ABCs:
 - Administer supplemental oxygen.
 - Severe poisonings usually require endotracheal intubation.
- 0.9% NS:
 - Hypotensive patients require at least 1–2 L IV fluid resuscitation.
 - Pressor support may be necessary for refractory hypotension.
- Activated charcoal effectively binds barbiturates and may decrease systemic

absorption.

ED TREATMENT/PROCEDURES

- Administer 1 dose of activated charcoal:
 - Utility greatest if given within 1 hr of ingestion
 - Ensure patient is awake and alert (or airway protected) prior to administration.
 - Consider “gut dialysis” with repeated dose activated charcoal (without sorbitol) given q2–4h (as long as bowel sounds are present).
- Rewarm patient if hypothermic (see “Hypothermia” chapter).
- Treat hypotension resistant to IV fluid bolus with vasopressors (dopamine, norepinephrine, epinephrine).
- Treat hyperkalemia (from muscle breakdown) with calcium, sodium bicarbonate, insulin and glucose, and/or potassium-binding agents.
- Repeat phenobarbital level in 2–4 hr to determine whether level is increasing.
- Consider hemodialysis if patient has
 - decreased or no renal function
 - prolonged coma
 - serum phenobarbital level > 100 mg/dL
 - refractory hypotension
- There is no role for urinary alkalinization

MEDICATION

First Line

- Activated charcoal: 1 g/kg PO
- Dopamine: 5–10 µg/kg/min titrating to desired effect (to max. of 20 µg/kg/min)
- Norepinephrine: 2–4 µg/min titrating to desired effect (to max. of 10 µg/min)

Second Line

Epinephrine: 0.1 µg/kg/min titrating to desired effect (to max. of 1 µg/kg/min)



FOLLOW-UP

DISPOSITION

Admission Criteria

ICU admission for:

- Coma
- Respiratory depression
- Hypotension
- Hypothermia

- Rhabdomyolysis

Discharge Criteria

Asymptomatic after a minimum of 6 hr of observation with 2 consecutive subtoxic phenobarbital levels before discharge

Issues for Referral

- If intentional overdose, will require psychiatric evaluation
- For nonintentional overdose, referral for adjustment in medications

FOLLOW-UP RECOMMENDATIONS

For nonintentional overdose, may need referral for adjustment in medications or change of medications to agents with a greater therapeutic window.

PEARLS AND PITFALLS

- Hypothermia may be pronounced:
 - Ensure accurate core temperature is measured.
- Check for rhabdomyolysis, since the patient may have been down for a while.
- Barbiturate poisoning can cause prolonged coma:
 - Ensure medication effects have resolved prior to making diagnosis of brain death.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Benzodiazepine, Poisoning
- Coma
- Hypothermia
- Rhabdomyolysis



CODES

ICD9

967.0 Poisoning by barbiturates

ICD10

- T42.3X1A Poisoning by barbiturates, accidental (unintentional), init
- T42.3X2A Poisoning by barbiturates, intentional self-harm, init
- T42.3X4A Poisoning by barbiturates, undetermined, initial encounter

BAROTRAUMA

Catherine M. Visintainer • Nicole L. Lunceford • Peter J. Park

BASICS

DESCRIPTION

Injury resulting from the expansion or contraction of gases in an enclosed space

ETIOLOGY

- Tissue damage results when a gas-filled space does not equalize its pressure with external pressure.
- Boyle's law: At a constant temperature, pressure (P) is inversely related to volume (V):
 - _ $PV = K$ (constant) or $P_1V_1 = P_2V_2$
 - _ As pressure increases/decreases, volume decreases/increases.
- Solid and liquid-filled spaces distribute pressure equally.
- Volume changes are greatest in the few feet nearest the surface.
- Gas-filled cavities in the body are subject to expansion/contraction:
 - _ External objects:
 - Air pockets in dive suit/mask expand and contract.
 - _ Paranasal sinus:
 - Barotrauma of descent
 - Pressure equalization impaired through nasal ostia resulting in negative pressure in sinus cavity
 - Frontal sinus most commonly affected
 - _ External ear:
 - Barotrauma of descent
 - Blockage of external auditory canal results in trapped air leading to a vacuum
 - _ Middle ear:
 - Barotrauma of descent
 - Most common type of barotraumas
 - Seen in 30% of inexperienced divers and 10% of experienced divers
 - Eustachian tube provides sole route of pressure equalization for middle ear.
 - Inadequate clearance via eustachian tube leads to increasingly negative pressure gradient across tympanic membrane (TM).
 - _ Inner ear:
 - Barotrauma of descent
 - Results from rapid development of pressure differential across middle and inner ear (Valsalva, Frenzel maneuvers, rapid descent)

- Increased pressure in inner ear may cause round or oval window to rupture.
- Frequently associated with middle ear barotrauma
- Teeth:
 - Entrapped gas within or around tooth
- GI:
 - Barotrauma of ascent
 - Swallowed air in GI tract expands as external pressure decreases.
- Pulmonary:
 - Barotrauma of ascent
 - Expansion of gas trapped in lungs (closed glottis, bronchospasm) leads to distention of alveoli
 - Can lead to alveolar rupture
 - Most common is pneumomediastinum
 - Potential arterial gas embolism (AGE) (see “Arterial Gas Embolism”)
 - Divers with decreased lung compliance/increased lung volumes at increased risk (chronic obstructive pulmonary disease [COPD], asthma)

DIAGNOSIS

SIGNS AND SYMPTOMS

- Facial:
 - Occlusive dive mask: Conjunctival hemorrhage, facial edema, and swelling
- Extremities:
 - Tight-fitting dive suit: Edema and erythema of the skin at locations of air pockets
- Paranasal sinuses (barosinusitis):
 - Sinus congestion, pain, epistaxis
 - Pain in maxillary teeth
 - Cheek/lip numbness from CN V neuropraxia
- External ear:
 - May result from tight-fitting hood, earplug, or earwax occluding canal
 - Auditory canal mucosa becomes edematous, then hemorrhagic, and ultimately tears
- Middle ear (barotitis media):
 - Begins as clogged sensation
 - Increasingly painful as pressure differential increases across TM
 - Progresses to rupture of TM
 - TM appearance:
 - Progresses from normal appearance to edema to hemorrhage to TM rupture (Teed classification)

- Inner ear:
 - Tinnitus, hearing loss, and vertigo
 - Similar symptoms to inner ear decompression illness (usually less vertigo)
- Teeth (barodontalgia):
 - Severe tooth pain: Possible air trapped in fillings
- GI (aerogastralgia):
 - Excessive belching
 - Flatulence
 - Abdominal distention
- Pulmonary (pulmonary barotrauma [PBT], or pulmonary overpressurization syndrome):
 - Localized pulmonary injury
 - Chest pain, cough, hemoptysis
 - Subcutaneous emphysema
 - Pneumomediastinum
 - Chest pain, neck fullness
 - Pneumothorax
 - Chest pain (pleuritic), dyspnea
 - Delayed symptoms include bull neck appearance, dysphagia, changes in voice character

History

Thorough dive history and timing of symptoms in relation to dive (ascent, descent, delayed)

Physical-Exam

- HEENT for tympanic membrane trauma/rupture
- Chest wall/neck exam for subcutaneous emphysema
- Lung exam for pneumothorax
- Neurologic exam for imbalance/ataxia representing inner ear pathology

ESSENTIAL WORKUP

Clinical diagnosis: Meticulous physical exam (as above) and thorough history should direct any workup

DIAGNOSIS TESTS & INTERPRETATION

Lab

ABG for pulmonary symptoms

Imaging

- Sinus imaging:
 - CT

– Plain films

- CXR for PBT
- Abdominal series (upright, decubitus) for free air from a ruptured viscus

DIFFERENTIAL DIAGNOSIS

- Decompression sickness
- Otitis media
- Otitis externa
- Sinusitis



TREATMENT

PRE HOSPITAL

- For barotrauma of *descent*, unless air-filled cavity has ruptured, no progression of disease on return to normal atmospheric pressure is to be expected.
- If patient requires air evacuation, maintain air cabin pressure at 1 atm or fly below 1,000 feet to avoid aggravating barotraumas.

INITIAL STABILIZATION/THERAPY

Airway, breathing, and circulation management (ABCs):

- 100% oxygen for ill-appearing patients
- Intubation for patients with subcutaneous emphysema of neck
- Immediate needle thoracostomy for evidence of tension pneumothorax

ED TREATMENT/PROCEDURES

- Establish IV access for unstable patients.
- Control bleeding from ear or nose.
- Tube thoracostomy for large pneumothorax
- Decongestants for middle ear or sinus congestion
- Antibiotics with TM or sinus rupture
- Analgesics

MEDICATION

- Amoxicillin: 250–500 mg (peds: 40 mg/kg/24h) PO TID
- Trimethoprim–sulfamethoxazole (Bactrim DS): 1 tablet double-strength (160 mg/800 mg) (peds: 40 mg/200 mg/5 mL, 5 mL/10 kg/dose) PO BID
- Oxymetazoline (Afrin) 0.05%: 2 or 3 drops/sprays per nostril BID for 3 days
- Pseudoephedrine (Sudafed): 60 mg (peds: 6–12 yr, 30 mg; 2–5 yr, 15 mg/dose) PO q4–6h



FOLLOW-UP

DISPOSITION

Admission Criteria

- PBTs
- Inner ear barotrauma with round window rupture or severe vertigo

Discharge Criteria

- Most non-PBT
- ENT follow-up for severe TM or sinus pathology

FOLLOW-UP RECOMMENDATIONS

ENT referral for ruptured TM or inner ear–related signs/symptoms

PEARLS AND PITFALLS

- Watch closely for development of decompression sickness in patients who present with barotraumas.
- Perform careful lung exam for signs of pneumothorax.
- Perform careful history in patients with PBT, any history of neurologic symptoms indicates AGE.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Arterial Gas Embolus
- Decompression Sickness
- Hyperbaric Oxygen Therapy



CODES

ICD9

- 993.2 Other and unspecified effects of high altitude

- 993.3 Caisson disease
- 993.9 Unspecified effect of air pressure

ICD10

- T70.3XXA Caisson disease [decompression sickness], initial encounter
- T70.9XXA Effect of air pressure and water pressure, unspecified, initial encounter
- T70.20XA Unspecified effects of high altitude, initial encounter

BARTHOLIN ABSCESS

Marilyn Althoff • Mark Mandell



BASICS

DESCRIPTION

- The Bartholin glands are located inferiorly on either side of vaginal opening:
 - _ Ducts open on sides of labial vestibule.
- Obstruction of duct produces a usually painless cyst:
 - _ Infection of cyst results in abscess formation.

EPIDEMIOLOGY

Prevalence

Most common in women aged 20–40 yr

ETIOLOGY

- Anaerobic and aerobic microflora normally found in vagina:
 - _ *Bacteroides* species
 - _ *Peptostreptococcus* species
 - _ *Escherichia coli*
 - _ Other gram-negative organisms
- Occasionally *Neisseria gonorrhoeae* and *Chlamydia trachomatis*



DIAGNOSIS

SIGNS AND SYMPTOMS

- Swollen, painful labia
- Tender, fluctuant mass on posterolateral margin of vestibule of vagina
- Warmth, erythema

History

Acute onset:

- Painful, unilateral labial swelling
- Pain with sitting, walking
- Dyspareunia

Physical-Exam

- Bartholin abscess:
 - _ Tender, fluctuant, unilateral labial mass
 - _ Surrounding erythema, warmth

- Fever uncommon
- Bartholin cyst:
 - Painless, unilateral labial mass

ESSENTIAL WORKUP

Diagnosis based on findings of tender, localized, fluctuant mass in region of Bartholin gland

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Culture material from abscess for gonorrhea and chlamydia.
- Culture cervix for gonorrhea and chlamydia.

Imaging

Generally not indicated

DIFFERENTIAL DIAGNOSIS

- Bartholin cyst
- Carcinoma of Bartholin gland (rare)
- Perineal hernia



TREATMENT

ED TREATMENT/PROCEDURES

- Prompt incision and drainage using local anesthesia with patient in lithotomy position
- Narcotic analgesia for patient comfort
- Alternative approaches include:
 - Simple incision and drainage
 - Word catheter method
 - Marsupialization
- Simple incision and drainage:
 - After local anesthesia, palpate abscess between thumb and index fingers.
 - Spread vulva apart and make stab incision on *mucosal* surface of abscess, parallel to hymenal ring.
 - When incising abscess, 2 tissue layers must be penetrated:
 - 1st the labial mucosa
 - Then abscess wall
 - Free flow of pus indicates penetration of abscess wall.
 - Pack wound with gauze.
 - Follow-up in 24–48 hr for removal of packing.

- Start sitz baths after 24 hr.
- Consider referral for marsupialization to avoid recurrence.
- Word catheter method:
 - Use small, inflatable, bulb-tipped Word catheter to treat abscess.
 - May avoid recurrence and make marsupialization unnecessary
 - Stab wound is made as with simple incision and drainage:
 - It should be just large enough to easily admit catheter so that balloon does not fall out after inflation.
 - After inserting bulb tip of catheter, inflate balloon by injecting 2–4 mL water using 25G needle (to minimize size of puncture):
 - Overinflation may cause patient discomfort
 - Remedied by withdrawing some water from balloon
 - Sitz baths may be started after 24 hr.
 - Follow-up in 2–4 days.
 - Leave catheter in place for 6–8 wk until epithelialization is complete; after device is removed, gland resumes normal function.
 - Common for catheter to fall out prematurely:
 - If this occurs, catheter may be reinserted or abscess can heal as with simple incision and drainage.
- Marsupialization:
 - Procedure allows for a permanent fistula by suturing wound edges of abscess cavity to edges of labial mucosa:
 - Technically more challenging in ED and better reserved for specialist.
 - Excise an ellipse of labial mucosa that overlays cyst cavity.
 - Incision and drainage of abscess
 - Evert edges of abscess and suture them to labial epithelium using absorbable suture:
 - Opening will shrink but remain patent.
 - Packing is not needed.
 - Start sitz baths in 24–48 hr.
 - Follow-up within 1 wk.
- Antibiotics not necessary after incision and drainage:
 - If mild cellulitis present or patient immunocompromised, broad-spectrum coverage may be started.
 - If sexually transmitted disease (STD) suspected, treat with antibiotics.

MEDICATION

First Line

Broad-spectrum coverage:

- Amoxicillin/clavulanic acid: 500–875 mg PO BID for 5 days with metronidazole 500 mg PO q8h for 5 days
- Ciprofloxacin: 500 mg PO BID for 5 days with metronidazole 500 mg PO q8h for 5

days

Second Line

Treat for STD if indicated



FOLLOW-UP

DISPOSITION

Admission Criteria

- Sepsis
- Significant cellulitis
- Evidence of necrotizing infection

Discharge Criteria

Well-appearing patients may be discharged with designated follow-up plan.

Issues for Referral

Patients should have gynecologic follow-up:

- Follow-up in 24–48 hr for removal of packing.
- Follow-up in 2–4 days after insertion of Word catheter.

FOLLOW-UP RECOMMENDATIONS

Continue sitz baths for at least 72 hr.

PEARLS AND PITFALLS

- Do not mistake a nontender Bartholin cyst, which does not require immediate treatment, for an inflamed abscess.
- Consider malignancy as an alternative cause of a mass, particularly in women > 40 yr.
- Incision should be on mucosal surface of abscess.

ADDITIONAL READING

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- Patil S, Sultan AH, Thakar R. Bartholin's cysts and abscesses. *J Obstet Gynaecol.* 2007;27:241–245.
- Pundir J, Auld BJ. A review of the management of diseases of Bartholin's gland. *J Obstet Gynaecol.* 2008;28:161–165.

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See Also (Topic, Algorithm, Electronic Media Element)

- Treatment of Chlamydia
- Treatment of Gonococcal Disease



CODES

ICD9

- 098.0 Gonococcal infection (acute) of lower genitourinary tract
- 616.3 Abscess of Bartholin's gland

ICD10

- A54.02 Gonococcal vulvovaginitis, unspecified
- N75.1 Abscess of Bartholin's gland

BATH SALTS – SYNTHETIC CATHINONES POISONING

Jami L. Hickey • Jenny J. Lu



BASICS

DESCRIPTION

“Bath salts”:

- General term for “designer drugs” containing synthetic cathinones:
 - 3,4 methylenedioxypropylamphetamine (MDPV) is most common in US
 - Also mephedrone, methylone, and many others
- Sold under numerous names including
 - Aura, Bliss, Bolivian Bath, Cloud 9, Ivory Snow, Ivory Wave, Vanilla Sky, White Dove, White Rush
 - Labeled “not for human consumption” to evade regulatory control
 - Falsely marketed as plant food, insect repellents, “bath salts”
- Substances may be powders, tablets, or crystals:
 - Ranging in color from white, yellow, brown, or gray
- May be ingested, snorted, smoked, injected
- Highly addictive CNS stimulant, often with hallucinogenic properties:
 - Many effects similar to cocaine, methamphetamine, or ecstasy
 - Severe delirium, psychosis, violence, multiorgan failure, DIC, myocardial infarction, stroke, and deaths have been reported

EPIDEMIOLOGY

Incidence and Prevalence Estimates

- 1st use in US reported in 2010
 - MDPV and mephedrone noted in Europe since 2004
- Called “America’s new drug problem” in 2011
 - Thousands of cases reported to poison control centers nationwide
- Immediate temporary classification (Fall 2011) as a DEA schedule I controlled substance
- Still available at retail shops or through the internet

ETIOLOGY

- MDPV is structurally similar to cathinone, an alkaloid derived from the khat plant (chewed socially and abused for centuries in East Africa and Arabian Peninsula)
- Drug chemical formulas change regularly to evade detection, compound identification, and classification as “illegal”
- Principal toxicity derives from effects on dopamine, norepinephrine, and serotonin receptors

- Effects from potential adulterants and contaminants in the drugs remain unknown



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Often unobtainable or incomplete
 - Friends, family, bystanders may provide information about patient behavior
 - High index of suspicion when signs and symptoms are present with no satisfactory alternative explanation

Physical-Exam

- No pathognomonic signs or symptoms
- Sympathomimetic toxidrome:
 - Hyperthermia
 - Tachycardia
 - Hypertension
 - Dysrhythmias
 - Diaphoresis
 - Mydriasis
 - Rhabdomyolysis
 - Respiratory distress
 - Hyperreflexia
 - Seizures
- Mental status and behavioral effects:
 - Psychomotor agitation
 - Hallucinations
 - Physical aggression
 - Psychosis
 - Paranoia
 - Excited delirium
 - Suicidal ideation
 - Panic attacks
 - Insomnia

ESSENTIAL WORKUP

Primarily focused on assessing severity of intoxication and excluding other medical or toxicologic causes of altered mental status

DIAGNOSIS TESTS & INTERPRETATION

Lab/Imaging

- No tests in current routine ED use to detect MDPV:
 - Samples of ingested substance, serum, or urine can be sent to reference labs
 - Results not available in ED setting
- Labs:
 - Urine and serum toxicology screens may detect coingestants
 - CBC, BMP, liver profile, PT/PTT
 - Lactate, pH
 - Total CK
 - Blood/urine culture if infectious process suspected
- Imaging:
 - Consider CT head if appropriate (e.g., trauma)
- ECG:
 - Evaluate QRS/QT intervals, dysrhythmias

DIFFERENTIAL DIAGNOSIS

- Other intoxications:
 - Cocaine
 - Amphetamines
 - Anticholinergic agents
 - Ecstasy
 - Ethanol
- Acute psychosis
- Serotonin syndrome
- Delirium from infectious or metabolic process



TREATMENT

PRE HOSPITAL

- Stabilize airway
- Vital signs
- IV access
- Fingertick glucose
- Oxygen administration if needed

INITIAL STABILIZATION/THERAPY

- Stabilize airway, establish IV, vital signs, cardiac monitoring
- Benzodiazepines are 1st-line medications
- Judicious use of physical restraints, if necessary, for prevention of harm to patient and staff

ED TREATMENT/PROCEDURES

- Supportive care is mainstay of treatment with continuous cardiac and temperature

monitoring:

- Fluid resuscitation
- Oxygen
- Benzodiazepines are 1st-line medications
- Aggressive cooling measures for hyperthermia:
 - Ice packs, cool mists, fans, cooling blankets, cool intravenous fluids
- Severe symptoms may necessitate intubation in rare cases:
 - Propofol for sedation
- Caution with antipsychotic administration which may lower seizure threshold, cause extrapyramidal symptoms, and dysrhythmias
- Poison Control Center/toxicology guidance (1-800-222-1222)

MEDICATION

- Ativan 2–4 mg increments IM or IV
- Valium 10–30 mg increments IM or IV



FOLLOW-UP

DISPOSITION

Admission Criteria

- All patients with symptoms should be admitted for monitoring
- Severe symptoms including uncontrollable hypertension, altered mental status, cardiovascular instability, and hyperthermia require ICU monitoring

Discharge Criteria

Only asymptomatic patients who remain asymptomatic after an adequate observation period (half-life of MDPV estimated at 1.88 hr with 6–8 hr duration of action) may be discharged; exact timing will vary on each case (consult your poison control center)

FOLLOW-UP RECOMMENDATIONS

Follow up with primary care after discharge

PEARLS AND PITFALLS

- A sympathomimetic toxidrome with delirium/psychosis should arouse suspicion for “bath salts” intoxication
- Severe hyperthermia should be aggressively controlled
- Focused supportive care is the mainstay of treatment, with benzodiazepines as initial therapy

ADDITIONAL READING

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- Spiller HA, Ryan ML, Weston RG, et al. Clinical experience with and analytical confirmation of “bath salts” and “legal highs” (synthetic cathinones) in the United States. *Clin Toxicol (Phila)*. 2011;49:499–505.



CODES

ICD9

969.6 Poisoning by psychodysleptics (hallucinogens)

ICD10

- T43.8X1A Poisoning by oth psychotropic drugs, accidental, init
- T43.8X4A Poisoning by oth psychotropic drugs, undetermined, init

BELL'S PALSY

Robert F. McCormack • Richard S. Krause



BASICS

DESCRIPTION

- Acute, *idiopathic* peripheral CN VII (facial nerve) palsy
- Complete recovery in 85% of cases without treatment
- Degree of deficit correlates with prognosis:
 - Complete lesions have poorest prognosis
 - Partial lesions often have excellent results
- Recovery usually begins within 2 wk (often taste returns 1st) and is complete by 2–3 mo:
 - Advanced age and slow recovery are poor prognosticators
- Affects men and women equally
- Age predominance between the 3rd and 5th decade (may occur at any age)
- Diabetes and pregnancy increase risk
- Incidence 15–40 per 100,000 per year
- The most common cause of facial nerve palsy in children

ETIOLOGY

- Idiopathic by definition, but viral cause (particularly herpes simplex) suspected
- Lyme disease, infectious mononucleosis (Epstein–Barr virus [EBV] infection), varicella-zoster infections, and others may cause peripheral 7th nerve palsy
- Mechanism: Edema and nerve degeneration within stylomastoid foramen
- Innervation to each side of forehead is from both motor cortices:
 - Unilateral cortical processes do *not* completely disrupt motor activity of forehead
- Only peripheral or brainstem lesion can interrupt motor function of just 1 side of forehead



DIAGNOSIS

SIGNS AND SYMPTOMS

History

Sudden onset of unilateral facial droop, incomplete eyelid closure, and loss of forehead muscle tone:

- Maximal deficit by 5 days in almost all cases (2 days in 50%)
- Tearing (68%) or dryness of eye (16%) and less frequent blinking on affected side
- Subjective “numbness” of the affected side

- Abnormal taste, drooling
- Hyperacusis (sensitivity to loud sounds)
- Fullness or pain behind mastoid
- Viral prodrome frequently reported

Physical-Exam

- Unilateral facial palsy including the forehead
- If forehead muscle tone is *not* lost, a central lesion is strongly implied (i.e., this is *not* Bell's palsy)
- Motor weakness isolated to 7th nerve distribution:
 - _ Involves both upper and lower face
- An otherwise normal neurologic exam including all cranial nerves and extremity motor function
- The Bell phenomenon (upward rolling of the eye on attempted lid closure) may be seen

ESSENTIAL WORKUP

Diagnosis is clinical and based on history and physical exam

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Not helpful in diagnosis of Bell's palsy
- Lyme titers are useful when Lyme disease is suspected or in endemic area
- Tests for mononucleosis (CBC, monospot) if EBV infection suspected

Imaging

- Not helpful in diagnosis of Bell's palsy unless a parotid tumor, mastoiditis, etc. are suspected

DIFFERENTIAL DIAGNOSIS

- Brainstem events (mass, bleed, infarct) affecting CN VII almost always involve CN VI (abnormal EOM) and may affect long motor tracts:
 - _ There have been (*rare*) case reports of *isolated* CN VII palsy from brainstem disease.
- Lyme disease: History of tick bite, erythema migrans rash, or endemic area
- Zoster (Ramsay Hunt syndrome): Look for herpetic vesicles, inquire about tinnitus or vertigo
- Infectious mononucleosis: Look for pharyngitis, posterior cervical adenopathy
- Tumors: Parotid, bone, or metastatic masses, acoustic neuroma (deafness)
- Trauma: Skull fracture or penetrating facial injury may damage CN VII
- Middle ear or mastoid surgery or infection, cholesteatoma
- Meningeal infection

- Guillain–Barré syndrome: Other neurologic deficits are present (e.g., ascending motor weakness or diminished deep tendon reflexes [DTRs])
- Basilar artery aneurysm; other CN deficits should be present
- Bilateral peripheral CN VII palsy: Consider multiple sclerosis, sarcoidosis, leukemia, and Guillain–Barré. Idiopathic (Bell's) palsy may be bilateral in rare cases
- Early HIV infection
- Bell's palsy may reoccur; treatment is unchanged



TREATMENT

PRE HOSPITAL

None

INITIAL STABILIZATION/THERAPY

Patients with an isolated peripheral CN VII palsy are stable.

ED TREATMENT/PROCEDURES

- Corneal damage may result from incomplete eyelid closure:
 - Lubricating and hydrating ophthalmic preparations are often needed
 - Eye patching at night
- Oral steroids may hasten recovery if started within 1 wk of onset (preferably w/in 72 hr):
 - Complications of therapy are rare
- Antiviral therapy (acyclovir or valacyclovir) with steroids may be effective in improving functional nerve recovery:
 - Initiate within 72 hr of symptom onset
 - No clear proven benefit
 - May be indicated for severe palsy
- Suspected Lyme disease should be treated with doxycycline or amoxicillin
- Surgical decompression may be indicated for complete lesions that do not improve; this is controversial

MEDICATION

First Line

- Lacri-Lube or artificial tears: At bedtime and PRN; dryness/irritation in affected eye (or equivalent)
- Prednisone: 30–40 mg PO BID for 7 days, (peds: 2 mg/kg/d PO [max. 60 mg])

Second Line

Valacyclovir 1 g PO TID for 7 days (peds: 20 mg/kg TID) may be useful in severe cases.



FOLLOW-UP

DISPOSITION

Admission Criteria

Isolated peripheral CN VII palsy does not require admission.

Discharge Criteria

Isolated peripheral CN VII palsy may be treated on outpatient basis.

FOLLOW-UP RECOMMENDATIONS

Follow-up should be within 1 wk.

PEARLS AND PITFALLS

- Motor weakness isolated to 7th nerve distribution:
 - _ Involves both upper and lower face
 - _ If tone is NOT lost on the forehead, it is *not* Bell's palsy.
- Otherwise normal neurologic exam including all cranial nerves and extremity motor function
- Protect the eye
- Steroids beneficial, antivirals controversial

ADDITIONAL READING

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CODES

ICD9

351.0 Bell's palsy

ICD10

G51.0 Bell's palsy

BENZODIAZEPINE POISONING

Michael E. Nelson

BASICS

DESCRIPTION

- Potentiates the activity of γ -aminobutyric acid (GABA), the major inhibitory neurotransmitter, by binding to its own specific site
- Also facilitates GABA binding to its site
- Results in chloride influx, membrane hyperpolarization, and inhibition of cellular excitation:
 - Benzodiazepines (BZDs) increase the frequency of chloride channel opening.
 - Depresses spinal reflexes and reticular activating system
- Rapidly absorbed from GI tract:
 - Highly protein bound
 - Large V_d
 - Hepatic metabolism
 - Duration of action is inversely proportional to lipophilicity with highly lipophilic drugs penetrating the CNS more rapidly.
 - Duration of lorazepam > diazepam > midazolam.
 - Synergistic with other sedative–hypnotic medications (e.g., ethanol, barbiturates, propofol)

DIAGNOSIS

SIGNS AND SYMPTOMS

- CNS:
 - Sedation/drowsiness
 - Slurred speech
 - Midrange or small pupils
 - Delirium
 - Coma
- Neuromuscular:
 - Incoordination
 - Slowed voluntary movements
 - Ataxia
 - Hypotension
 - Hyporeflexia/areflexia
- Cardiovascular:
 - Mild depression
 - Rarely lethal if ingested alone

- Respiratory:
 - Mild depression but less than barbiturates
 - Short acting and IV have higher depression
- GI:
 - Nausea, vomiting, diarrhea
- Other:
 - Hypothermia
 - Complications may include cerebral hypoxia, rhabdomyolysis, pressure-induced neuropathies.
 - No long-term organ toxicity

Pediatric Considerations

Rarely may cause paradoxical restlessness and agitation

ESSENTIAL WORKUP

Diagnosis based on:

- History of ingestion or recent injection
- Clinical findings associated with CNS depression
- No response to naloxone

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Pulse oximetry
- Electrolytes, BUN, creatinine, serum glucose
- Thyroid studies
- Urinalysis (UA) for myoglobin if coma present or down for prolonged period of time
- ABG
- Qualitative urine screen:
 - May confirm exposure, but does not indicate or measure intoxication or correlate clinical state.
 - False-negative test results reported
 - Qualitative immunoassays detect only BZDs that are metabolized to oxazepam.
 - BZDs that do not produce this metabolite (clonazepam, lorazepam, midazolam, alprazolam) are not detected on qualitative screen.
 - Serum levels not acutely practical
 - Clinical signs and symptoms more important than theoretic LD₅₀ or serum levels
- Alcohol(s) level
- Barbiturate level (e.g., Phenobarbital)

- Acetaminophen and salicylate levels
- Pregnancy test

Imaging

- EKG
- CXR for aspiration pneumonia
- Consider CT head

Diagnostic Procedures/Surgery

Core body temperature

DIFFERENTIAL DIAGNOSIS

- Drugs and toxins causing decreased level of consciousness:
 - Hypoglycemics
 - Other sedative–hypnotics (barbiturates, chloral hydrate, GHB, bromides)
 - Antidepressant–antipsychotics
 - Narcotics
 - Anticonvulsants
 - Carbon monoxide/cyanide
 - Alcohols
- Nontoxic medical conditions:
 - Hypoxemia
 - Hypothermia
 - Head trauma (intracranial bleeding)
 - Infection (meningitis or encephalitis)
 - Electrolyte and metabolic abnormalities



TREATMENT

PRE HOSPITAL

- Attention to airway and breathing
- Cardiac monitor
- IV access
- Rapid glucose determination
- Obtain pill bottles/pills in suspected overdose

INITIAL STABILIZATION/THERAPY

- ABCs:
 - Secure airway and assist ventilation with supplemental oxygen to prevent hypoxemia and shock.
 - IV access with 0.9% NS
 - Cardiac monitor

- Administer naloxone, thiamine, and dextrose if altered mental status/comatose.

ED TREATMENT/PROCEDURES

- Consider gastric lavage when presenting within 1 hr of life-threatening ingestion with protected airway but should be done cautiously
- Activated charcoal (AC) PO or via nasogastric tube (NGT) if airway protected
- No role for diuresis, dialysis, or charcoal hemoperfusion
- Flumazenil (FZ):
 - _ Competitive BZD-receptor inhibitor
 - _ Rapidly reverses BZD-induced coma
 - _ Onset within 1–2 min; peak at 6–10 min; duration 1–2 hr (repeated dosing may be indicated)
 - _ Efficacy dependent on dose of BZD being antagonized and dose of FZ used
 - _ Do not administer empirically as part of “coma cocktail” or unknown ingestion
 - _ May help avert need of airway intubation but has not consistently reversed respiratory depression
 - _ May be beneficial in shortening hospital stay or as diagnostic maneuver
 - _ Indications include isolated BZD overdose in nonhabituated user with CNS depression, normal EKG, normal vital signs, and normal neurologic exam.
 - _ Most useful to reverse iatrogenic poisoning (conscious sedation)
 - _ Contraindications include:
 - Coingestions that might lower seizure threshold (tricyclic antidepressants [TCAs])
 - Seizure history or activity
 - Allergy
 - Neuromuscular blockade
 - Do not use if hypotension, hypoxia, dysrhythmias, or increased intracranial pressure is present.
 - May precipitate withdrawal state including seizures, for which BZDs can no longer be used to treat

MEDICATION

- AC: 1 g/kg PO/NG (ideal is 10:1 ratio of AC:dose of drug)
- Dextrose: D₅₀W 1 ampule: 50 mL or 25 g (peds: D₂₅W 2–4 mL/kg) IV if hypoglycemic
- FZ (Romazicon):
 - _ Initial: 0.2 mg IV over 30 sec (adult)
 - _ If no response: 0.3 mg IV after 30 sec
 - _ If still no response: 0.5 mg IV and repeat q1min if needed, to max. dose of 3 mg
 - _ Continuous infusion at 0.1–1 mg/h if multiple repeated doses required to maintain response. Continuous infusion not FDA approved

- Pediatric dosing: Titrate to max. cumulative dose of 0.05 mg/kg/d. Continuous infusion at 0.005–0.01 mg/kg/h has been used.
- Only use in selected patients (see above) as may precipitate seizures or dysrhythmias
- Monitor after use for re sedation (occurs between 20 and 120 min after use)
- Naloxone (Narcan): 0.4–2 mg (peds: 0.1 mg/kg) IV or IV initial dose
- Thiamine (vitamin B₁): 100 mg IV/IM



FOLLOW-UP

DISPOSITION

Admission Criteria

- Persistent or profound CNS depression
- Cardiovascular or respiratory compromise
- Coingestants with potential delayed toxicity

Discharge Criteria

- Can discharge after 4-hr observation period if no signs or symptoms of BZD poisoning.
- If FZ administered, observe for additional 2–4 hr for recurrent sedation.

Issues for Referral

Psychiatry consultation for intentional overdoses.

FOLLOW-UP RECOMMENDATIONS

Habituated patients may experience BZD withdrawal after cessation:

- Autonomic instability, tremor, paresthesias, seizures

PEARLS AND PITFALLS

IV formulations of certain BZDs (e.g., lorazepam) may contain propylene glycol diluent that can produce elevated osmolar gap and anion gap metabolic acidosis.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Barbiturate Poisoning

The author would like to provide special thanks to the author of the prior edition, Matthew Valento.



CODES

ICD9

969.4 Poisoning by benzodiazepine-based tranquilizers

ICD10

- T42.4X1A Poisoning by benzodiazepines, accidental, init
- T42.4X2A Poisoning by benzodiazepines, intentional self-harm, init
- T42.4X4A Poisoning by benzodiazepines, undetermined, init encntr

BETA-BLOCKER POISONING

Christopher S. Lim • Steven E. Aks



BASICS

DESCRIPTION

Normal Physiology

- Cardiovascular: β_1 -receptors:
 - ATP converted to cAMP by adenylyl cyclase with stimulation of β -receptors.
 - cAMP activates protein kinase, which phosphorylates proteins of the sarcoplasmic reticulum.
 - Sarcoplasmic reticulum releases calcium.
 - Excitation–contraction coupling occurs.
- Effects of β -blockers:
 - Cardiovascular:
 - Decreased excitation/contraction
 - Membrane stabilizing activity
 - Sodium channel blockade causes a prolongation of the QRS complex (with some agents).
 - Prolongation of QTc interval leading to ventricular dysrhythmias (with some agents)
 - Intrinsic sympathomimetic activity
 - Partial agonist properties (with some agents)
 - Neurologic:
 - CNS effects with the lipophilic agents (propranolol, metoprolol, labetalol)



DIAGNOSIS

SIGNS AND SYMPTOMS

- Cardiovascular:
 - Hypotension
 - Bradycardia
 - Cardiac conduction delays
 - Heart block
 - Heart failure
 - Electrical mechanical dissociation
 - Loss of β -selectivity in overdose settings
- Neurologic:
 - Coma

- Seizures
- Pulmonary:
 - Bronchospasm
 - Pulmonary edema
- Metabolic:
 - Hypoglycemia

History

- Inquire about risk of medication error.
- Inquire about risk of suicidal ideation with intent.
- Inquire about possible exposure to medications with a pediatric patient.

Physical-Exam

- Hypotension
- Bradycardia
- Dysrhythmias

ESSENTIAL WORKUP

- With unknown ingestion: Suspect β -blocker poisoning with bradycardia/hypotension.
- ECG:
 - Conduction delays
 - 1st-, 2nd-, or 3rd-degree heart block
 - Bradycardia

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC
- Electrolytes, BUN, creatinine, glucose
- Toxicology screen if coingestants suspected

DIFFERENTIAL DIAGNOSIS

- Calcium-channel blocker toxicity
- Clonidine toxicity
- Digitalis toxicity
- Acute myocardial infarction with heart block



TREATMENT

PRE HOSPITAL

- Transport pills and pill bottles when overdose suspected.

INITIAL STABILIZATION/THERAPY

- ABCs:
 - Airway protection as indicated by mental status
 - Supplemental oxygen as needed
 - 0.9% NS IV access
 - Close hemodynamic monitoring
- Naloxone and thiamine if altered mental status
- Bedside glucose determination, treat hypoglycemia with D₅₀W
- Treat prolonged seizures with benzodiazepines

ED TREATMENT/PROCEDURES

Goals

- Heart rate > 60 beats per minute
- Systolic BP > 90 mm Hg
- Adequate urine output
- Improving level of consciousness

GI Decontamination

- Syrup of ipecac: Contraindicated in the prehospital and ED setting.
- Consider lavage with Ewald tube if ingestion within 1 hr:
 - Propranolol may cause esophageal spasm producing difficulty with passage and removal of gastric lavage tube.
- Activated charcoal helpful especially in the presence of coingestants.

Bradycardia/Hypotension

- Atropine:
 - Initial agent
 - Low success rate
- Glucagon:
 - Initial drug of choice for β -blocker-induced hemodynamic instability
 - Administer if atropine does not increase heart rate.
 - Promotes cAMP production through a receptor site other than the β -receptor
 - May cause nausea and vomiting
 - Mix with NS or D₅W
- IV fluids:
 - Administer cautiously in the hypotensive patient
 - Swan-Ganz catheter or central venous pressure (CVP) monitoring to help follow volume status
- Amrinone:
 - Selective phosphodiesterase inhibitor
 - Indirectly increases cAMP leading to increased inotropy

- Use in conjunction with glucagon to treat symptomatic sustained bradycardia.
- Vasopressor agents:
 - Initiate when symptomatic hypotension/bradycardia persists after atropine/glucagon.
 - Use invasive monitoring to help guide therapy.
 - Utility may be limited owing to β -blockade:
 - Higher doses may be required.
 - Isoproterenol (nonselective β -agonist):
 - Titrate for BP and heart rate.
 - Epinephrine (potent α - and β -receptor agonist):
 - BP increases as a result of direct myocardial stimulation, increase in heart rate, and vasoconstriction.
 - Use if no BP response with isoproterenol
 - High-dose dopamine
- Sodium bicarbonate:
 - In theory, this is used if there is evidence of prolongation of QRS > 100 ms owing to some of the β -blockers also causing sodium channel blockade leading to a prolonged QRS.
 - Not routinely administered for all β -blocker toxicities
- Electrical pacing: When other treatment options have failed

Experimental Treatment Options

- Consult with local poison center
- High-dose insulin:
 - Promotes more efficient myocardial metabolism
 - Hypoglycemia commonly seen in β -blocker overdose, will require frequent monitoring of glucose concentration
 - Perform in consultation with local poison center
- IV fat emulsion therapy (20% intralipid)

Enhanced Elimination

- Hemodialysis helpful with water-soluble β -blocking agents:
 - Nadolol
 - Atenolol
 - Sotalol
- IV fat emulsion (20% Intralipid):
 - Potential treatment in the future

MEDICATION

- Activated charcoal: 1 g/kg PO
- Amrinone: Loading dose 0.75 mg/kg; maintenance drip 2–20 μ g/kg/min; titrate for effect

- Atropine: 0.5 mg (peds: 0.02 mg/kg) IV; repeat 0.5–1 mg IV (peds: 0.04 mg/kg)
- Dopamine: 2–20 µg/kg/min IV
- Dextrose: D₅₀W 1 ampule (50 mL or 25 g; peds: D₂₅W 2–4 mL/kg) IV
- Epinephrine: 2 µg/min (peds: 0.01 mg/kg [0.1 mL/kg 1:10,000]); titrate to effect
- Glucagon: 3–5 mg IV over 1–2 min (peds: 0.03–0.1 mg/kg) bolus followed by 70 µg/kg/h infusion
- Insulin (regular insulin): 1 IU/kg bolus IV followed by 0.5–1 IU/kg/h titrated to clinical response (be sure to supplement with dextrose)
- Isoproterenol: 5 µg/min IV and titrate for heart rate effect
- Norepinephrine: Start 2–4 µg/min IV, titrate up to 1–2 µg/kg/min IV
- Sodium bicarbonate: 1 mEq/kg IVP

First Line

- IV fluids
- Glucagon
- Vasopressor agents

Second Line

- Sodium bicarbonate
- Hemodialysis



FOLLOW-UP

DISPOSITION

Admission Criteria

- ICU admission for decreased level of consciousness or hemodynamic instability (bradycardia, conduction delays, hypotension)
- Observation and monitoring for 24 hr for long-acting or sustained-release preparations owing to the potential delay in symptoms

Discharge Criteria

Asymptomatic 8–10 hr after ingestion of short- or immediate-release preparation

FOLLOW-UP RECOMMENDATIONS

- Psychiatric evaluation for all suicidal patients
- Poison prevention guidance for parents of pediatric accidental ingestion

PEARLS AND PITFALLS

- Consider β-blocker toxicity in patients who present with hypotension and bradycardia.

- Wide complex QRS dysrhythmias should be treated with sodium bicarbonate.

ADDITIONAL READING

- Harvey MG, Cave GR. Intralipid infusion ameliorates propranolol-induced hypotension in rabbits. *J Med Toxicol.* 2008;4:71–76.
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- Shepherd G. Treatment of poisoning caused by β -adrenergic and calcium-channel blockers. *Am J Health Sys Pharm.* 2006;63:1828–1835.



See Also (Topic, Algorithm, Electronic Media Element)

Calcium Channel Blocker, Poisoning



CODES

ICD9

971.3 Poisoning by sympatholytics [antiadrenergics]

ICD10

- T44.7X1A Poisoning by beta-adrenocpt antagonists, accidental, init
- T44.7X4A Poisoning by beta-adrenocpt antagonists, undetermined, init
- T44.7X5A Adverse effect of beta-adrenoreceptor antagonists, initial encounter

BIOLOGIC WEAPONS

Brigham R. Temple

BASICS

DESCRIPTION

- Defined as naturally occurring organisms or toxins that are purified and prepared for mass dissemination with intent of causing mass morbidity, mortality, and social disruption.
- Organisms include bacteria, viruses, and fungi.
- Over 400 potential or actualized etiologic agents capable of being used as biologic weapon:
 - Characterized by their relatively low cost compared with other weapons of mass destruction (WMD), high potency, and their ability to be delivered in a stealthy manner
 - Stealth quality of biologic weapons comes from organism's natural incubation period.
- Easy to conceal and difficult to detect:
 - Agents often invisible to naked eye, odorless, and tasteless
- Patients typically present to various health care facilities with host of common complaints, adding to delay in recognition of covert release of biologic weapon.
- Victims of biologic warfare agents are exposed either via direct cutaneous contact with agent, respiratory inhalation of aerosolized agent, or via GI tract after poisoning of food or water source.

ETIOLOGY

- Bacteria:
 - Anthrax: *Bacillus anthracis*
 - Plague: *Yersinia pestis*
 - Cholera: Infection from *Vibrio cholerae*:
 - Presents with severe GI symptoms and rapidly leads to profound dehydration
 - Tularemia: *Francisella tularensis*
 - Brucellosis: Organism in the *Brucella* genus
 - Q fever: *Coxiella burnetii*
- Viruses:
 - Smallpox: Variola virus
 - Viral encephalitides: Members of *Alphavirus* genus (Venezuelan equine encephalitis, Eastern equine encephalitis, and Western equine encephalitis)
 - Viral hemorrhagic fevers: From 4 families of viruses, includes illnesses such as Ebola, Marburg, Lassa, and dengue fever

- Toxins:
 - Ricin
 - Staphylococcal enterotoxin B
 - Botulinum toxin
 - Mycotoxins

DIAGNOSIS

SIGNS AND SYMPTOMS

- Health care providers need to be alert to detect illness patterns and diagnostic clues that indicate biologic weapon release.
- Indications of intentional release of agent include:
 - Geographic clustering of illnesses with individuals who live, work, or attended event in close proximity (if multiple people who work in same office develop pneumonia, it could potentially represent respiratory pathogen release)
 - Unusual age distribution for common illness (chickenpox-like illness among adult patients could represent smallpox release)
 - ≥ 2 patients presenting with similar unexplained illnesses (2 patients presenting with flaccid paralysis could represent botulinum toxin release)
 - Single case of illness caused by uncommon agent (smallpox, inhalational anthrax)
 - High volume of patients with similar presentation of symptoms associated with escalating morbidity and mortality

Anthrax

- Inhalational anthrax:
 - Fever
 - Chills
 - Fatigue, malaise, lethargy
 - Cough, usually dry or minimally productive
 - Nausea or vomiting
 - Dyspnea
 - Diaphoresis
 - Chest pain
 - Myalgias
 - Tachycardia
 - Fever
 - Meningeal signs
- Cutaneous anthrax:
 - Skin lesion:
 - Painless pruritic papule

- Turning into vesicle that ruptures forming necrotic ulcer
- Black eschar
- Surrounding gelatinous nonpitting edema

Plague

- Abrupt onset
- Fever, chills
- Cough, hemoptysis, dyspnea
- Headache
- Vomiting
- Swollen tender lymph nodes (buboes)
- Skin lesions at site of inoculation (i.e., flea bite)
- Confusion
- Abdominal pain
- Oliguria
- Obtundation
- Extensive ecchymosis
- Acral gangrene (digits, nose, penis)

Tularemia

- See “Tularemia” chapter.
- Typhoidal:
 - Most likely form of disease when weaponized and delivered by aerosol
 - Fever, headache, malaise
 - Nonproductive cough
 - 35% mortality if untreated

Q fever

- Incubation period 10–40 days
- Flu-like symptoms and pleuritic chest pain for 2–10 days
- CXR shows patchy infiltrates
- Definitively diagnosed serologically
- Mortality:
 - <1% even if untreated

Brucellosis

- Incubation period 3–60 days
- Flu-like symptoms and neuropsychiatric symptoms (headache, depression, fatigue, and irritability)
- Focal infection of joints and GU tract may cause localized pain, particularly back pain.
- Diagnosis by combination of serologic testing and cultures of blood or bodily

fluids.

- Mortality: < 2%

Smallpox

- Incubation period 7–17 days (average is 12 days)
- Flu-like symptoms (fever, fatigue, myalgias, headache) for ~ 2–3 days followed by characteristic rash:
 - Progresses from macules to papules to pustular lesions and crusted lesions
 - Starts on face and extremities (including palms/soles) and spreads to trunk in 1 wk
 - Scabs over in 1–2 wk
- Mortality:
 - 30% if untreated

Hemorrhagic Fevers

- See “Hemorrhagic Fever” chapter
- Incubation period 1–3 wk
- Starts as flu-like syndrome with fever, malaise, myalgias, headache, and sore throat
- Afterward, infectious gastroenteritis syndrome, rash, and renal/hepatic dysfunction
- Finally, hemorrhagic symptoms develop around the 5th day followed by shock and death:
 - Mortality in 50–90% for Ebola if untreated

ESSENTIAL WORKUP

Suspect bioterrorism if:

- Multiple cases of relatively young, healthy patients who present with flu-like syndrome and within days deteriorate rapidly
- Typical cutaneous lesions appear

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC
- Electrolytes, BUN, creatinine
- ABG
- Cerebrospinal fluid (CSF):
 - Anthrax: 50% with inhalation anthrax develop hemorrhagic meningitis.
- Coagulation studies:
 - Plague: Disseminated intravascular coagulation (DIC)
- Blood cultures
- Wound cultures

- Alert lab personnel to potential concerns of clinicians.

Imaging

CXR:

- Anthrax: Mediastinal widening, pulmonary infiltrate/consolidation, pleural effusion
- Plague: Bronchopneumonia

DIFFERENTIAL DIAGNOSIS

- Anthrax:
 - Influenza
 - Bacterial pneumonia, bacterial meningitis
 - Brown recluse spider bite
 - Tularemia
 - Streptococcal/staphylococcal skin infection
- Plague:
 - Tularemia, catscratch disease
 - Lymphogranuloma venereum, chancroid
 - Tuberculosis
 - Streptococcal adenitis
 - Meningitis, encephalitis, sepsis
- Smallpox:
 - Varicella
 - Rash starts centrally on trunk and spreads outward:
 - Lesions in different stages of development
 - Rarely involves palms or soles
 - Disseminated molluscum contagiosum
 - Monkeypox, drug eruptions
- Toxins:
 - Staphylococcal enterotoxin B:
 - Most common cause of food poisoning
 - Can be aerosolized in addition to being placed in food or water reservoir
 - When inhaled, produces febrile type of illness that can progress to septic shock picture
 - Ricin:
 - Plant protein derived from castor beans
 - Causes rapid progression from upper respiratory congestion to cardiopulmonary collapse
 - Ingestion is less toxic because GI tract does not absorb it well, but it can lead to local cytotoxic death, shock, and death.
 - Botulinum toxin:
 - Initially symptoms include cranial nerve dysfunction with descending

paralysis that leads to respiratory failure.

– **Mycotoxins:**

- Highly toxic compounds produced by certain species of fungus
- Dermal, respiratory, or GI contact can rapidly lead to multiorgan system failure and death.



TREATMENT

PRE HOSPITAL

Universal precautions with N-95 mask

INITIAL STABILIZATION/THERAPY

- ABCs
- 0.9% NS fluid bolus for hypotension
- Supplemental oxygen for hypoxemia
- Vasopressors for persistent hypotension
- Respiratory and contact isolation for suspected cases

ED TREATMENT/PROCEDURES

- All treatments include:
 - Control fever with acetaminophen.
 - Initiate therapy for specific disease.
- Anthrax:
 - Initiate antibiotics:
 - IV for inhalational or severe cutaneous
 - Antibiotic choice depends on susceptibility.
 - Antibiotic options:
 - Ciprofloxacin: 1st line
 - Doxycycline
 - Rifampin
 - Clindamycin
 - Vancomycin
- Plague:
 - Antibiotics initiated within 24 hr minimizes mortality.
 - 1st-line agents: Streptomycin or gentamicin
 - Add chloramphenicol if signs of meningitis or unstable patient
 - Prophylaxis: Doxycycline or ciprofloxacin
- Brucellosis:
 - Supportive therapy
 - Start doxycycline 100 mg PO BID for 6 wk with the addition of streptomycin 1 g per day IM for the 1st 2–3 wk or rifampin 900 mg per day for 6 wk.
- Q fever:

- Recovery occurs within 2 wk without treatment.
- Doxycycline shortens duration of illness.
- Smallpox:
 - Supportive therapy
 - Vaccine given within 4 days of initial exposure decreases chances of contracting smallpox or developing severe symptoms.
 - Vaccinate medical staff caring for patient.
 - Treat secondary bacterial infection.
- Tularemia:
 - See “Tularemia.”
- Hemorrhagic fevers:
 - See “Hemorrhagic Fever.”

MEDICATION

- Chloramphenicol: 25 mg/kg IV q6h
- Ciprofloxacin: 400 mg IV q12h or 500 mg PO BID (peds: 15 mg/kg BID PO)
- Clindamycin: 900 mg IV q12h
- Doxycycline: 100 mg (peds: ≥ 45 kg, 100 mg; if weight ≤ 45 kg, 2.2 mg/kg IV) PO/IV q12h
- Gentamicin: 5 mg/kg IM or IV q24h (peds: 2.5 mg/kg IV/IM q8h)
- Rifampin: 10 mg/kg IV not to exceed 600 mg/d
- Streptomycin: 1 g (peds: 20–40 mg/kg) IM q12h
- Vancomycin: 1 g IV q12h



FOLLOW-UP

DISPOSITION

Admission Criteria

- Decision to treat patient as inpatient vs. outpatient will have to be made in context of overall disaster.
- Toxic or hypoxic patients require admission.
- Respiratory isolation

Discharge Criteria

Mild, noncontagious illness

Issues for Referral

- Contact local and state health departments for suspected or confirmed illness related to biologic weapons.
- Infectious disease and toxicology consult for suspected illness

FOLLOW-UP RECOMMENDATIONS

- Postexposure prophylaxis and vaccinations should be continued based on the causative agent.
- Exposed staff should have follow-up with employee health and infection control prior to returning to work.

PEARLS AND PITFALLS

- Early diagnosis is difficult, and a high index of suspicion is required.
- Failing to use personal protective equipment to protect self and staff is a pitfall.
- Suspect biologic weapons etiology when there is geographic clustering of patients who live, work, or attended an event in close proximity.
- Initiate therapy or prophylaxis early in suspected illness.

ADDITIONAL READING

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- Franz DR, Jahrling PB, Friedlander AM, et al. Clinical recognition and management of patients exposed to biological warfare agents. *JAMA.* 1997;278(5):399–411.
- US Army Medical Research Institute of Infectious Diseases. *Medical Management of Biological Casualties Handbook.* 6th ed. Fort Detrick, Frederick, MD, April 2005.

Useful Websites

- emergency.cdc.gov/bioterrorism/
- sis.nlm.nih.gov/enviro/biologicalwarfare.html



See Also (Topic, Algorithm, Electronic Media Element)

- Botulism
- Hemorrhagic Fever
- Tularemia



CODES

ICD9

- V01.0 Contact with or exposure to cholera
- V71.82 Observation and evaluation for suspected exposure to anthrax
- V71.83 Observation and evaluation for suspected exposure to other biological agent

ICD10

- Z03.818 Encounter for observation for suspected exposure to other biological agents ruled out
- Z20.09 Contact with and (suspected) exposure to other intestinal infectious diseases
- Z20.810 Contact with and (suspected) exposure to anthrax

BIPOLAR DISORDER

Paul H. Desan • Gary S. Sachs

BASICS

DESCRIPTION

- **Mania:**
 - Presentation is diverse and may be difficult to recognize as mania:
 - Simple irritability
 - Cheerfulness
 - Psychosis
 - Delirium
 - Agitation
 - Full extent of pathology often revealed only by outside informants
 - Onset gradual or acute, duration several weeks or months; rarely may be chronic
- **Hypomania:**
 - Milder symptoms without marked impairment
- **Mixed mood:**
 - Simultaneous symptoms of mania and depression
 - Treat in ED as for mania
- **Bipolar disorder:**
 - Formerly manic depressive disorder
 - Defined as one or more episodes of hypomanic, manic, or mixed mood
 - Possibly with episodes of depressed mood
 - Bipolar II is used to denote cases where hypomania has occurred in the course of the disorder but never mania.
 - Typically begins in the teens or 20s
 - Episodes of abnormal mood may be mild or severe, brief or prolonged, infrequent or chronic, chiefly elevated or chiefly depressed in character.
 - Bipolar disorder may be readily responsive to treatment or nearly intractable.
- **Schizoaffective disorder:**
 - Characterized by episodes of altered mood, but psychotic features present even when mood is normal

ETIOLOGY

- Typically, a primary psychiatric disorder, with genetic association
- May be secondary to medical disorder (e.g., drug toxicity, endocrine, neurologic process)
- Particularly likely to be secondary if

- 1st episode
- patient > 40 yr
- atypical or mixed presentation
- abnormal sensorium

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- **Psychiatric history:**
 - Recent symptoms of mania (often collateral sources critical): Elevated, expansive, or irritable mood; increased energy and activity; decreased need for sleep; irresponsibility, disregard for negative consequences of actions; talkativeness; distractibility; fast thoughts; grandiosity, overconfidence
 - Past mania or depression
 - Noncompliance with mood stabilizer
 - Recent initiation or discontinuation of antidepressant
 - Recent substance abuse
 - Bipolar family history
- **Medical history:**
 - Endocrine, metabolic, or neurologic disorders
 - Current or recent medications

Physical-Exam

- **Appearance:**
 - Hyperactive, if not agitated
 - Talkative, often with loud, rapid, or “pressured” speech
- **Affect:**
 - Irritable, argumentative, often multiple recent arguments or fights
 - Less commonly euphoric or expansive
 - Often labile with depressed or tearful intervals (may confound diagnosis)
 - Patient likely to describe mood as tense, irritable, or depressed rather than euphoric
- **Neurovegetative:**
 - Increased energy, engaged in multiple goal-directed activities many hours per day
 - Racing thoughts
 - Decreased sleep
- **Thought process:**
 - Rapid, distractible, may be incoherent, delirious
- **Thought content:**

- Psychosis possible, either mood congruent (e.g., delusions of grandeur or power) or mood incongruent (may be indistinguishable from other psychotic disorders)
- Judgment:
 - Inflated self-esteem, perhaps to grandiose or psychotic extent
 - Uncharacteristic, irresponsible behavior, such as financial or sexual indiscretions, with inability to recognize negative consequences of actions.
 - Substance abuse is frequent during mania.
- Sensorium:
 - Typically normal
 - Confusion or delirium possible

ESSENTIAL WORKUP

- Physical and neurologic exam; vital signs
- Mania may present as delirium and need workup of full differential diagnosis of delirium.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Toxicology screen (urine or serum)
- Blood alcohol level
- Electrolytes
- Blood glucose
- CBC
- TSH
- Lithium, carbamazepine, valproate serum levels, if relevant
- Other tests as suggested by history or physical exam

Imaging

CT head only with suspicion of neurologic etiology

DIFFERENTIAL DIAGNOSIS

- Primary mania of bipolar or schizoaffective disorder
- Psychosis
- Agitated depression
- Personality disorders:
 - Borderline
 - Narcissistic
 - Antisocial
- Attention deficit disorder
- Conduct or intermittent explosive disorders
- Organic brain syndrome

- Intoxication or withdrawal from alcohol or sedative hypnotics
- Intoxication with cocaine, amphetamines, phencyclidine, or other sympathomimetics
- Accidental or deliberate toxic overdose
- Treatment with antidepressants or electroshock therapy in susceptible individuals
- Recent discontinuation of antidepressant medication
- Corticosteroid or thyroid hormones
- Anticholinergics
- Treatment of Parkinson disease
- Cyclobenzaprine (Flexeril)
- Endocrine or metabolic disorders (particularly thyroid disease)
- Encephalitis
- Meningitis
- Postictal states
- MS
- Postcerebrovascular accident
- CNS tumors
- CNS vasculitis
- General paresis



TREATMENT

INITIAL STABILIZATION/THERAPY

- High violence potential:
 - Quiet environment
 - Prompt evaluation
 - Nonconfrontational manner
 - Adequate security backup
 - Physical restraint and sedation, as needed
- For cooperative, but agitated patient:
 - PO neuroleptics (e.g., haloperidol, consider olanzapine or chlorpromazine as alternate) or PO benzodiazepines (e.g., lorazepam)
- For uncooperative agitated patient:
 - Synergistic combination of IM, IV, or PO haloperidol and lorazepam widely used (some authorities favor monotherapy with benzodiazepine or neuroleptic):
 - Bzotropine for prevention of acute dystonic reaction to haloperidol is not usually required when concurrent benzodiazepine is given.
 - Consider lorazepam, olanzapine, ziprasidone, or chlorpromazine IM as alternative.

ED TREATMENT/PROCEDURES

- Outpatient management:
 - Neuroleptics for symptomatic treatment, on temporary or continuing basis
 - Agents for sleep
 - Discontinuation of antidepressant if related to present hypomania or mania
 - Initiation or restart of mood-stabilizer therapy:
 - Action of mood-stabilizing agents requires days or weeks, even after full serum level attained.
- Inpatient management:
 - Sedation or initiation of mood stabilizer in consultation with admitting psychiatrist

MEDICATION

- Acute agitation:
 - Lorazepam: 2 mg PO/IM (lower dose in mild agitation or in frail or elderly); may repeat q30min, generally not to exceed 12 mg/24h
 - Haloperidol: 5 mg PO (lower dose in mild agitation or in frail or elderly); may repeat q30min, generally not to exceed 20 mg/24h
 - Synergistic combination of haloperidol, 5 mg IM/IV/PO + lorazepam 1–2 mg IM/IV/PO, repeat q30min, as required (doses may be smaller in elderly or frail patients)
 - Olanzapine 10 mg IM, ziprasidone 10 mg IM, aripiprazole 9.75 mg IM or chlorpromazine 50 mg IM may be useful parenteral alternatives, perhaps at a lower dose in frail or elderly (avoid chlorpromazine in hypotension; ziprasidone may have more QT prolonging effect than other neuroleptics but the clinical relevance of such effect at this dose is unclear).
- Typical outpatient medications:
 - Aripiprazole: 5–20 mg PO QD
 - Benztropine: 1 mg PO BID
 - Carbamazepine: 400–2,000 mg/d (often in div. doses or in sustained-release dose forms)
 - Clonazepam: 0.5–2 mg PO QHS or 0.5–2 mg PO BID
 - Haloperidol: 0.5–5 mg PO BID
 - Lamotrigine: 25–200 mg/d in 1 or 2 div. doses (typically up to 100 mg/d in patients taking valproate, up to 500 mg/d in patients taking carbamazepine or certain other cytochrome inducers, but not valproate)

ALERT

- Lamotrigine must be started by a gradual dose escalation schedule specified by manufacturer to avoid increased risk of severe dermatologic reactions; if resumed after discontinuation for more than 5 half-lives (about 5 days), the gradual dose escalation schedule must be used again (half-life is shorter with certain antiepileptics, OCPs, rifampin; see prescribing literature).
 - Lithium: 600–3,000 mg/d (often in div. doses or in sustained-release dose

- forms; in acute mania, initiate at 300 mg PO TID)
- Olanzapine: 1.25–30 mg/d, QHS or in div. doses
- Perphenazine: 4–32 mg/d PO QHS or in div. doses
- Quetiapine: 50–400 mg PO QHS or 100–400 PO BID; quetiapine XR PO 50–800 mg QHS
- Risperidone: 0.5–6 mg/d PO QHS or in div. doses
- Valproate (e.g., Depakote): 750–3,000 mg/d (often in div. doses; in acute mania, initiate at 250 mg PO TID)

Pregnancy Considerations

The safety of psychotropic medications in pregnancy is a complex issue: Lithium, valproate, and carbamazepine are Pregnancy Category D and pose particular risks, highest in early pregnancy.

FOLLOW-UP

DISPOSITION

Admission Criteria

- Involuntary hospitalization is required by danger to self:
 - Suicidal risk, especially if mixed or labile mood or psychotic
 - Unsafe behaviors due to impaired judgment
 - Medically unstable
 - Hospitalization diagnostically required
- Involuntary hospitalization also required by:
 - Risk of behaviors dangerous to others
 - Inability to care for self (unable to obtain basic needs, such as food, clothing, or shelter)

Discharge Criteria

- Patients with mild symptoms may be discharged on medications noted above if:
 - necessary supports to ensure safety are in place.
 - patient is compliant with treatment plan.
 - consultation with outpatient psychiatrist is available within 1–3 days.
- Some patients who are not legally committable may refuse treatment; explain availability of future treatment to patient and any involved friends or family.

PEARLS AND PITFALLS

- Manic patients are more likely to appear dysphoric or irritable, rather than “happy.”
- Patients presenting with depression should be asked about features suggesting

mania and hypomania; 70% of bipolar patients have previously been misdiagnosed.

- Individuals with bipolar disorder are at high risk for addiction, further complicating treatment.
- Prompt recognition of the earliest signs of mania may allow prevention of a full episode.
- Bipolar disorder in children frequently manifests as behavioral disinhibition or irritability.

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See Also (Topic, Algorithm, Electronic Media Element)

(Topic, Algorithm, Electronic Media Element) Medical vs. Psychiatric

- Delirium
- Depression
- Dystonic Reaction
- Psychiatric Commitment
- Psychosis, Acute
- Psychosis, Medical vs. Psychiatric



CODES

ICD9

- 296.00 Manic disorder, single episode, unspecified degree
- 296.50 Bipolar affective disorder, depressed, unspecified degree
- 296.80 Bipolar disorder, unspecified

ICD10

- F31.9 Bipolar disorder, unspecified
- F31.10 Bipolar disorder, current episode manic without psychotic features, unspecified
- F31.30 Bipolar disorder, crnt epsd depress, mild or mod severt, unsp

BITE, ANIMAL

Daniel T. Wu

BASICS

DESCRIPTION

- Most bites are from provoked animals.
- Dog bite wounds:
 - Large dogs inflict the most serious wounds (pit bulls cause the most human fatalities).
 - Most fatalities in children (70%) due to bites to face/neck
 - Dogs of family or friends account for most bites.
- Cat bite wounds:
 - Majority from pets known to victim
 - 50% infection rate in those seeking care
 - Puncture wounds most frequent due to sharp thin teeth causing deep inoculation of bacteria
- Catscratch disease (CSD):
 - 3 of the following 4 criteria:
 - Cat contact, with presence of scratch or inoculation lesion of the skin, eye, or mucous membrane
 - Positive CSD skin test result
 - Characteristic lymph node histopathology
 - Negative results of lab studies for other causes of lymphadenopathy
- Rat bite wounds:
 - Occur in lab personnel or children of low socioeconomic class
 - Rat-bite fever (RBF), rare in US but high mortality rate
 - Rat bites rarely transmit rabies, and prophylaxis not routine

ETIOLOGY

- Dog and cat bites:
 - *Pasteurella multocida* is the major organism in both:
 - Twice as likely to be found in cat bites than dog bites
 - Gram-negative aerobe found in up to 80% of cat infections
 - Infection appears in < 24 hr
 - *Staphylococcus* or *Streptococcus*:
 - Infection appears in > 24 hr
 - Other organisms include anaerobes and *Capnocytophaga canimorsus* (dogs).
- Catscratch disease:
 - Caused by *Bartonella henselae*
- Rat bites:

- Caused by *Spirillum minus* and *Streptobacillus moniliformis* (RBF)



DIAGNOSIS

SIGNS AND SYMPTOMS

- Distribution of mammalian bites:
 - Dog bites represent 80–90% of all bites.
 - Cat bites represent 5–15% of all bites.
 - Human bites represent 2–5% of all bites (see “Human Bite” chapter).
 - Rat bites represent 2–3% of all bites.
- Dog bites:
 - Appearance:
 - Crush injuries (most common), tears, avulsions, punctures, and scratches
 - Low rates of infection compared with cat and human bites
 - Infections usually present with:
 - cellulitis
 - malodorous gray discharge
 - fever
 - lymphadenopathy
- Cat bites:
 - Appearance:
 - Puncture wounds (most common)
 - Abrasions
 - Lacerations
 - High infection rates (30–50%) due to deeper puncture wounds
- Catscratch disease:
 - From the bite/scratch of a cat, dog, or monkey
 - Small macule or vesicle that progresses to a papule:
 - Begins several days (3–10) after inoculation
 - Resolves within several days or weeks
 - Regional lymphadenopathy occurs 3 wk postinoculation
 - Tender
 - Nonsuppurative
 - Resolves after 2–4 mo
 - Low-grade fever, malaise, headache
- Rat-bite fever:
 - Does not have to involve a bite. Can occur from handling of rats
 - *S. moniliformis*:
 - Begins several days (2–10) after exposure
 - Common in US
 - Fever, rigors, migratory polyarthralgias, headaches, nausea, and

vomiting

– *S. minus*

- Incubation period from 1–3 wk
- More common in Asia
- Arthritis not common

History

- Animal's behavior, provocation, location, ownership
- Time since attack
- Past medical history: Conditions compromising immune function, allergies, and tetanus status

Physical-Exam

- Record the location and extent of all injuries.
- Document any swelling, crush injuries, or devitalized tissue.
- Note the range of motion of affected areas.
- Note the status of tendon and nerve function.
- Document any signs of infection, including regional adenopathy.
- Document any joint or bone involvement.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Aerobic and anaerobic cultures from any infected bite wound
- Cultures not routinely indicated if wounds not clinically infected
- Catscratch disease:
 - Presence of elevated titers of *B. (Rochalimaea) henselae*, or
 - Positive reaction to catscratch antigen (CSA):
 - Inject 0.1 mL CSA IM
 - Induration at the site 48–72 hr later equal to or exceeding 5 mm is positive

Imaging

Plain radiograph indications:

- Fracture
- Suspect foreign body (e.g., tooth)
- Baseline film if a bone or joint space has been violated in evaluating for osteomyelitis
- For infection in proximity to a bone or joint space

DIFFERENTIAL DIAGNOSIS

- Human bite injuries: Human teeth cause crush injuries and animal teeth cause more punctures and lacerations.

- Bite injuries from other animals
- CSD-caused lymphadenopathy:
 - Reactive hyperplasia (leading cause of lymphadenopathy in children <16 yr)
 - Infection, chronic lymphadenitis, drug reaction, malignancy, and congenital conditions



TREATMENT

PRE HOSPITAL

Apply pressure to any bleeding wound

INITIAL STABILIZATION/THERAPY

- Achieve hemostasis on any bleeding wound.
- Airway stabilization if bite located on face or neck

ED TREATMENT/PROCEDURES

- Wound irrigation:
 - Copious volumes of normal saline irrigation with an 18G plastic catheter tip aimed in the direction of the puncture.
 - Avoid injection of saline through tissue planes due to force of irrigation.
- Débridement:
 - Remove foreign material, necrotic skin tags, or devitalized tissues.
 - Do not débride puncture wounds.
 - Remove any eschar present so underlying pus may be expressed and irrigated.
- Wound closure:
 - Closing wounds increases risk of infection and must be balanced with scar formation and effect of leaving wound open to heal secondarily.
 - Do not suture infected wounds or wounds > 24 hr after injury.
 - Repair of wounds > 8 hr: Controversial
 - Close facial wounds (warn patient of high risk of infection).
 - Infected wounds, those presenting > 24 hr after the event, and deep hand wounds should be left open.
 - May approximate the wound edges with Steri-Strips and perform a delayed primary closure.
- Antibiotic indications:
 - Infected wounds
 - Cat bites
 - Hand injuries
 - Severe wounds with crush injury
 - Puncture wounds

- Full-thickness puncture of hand, face, or lower extremity
- Wounds requiring surgical débridement
- Wounds involving joints, tendons, ligaments, or fractures
- Immunocompromised patients
- Wounds presenting > 8 hr after the event
- Elevate injured extremity
- Tetanus prophylaxis
- Rabies immunoprophylaxis:
 - Not required if rabies not known or suspected
 - Rodents (squirrels, hamsters, rats, mice) and rabbits rarely transmit the disease.
 - Skunks, raccoons, bats, and foxes represent the major reservoir for rabies.
 - See “Rabies” chapter for treatment guidelines.
- Catscratch disease:
 - Analgesics
 - Apply local heat to affected nodes.
 - Avoid lymph node trauma.
 - Disease usually self-limiting
 - Antibiotics controversial, consider if severe disease is present or immunocompromised victim
- Rat-bite fever:
 - High mortality (10%)
 - IV penicillin or doxycycline

MEDICATION

First Line

- Amoxicillin/clavulanic acid (Augmentin): 500/125 mg (peds: 40 mg/kg/24h) q8h PO
- Ampicillin–sulbactam (Unasyn): 3 g q6h IV
- Penicillin 1–2 million units q6h IV (peds 20,000–50,000 U/kg/d div. q4h IV)
- Piperacillin–Tazobactam (Zosyn): 4.5 g q8h IV
- Ticarcillin–clavulanate (Timentin): 3.1 g q4h IV
- Ceftriaxone (Rocephin): 1 g/d plus Metronidazole (Flagyl): 500 mg q8h

Second Line

- 2 drug therapy: 1 of the following below + anaerobic coverage:
 - Trimethoprim–sulfamethoxazole (Septra DS): 1 tablet q12h (peds: 8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per day divided into 2 daily doses) PO
 - Penicillin (Penicillin VK): 500 mg (peds: 50 mg/kg/24h) PO q6h
 - Ciprofloxacin (Cipro): 500–750 mg q12h PO or 400 mg q12h IV
 - Doxycycline: 100 mg PO BID

- + (anaerobic coverage):
 - Clindamycin (Cleocin): 150–450 mg (peds: 8–20 mg/kg/24h) PO q6h or 600–900 mg (peds: 20–40 mg/kg/24h) IV q8h
 - Metronidazole (Flagyl): 500 mg PO TID (peds: 10 mg/kg/dose TID)



FOLLOW-UP

DISPOSITION

Admission Criteria

- All bites:
 - Infected wounds at presentation
 - Severe/advancing cellulitis/lymphangitis
 - Signs of systemic infection
 - Infected wounds that have failed to respond to outpatient (PO) antibiotics
- Catscratch disease:
 - Prolonged fever, systemic symptoms, and/or marked lymphadenopathy

Discharge Criteria

- Healthy patient with localized wound infection:
 - Discharge on antibiotics with 24-hr follow-up.
- Noninfected wounds:
 - 48-hr follow-up

FOLLOW-UP RECOMMENDATIONS

- Hand specialist referral/follow-up for infected hand wounds.
- Healthy patient with localized wound infection: Discharge on antibiotics with 24-hr follow-up.
- 48-hr follow-up for noninfected wounds

PEARLS AND PITFALLS

Animal bites must be reported to authorities in many localities.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Rabies



CODES

ICD9

- 873.40 Open wound of face, unspecified site, without mention of complication
- 874.8 Open wound of other and unspecified parts of neck, without mention of complication
- 882.0 Open wound of hand except finger(s) alone, without mention of complication

ICD10

- S01.80XA Unspecified open wound of other part of head, init encntr
- S11.90XA Unsp open wound of unspecified part of neck, init encntr
- S61.409A Unspecified open wound of unspecified hand, init encntr

BITE, HUMAN

Daniel T. Wu



BASICS

DESCRIPTION

- 3rd most common bite (after dogs and cats)
- Most bites (up to 75%) occur during aggressive acts.
- 15–20% are related to sexual activity (love nips).
- 2 types of bites:
 - Occlusional bites: Laceration or crush injury to affected body part:
 - Occurs when human teeth bite into the skin
 - More prone to infection than animal bites
 - Clenched-fist injuries (CFIs) (CFIs; most serious type): Present as small wounds over metacarpophalangeal joints in dominant hand (fight bites):
 - Sustained from a clenched fist striking the mouth and teeth of another person
- With joint relaxation from the clenched position:
 - Puncture site sealed
 - Oral bacteria inoculated in the anaerobic setting within the joint
 - Bacterial inoculation carried by the tendons deeper into the potential spaces of the hand
 - Increases chances for a more extensive infection

ETIOLOGY

- Aerobic and anaerobic organisms:
 - Most common:
 - *Streptococcus*
 - *Staphylococcus*
 - Others:
 - *Eikenella corrodens*
 - *Haemophilus influenzae*
 - *Peptostreptococcus*
 - *Corynebacterium*
 - *E. corrodens* exhibits synergism with *Streptococcus*, *Staphylococcus aureus*, *Bacteroides*, and gram-negative organisms
- Although rare, case reports of viral transmission via bites (hepatitis, HIV, and herpes)



DIAGNOSIS

SIGNS AND SYMPTOMS

- Location:
 - Upper extremities (60–75%)
 - Head and neck (15–20%)
 - Trunk (10–20%)
 - Lower extremities (~ 5%)
- Frequent complications:
 - Cellulitis
 - Serious deep-space infections (septic arthritis and osteomyelitis)
 - Fractures and tendon injuries
 - Hand bites have highest rates of infection.

History

- Time of injury
- Patient allergies
- Relevant medical history (immune status)
- Last tetanus shot
- HIV, hepatitis B status of person inflicting bite

Physical-Exam

- Record the location and extent of all injuries.
- Document any swelling, crush injuries, or devitalized tissue.
- Note the range of motion of affected areas.
- Note the status of tendon and nerve function.
- Document any signs of infection, including regional adenopathy.
- Document any joint or bone involvement.

ESSENTIAL WORKUP

Careful physical exam for involvement of deep structures and foreign bodies:

- Examine the deepest part of clenched-fist bites while putting the fingers through full range of motion to check for extensor tendon lacerations and joint violation.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Aerobic and anaerobic cultures from any infected bite wound
- Cultures not indicated if wounds not clinically infected
- CBC if signs of significant infection.
- Electrolytes, glucose, BUN, and creatinine:
 - For diabetic patients or those with significant infections

Imaging

- Generally not helpful

- Plain radiograph indications:
 - Fracture
 - Suspect foreign body (e.g., tooth)
 - Baseline film if a bone or joint space has been violated in evaluating for osteomyelitis
 - For infection in proximity to a bone or joint space
- Ultrasound can be useful in differentiating abscess from cellulitis

DIFFERENTIAL DIAGNOSIS

Bite injuries from animals:

- Sharper teeth cause more punctures and lacerations than human teeth, which usually cause more crush-type injuries.

Other Considerations

- In suspected sexual abuse:
 - Check for a central area of bruising or “hickey” from suction
- Linear abrasions or bruises on both the dorsal and palmar/plantar surfaces of the hand or foot:
 - Highly suggestive of bite marks
 - Lesions on one extremity should prompt a search for lesions on the other extremities.
- An intercanine distance of > 3 cm indicates permanent dentition (present only if the attacker is > 8 yr)
- If abuse suspected:
 - Rub a saline-moistened swab in the wound to collect any saliva and then place in a paper envelope for analysis.
 - Obtain photographs.
 - Notify authorities.



TREATMENT

PRE HOSPITAL

Control bleeding with direct pressure.

INITIAL STABILIZATION/THERAPY

ABCs: Ensure patent airway and adequate peripheral tissue perfusion

ED TREATMENT/PROCEDURES

- Wound irrigation:
 - Copious volumes of normal saline irrigation with an 18G needle or plastic catheter tip aimed in the direction of the puncture
 - Care should be taken not to inject fluid into the tissues.

- Débridement:
 - Remove any foreign material, necrotic skin tags, or devitalized tissues.
 - Do not débride puncture wounds.
 - Remove any eschar present so that underlying pus may be expressed and irrigated.
- Clenched-fist injuries:
 - Immobilize
 - Splint in a position of function that maintains the maximal length of ligaments and intrinsic muscles.
 - Use a bulky hand dressing
 - Consultation with hand surgeon regarding operative irrigation/exploration of wound
 - Elevation for several days until any edema resolved
 - Sling for outpatients
 - Place the hand in a tubular stockinette attached to an IV pole for inpatients.
 - Administer antibiotics
- Do not perform primary repair of avulsion wounds.
- Wound closure:
 - Closing wounds increases risk of infection and must be balanced with scar formation and effect of leaving wound open to heal secondarily.
 - Do not suture infected wounds or wounds > 24 hr after injury.
 - Repair of wounds > 8 hr after bite: Controversial.
 - Close facial wounds up to 24 hr after bite (warn patient of high risk of infection).
 - Infected wounds and those presenting > 24 hr should be left open.
 - May approximate the wound edges with Steri-Strips and perform a delayed primary closure.
 - Do not suture CFIs.
- Prophylactic antibiotics controversial for low-risk bites
- Antibiotics for outpatients with:
 - Moderate to severe injuries with crush injury or edema
 - Involvement of the bone or a joint
 - Hand bites
 - Wounds near a prosthetic joint
 - Underlying disease (diabetes, prior splenectomy, or immunosuppression) that increases the risk of developing a more serious infection
- Tetanus prophylaxis
- Refer for possible testing/surveillance for HIV infection.

MEDICATION

First Line

- Amoxicillin/clavulanic acid (Augmentin): 500/125 mg (peds: 40 mg/kg/24h) q8h

PO

- Ampicillin–sulbactam (Unasyn): 3 g q6h IV
- Piperacillin–Tazobactam (Zosyn): 4.5 g q8h IV
- Ticarcillin–clavulanate (Timentin): 3.1 g q4h IV
- Ceftriaxone (Rocephin): 1 g/d plus Metronidazole (Flagyl): 500 mg q8h

Second Line

- 2 drug therapy: 1 of the following below + anaerobic coverage:
 - Trimethoprim–sulfamethoxazole (Septra DS): 1 tablet q12h (peds: 8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per day divided into 2 daily doses) PO
 - Penicillin (Penicillin VK): 500 mg (peds: 50 mg/kg/24h) PO q6h
 - Ciprofloxacin (Cipro): 500–750 mg q12h PO or 400 mg q12h IV
 - Doxycycline: 100 mg PO BID
- + (anaerobic coverage):
 - Clindamycin (Cleocin): 150–450 mg (peds: 8–20 mg/kg/24h) PO q6h or 600–900 mg (peds: 20–40 mg/kg/24h) IV q8h
 - Metronidazole (Flagyl): 500 mg PO TID (peds: 10 mg/kg/dose TID)



FOLLOW-UP

DISPOSITION

Admission Criteria

- Infected wounds at presentation
- Severe/advancing cellulitis/lymphangitis
- Signs of systemic infection
- Infected wounds that have failed to respond to outpatient (PO) antibiotics

Discharge Criteria

- Healthy patient with localized wound infection:
 - Discharge on antibiotics with 24-hr follow-up.
- Noninfected wounds
 - 48-hr follow-up

Geriatric Considerations

- Human bite marks rarely occur accidentally; good indicators of inflicted injury.
- Consider elder abuse.

Pediatric Considerations

- Human bite marks rarely occur accidentally; good indicators of inflicted injury.
- If intercanine distance > 3 cm, bite likely from an adult. Consider child abuse.

Issues for Referral

Suspected child abuse

FOLLOW-UP RECOMMENDATIONS

- Hand specialist referral/follow-up for infected hand wounds
- Healthy patient with localized wound infection: Discharge on antibiotics with 24-hr follow-up.
- 48-hr follow-up for noninfected wounds

PEARLS AND PITFALLS

- Examine the deepest part of clenched-fist bites while putting the fingers through full range of motion to check for extensor tendon lacerations and joint violation.
- Obtain hand consultation for operative irrigation for all patients with clenched-fist lacerations due to the high rate of infection.
- An intercanine distance of > 3 cm indicates permanent dentition (present only if the attacker is > 8 yr).

ADDITIONAL READING

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- Smith PF, Meadowcroft AM, May DB. Treating mammalian bite wounds. *J Clin Pharm Ther.* 2000;25:85–99.



See Also (Topic, Algorithm, Electronic Media Element)

Bite, Mammal



CODES

ICD9

- 879.8 Open wound(s) (multiple) of unspecified site(s), without mention of complication
- 882.0 Open wound of hand except finger(s) alone, without mention of complication

- 882.1 Open wound of hand except finger(s) alone, complicated

ICD10

- S11.90XA Unsp open wound of unspecified part of neck, init encntr
- S21.90XA Unsp open wound of unspecified part of thorax, init encntr
- S61.409A Unspecified open wound of unspecified hand, init encntr

BLADDER INJURY

Mary E. Johnson



BASICS

DESCRIPTION

- Blunt trauma is the most common mechanism.
- 10% of pelvic fractures have serious bladder injury.
- 80–90% of bladder ruptures have pelvic fracture.
- Mortality: 17–22% overall; 60% if combined intraperitoneal/extraperitoneal rupture

ETIOLOGY

- Mechanism:
 - Trauma, 82%
 - Blunt trauma: Motor vehicle accident (MVA; 87%), falls (7%), assault (6%)
 - Penetrating: Gunshot wound (GSW) (85%), stabbings (15%)
 - Iatrogenic 14%: TURP and urologic procedures, gynecologic procedures, obstetric procedures, abdominal procedures, hernia repair, intrauterine device (IUD), orthopedic hip procedures, biopsies, indwelling Foley
 - Intoxication 2.9%
 - Spontaneous <1%
- Classification:
 - Extraperitoneal bladder rupture (62%):
 - Associated with pelvic fractures
 - Caused by blunt force or fracture fragments
 - Intraperitoneal bladder rupture (25%):
 - Direct compression of distended bladder
 - Caused by rupture of the dome of the bladder
 - Combined extraperitoneal and intraperitoneal rupture (12%):
 - Highest mortality owing to associated injuries
 - Bladder contusion:
 - Damage to endothelial lining or muscularis layer with intact bladder wall
 - Gross hematuria after extreme physical activity (long-distance running)
 - Gross hematuria with normal imaging
 - Usually resolves without intervention

Pediatric Considerations

- In children, the bladder is an intra-abdominal organ and descends into the pelvis

by age 20 yr.

- Intraabdominal rupture is more common in children than adults because the bladder is an abdominal organ.
- Bladder injury is more common in children than in adults because the pediatric bony pelvis is less rigid and transmits more force to adjacent structures.



DIAGNOSIS

SIGNS AND SYMPTOMS

Triad:

- Gross hematuria
- Suprapubic pain
- Difficulty voiding

History

Establish potential mechanism.

Physical-Exam

Evaluate urethral meatus—if blood is present, do not insert Foley catheter until retrograde urethrogram (RUG) is performed (concomitant urethral and bladder injuries occur in 10–29% of patients).

ESSENTIAL WORKUP

- History of trauma or procedures
- Evaluate urethral meatus for blood.
- Urinalysis (UA)
- Retrograde cystography

DIAGNOSIS TESTS & INTERPRETATION

Lab

- UA:
 - Gross hematuria in 95–100% of patients with significant bladder or urethral trauma
 - Microscopic hematuria in 5%
- BUN and creatinine:
 - The BUN can be elevated from resorption of urine within the peritoneum.
- Electrolytes:
 - Hyperkalemia and hyponatremia may result from resorption of urine within the peritoneum.

Imaging

- Retrograde cystography and retrograde CT cystography are the methods of choice to diagnose a ruptured bladder. Both studies have reported sensitivity and specificity of 95% and 100% respectively.
- If urethral injury is suspected, the cystogram is performed after a RUG.
- Cystography technique:
 - Kidneys/ureter/bladder (KUB) scout film
 - Infuse 100 mL of diluted contrast via Foley into bladder. Contrast material needs to be diluted: 30% or 6:1 saline; otherwise it is too dense.
 - Plain film is repeated to evaluate early extravasation.
 - If initial film is normal, fill rest of bladder with diluted contrast:
 - Min. 300–350 mL total for adult
 - 3–5 mL/kg or 60 mL + (age in yr × 30) for children or until discomfort
 - It is essential to have a bladder full of contrast for diagnosis; it is not sufficient to place contrast and clamp Foley in antegrade fashion.
 - Cystogram films taken in AP, lateral, and oblique views (oblique may be difficult in trauma and CT is often used)
 - Empty bladder and obtain a postdrainage film unless CT cystography obtained.
 - Postdrainage film is essential without CT cystography—10% of bladder ruptures are seen only on postdrainage film; a distended bladder may hide extravasation.
- Cystography interpretation:
 - Extraperitoneal rupture: Tear drop- or star-shaped form
 - Intraperitoneal rupture: Outlining of bowel or contrast within the paracolic gutters

Diagnostic Procedures/Surgery

- FAST scan:
 - Free pelvic fluid should raise concern for bladder injury.

DIFFERENTIAL DIAGNOSIS

- Peritoneal trauma
- Urethral trauma
- Renal or ureteric trauma



TREATMENT

PRE HOSPITAL

Do not attempt bladder catheterization in the field.

INITIAL STABILIZATION/THERAPY

- ABCs
- Early urologic consultation

ED TREATMENT/PROCEDURES

- Urologic consultation is needed when bladder rupture is diagnosed.
- Extraperitoneal nonpenetrating ruptures may be managed by catheter drainage:
 - 20F Foley or larger for 14 days
 - 80% of lacerations will seal in 3 wk.
 - If patient is undergoing abdominal or pelvic surgery for other injury, surgical repair is recommended.
- Intraperitoneal ruptures require surgical exploration.
- Bladder contusions do not need any specific interventions.

MEDICATION

Broad-spectrum antibiotics for intraperitoneal rupture



FOLLOW-UP

DISPOSITION

Admission Criteria

- Concurrent major trauma requiring admission or observation
- Surgical intervention required

Discharge Criteria

- Bladder contusion with no rupture or other major trauma requiring admission
- Most cases of bladder rupture will require admission; discharge only after clearance by urology and no other associated injuries.

Issues for Referral

Any bladder injury managed as an outpatient should have urologic referral.

FOLLOW-UP RECOMMENDATIONS

Follow-up to be arranged with urology:

- Extraperitoneal bladder rupture with Foley catheter management will have Foley removal in 14 days.

PEARLS AND PITFALLS

- Any free fluid on CT or US exam should raise suspicion for bladder injury.
- Unresponsive, altered, and intoxicated patients warrant careful exam.
- Penetrating injuries to lower abdomen with any degree of hematuria warrant cystography.

ADDITIONAL READING

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- Ramchadani P, Buckler PM. Imaging of genitourinary trauma. *AJR Am J Roentgenol*. 2009;192(6):1514–1523.
- Uptodate.com
- Wein AJ, Kavoussi LR, Novick AC, et al., eds. *Cambell-Walsh Urology*. 10th ed. Philadelphia, PA: WB Saunders; 2012.



See Also (Topic, Algorithm, Electronic Media Element)

- Pelvic Fracture
- Urethral Trauma
- Trauma, Multiple



CODES

ICD9

- 665.50 Other injury to pelvic organs, unspecified as to episode of care or not applicable
- 867.0 Injury to bladder and urethra, without mention of open wound into cavity
- 867.1 Injury to bladder and urethra, with open wound into cavity

ICD10

- S37.20XA Unspecified injury of bladder, initial encounter
- S37.23XA Laceration of bladder, initial encounter
- S37.29XA Other injury of bladder, initial encounter

BLOW-OUT FRACTURE

Shari Schabowski



BASICS

DESCRIPTION

- Defined as an orbital floor fracture without orbital rim involvement
- Results from sudden blunt trauma to the globe:
 - Typically caused by the force of a projectile $>$ half the size of the fist
- Force transmitted through the noncompressible structures of the globe to the weakest structural point: The orbital floor
- Transmitted force “blows out” or fractures the orbital floor.
- Orbital floor serves as roof to air-filled maxillary and ethmoid sinuses:
 - Communication between the spaces results in orbital emphysema.
- Orbit contains fat, which holds the globe in place:
 - Orbital floor fracture may result in herniation of the fat on the inferior orbital surface into the maxillary or ethmoid sinuses.
 - Leads to enophthalmos owing to orbital volume loss and sinus congestion; fluid collection may occur secondary to edema and bleeding.
- Infraorbital nerve runs through the bony canal 3 mm below the orbital floor:
 - Injury may result in hypoesthesia of the ipsilateral cheek and upper lip.
 - To distinguish facial hypoesthesia related to local swelling from nerve injury: Test for sensation on the ipsilateral gingiva, which is within the infraorbital nerve distribution.
- Inferior rectus and the inferior oblique muscle run along the orbital floor:
 - Restriction of these extraocular muscles may occur because of entrapment within the fracture, contusion, or cranial nerve dysfunction.
 - Typically manifests as diplopia on upward gaze
 - Inability to elevate the affected eye normally on exam
- Medial rectus located above the ethmoid sinus:
 - Less commonly entrapped
 - Diplopia on ipsilateral lateral gaze

ETIOLOGY

Caused by a projectile which strikes the globe. The force is transmitted through the noncompressible structures of the globe to the weakest structural point: the orbital floor resulting in a blow out fracture.

Pediatric Considerations

- Orbital roof fractures with associated CNS injuries more common in children
- Orbital floor fractures: Unlikely before 7 yr of age:

- Orbital floor is not as weak a point in the orbit due to lack of pneumatization of the paranasal sinuses.
- Unfortunately fractures can occur in children and may result in unrecognized entrapment of the rectus muscle labeled the “white-eyed” fracture:
 - These children may present with marked nausea, vomiting, headache, and irritability suggestive of a head injury that commonly distracts from the true diagnosis.

DIAGNOSIS

SIGNS AND SYMPTOMS

- Periorbital tenderness, swelling, and ecchymosis
- Impaired ocular mobility or diplopia:
 - Restricted upward gaze owing to inferior rectus entrapment
 - Restricted ipsilateral lateral gaze with medial rectus entrapment
- Infraorbital hypoesthesia:
 - Caused by compression/contusion of infraorbital nerve
 - May extend to upper lip
- Enophthalmos:
 - Globe set back owing to orbital fat displaced through fracture
- Periorbital emphysema:
 - From the ethmoid or maxillary sinus
- Epistaxis
- Normal visual acuity:
 - If not, consider more extensive injuries
- No orbital rim step off

Associated Severe Injuries

- Ocular injuries:
 - Ruptured globe:
 - Incidence up to 30% of blow-out fractures
 - Ophthalmologic emergency
 - Retrobulbar hemorrhage
 - Emphysematous optic nerve compression
- Cervical spine or intracranial injuries
- Commonly associated injuries:
 - Subconjunctival hemorrhage
 - Corneal abrasion/laceration
 - Hyphema
 - Traumatic mydriasis
 - Traumatic iridocyclitis (uveitis)
- Less common:

- Iridodialysis
- Retinal detachment
- Vitreous hemorrhage
- Optic nerve injury
- Associated fractures:
 - Nasal bones
 - Zygomatic arch fracture
 - Le Fort fracture
- Late complications:
 - Sinusitis
 - Orbital infection
 - Permanent restriction of extraocular movement
 - Enophthalmos

History

Struck in the eye with a projectile. Paintball, handball, racquetball, baseball, rock, or possibly fist. Larger-sized projectiles will likely be blocked by the orbital rim. Seen frequently after MVCs which are the most common cause of maxillofacial trauma.

Physical-Exam

- Thorough ophthalmologic exam:
 - Palpate bony structures of the orbit for evidence of step off.
 - Careful attention not to place pressure on the globe until ruptured globe excluded:
 - Desmarres lid retractors may be necessary to evaluate the eye with swollen lid.
- Document pupillary response
- Visual acuity (should not be affected):
 - Handheld visual acuity Rosenbaum card is most useful with injuries.
- Test extraocular movements for disconjugate gaze or diplopia.
- Test sensation in inferior orbital nerve distribution.
- Examine lid and adnexa:
 - Orbital emphysema may be present.
- Slit-lamp and fundoscopic exam to identify associated injuries.
- Full physical exam to identify associated injuries and neurologic impairment.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Preoperative lab studies if indicated
- Pregnancy testing prior to radiography

Imaging

- If CT unavailable or contraindicated, plain radiographs will provide important information:
 - _ Facial films
 - _ Orbits
 - _ Waters view and exaggerated Waters view:
 - Classic “teardrop sign” illustrates herniated mass of orbital contents in the ipsilateral maxillary sinus.
 - Opacification of or air–fluid level in the ipsilateral maxillary sinus (less specific)
 - Orbital floor bony fracture
 - Lucency in orbits consistent with orbital emphysema
- CT-preferred modality:
 - _ Defines involved anatomy
 - _ Obtain axial and coronal 1.5-mm cuts:
 - Reconstruction of coronals not preferred but acceptable if positioning impossible

Diagnostic Procedures/Surgery

Forced duction test:

- Distinguishes nerve dysfunction from entrapment
- Topical anesthesia applied to the conjunctiva on the opposite side, and the globe is pulled away from the expected point of entrapment; if the globe is not mobile, the test is positive—defining physical entrapment.

Pediatric Considerations

- Orbital CT: Study of choice:
 - _ Plain films less helpful
- Essential to identify entrapment early as long-term outcome will likely be affected if left undiagnosed:
 - _ Early surgical intervention for entrapment may significantly improve outcome.

DIFFERENTIAL DIAGNOSIS

- Cranial nerve palsy
- Orbital cellulitis
- Periorbital cellulitis
- Periorbital contusion/ecchymosis
- Retrobulbar hemorrhage
- Ruptured globe



TREATMENT

PRE HOSPITAL

- Metal protective eye shield if possible globe injury
- Place in supine position.

INITIAL STABILIZATION/THERAPY

Initial approach and immediate concerns:

- Assess for associated intracranial or cervical spine injuries.
- Rule out ruptured globe.
- Test visual acuity:
 - _ Decreased visual acuity suggestive of associated with more extensive injuries

ED TREATMENT/PROCEDURES

- After globe rupture is excluded, apply cool compresses for the 1st 24–48 hr to decrease swelling to minimize or reverse herniation and avoid surgical intervention.
- Avoid Valsalva maneuvers and nose blowing to prevent compressive orbital emphysema.
- Prophylactic antibiotics to prevent infection
- Nasal decongestants if no contraindication
- Analgesics as needed
- Tetanus prophylaxis

MEDICATION

- Antibiotics are recommended prophylactically to prevent sinusitis and orbital cellulitis:
 - _ Cephalexin 250 mg q6h for 10 days
- Systemic corticosteroids have been advocated to speed up the resorption of edema in order to more accurately assess any muscle entrapment and orbital damage:
 - _ Prednisone (60–80 mg/d) within 48 hr of the injury and continued for 5 days
- Nasal decongestants may be beneficial if not contraindicated:
 - _ Phenylephrine nasal spray: BID for 2–4 days



FOLLOW-UP

DISPOSITION

Admission Criteria

- Rarely indicated
- 85% resolve without surgical intervention.
- Consultation with facial trauma service in ED and consideration for admission if:
 - _ 50% of floor fractured
 - _ Diplopia or entrapment is identified

- Particularly in children
- Enophthalmos > 2 mm or more

Discharge Criteria

In most cases, observe for 10–14 days until swelling resolves, then follow up with facial trauma surgeon to determine need for surgical intervention.

FOLLOW-UP RECOMMENDATIONS

Symptoms should improve over time:

- If at any point patient develops increased swelling, tenderness, redness, or pain around the eye, they should return to ED for re-evaluation.
- If any visual disturbance, visual loss, or increased eye pain return to ED for re-evaluation.

PEARLS AND PITFALLS

- Be hypervigilant in checking pupillary response and visual acuity:
 - Abnormal results may be the 1st sign of serious complications:
 - Globe rupture
 - Optic nerve injury possibly stemming from emphysematous or retrobulbar compression
- Careful evaluation for entrapment:
 - Essential for all, but particularly children, to exclude white-eyed fracture and its long-term complications
- The oculocardiac (Aschner) reflex may be associated with this injury. It manifests as a decrease in pulse rate associated with traction applied to extraocular muscles and/or compression of the eyeball:
 - May be seen more commonly in children
 - Treated by release of pressure and in some cases may require atropine

ADDITIONAL READING

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- Gosau M, Schöneich M, Draenert FG, et al. Retrospective analysis of orbital floor fractures – complications, outcomes and review of the literature. *Clin Oral Investig*. 2011;15(3):305–313.
- Higashino T, Hirabayashi S, Eguchi T, et al. Straightforward factors for predicting the prognosis of blow-out fractures. *J Craniofac Surg*. 2011;22(4):1210–1214.



See Also (Topic, Algorithm, Electronic Media Element)

- Facial Fractures
- Globe Rupture
- Iritis
- Oculomotor Nerve Palsy
- Periorbital and Orbital Cellulitis



CODES

ICD9

- 376.52 Enophthalmos due to trauma or surgery
- 802.6 Closed fracture of orbital floor (blow-out)
- 802.7 Open fracture of orbital floor (blow-out)

ICD10

- H05.429 Enophthalmos due to trauma or surgery, unspecified eye
- S02.3XXA Fracture of orbital floor, init encntr for closed fracture
- S02.3XXB Fracture of orbital floor, init encntr for open fracture

BOERHAAVE SYNDROME

Lauren M. Smith • Edwin R. Malone



BASICS

DESCRIPTION

- Spontaneous esophageal rupture from sudden combined increase in intra-abdominal pressure and negative intrathoracic pressure
 - Causes complete, full-thickness (transmural), longitudinal tear in esophagus
- Esophagus has no serosal layer (which normally contains collagen and elastic fibers):
 - Results in weak structure vulnerable to perforation and mediastinal contamination
 - Esophageal wall is further weakened by conditions that damage mucosa (i.e., esophagitis is of various causes).
- Majority of perforations occur at left posterolateral wall of the lower third esophagus.
- Significant morbidity/mortality (most lethal GI tract perforation):
 - Owing to explosive nature of tear
 - Owing to almost immediate contamination of mediastinum with contents of esophagus
 - Overall mortality can approach 20%
 - Mortality can double if treatment is delayed > 24 hr from rupture
 - Cervical rupture associated with the lowest mortality, followed by abdominal and thoracic rupture, respectively

ETIOLOGY

- Associated with:
 - Forceful vomiting and retching (most common)
 - Heavy lifting
 - Seizures
 - Childbirth
 - Blunt trauma
 - Induced emesis
 - Caustic ingestions
 - Laughing
 - History of Barrett ulcer
 - History of HIV/AIDS
 - History of pill esophagitis
- Common in middle-aged men
- Medical procedures cause over 50% of all perforations.

Pediatric Considerations

- Described in female neonates but rarely seen
- Consider caustic ingestions

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Often no classic symptoms
- Most common symptoms:
 - Chest or epigastric pain after vomiting/retching
- Mackler triad:
 - Vomiting/retching
 - Chest pain
 - Subcutaneous emphysema
- Retrosternal chest pain present in most patients:
 - Often pleuritic
 - Radiates to back or left shoulder
 - Worsens with swallowing
- Odynophagia
- Swallowing may precipitate coughing
- Frequently, a history of alcoholism or heavy alcohol ingestion may be elicited

ALERT

The vague nature of symptoms often lead to a delay in outcome and poorer prognosis

Physical-Exam

- Dyspnea
- Diaphoresis
- Subcutaneous emphysema in neck and chest wall
- Mediastinal crackling on auscultation (Hamman crunch)
- Pleural effusions
- Tachypnea
- Fever
- Shock, in more severe cases
- If untreated, mediastinitis will develop and abscesses will form.
- Not usually associated with bleeding

ESSENTIAL WORKUP

- Upright chest radiographs (preferably posteroanterior and lateral views if tolerated) evaluating for:
 - Pneumomediastinum

- SC emphysema
- Pleural effusion (left side)
- Pneumothorax
- Widened mediastinum
- Hydropneumothorax
- Empyema
- Free peritoneal air
- Naclerio “V” sign:
 - V-shaped radiolucency seen through the heart (air in left lower mediastinum)
- Contrast esophagram identifies leak in esophagus:
 - Aids in decision of which type of surgical approach
 - Controversy exists regarding contrast use, water-soluble vs. barium
 - Water-soluble contrast material was thought to be less toxic if extravasated into the mediastinum; however, if aspirated may cause necrotizing pneumonitis and has a higher rate of false negatives
 - Barium, more sensitive for diagnosing perforation, but more irritating to the mediastinum
 - If esophagus is intact, use barium contrast for better detail

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC
- PT/PTT/INR
- Blood cultures
- Pleural effusion:
 - Amylase content
 - pH (< 6)
 - Undigested food particles
- ECG

Imaging

- CXR
- Endoscopy:
 - Controversial because this may extend perforation and/or introduce air into mediastinum
- CT chest:
 - Sensitive for identifying free air, periesophageal fluid, mediastinal widening, air or fluid in pleural spaces; however, does not isolate lesion
 - Indicated if esophagram cannot be obtained
 - Evaluates other intrathoracic structures

DIFFERENTIAL DIAGNOSIS

- Cholecystitis
- Dissecting aortic aneurysm
- Intestinal obstruction
- Lung abscess
- Mesenteric thrombosis
- Myocardial infarction
- Pneumothorax
- Pericarditis
- Pneumonia
- Pancreatitis
- Pulmonary thromboembolism
- Ruptured abdominal viscus
- Spontaneous pneumomediastinum (clinically benign)



TREATMENT

PRE HOSPITAL

- Airway control must be established if patient unresponsive or airway patency in jeopardy.
- Establish 2 large-bore intravenous catheters and treat hypotension with 0.9% NS.
- Avoid opiates until patient is in ED to avoid complication of hypotension.

INITIAL STABILIZATION/THERAPY

- ABCs
- Airway control: 100% oxygen or intubate patient if unresponsive or airway patency is in jeopardy.
- Establish intravenous access and treat hypotension:
 - Administer 1 L (20 mL/kg) bolus with 0.9% NS (or lactated Ringer solution).
 - Initiate dopamine if blood pressure does not respond to fluids.
 - Central catheter placement if condition of patient remains unstable for more efficient delivery of fluids and monitoring of central venous pressure

ED TREATMENT/PROCEDURES

- NPO
- Careful placement of a nasogastric tube to decompress the stomach
- Bladder catheter to monitor urine output
- Expedient diagnosis to decrease incidence of morbidity/mortality
- Prompt surgical consultation
- Definitive treatment:
 - Surgical repair
 - Endoscopic stent placement, considered in appropriate patients

- Conservative management, may be considered in patients with a contained perforation
- Initiate broad-spectrum antibiotics directed against oral microflora and gastrointestinal pathogens:
 - Ampicillin/sulbactam + gentamicin
 - Imipenem/Cilastatin

MEDICATION

- Ampicillin/sulbactam: 3 g IV q6h
- Dopamine: 2–20 $\mu\text{g}/\text{kg}/\text{min}$ IV per bolus
- Gentamicin: 2 mg/kg load, then 1.7 mg/kg IV q8h or 5–7 mg/kg IV QD (assuming normal renal function)
- Imipenem/cilastatin: 250–500 IV q6h



FOLLOW-UP

DISPOSITION

Admission Criteria

All cases of Boerhaave syndrome must be admitted to surgical ICU:

- Cervical esophageal perforations may be treated by drainage alone.
- All thoracic and abdominal perforations require surgical intervention.

Discharge Criteria

None

Issues for Referral

Thoracic or general surgeon must be consulted for admission and possible operative intervention.

FOLLOW-UP RECOMMENDATIONS

As per surgeon recommendations

PEARLS AND PITFALLS

- Chest radiographs done immediately after injury may be normal.
- Left pleural space involvement is usually associated with a distal esophageal perforation.
- Right pleural space involvement is usually associated with proximal esophageal perforations.
- If esophagram is negative and there is high suspicion, repeat with patient in left and right decubitus positions.

- Immediate surgical consultation is the keystone of management.
- Significant increases in mortality are seen with delay in diagnosis and management.

ADDITIONAL READING

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CODES

ICD9

530.4 Perforation of esophagus

ICD10

K22.3 Perforation of esophagus

BOTULISM

Philip Shayne • Jean Wheeler



BASICS

DESCRIPTION

- Rare in US, causing < 200 cases/yr; however, has significant bioterrorism potential.
- Caused by a polypeptide, heat-labile exotoxin produced by *Clostridium botulinum*:
 - Most potent poison known
- Toxin blocks neuromuscular transmission in cholinergic nerve fibers.
- Symptoms occur by inhibition of acetylcholine release from presynaptic nerve membranes:
 - Damage is permanent.
 - Recovery is by formation of new synapses through sprouting from the axon.
- Onset: 12–72 hr after exposure; may be up to 1 wk after exposure:
 - Death can occur 24 hr after onset of symptoms.
- Slow recovery; symptoms often persist for months
- Mortality:
 - Untreated: 60–70%
 - With supportive care: 3–10%
- 3 major types: Food-borne botulism, wound botulism, and infantile botulism (see “Pediatric Considerations”). Absorbed through mucosal surfaces or nonintact skin
- Food-borne botulism:
 - Occurs by ingestion of preformed toxin; from improperly canned food, improper refrigeration
 - Conditions required for exposure:
 - Food product contaminated with *C. botulinum* bacilli or spores
 - Proper conditions for germination of spores exist.
 - Time and conditions permit production of toxin before eating.
 - Food not heated sufficiently to destroy botulism toxin
 - Toxin-containing food ingested by susceptible host
- Wound botulism:
 - Clinical evidence of botulism after trauma with a resultant infected wound and no history suggestive of food-borne illness
 - Botulinum isolated in about 50%
 - Wounds usually contaminated with soil
 - Majority of US cases from IV drug use
- Other types:
 - Adult intestinal toxemia botulism:
 - Seen in adults with functional or structural GI abnormalities, are

- immunocompromised or with prolonged antibiotic use
- Predisposes to *Clostridial* colonization
- May have sporadic or recurrent botulism with no known source and even after immunoglobulin treatment
- Iatrogenic botulism:
 - Doses found in cosmetic applications are insufficient to cause systemic symptoms.
 - No known recent cases from medical use.
 - Symptoms would be expected to be classic.
- Inhalation botulism:
 - Aerosolization of toxin may have bioterrorism applications. Last reported naturally occurring case in 1962 from the disposal of animal remains.

Pediatric Considerations

- Infantile botulism occurs from the ingestion of *C. botulinum* spores, which germinate in the gut and produce the toxin.
- Accounts for 50–76% of botulism cases
- 90% occur in children <6 mo:
 - Associated with patient or family exposure to soil, dust, or agricultural industry.
 - May also be associated with weaning from breast milk, which may alter intestinal flora and increase susceptibility to *Clostridia* infection.
- Usually presents with change in stool pattern or constipation, progressing over several days to symptoms of bulbar weakness, then descending flaccid paralysis.
- Slower onset is attributed to the toxin being produced locally as opposed to being ingested in 1 dose.
- *C. botulinum* spores found in honey:
 - Honey not recommended for children <1 yr.

ETIOLOGY

- *C. botulinum* is a large spore-forming, usually gram-positive, strictly anaerobic bacilli ubiquitous in nature.
- Each strain produces antigenically distinct toxins, designated types A to G:
 - Types A, B, E, and rarely F are responsible for most human cases.

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Ingestions/food history for previous 4–5 days:

- Exposures traditionally from home-processed fruit or vegetable products
- In prison populations ingestion of “pruno” (alcohol product created by prisoners using leftover food products)
- Immune status (AIDS, cancer, chronic illness)
- IV drug use

Physical-Exam

- Food-borne botulism (classic botulism):
 - Bulbar weakness is invariably the initial presentation: Diplopia, dysphagia, dysarthria, and dysphonia
 - Subsequent symmetric, descending weakness or paralysis of the extremities (hallmark of the disease)
 - No sensory deficit
 - May have progressively diminishing deep tendon reflexes
 - Patient remains awake/alert; mentation unaffected.
 - Ventilatory insufficiency from weakness of respiratory muscles
 - Autonomic dysfunction (sympathetic and parasympathetic):
 - Dry mouth
 - Blurred vision
 - Orthostatic hypotension
 - Constipation
 - Urinary retention
 - Nausea and vomiting with food-borne botulism only
 - Afebrile
- Wound botulism:
 - Finding similar to food-borne botulism
 - May be febrile as a result of soft-tissue infection
- Infantile botulism:
 - Constipation
 - Weakness
 - Poor suck
 - Weak cry
 - Lethargy
 - Hypotonia
 - Flaccid facial expression
 - Respiratory difficulty
- Inhalation botulism:
 - Similar to food-borne botulism with absence of GI symptoms

ESSENTIAL WORKUP

- Diagnosis is entirely clinical.
- Workup focuses on differentiation from other conditions causing general paralysis.
- If diagnosis is suspected, immediately notify state health department or CDC (770-

488-7100 for adults or 1-510-231-7600 for infant cases).

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC
- Electrolytes, BUN/creatinine, and glucose:
 - Check for hypokalemia.
- Arterial blood gas (ABG):
 - For signs of respiratory insufficiency
- Confirmatory testing via mouse assay performed by select state and federal labs, using samples from:
 - Blood
 - Feces
 - Gastric contents
 - Suspected food and containers
 - Takes between 6–96 hr for results
- Anaerobic blood cultures:
 - May detect bacterium
- Nasal swab for ELISA test:
 - For inhalation botulism, as less reliably detected in sera and stool than other forms
 - Sample needs to be collected within 24 hr of exposure

Imaging

CT/MRI of brain:

- Normal

Diagnostic Procedures/Surgery

- CSF testing:
 - Normal
 - Helps differentiate from Guillain–Barré syndrome (which has markedly elevated CSF protein)
- Electrophysiologic studies:
 - Normal nerve conduction with diminished evoked muscle action potential
- Edrophonium testing may be positive, but not to the degree seen in myasthenia gravis.

DIFFERENTIAL DIAGNOSIS

- Myasthenia gravis (less acute)
- Lambert–Eaton myasthenic syndrome (less acute)
- Polio (fever and asymmetric)
- Guillain–Barré (simultaneous sensory findings and elevated spinal fluid protein)

- Tick paralysis
- Magnesium intoxication
- Hypokalemic periodic paralysis
- Diphtheritic neuropathy
- Rare basilar stroke syndromes with bulbar palsy

Pediatric Considerations

- Often misdiagnosed as dehydration, sepsis, or Reye syndrome
- Other diagnoses include inborn errors of metabolism, Guillain–Barré syndrome, and spinal muscle atrophy.



TREATMENT

ALERT

Death is invariably from progressive ventilatory failure:

- Intubate as soon as respiratory insufficiency noted, clinically and/or in conjunction with ABG.
- May require several weeks of ventilatory support

PRE HOSPITAL

- Transcutaneous pacing for unstable type II 2nd- or 3rd-degree block
- Atropine:
 - Avoid with type II 2nd-degree block because it may precipitate complete heart block
 - Contraindicated in 3rd-degree heart block with a widened QRS complex
- Attempts should be made at preventing increases in vagal tone.

INITIAL STABILIZATION/THERAPY

- Early intubation and ventilatory support is the key to survival.
- Respiratory difficulties occur rapidly.

ED TREATMENT/PROCEDURES

- Bivalent AB antitoxin:
 - IV administration as soon as the diagnosis is made and initial samples are collected, without waiting for lab confirmation
 - Before use assess hypersensitivity with skin test using horse serum or antitoxin
 - Using recommended dose < 1% will have hypersensitivity reaction
- With wound botulism perform wound débridement even if it appears to be healing.
- Antibiotics for specific infectious complications
- Standard precautions only; no evidence of person-to-person transmission
- If environmental exposure, wash clothing and skin with soap and water

MEDICATION

- ABE antitoxin formulations no longer used because of declines in titer to type E toxin
- 1st-line treatment:
 - Baby BIG human-derived antitoxin to types A and B licensed by USFDA for treatment of infant botulism and distributed by CA Dept. of Public Health (510-231-7600). www.infantbotulism.org/
 - Heptavalent antitoxin (H-Bat) available from CDC as an investigational use drug protocol and emergency therapeutic use. Not for infant botulism.

Pediatric Considerations

- Baby BIG halves average hospital stay from 6–3 wk:
 - Adult equine antitoxin should not be used on pediatric patients
- Antibiotics:
 - Ineffective in eradicating organism from the intestine
 - Release of toxin in the gut through bacterial cell lysis may worsen neurologic symptoms.

Second Line

Pentavalent toxoid for lab workers



FOLLOW-UP

DISPOSITION

Admission Criteria

Admit patients with suspected botulism poisoning to monitored bed:

- ICU admission for any respiratory deficiency

Discharge Criteria

Clinical course of botulism poisoning is unpredictable; it can become rapidly progressive and fatal:

- Discharge patients only after a prolonged period of progressive recovery from symptoms.

FOLLOW-UP RECOMMENDATIONS

- Physical medicine and rehabilitation:
 - Residual weakness can last for up to 1 yr
- Mental health:
 - Patients and their families often experience stress and depression with the prolonged recovery.

PEARLS AND PITFALLS

- Botulism is a public health emergency; early consultation with state and federal health departments is required.
- Suspect botulism if there are more than 2 cases; other conditions in the differential do not produce outbreaks.
- Antitoxin does not reverse paralysis but only halts its progression. Therefore, administer antitoxin once diagnosis is suspected. Do not wait until signs of respiratory compromise are present.
- Initial signs of respiratory distress may not be clinically apparent secondary to paralysis.
- Bulbar palsy at presentation may be mistaken for altered mental status.

ADDITIONAL READING

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CODES

ICD9

- 005.1 Botulism food poisoning
- 040.41 Infant botulism
- 040.42 Wound botulism

ICD10

- A05.1 Botulism food poisoning

- A48.51 Infant botulism
- A48.52 Wound botulism

BOWEL OBSTRUCTION (SMALL AND LARGE)

Jenny J. Lu

BASICS

DESCRIPTION

- Obstruction of normal intestinal flow from mechanical or nonmechanical causes
- Small-bowel obstruction (SBO):
 - 20% of acute surgical admissions
 - Adhesions: Most common cause (60%)
 - Neoplasms
 - Hernias
 - Strictures: Inflammatory bowel disease
 - Trauma: Bowel wall hematoma
 - Miscellaneous (e.g., ascaris infection)
- Large-bowel obstruction (LBO):
 - Disease primarily of the elderly
 - Carcinoma (60%)
 - Diverticular disease (20%)
 - Volvulus (5%)
 - Colitis (e.g., ischemic, radiation)
 - Crohn's disease
 - Foreign bodies
- Functional, nonmechanical:
 - Paralytic ileus (e.g., electrolyte abnormalities, injury)
 - Pseudo-obstruction (i.e., Ogilvie syndrome [e.g., operative and nonoperative trauma] 11%)

ETIOLOGY

- Obstruction leads to proximal dilatation of intestines due to swallowed air and accumulated GI secretions, leading to increased intraluminal pressures.
- Retrograde peristalsis causes vomiting.
- Distended bowel becomes progressively edematous, and additional intestinal secretions cause further distention and 3rd spacing of fluid into the intestinal lumen.
- Obstruction may lead to intestinal wall ischemia (strangulated obstruction), resulting in increased aerobic and anaerobic bacteria, and methane and hydrogen production. Peritonitis, sepsis, and death may follow.
- Mortality is 100% in untreated strangulated obstruction, 8% if treated surgically within 36 hr, but 25% if surgery delayed after 36 hr.

SIGNS AND SYMPTOMS

History

- Previous surgery, malignancy, hernias, colonoscopy history, significant family history
- Abdominal pain:
 - Intermittent when early
 - Symptoms may be vague in elderly or altered patients
 - Constant with strangulated obstruction
- Vomiting:
 - Bile-stained emesis with proximal obstruction
 - Feculent emesis with distal obstruction
- Obstipation, constipation, diarrhea
- Stool caliber changes, weight loss

Physical-Exam

- Vital signs:
 - Tachycardia, hypotension with significant volume depletion
 - Fever with strangulation or perforation
 - Hypothermia with sepsis
- Abdominal exam:
 - Distention
 - Variable tenderness, often diffuse
 - Hyperactive and high-pitched bowel sounds when early; hypoactive when late
 - Consider ischemic or gangrenous bowel if pain out of proportion to exam.
 - Peritoneal signs indicate strangulation or perforation.
- Hernia (ventral, inguinal, femoral)
- Digital rectal exam:
 - Rectal mass
 - Blood in stool, gross or occult

Geriatric Considerations

- Abdominal pain variable in elderly, may be vague
- Nausea/vomiting and abdominal pain are common symptoms in elderly patients with acute myocardial infarctions:
 - Abdominal distention, obstipation, and colicky pain suggest GI cause.

Pediatric Considerations

- Intussusception:

- Leading cause of intestinal obstruction in infants
- Most common between 3 and 12 mo of age
- Incarcerated inguinal/umbilical hernia
- Malrotation with volvulus:
 - Can occur as early as 3–7 days of age
 - “Double bubble” sign seen on plain radiograph owing to partial obstruction of duodenum, resulting in air in stomach and in 1st part of duodenum
- Pyloric stenosis:
 - Progressive, projectile, nonbilious postprandial vomiting
 - Male/female ratio: 5:1 incidence
 - Onset usually 2–5 wk of age
- Other causes include duodenal atresia, Hirschsprung, and imperforate anus.

ESSENTIAL WORKUP

Careful history and physical exam

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC:
 - Leukocytosis common
- Electrolytes, BUN/creatinine, glucose:
 - Hypokalemia
 - Hypochloremic metabolic alkalosis
 - Prerenal azotemia
- Lactate
- Amylase/lipase
- Liver enzymes/function to exclude hepatic/biliary pathology
- Stool heme test
- Urinalysis
- Type and crossmatch
- PT/PTT
- ECG in patients at risk of coronary artery disease

Imaging

- Upright CXR:
 - Evaluate for pulmonary pathology.
 - Check for free air beneath diaphragm.
- Plain abdominal radiographs, supine and upright (75% sensitivity; 53% specificity):
 - Distended loops of bowel (normal small bowel < 3 cm in diameter)
 - Distended cecum > 13 cm indicates potential for perforation.
 - Air–fluid levels

- “String of pearls” sign if small bowel loops nearly completely fluid filled
- Less helpful for distinguishing strangulation
- Abdominal CT:
 - Sensitivity:
 - 90% for SBO; 91% for LBO
 - Detects neoplastic causes and stages malignancy
 - Effective in defining location of obstruction
 - More helpful than plain radiographs in identifying early strangulation (with IV contrast)
 - Exclude other incidental findings/causes
 - Has decreased use of contrast enemas due to ease of use
- MRI:
 - Sensitivity approached that of CT
 - Availability variable
- US:
 - More sensitive and specific than plain films for SBO but not as accurate as CT

Diagnostic Procedures/Surgery

Upper GI/barium enemas/endoscopy:

- If carcinoma or mass lesion suspected as cause
- Use decreased with availability of CT scan
- May be painful or difficult in sick patients

DIFFERENTIAL DIAGNOSIS

- Paralytic ileus
- Pseudo-obstruction (Ogilvie)
- Perforated ulcer
- Pancreatitis
- Cholecystitis
- Colitis
- Mesenteric ischemia



TREATMENT

PRE HOSPITAL

Establish IV access for patients with dehydration, vomiting, or significant abdominal pain.

INITIAL STABILIZATION/THERAPY

- ABCs
- 0.9% normal saline (NS) or lactated ringers (LR) IV fluid resuscitation for

significant volume depletion and strangulated or perforated bowel:

- Adults: 1 L bolus
- Peds: 20 mL/kg bolus
- Correct electrolyte abnormalities, especially hypokalemia.

ED TREATMENT/PROCEDURES

- IV fluids (isotonic saline or lactated Ringer's)
- Nasogastric tube (NGT)
- Foley catheter to monitor urine output
- Surgical consultation
- Antibiotics for suspected strangulated/perforated bowel:
 - Antibiotic choices should cover gram-negative aerobic and anaerobic organisms:
- Analgesics
- Antiemetics
- Treat underlying etiology, appropriate steroids for inflammatory bowel disease, radiation enteritis

MEDICATION

- Antibiotic choices (broad spectrum, for suspected ischemia):
 - Combination therapy:
 - Metronidazole (Flagyl): 1 g IV, then 500 mg IV q6h (peds: 7.5–30 mg/kg/24h IV div. q6–8h)
 - Ciprofloxacin (Cipro): 400 mg IV q12h
 - Ceftriaxone (Rocephin): 1–2 g (peds: 25–75 mg/kg/d IV up to 2 g div. q12–24h) IV q24h
 - Single therapy:
 - Piperacillin–tazobactam (Zosyn): 3.375 g (peds: 150–400 mg/kg/24h IV div. q6–8h) IV q4–6h
 - Ampicillin–sulbactam (Unasyn): 1.5–3 g (peds: 100–400 mg/kg/24h IV div. q6h) IV q6h
 - Meropenem (Merrem): Adult: 1 g (peds: 60–120 mg/kg/24h IV q8h) IV q8h
 - Imipenem–cilastatin (Primaxin): 250–1,000 mg (peds: 50–100 mg/kg/24h IV q6–12h) IV q6–8h
- Analgesics:
 - Morphine: 2–10 mg/dose (peds: 0.1–0.2 mg/kg IV/IM/SC q2–4h) IV/IM/SC q2–6h PRN
- Antiemetics:
 - Ondansetron (Zofran): 4 mg (peds: 0.1 mg/kg IV div. q8h) IV q4–8h PRN
 - Promethazine (Phenergan): 12.5–25 mg (peds: > 2 yr: 0.25–1 mg/kg/d IV/IM/PR div. q4–6h PRN) IV/IM/SC q4h



FOLLOW-UP

DISPOSITION

Admission Criteria

All patients with suspected/confirmed intestinal obstruction should be admitted with early surgical consultation.

Discharge Criteria

Normal lab/radiology results with resolution of symptoms and no further suspicion for intestinal obstruction.

Issues for Referral

Surgery consult for patients with suspected bowel obstruction

FOLLOW-UP RECOMMENDATIONS

Discharged patients:

- Normal lab and radiologic studies
- Timely appointment for re-evaluation
- Explicit instructions detailing signs/symptoms to return to emergency department

PEARLS AND PITFALLS

- Carefully examine patient with history of vomiting for incarcerated hernias.
- Failure to diagnose strangulated bowel obstruction:
 - Symptoms potentially vague in very old and very young and in altered patients
- Failure to adequately replete fluid losses and electrolyte imbalances

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Abdominal Pain
- Gastric Outlet Obstruction
- Pyloric Stenosis
- Vomiting



CODES

ICD9

- 560.9 Unspecified intestinal obstruction
- 560.81 Intestinal or peritoneal adhesions with obstruction (postoperative) (postinfection)
- 560.89 Other specified intestinal obstruction

ICD10

- _ K56.5 Intestinal adhesions w obst (postprocedural) (postinfection)
- _ K56.60 Unspecified intestinal obstruction
- _ K56.69 Other intestinal obstruction

BRADYARRHYTHMIAS

Benjamin S. Heavrin



BASICS

DESCRIPTION

- Ventricular heart rate < 60 beats/ min:
 - Sinus bradycardia can be normal variant.
 - All other rhythms are pathologic.
- May be asymptomatic or have hypotension, altered mental status, fatigue, nausea, syncope.
- Treatment varies based on ECG findings and clinical status.

ETIOLOGY

- Idiopathic:
 - Healthy athletes
- Intrinsic cardiac disorders:
 - Sinus node dysfunction such as sick sinus syndrome (may alternate with tachycardia)
 - Atrioventricular block:
 - Junctional or ventricular escape rhythm
 - Infiltrative disease:
 - Amyloidosis, sarcoidosis, hemochromatosis
 - Collagen vascular disease:
 - Systemic lupus erythematosus (SLE), scleroderma, rheumatoid arthritis
 - Anatomic abnormalities:
 - Congenital, postsurgical, post-transplant, postradiation
 - Muscular disorders:
 - Myotonic muscular dystrophy
 - Trauma with myocardial contusion
- Extrinsic disorders:
 - Cardiac injury and infarction:
 - RCA infarction can cause sinus bradycardia.
 - LAD infarction can cause high-grade block.
 - Acidemia
 - Medication and toxin effects:
 - β -Blockers, calcium channel blockers, digoxin, clonidine, antiarrhythmics, lithium, organophosphate
 - Electrolyte abnormalities:
 - Hypo-/hyperkalemia, hypoglycemia, hypo-/hypercalcemia, hypermagnesemia

- Vital sign abnormalities:
 - Hypoxia, hypothermia, hypotension, HTN
- Endocrine abnormalities:
 - Hypothyroidism
- Infectious disease:
 - Lyme disease, Chagas disease, diphtheria, endocarditis, myocarditis
- Neurologic disorders:
 - Increased intracranial pressure, increased vagal tone, carotid sinus hypersensitivity, spinal cord injury
 - Can be triggered by micturition, defecation, coughing, vomiting, ocular pressure, or other Valsalva maneuvers

Pediatric Considerations

Hypoxia is the most common etiology in children.

Pregnancy Considerations

Maternal SLE can result in congenital complete heart block.



DIAGNOSIS

SIGNS AND SYMPTOMS

- Often asymptomatic
- Lightheadedness, confusion, fatigue, decreased level of consciousness
- Dyspnea, cyanosis, pallor
- Chest pain/pressure, diaphoresis
- Hypotension
- Syncope
- Hypothermia
- Cardiac arrest

History

- Medication changes, especially cardiac
- Urine output:
 - Hypokalemia with diuretics
 - Hyperkalemia with renal failure
- Trauma:
 - Intracranial injury
 - Myocardial contusion
- Activity at time of symptom onset:
 - Increased vagal tone

Physical-Exam

- Respiratory status
- Perfusion status, pulses
- Regular vs. irregular cardiac rhythm
- Mental status, thorough neuro exam
- Body habitus, skin/hair/nails
- Temperature

ESSENTIAL WORKUP

- ECG and continuous cardiac monitoring
- Pulse oximetry
- BP monitoring
- Glucose and electrolytes

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Serum glucose
- Serum electrolytes
- BUN and creatinine
- Cardiac enzymes
- Digoxin level
- Thyroid function tests
- ANA, RF, other rheumatologic testing
- Lyme titers
- Iron levels

Imaging

- CXR
- CT head if patient has altered mental status

Diagnostic Procedures/Surgery

EKG:

- Sinus bradycardia:
 - P wave before every QRS, QRS after every P wave, usually narrow QRS
- Sinoatrial block: Abnormal conduction between sinus node and atrium
- Sinus arrest:
 - No sinus activity, no P waves
- Atrioventricular block: Abnormal conduction between atria and ventricles:
 - 1st degree: PR > 0.2 sec, every P wave conducts a QRS complex
 - 2nd-degree type I, Mobitz I, Wenckebach: Progressive prolongation of PR interval with eventual dropped QRS, grouped beats
 - 2nd-degree type II, Mobitz II: Stable PR interval and intermittent dropped QRS, high risk of degeneration into 3rd-degree block

- 3rd-degree, complete heart block: Complete dissociation of atrial and ventricular activity, constant P-P interval and constant R-R interval, but no relation between the 2, unstable rhythm
- Junctional rhythm:
 - Loss of atrial conduction, AV pacemaker “escapes” at 40–60 bpm
 - Retrograde P waves may occur before, during, or after QRS, and QRS can be any duration
- Idioventricular rhythm:
 - Loss of both SA and AV nodal activity, bundle of His or Purkinje network takes over at 30–40 bpm
 - QRS always > 0.12 sec
 - Preterminal rhythm

DIFFERENTIAL DIAGNOSIS

- Normal variant
- Cardiac ischemia
- Medication toxicity
- Pacemaker malfunction
- Hypoxia
- Hypothermia
- Electrolyte abnormality
- Renal failure
- Hypothyroidism
- Infection
- Rheumatologic disease
- Neuromuscular disease
- Increased intracranial pressure
- Myocardial contusion



TREATMENT

PRE HOSPITAL

- Treat the patient, not the heart rate
- Oxygen:
 - For all patients, especially children
- If hypothermic, warm the patient and give magnesium:
 - Do NOT pace; move patient gently as rough handling can induce v-fib.
- Atropine or epinephrine:
 - Only with hypotension or altered mental status
 - Often ineffective or harmful in 3rd-degree block
- Transcutaneous pacing:
 - If other measures ineffective

INITIAL STABILIZATION/THERAPY

- ABCs
- Oxygen therapy
- Apply pacing pads and continuous cardiac monitoring
- IV access

ED TREATMENT/PROCEDURES

- Asymptomatic bradycardia:
 - Monitor while continuing workup
- Symptomatic or unstable bradycardia:
 - Oxygen
 - Atropine:
 - Symptomatic sinus bradycardia and symptomatic 1st- and 2nd-degree type I AV blocks
 - Usually ineffective for high-grade AV blocks
 - Epinephrine
 - Transcutaneous pacing
 - Transvenous pacing if transcutaneous pacing unsuccessful
- Find and treat underlying cause:
 - Hypoglycemia:
 - D50
 - Hypocalcemia:
 - Calcium gluconate
 - Hypercalcemia:
 - NS +/- Lasix
 - β -Blocker or calcium channel blocker overdose:
 - Glucagon, calcium gluconate, insulin, D50, intralipid emulsion
 - Hyperkalemia:
 - IV calcium, insulin with D50, albuterol, bicarb if acidotic, Lasix, Kayexalate, dialysis
 - Hypokalemia:
 - Potassium
 - Digoxin toxicity:
 - Digibind (Digoxin immune Fab)
 - MI:
 - ASA, Plavix, heparin, statin, cath lab
 - Hypothyroidism:
 - Levothyroxine
 - Hypothermia:
 - Warm O₂, warm IVF, Bair Hugger, blankets, warming lights, consider warm bladder and gastric irrigation, cardiopulmonary bypass
 - Infection:
 - Targeted antibiotics, antivirals, or antifungals

- Myocardial contusion:
 - Supportive care
- Increased intracranial pressure:
 - Mannitol, neurosurgical consult
- Pacemaker malfunction:
 - Interrogate pacemaker, cardiology consult
- Idiopathic:
 - Cardiology consult for ICU admission and pacemaker placement

MEDICATION

- Atropine: 0.5–1 mg (peds: 0.02 mg/kg; min. 0.1 mg) IV q3–5 min; max. 3 mg or 0.04 mg/kg
- Calcium gluconate: 1,000 mg (peds: 60 mg/kg) IV q3–5min, max. 3 g
- D50: 1–2 amps (peds: D10 or D25 2–4 mL/kg) IV
- Digoxin immune Fab: Dose varies with amount of digoxin ingested, average 6 vials (peds: Average dose, 1 vial) IV bolus; see package insert
- Epinephrine: 0.1–0.5 mg (peds: 0.01–0.03 µg/kg/min) IV q3–5min; infusion 2–10 µg/min (peds: 0.1–1 µg/kg/min) IV
- Glucagon: 3–5 mg (peds: 0.05 mg/kg) IV, can repeat once; infusion 1–5 mg/h (peds: 0.07 mg/kg/h) IV for BB or CCB overdose
- Insulin regular: 10 U (peds: 0.1 U/kg) IV × 1 with glucagon for BB or CCB overdose. Higher doses may be appropriate after tox. consult.

First Line

Atropine, epinephrine, pacing

Second Line

Treatment for specific disorders



FOLLOW-UP

DISPOSITION

Admission Criteria

- ICU:
 - Hemodynamically unstable bradycardia
 - 2nd-degree type II or 3rd-degree block
 - Transcutaneous or transvenous pacer
 - Pressors
 - Acute myocardial infarction or ischemia
- Telemetry:
 - Hemodynamically stable bradycardia

Discharge Criteria

Asymptomatic sinus bradycardia

Issues for Referral

- All patients without existing primary care physicians should be referred to a generalist for follow-up as needed.
- 1st- and 2nd-degree type I AV block need cardiology referral.
- Severe endocrine, rheumatologic, infectious, renal, or neurologic disorders require appropriate specialty referral.

FOLLOW-UP RECOMMENDATIONS

- Minor lab abnormalities that do not require admission require PCP follow-up.
- All patients except asymptomatic sinus bradycardia require cardiology follow-up.
- Specific disorders require appropriate specialty follow-up.

PEARLS AND PITFALLS

- Asymptomatic sinus bradycardia is the ONLY potentially “normal” bradycardia. All others require treatment or follow-up.
- O₂, O₂ sat, IV, ECG, cardiac monitor for all patients.
- Pediatric bradycardia is likely secondary to hypoxia.
- Have pacing pads available for all symptomatic patients.
- The most important treatment targets the underlying cause.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Acute Coronary Syndrome
- β -Blocker Overdose
- Calcium Channel Blocker Overdose
- Digoxin Overdose
- Hyperkalemia
- Hypothermia
- Pacemaker



CODES

ICD9

- 427.81 Sinoatrial node dysfunction
- 427.89 Other specified cardiac dysrhythmias

ICD10

- I49.5 Sick sinus syndrome
- I49.8 Other specified cardiac arrhythmias

BRONCHIOLITIS

Suzanne Schuh

BASICS

DESCRIPTION

Lower respiratory tract infection by airway inflammation and bronchoconstriction with wheezes/tachypnea and respiratory distress and upper respiratory prodrome

ETIOLOGY

- Respiratory syncytial virus (RSV) in 85–90% of cases
- Influenza
- Parainfluenza
- Adenovirus
- Normally occurs during the winter months

DIAGNOSIS

SIGNS AND SYMPTOMS

- Age < 2 yr (usually 1 yr or younger)
- Nasal congestion, often with marked rhinorrhea
- Cough, sometimes associated with vomiting
- Wheezing
- Crackles, rhonchi
- Respiratory distress manifested by tachypnea, nasal flaring, retractions, grunting. Often progressive over a period of 1–3 days
- Fever usually < 39.5°C
- Hypoxemia may be present (usually mild). Cyanosis rare
- Decreased fluid intake common, frank dehydration uncommon
- Apnea may occur, particularly in young infants with history of prematurity
- Synagis, an RSV specific immunoglobulin, may be administered IM monthly during winter months in high-risk children. This reduces risk of RSV infection.

ESSENTIAL WORKUP

- Clinical diagnosis
- Defining viral cause may be useful for cohorting in hospital if admitted.
- Assess ventilation clinically.
- Pulse oximetry:
 - Confirms proper oxygenation on continuing basis
 - Follows trends over the course of illness

DIAGNOSIS TESTS & NTERPRETATION

Lab

- Most patients need no specific tests beyond oximetry.
- Nasopharyngeal aspirate/wash:
 - Viral cultures
 - Fluorescent antibodies
 - Commercial kits are available.
 - Consider when:
 - Clinical symptoms suggestive of other cause (pertussis, chlamydia)
 - Critically ill child
 - Febrile child < 3 mo old with bronchiolitis (consider UTI as coexistent cause of fever)
 - Coexisting signs suggesting significant bacterial infection (positive aspirate does not exclude coexisting significant bacterial infection but such infections are uncommon)
 - Bronchopulmonary dysplasia or chronic lung disease
 - Coexistent cardiac disease
 - Prematurity
 - Other conditions warranting antiviral therapy (rare)

Imaging

CXR:

- Usually hyperinflation, airway disease, atelectasis, variable infiltrate:
 - Atelectasis in young infants indicates more severe disease.
- Minority have airway + airspace disease; pneumonia usually viral
- Rarely changes management acutely
- Consider when:
 - Need to exclude other diagnoses such as CHF, aspiration, congenital airway anomaly (rare)
 - Chronic course with lack of resolution over 2–3 wk
 - Critically ill infants with impending respiratory failure
 - Atypical presentation in toxic or deteriorating child
 - Not routinely indicated in typical clinical presentation

Diagnostic Procedures/Surgery

- Septic workup in febrile bronchiolitis < 28 days of age if respiratory status permits
- In febrile infants 1–3 mo of age, consider catheterized urine culture
- Oximetry during significant distress

DIFFERENTIAL DIAGNOSIS

- Asthma/recurrent virus-induced wheezing: Severe bronchiolitis requiring hospitalization, and family history of atopy are risk factors for future asthma.
- Pertussis: No respiratory distress between coughing spasms, no wheezing
- Bacterial pneumonia: Often toxic appearance, no wheezing, isolated airspace

- disease (consolidation) with no airway abnormality on chest radiograph
- Foreign body: Sudden onset of symptoms, usually afebrile
- CHF: Pre-existing clinical red flags (failure to thrive [FTT], feeding problems)



TREATMENT

PRE HOSPITAL

ALERT

- Young infants have limited respiratory reserve and decompensate rapidly with little warning.
- Monitor cardiorespiratory status and oxygenation.
- Supplemental oxygen if saturation $< 90\text{--}92\%$ (sea level) and/or severe distress
- Watch for apneic pauses:
 - Greatest risk of developing high-risk outcomes in children < 7 wk, weight < 4 kg, respiratory rate $> 80/\text{min}$, heart rate $> 180/\text{min}$, comorbidities, premature
 - Bag-mask ventilation if recurrent apneas

INITIAL STABILIZATION/THERAPY

- Pediatric advanced life support: Airway, ventilation, and fluid hydration
- Emergent intubation if recurrent apneas, impending respiratory failure

ED TREATMENT/PROCEDURES

- Supplemental oxygen if oxygen saturation $< 90\text{--}92\%$ (sea level)
- Parenteral hydration if dehydration or severe respiratory distress. Many children may improve their intake once respiratory status has improved.
- Many children with bronchiolitis do not benefit from pharmacotherapy.
- Bronchodilators (albuterol, racemic epinephrine, l-epinephrine, levalbuterol):
 - Should not routinely be used alone without determination of efficacy
 - Some clinicians administer on trial basis with 2–3 consecutive treatments in those with moderate to severe distress and continue as part of management if there is a clear decrease in the work of breathing.
 - Often utilized in significantly ill children
- Steroids:
 - On their own do not change clinical course or hospitalizations in the majority of patients without prior atopic or family history.
 - 2 doses of 1:1,000 l-epinephrine 30 min apart in the ED + 6 daily doses of oral dexamethasone may be useful in moderate to severe distress—reduces admissions by 35% by day 7, shortens time to discharge and duration of symptoms
 - Conflicting evidence with another recent dexamethasone trial showing no benefit when used alone—synergy between steroids and epinephrine likely

critical for efficacy

- Often used empirically in children with past or family history of atopy. Prednisolone common for this usage.
- Albuterol–dexamethasone combination efficacy not confirmed in a big trial.
- Bronchodilators alone after discharge not effective unless there was demonstrated effectiveness prior to discharge.
- Antibiotics:
 - Not generally indicated since viral etiology
 - Consider if associated signs of focal bacterial disease (otitis, focal pneumonia), radiographic evidence of isolated lobar consolidation without airway disease (usually bacterial pneumonia rather than bronchiolitis), significant toxicity, sepsis
- Ribavirin:
 - No role in ED management and rarely used in the inpatient setting

MEDICATION

- Albuterol: 2.5 mg/3 mL, 2–3 doses via nebulizer or 400 mcg via MDI/spacer 20–30 min apart in the ED. A therapeutic trial can be considered but continue only if there is a clear improvement in the work of breathing. Does not change overall disease outcomes.
- Levalbuterol: 1.25 mg/dose, 2–3 doses via nebulizer, 20–30 min apart in the ED (see above).
- l-epinephrine: 3 mL (1:1,000 solution), 2 doses via nebulizer 30 min apart in the ED or with
- Dexamethasone: 1 mg/kg/dose PO in the ED, then 0.15 mg/kg daily for 5 days
- Prednisolone (15 mg/5 mL): 1–2 mg/kg/d PO BID/3–5 d
- Comment 1: Most children require no medications. Bronchodilators alone rarely change outcomes. Initial trial of albuterol should be extended only if clear clinical improvement. Epinephrine–dexamethasone combination shown to decrease hospitalizations by day 7 of illness and may warrant consideration.
- Comment 2: Although no trial to date has identified any pharmacotherapeutic agent to change the course of the disease, a recent meta-analysis found that (a) inhaled epinephrine alone and epinephrine + oral dexamethasone appear to have half the odds of hospitalization compared to placebo and (b) salbutamol does not reduce hospitalizations in bronchiolitis.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Need for supplemental oxygen (oxygen saturation on room air is < 90–92% at sea

level)

- Some institutions have developed protocols that allow full-term, stable children >6 months of age, who are well hydrated, have compliant parents, and good follow-up to be discharged on minimal oxygen after a prolonged period of observation in the ED.
- Inability to self-hydrate
- Marked increase work of breathing (tachypnea with retractions or accessory muscle use)
- Apnea
- Severe underlying chronic lung disease or cardiac disease
- Persistent marked respiratory distress 4 hr after a trial of epinephrine and dexamethasone
- Significant comorbidity/suspicion of alternative diagnosis/underlying systemic disease/immunodeficiency or immunosuppressive therapy
- Strongly consider in infants <7 wk, weight <4 kg, respiratory rate >80/min, heart rate >180/min, comorbidities, or prematurity
- Caretaker noncompliant or unable to monitor child closely

Discharge Criteria

- Feeding reasonably well
- Acceptable room air saturation (see above)
- Absence of significant respiratory distress
- Follow-up available within 24 hr
- Compliant home environment
- Discharge instructions:
 - Symptoms may persist for 2–3 wk
 - Frequent small feeds
 - Bronchodilators after discharge not uniformly beneficial

FOLLOW-UP RECOMMENDATIONS

Because of the progressive nature of bronchiolitis close follow-up is required, particularly early in the illness alerting parents to the likelihood of worsening respiratory distress, dehydration, and apnea.

PEARLS AND PITFALLS

Infants with bronchiolitis often present with respiratory distress associated with hypoxia, dehydration, and/or apnea. Aggressive monitoring may be warranted.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Asthma, Pediatric



CODES

ICD9

- 466.1 Acute bronchiolitis
- 466.11 Acute bronchiolitis due to respiratory syncytial virus (RSV)
- 466.19 Acute bronchiolitis due to other infectious organisms

ICD10

- J21.0 Acute bronchiolitis due to respiratory syncytial virus
- J21.1 Acute bronchiolitis due to human metapneumovirus
- J21.9 Acute bronchiolitis, unspecified

BRONCHITIS

Robin R. Hemphill

BASICS

DESCRIPTION

- Hyperemia and edema of the mucous membranes
- Production of mucopurulent exudates
- Impairment of the productive function of the cilia, lymphatics, and phagocytes
- Airway obstruction from:
 - Edema
 - Secretions
 - Bronchial muscle spasm

ETIOLOGY

- Viral infections are the primary cause of bronchitis:
 - Parainfluenza
 - Influenza A and B
 - Respiratory syncytial virus
 - Human metapneumovirus
 - Echovirus
 - Coronavirus
 - Adenovirus
 - Coxsackievirus
 - Rhinovirus
 - Measles and herpes viruses (can cause severe viral bronchitis)
- Particularly severe or long-lasting bronchitis:
 - *Mycoplasma pneumoniae*
 - *Chlamydia pneumoniae*
 - *Bordetella pertussis*:
 - Rates of pertussis are increasing, even in the fully immunized population (little protection remains after 10 yr).
- Other bacteria have not been conclusively proven to cause bronchitis except in those with chronic lung disease.

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Complaints that may precede upper respiratory tract infection (URTI) symptoms:

- Malaise
- Chills
- Myalgias
- Coryza (rhinitis)
- Sore throat
- Onset of URTI symptoms:
 - Mild dyspnea
 - Cough, initially dry and nonproductive
 - Cough, later becomes mucoid or mucopurulent
 - Chest pain or burning related to cough
 - Initial symptoms improve after 3–5 days, with 1–3 wk of residual cough and malaise

Physical-Exam

- Fever, not usually above 102°F (38.5°C)
- Tachypnea
- Mild hemoptysis
- Ronchi (wheezing)
- Rales (crackles)

ESSENTIAL WORKUP

- Influenza A and B testing if identification of these organisms is required for treatment or reporting
- Evaluate for pertussis:
 - Acute cough illness lasting 14 days or more in a person with paroxysmal cough, post-tussive vomiting, or inspiratory whoop
 - 14 days or more of cough within an outbreak setting

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Influenza A and B testing may help immediately confirm clinical suspicion.
- In most cases, no specific test will help make the diagnosis immediately.
- Viral or bacterial cultures are rarely helpful.
- CBC may show leukocytosis, but is a nonspecific finding.
- Pertussis may be confirmed using PCR testing, but diagnosis will be delayed.

Imaging

CXR:

- No evidence of consolidation
- Indications:
 - Shortness of breath
 - Hypoxia

- Chest pain
- Heart rate > 100 beats/min
- Respiratory rate ≥ 24 breaths/min
- Temperature ≥ 38°C
- Focal findings on chest exam
- Elderly patient with multiple comorbid conditions
- Hypoxia
- 14 days or more of cough

Diagnostic Procedures/Surgery

Pulmonary function tests are frequently abnormal.

DIFFERENTIAL DIAGNOSIS

- Acute and subacute < 8 wk:
 - Pneumonia
 - Reactive airway disease
 - Aspiration
 - Acute sinusitis
 - Bacterial tracheitis
 - Occupational exposure
- Chronic > 8 wk:
 - Asthma
 - GERD
 - Chronic bronchitis
 - Bronchiectasis
 - ACE inhibitor use
 - Bronchogenic carcinoma
 - Carcinomatosis
 - Sarcoidosis
 - Left ventricular failure
 - Aspiration syndrome
 - Psychogenic/habit



TREATMENT

PRE HOSPITAL

- Maintain adequate oxygenation
- Bronchodilators if wheezing is present

INITIAL STABILIZATION/THERAPY

- Aggressive initial management of these patients is seldom required.
- Administer oxygen if the patient is hypoxic.

- Fluids may be administered if the patient is dehydrated.

ED TREATMENT/PROCEDURES

- Bronchitis is usually a viral process, but may be bacterial and there is no practical test to distinguish between the 2:
 - Because this is usually a viral process, treatment is symptomatic:
 - Cough suppressants may be considered.
 - β -Adrenergic inhaler for patients with evidence of airflow obstruction
- Amantadine may be used in known outbreaks of influenza A, although local patterns of resistance should be reviewed.
- Oseltamivir (Tamiflu) and zanamivir (Relenza) may be considered in patients with recent onset of influenza.
- Antibiotics:
 - Generally, antibiotics are not indicated (even when secretions are purulent).
 - In healthy patients with no underlying lung disease, antibiotics may help some patients get better slightly faster, but weighed against the many people it does not help, cost, side effects, and resistance, antibiotics are not recommended.
 - Consider use in those patients who have recurrence of fever after initial improvement.
- Symptomatic control with antipyretics and analgesics
- Although patients should be encouraged to stop smoking, the use of tobacco is not an indication for antibiotics unless the patient has a known history of emphysema.

ALERT

Be aware that respiratory viruses can cause significant morbidity in immunocompromised patients and their care should be discussed with their primary care physician.

Pediatric Considerations

- Aggressive initial management of these patients is seldom required.
- Administer oxygen if the patient is hypoxic.
- Fluids may be administered if the patient is dehydrated.
- Repeated bouts in children should lead to referral for complete evaluation of the respiratory tract.

MEDICATION

- Albuterol Inhaler may be used for those with evidence of airflow obstruction.
- Amantadine: 100 mg/d PO, must be given within 48 hr of symptom onset
- Oseltamivir (Tamiflu) and zanamivir (Relenza) within 48 hr of symptom onset for influenza-related bronchitis:
 - Zanamivir: 10 mg inhalation q12h (peds: >7 yr 10 mg or 2 inhalations q12h) \times 5 d

– Oseltamivir: 75 mg PO BID (peds: 2 mg/kg) × 5 d

- Erythromycin should be given to proven cases of pertussis and to household contacts of those with proven pertussis.
- Yearly influenza vaccinations should be encouraged in health care providers and in the high-risk populations (elderly, immunocompromised, chronic lung disease).

Geriatric Considerations

- Use of acetaminophen rather than aspirin for analgesia
- Antibiotic considerations are the same as in adults.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Underlying significant cardiopulmonary compromise
- Significant hypoxia
- Ill patient with unclear diagnosis

Discharge Criteria

- No pulmonary compromise should be present.
- Instruct patients, particularly high-risk patients, to return if no improvement or worsening of symptoms occurs.
- Bed rest
- Fluids
- Aspirin or acetaminophen

FOLLOW-UP RECOMMENDATIONS

- No follow-up is needed in those patients that improved.
- Patients should be instructed to return to the ED for onset of shortness of breath and should see their doctor if not improved after 2–3 wk.

PEARLS AND PITFALLS

Patients with high fever or significant pulmonary symptoms should be evaluated for pneumonia.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Cough
- Dyspnea
- Pneumonia, Adult
- Pneumonia, Pediatric



CODES

ICD9

- 466.0 Acute bronchitis
- 466.11 Acute bronchiolitis due to respiratory syncytial virus (RSV)
- 490 Bronchitis, not specified as acute or chronic

ICD10

- J20.4 Acute bronchitis due to parainfluenza virus
- J20.5 Acute bronchitis due to respiratory syncytial virus
- J20.9 Acute bronchitis, unspecified

BRUGADA SYNDROME

Edward Ullman • John W. Hardin



BASICS

DESCRIPTION

- Inherited heart disease due to mutations of cardiac Na⁺ channels without structural abnormalities
- Very high risk of sudden cardiac death in the form of ventricular fibrillation
- 2-yr mortality ~ 30%
- Suspected in 40–60% of what was previously known as idiopathic ventricular fibrillation
- Higher prevalence in men of Southeast Asian descent, but all ages, genders, races can be affected

ETIOLOGY

- Inherited:
 - Autosomal dominant in 50%
 - Variable penetration
- Cardiac Na⁺ channel:
 - >70 described mutations
 - Variable penetrance
 - SCN5A mutations account for 20%



DIAGNOSIS

SIGNS AND SYMPTOMS

- Most commonly presents as episodes of sudden death (in ventricular fibrillation) or as syncope or near-syncope in self-terminating episodes of polymorphic ventricular tachycardia

History

- HPI:
 - Episodes of syncope or near-syncope
 - Palpitations
 - Cardiac arrest
 - Concomitant illness, fever, metabolic, or electrolyte disorders
 - Cocaine use
 - TCA and psychotropic drugs
 - Nocturnal agonal respirations
- Family history:

- History of drowning due to syncope or dysrhythmias while submerged
- History of early and/or sudden cardiac death
- Known relatives with Brugada syndrome

Physical-Exam

- Complete physical exam, with special attention to other causes of syncope or dysrhythmia:
 - Abnormal heart sounds
 - Pectus excavatum (normal variant EKG changes)
 - Athletes
 - Pacemaker in situ

ESSENTIAL WORKUP

- A 12-lead EKG is imperative
- Detailed HPI and family history
- Toxicology screen

DIAGNOSIS TESTS & INTERPRETATION

EKG Diagnostic Criteria

- Basic:
 - Right bundle branch block (RBBB) or Incomplete right bundle branch block (IRBBB) with ST-segment elevation in the right precordial leads only
- Morphology of QRS-T in V1–V3
 - ST-elevation
 - Sometimes only in V1 and very rarely in V3
- Type 1 (coved pattern):
 - Initial ST-elevation ≥ 2 mm, slowly descending, concave with respect to the isoelectric line
 - Negative symmetric T-wave
 - No clear r'
 - QRS duration mismatch between V1 and V6
- Type 2 (saddle back pattern):
 - High r' take-off is ≥ 2 mm with respect to the isoelectric line
 - Followed by ST-elevation – convex with respect to the isoelectric line
 - QRS duration mismatch between V1 and V6

Lab

- Serum:
 - Chemistries to rule out underlying electrolyte causes of dysrhythmia or syncope
 - Cardiac biomarkers (troponin, CK-MB) for ischemia
 - D-dimer in the appropriate population (Wells, PERC) if considering

- pulmonary embolism
- CBC for evaluation of syncope

Imaging

- CXR:
 - Evaluate for cardiomegaly
- CT-angiogram of the chest:
 - If considering pulmonary embolism as a cause

Diagnostic Procedures/Surgery

- Electrophysiology lab
 - Drug challenge with sodium channel blockers (type 1a and 1c)
- AICD placement
 - Mortality reduced to 0% in this group

DIFFERENTIAL DIAGNOSIS

- Syncope:
 - Primary cardiogenic
 - Vasovagal
 - Neurogenic
 - Hypovolemia
 - Pregnancy
- Dysrhythmias:
 - Paroxysmal atrial fibrillation
 - Atrial fibrillation with rapid ventricular response
 - Wolff–Parkinson–White syndrome
 - Lown–Ganong–Levine syndrome
 - Ventricular tachycardia
 - Multifocal atrial tachycardia
 - Spontaneously terminating ventricular fibrillation
 - Symptomatic bradycardia
 - High-grade heart blocks
 - Long QT syndromes
 - Overdose especially TCA
- EKG mimics:
 - Isolated RBBB
 - Athletes
 - Septal hypertrophy
 - Pectus excavatum
 - Arrhythmogenic right ventricular dysplasia
 - STEMI
- Other systemic illness:
 - Electrolyte disturbances

- Pericarditis
- Myocarditis
- Myopericarditis
- Pulmonary embolism



TREATMENT

PRE HOSPITAL

- Airway, breathing, and circulation management
- ACLS protocol for arrest/dysrhythmias

INITIAL STABILIZATION/THERAPY

- Airway, breathing, and circulation management
- Start or continue ACLS algorithms

ED TREATMENT/PROCEDURES

- Cardiac monitoring at all times
- Cardiology consult:
 - For electrophysiology evaluation
- Defibrillator/pacing pads
- Correct underlying disease processes:
 - Replete electrolytes
 - Correct metabolic derangements
 - Asymptomatic patients' management controversial. Current consensus states EP evaluation but not supported in literature.

MEDICATION

- ACLS medications per protocol
- Antiarrhythmics usually not helpful

Pediatric Considerations

- PALS/defibrillation
- Appropriate weight-based medication and energy (joule) adjustments



FOLLOW-UP

DISPOSITION

Admission Criteria

- EKG findings concerning for Brugada in the appropriate clinical setting
- Unexplained syncope
- Inability to obtain rapid cardiology follow-up

- Ongoing dysrhythmias even if they are spontaneously terminating

Discharge Criteria

- Hemodynamically stable
- Asymptomatic
- Cardiology clearance
- Appropriate AICD intervention after an event
 - After interrogation of AICD

FOLLOW-UP RECOMMENDATIONS

- All patients with concerning EKG findings and history should be referred to EP for additional evaluation

PEARLS AND PITFALLS

- Consider in any episodes of sudden cardiac death or syncope, especially in the setting of family history of the same
- The EKG is diagnostic showing RBBB or IRBBB with ST-segment elevation in the right precordial leads only
- Beware of EKG mimics which can have similar presentation – typically mimics will have concordant QRS duration in V1 and V6 whereas Brugada QRS changes should be isolated to V1–V3
- Have a low threshold for cardiology consultation given high risk of death
- AICD implantation is definitive treatment, almost eliminating risk of sudden cardiac death
- Antiarrhythmic agents have not been found to be helpful
- The Brugada pattern may be “unmasked” in systemic illness, even if resolution of the EKG occurs, the patient should still have EP follow-up

ADDITIONAL READING

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ICD9

746.89 Other specified congenital anomalies of heart

ICD10

- I49.8 Other specified cardiac arrhythmias
- Q24.8 Other specified congenital malformations of heart

BUNDLE BRANCH BLOCKS

Annette Dorfman • James Scott



BASICS

DESCRIPTION

- Blockage of intraventricular electrical impulses through the right and left bundles
- Complete bundle branch block:
 - Absence or delay of conduction down one bundle, with normal conduction down the other bundle
 - Affected ventricle depolarizes from muscle to muscle in a slower and more disorganized fashion.
 - Quasi-random signal (QRS) complex at 120 msec or longer
- Incomplete bundle branch block:
 - Delayed depolarization, but less than complete bundle branch block
 - QRS complex duration 100–120 msec
- Right bundle branch block (RBBB):
 - Delayed depolarization of the right ventricle
- Left bundle branch block (LBBB):
 - Delayed depolarization of the left ventricle
 - LBBB can be caused by delay of conduction in main left bundle or delay in both fascicles of the left bundle.
 - Causes early activation of the right side of the septum and the right ventricular myocardium (so explaining loss of “septal Q” on ECG)
 - Left bundle branches into 2 fascicles:
 - Left anterior fascicle: Initial septal activation proceeds inferiorly, anteriorly, and to the right.
 - Left posterior fascicle: Isolated blockage rare; activation begins in the midseptum and finishes in inferior and posterior walls.
- Bifascicular block:
 - RBBB with concomitant block of the left anterior or left posterior fascicle

ETIOLOGY

- Myocardial infarction
- Cardiomyopathy
- Hypertension
- Age-related fibrosis of Purkinje fibers
- Valvular disease
- Exercise induced
- Congenital/atrial septal defect
- Brugada syndrome (RBBB): Cause of sudden cardiac death in otherwise healthy

patients.

- Chagas disease (especially Central/South America)
- Postoperative, following cardiac surgery
- Drugs:
 - β -Blockers
 - Calcium blockers
 - Tricyclic antidepressants
 - Type Ia and Ic antiarrhythmics
 - Digitalis



DIAGNOSIS

SIGNS AND SYMPTOMS

- Asymptomatic
- RBBB: Split S_2 that persists with expiration
- LBBB: Reversed/paradoxical split S_2
- Syncope
- Chest pain

ESSENTIAL WORKUP

ECG:

- RBBB:
 - Complete: QRS complex ≥ 0.12 sec
 - Incomplete: QRS complex duration 0.10–0.12 sec
 - rsr' , rsR' , rSR' in V_1 or V_2 (*M* shape)
 - Wide and deep S-wave in V_5 – V_6
 - Brugada syndrome: RBBB and ST-segment elevation in V_1 – V_3
- LBBB:
 - Broad slurred R-waves in leads V_5 – V_6 , aVL, and I
 - Small/absent R-wave in V_1 – V_2 and deep S-waves
 - Absence of normal Q-waves in leads V_5 – V_6 and I
- Left anterior fascicular block:
 - QRS complex < 120 msec, axis 45° – 90°
 - Deep S-wave in leads II, III, aVF, qR in leads aVL and I
- Left posterior fascicular block:
 - QRS < 120 msec, axis $\geq 120^\circ$
 - RS-waves in leads I and aVL, qR in leads II, III, and aVF
 - Exclusion of other things causing right axis deviation (right ventricle overload, right ventricular hypertrophy, lateral infarction)

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Electrolytes if hyperkalemia, hypercalcemia are suspected
- Cardiac enzymes if ischemia is suspected

Imaging

- CXR:
 - May reveal cardiac enlargement or CHF
- Electrophysiologic testing:
 - Especially for unexplained syncope in patient with structural heart disease, as part of inpatient workup

DIFFERENTIAL DIAGNOSIS

- Ventricular tachycardia
- MI:
 - Criteria for diagnosing MI with LBBB (Sgarbossa criteria) include any of the following:
 - ST-segment elevation ≥ 1 mm concordant with QRS
 - ST-segment elevation ≥ 5 mm discordant with QRS
 - ST-segment depression ≥ 1 mm in leads V_1 – V_3
- Hyperkalemia
- Ventricular hypertrophy
- Drug effects (see “Etiology” section)



TREATMENT

PRE HOSPITAL

Cautions:

- Monitor: Difficult to diagnose from single lead
- Avoid confusing with ventricular tachycardia or ischemia, use.
- Treat patient; bundle branch block requires no specific therapy.

INITIAL STABILIZATION/THERAPY

- Standard treatment for symptoms of ischemia, dyspnea, and syncope
- Symptomatic bifascicular block and high-degree atrioventricular block:
 - Apply transcutaneous pacing pads to back and chest.
 - IV sedation and analgesia
 - Gradually increase current until capture is achieved.

ED TREATMENT/PROCEDURES

- Asymptomatic: None
- Thrombolysis or cardiac catheterization for symptoms suggestive of myocardial infarction and new bundle branch block

- Transvenous pacemaker indications:
 - Bifascicular block and type II 2nd- or 3rd-degree atrioventricular block
 - Alternating LBBB and RBBB



FOLLOW-UP

DISPOSITION

Admission Criteria

- Suspected myocardial ischemia
- Syncope
- Dysrhythmias
- Bundle branch block with high-degree atrioventricular block

Discharge Criteria

Asymptomatic or incidental finding of bundle branch block

Issues for Referral

At discharge, patient should be referred to cardiologist for evaluation of underlying disease.

FOLLOW-UP RECOMMENDATIONS

- Reassure patients that usually no treatment is needed.
- Instruct patient to return or call for help if:
 - Dizziness
 - Fainting
 - Palpitations

PEARLS AND PITFALLS

- Myocardial ischemia should be considered in all patients who develop new conduction abnormalities.
- Specific criteria can be used to diagnose cardiac ischemia in patients with a bundle branch block.

ADDITIONAL READING

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- Prineas RJ, Le A, Soliman EZ, et al. United States national prevalence of electrocardiographic abnormalities in black and white middle-age (45- to 64-year) and older (≥ 65 -year) adults (from the Reasons for Geographic and Racial Differences in Stroke Study). *Am J Cardiol.* 2012;109(8):1223–1228.
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CODES

ICD9

- 426.3 Other left bundle branch block
- 426.4 Right bundle branch block
- 426.50 Bundle branch block, unspecified

ICD10

- I44.7 Left bundle-branch block, unspecified
- I45.4 Nonspecific intraventricular block
- I45.10 Unspecified right bundle-branch block

BURNS

Gabriel Wardi • Anthony J. Medak

BASICS

DESCRIPTION

Burns represent an acute injury to the flesh.

ETIOLOGY

Burns can be classified into 7 categories

- Scalds: Hot liquids, grease, or steam
- Contact: Hot or cold surfaces
- Thermal: Fire or flames
- Radiation burns
- Chemical burns
- Electrical burns
- Friction burns: Road rash, rope burns

DIAGNOSIS

SIGNS AND SYMPTOMS

- Most burns will have external signs of trauma to the skin
- Inhalation injury
 - _ Facial burns, pharyngeal injection
 - _ Singed nasal hair/eyelashes
 - _ Carbonaceous sputum
 - _ Change in respiratory mechanics (wheezing, coughing, tachypnea)
- Electrical and chemical burns may have minimal external findings (entry/exit wounds)

History

- AMPLE history, source of fire (plastic, wood, chemical, etc.), the location and surroundings, any explosions
- Medical/surgical/social history, medications, allergies, tetanus immunization status
- Carbon monoxide (CO) poisoning (most common cause of death in fires) from exposure to wood-based fires/combustion:
 - _ Pulse oximetry unreliable in CO poisoning
- Cyanide poisoning from burning wool, silk, nylon, and polyurethane found in furniture/paper

Physical-Exam

- Focus on airway 1st, then secondary survey for concurrent injuries
- Evaluate the face, oropharynx, and nares for signs of inhalation injury
- Assess need for immobilization of cervical spine (explosion or falls)
- Eye exam for corneal burns
- Estimate severity of partial- and full-thickness burns by assessing size/depth of burn

Pediatric Considerations

Specific patterns of injury may indicate nonaccidental injury (immersion wounds, cigarette burns, etc.)

ESSENTIAL WORKUP

- Severity of the burn should be assessed by determining the size and depth of the burn
- Size is reported as percent involvement of total body surface area (TBSA) in 1 of 3 ways:
 - 1: Rule of nines (applies only to adults)
 - TBSA of body parts is estimated by multiples of 9%
 - Adult estimates of percentage of TBSA
 - Head and neck: 9
 - Arms: Right, 9; left, 9
 - Legs: Right, 18; left, 18
 - Trunk: Front, 18; back, 18
 - Perineum, palms: 1
 - In infants and children, the head contributes more to the percentage of TBSA and legs contribute less.
 - Infants/children
 - Head and neck: 18
 - Arms: Right, 9; left, 9
 - Legs: Right, 14; left, 14
 - Trunk: Front, 18; back, 18
 - 2: Lund and Browder chart; divides body into areas and assigns percentage of BSA based on age; produces more reliable results than rule of 9s
 - 3: Palm surface area; patient's palm and fingers represent ~1% of TBSA
 - Helpful in assessing irregular/scattered burns
 - Superficial or 1st-degree (epidermis only) when: Local erythema, minimal swelling/pain, no initial blisters (may occur in 2–3 days); healing occurs in several days, no scar
 - Partial-thickness or 2nd-degree burns (epidermis and dermis): Superficial partial-thickness or deep partial-thickness burns:
 - Superficial partial-thickness burns (epidermis and superficial dermis)
 - Often seen in scald burns

- Generally with blister formation, underlying skin is pink, moist, painful, good capillary refill, sensation intact
- Heals in 14–21 days, generally no scars
- Deep partial-thickness burns (epidermis and deep dermis)
 - Skin may be blistered, with white to yellow dermis; absent capillary refill/pain sensation
 - Heals via epithelialization within 3–12 wk, although may cause severe scarring and contractures requiring surgery
- Full-thickness or 3rd-degree burns (through epidermis and dermis)
 - Skin is charred, leathery, and pale, no blisters, sensation is absent
 - Will not heal spontaneously; will require surgical repair and skin grafting
- Full-thickness burns or 4th-degree burns (damage to underlying structures)
 - Full-thickness + involvement of underlying fascia, muscle, bone, and other tissues
 - Requires extensive débridement/grafting
 - Resultant disability

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Severe burns: CBC, serum electrolytes, glucose, BUN, creatinine, PT/PTT, type and crossmatch, and pregnancy test (if indicated)
- Blood gas with CO level for closed space or suspected inhalation exposures
- Cyanide level (if indicated)

Imaging

CXR

Diagnostic Procedures/Surgery

- Bronchoscopy to assess for inhalation injury
- ECG, especially in electrical burns, elderly patients

DIFFERENTIAL DIAGNOSIS

- Electrical injury
- Chemical injury
- Associated trauma or intoxication



TREATMENT

PRE HOSPITAL

- Stop the burning process, remove smoldering/contaminated clothes/jewelry
- Keep patient warm, cool affected areas

- Establish patent airway, frequent reassessment, start 100% oxygen
 - Early intubation for respiratory distress
- Early IV fluid therapy, especially if >20% TBSA
- Institute pain relief, with narcotics if possible
- Protect the wound with clean sheets
- Transport to burn center (for major burns) if transport time <30 min
- Spinal immobilization if mechanism is concerning

INITIAL STABILIZATION/THERAPY

- Airway control is paramount
 - Early intubation for patients with signs of upper airway involvement, significant nasolabial burns, or circumferential neck burns
- IV access, supplemental 100% oxygen, monitor, pulse oximetry
- Evaluation for concurrent injuries
- Provide adequate analgesia
- Early fluid resuscitation is essential

ED TREATMENT/PROCEDURES

- Fluid resuscitation: Partial- and full-thickness burns (>20% TBSA)
 - Parkland formula (not for pediatric patients): 4 mL of lactated Ringer solution (preferred) or normal saline (NS) per kilogram per percentage of BSA burned
 - 1/2 in the 1st 8 hr and the remaining 1/2 over the next 16 hr
 - Example: 70-kg patient with a 40% TBSA burn requires $4 \text{ mL} \times 70 \text{ kg} \times 40\% = 11,200 \text{ mL}$ over 24 hr, with 5,600 mL over 1st 8 hr or 700 mL/hr
 - After the initial resuscitation, burns with >20% TBSA should have IV fluid therapy guided by invasive hemodynamic monitoring or urine output
 - Maintain urine output of 0.5 mL/kg/hr for adults and 1 mL/kg/hr for children
- Escharotomy
 - Circumferential burn eschar may lead to vascular or respiratory compromise
 - Indications: Circulatory compromise of limb that does not improve with elevation, or respiratory compromise in circumferential chest wall burn
 - Extremities: Incisions on extremities should be made medially and laterally along the long axis of the limb just superficial to the subcutaneous fat layer through the entire length of the burn eschar
 - Chest wall: Make longitudinal incisions at anterior axillary line from the 2nd rib to the level of the 12th rib; connect with 2 transverse incisions across the chest
- Wound care:
 - Cover the wounds with Polysporin or bacitracin ointment and nonadherent

- dressings
- Use silver sulfadiazine in contaminated/dirty wounds (avoid if transferring to burn center as interferes with later assessment of burn)
- Do not delay transfer to burn unit for wound care
- Prophylactic antibiotics not indicated
- Outpatient management of minor burns
 - Sterile technique for cleansing and débridement
 - Pain control is almost always needed
 - Remove loose, necrotic skin; débride broken, tense, or infected blisters
 - Topical antibacterial agents (e.g., silver sulfadiazine, bacitracin, mafenide acetate) recommended in deep partial-thickness or full-thickness burns only
 - Consider collagenase (Santyl) in partial thickness wounds with eschar present; no need in 3rd-degree wounds; this lacks antibiotic properties
 - 3-layer burn dressings should keep the wound moist and absorb exudate
 - Inner layer should be nonadherent porous mesh gauze saturated with nonpetroleum-based lubricant, or a mild ointment (bacitracin or Polysporin) under a nonadherent porous gauze
 - 2nd layer should be fluffed coarse-mesh gauze
 - Outer wrap should keep the dressing in place without constricting
 - Dressings should be changed at least daily
 - Silver wound dressings (Silverlon and Acticoat)
 - Thin coating of metallic silver applied to knitted fabric backing
 - Requires dressing to remain moist
 - May leave in place for up to 3 days
 - Even minor burns need tetanus if indicated

Pediatric Considerations

- Parkland formula underestimates fluid requirements in children; the Galveston formula is more accurate
 - $5,000 \text{ mL/m}^2 \text{ BSA burned} + 2,000 \text{ mL/m}^2$
- Use 5% dextrose in lactated Ringer solution IV over the 1st 24 hr; give 1/2 in the 1st 8 hr and the other 1/2 over the next 16 hr
 - Titrate to goal urine output of 1 mL/kg/hr
- Consider nonaccidental trauma, particularly with burns on the back of hands or feet, buttocks, perineum, or legs
- Avoid hypothermia
 - Children have greater body surface area/mass ratio and lose heat more rapidly
- Avoid hypoglycemia
 - Children are more prone to hypoglycemia due to limited glycogen stores

Pregnancy Considerations

- Significant morbidity to mother and child
- Fluid requirements may exceed estimations
- Fetal monitoring and early obstetric consultation recommended

MEDICATION

- Bacitracin ointment: Apply 1–4 times per day
- Mafenide (Sulfamylon) acetate cream: Apply 1 or 2 times per day
- Narcotics, especially for débridement of blisters and larger, severe burns
- Silverlon and Acticoat: Cut sheet to size of burn; moisten with sterile water
- Silver sulfadiazine cream: Apply 1–2 times per day
- Tetanus toxoid or immunoglobulin: 0.5 mL IM; 250 U IM once along with toxoid
 - Santyl: apply to eschar/wound bed once daily



FOLLOW-UP

DISPOSITION

Admission Criteria

- Injuries requiring admission
 - Partial-thickness burns of noncritical areas (excludes eyes, ears, face, hands, feet, or perineum) involving 10–20% of BSA in adults (>10 yr and <50 yr)
 - Partial-thickness burns of noncritical areas involving 5–10% of BSA in children <10 yr
 - Suspicion of nonaccidental trauma
 - Patients unable to care for wounds in outpatient setting (e.g., homeless patients)
- Injuries requiring transfer/admission to a burn center
 - Partial-thickness or full-thickness burns involving $\geq 10\%$ of BSA
 - Full-thickness burns involving $> 5\%$ of BSA
 - Partial-thickness and full-thickness of face, hands, feet, genitalia, perineum, or major joints
 - Electrical burns, including lightning injury
 - Significant chemical burns
 - Inhalation injury
 - Patients with pre-existing illness that could complicate management
 - Patients with concomitant trauma or social barriers

Discharge Criteria

Partial-thickness burns of $< 10\%$ of BSA in adults ($< 5\%$ in children or the elderly) involving noncritical areas only. Patients must be reliable, able to manage wounds as outpatients and obtain follow-up.

Issues for Referral

Maintain low threshold for referral to burn specialist whenever there is raised concern regarding cosmesis, involvement of hands/face/eyes/perineum, or if burn is overlying a joint.

FOLLOW-UP RECOMMENDATIONS

1–2 days after the injury to assess for early infection, saturation of dressings, pain control

PEARLS AND PITFALLS

- Early IV fluid rehydration is essential
- Intubate early for signs of respiratory distress; must recognize potential for airway involvement
- Early pain control in all burns
- Monitor for hypoglycemia in children

ADDITIONAL READING

- Committee on Trauma, American College of Surgeons. Guidelines for the operation of burn units. *Resources for Optimal Care of the Injured Patient*; 2006:79–86.
- Pham TN, Cancio LC, Gibran NS. American Burn Association practice guidelines burn shock resuscitation. *J Burn Care Res*. 2008;29(1):257–266.
- Toon MH, Maybauer MO, Greenwood JE, et al. Management of acute smoke inhalation injury. *Crit Care Resusc*. 2010;12:53–61.
- Cancio LC, Lundy JB, Sheridan RL. Evolving changes in the management of burns and environmental injuries. *Surg Clin North Am*. 2012;92(4):959–986.



CODES

ICD9

- 949.0 Burn of unspecified site, unspecified degree
- 949.1 Erythema [first degree], unspecified site
- 949.2 Blisters, epidermal loss [second degree], unspecified site

ICD10

- T20.00XA Burn of unsp degree of head, face, and neck, unsp site, init
- T30.0 Burn of unspecified body region, unspecified degree
- T30.4 Corrosion of unspecified body region, unspecified degree

BURSITIS

Patrick H. Sweet

BASICS

DESCRIPTION

- Bursae are synovial fluid-filled sacs:
 - ~150 are located between bones, ligaments, tendons, muscles, and skin.
- They provide lubrication to reduce friction during movement.
- Bursitis is inflammation of a bursa caused by trauma and overuse, infection, crystal deposition, or systemic disease.
- Chronic bursitis can lead to proliferative changes in the bursa.
- Commonly affected sites:
 - Shoulder (subacromial)
 - Elbow (olecranon): Usually secondary to trauma
 - Hip (greater trochanter, ischial, iliopsoas): More common in elderly
 - Knee (prepatellar and pes anserine): Secondary to trauma or arthritis
 - Foot (calcaneal): Almost always secondary to improperly fitting shoes/heels

ETIOLOGY

- Trauma (most common cause):
 - Specific traumatic event or repetitive use of associated joints
- Infection: Secondary to direct penetration; may be obvious or microscopic:
 - Higher risk with diabetes, chronic alcohol abuse, uremia, gout, immunosuppression
 - 90% caused by *Staphylococcus* spp.
- Crystal deposition: Calcium phosphate, urate
- Systemic disease: Rheumatoid arthritis, gout, ankylosing spondylitis, psoriatic arthritis, lupus, rheumatic fever

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Acute or chronic
- History of trauma, overuse, or prolonged pressure
- Pain with increased activity at respective joint or with pressure
- Functional complaints (e.g., limping)
- History of localized swelling
- Screen for symptoms of systemic disease

- History of gout or pseudogout or rheumatologic disease
- History of recent procedure at bursa (e.g., aspiration, injection, etc.)

Physical-Exam

- Tenderness to palpation is minimal to mild in aseptic bursitis.
- Localized pain with movement
- Often reduced active range of motion (ROM) with preserved passive ROM
- Localized swelling, particularly with superficial bursae
- Skin trauma overlying bursa
- Warmth and erythema*
- May be febrile in septic bursitis

*NB: The constellation of erythema, warmth, swelling, and exquisite tenderness are common in septic bursitis.

ESSENTIAL WORKUP

- Full assessment of adjacent musculoskeletal structures
- Any suspicion of infection warrants aspiration of bursae (especially olecranon and prepatellar bursae).
- Lateral approach to prevent a needle tract directly over lines of tension of the joint
- Aspiration of hip and other deep bursae may be guided in ED by US or deferred to consultants.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Serum labs:
 - Suspected infection: CBC with differential
 - Evaluation of related systemic disease (e.g., uric acid level for gout); ESR and CRP in rheumatologic disease
 - Send serum glucose if bursal fluid aspiration is done
- Bursal fluid analysis:
 - Send fluid for complete cell count with differential, glucose, total protein, crystal determination, Gram stain, and culture.
 - Cultures must always be sent.
 - Normal fluid: Fluid is clear yellow with 0–200 WBCs, 0 RBCs, low protein, and glucose is same as serum.
 - Traumatic bursitis: Fluid is bloody/xanthochromic with < 1,200 WBCs, many RBCs, low protein, and normal glucose.
 - Septic bursitis: Fluid is cloudy yellow with > 50,000 WBCs, few RBCs, slightly increased protein, and decreased glucose; bacteria on Gram stain.
 - Rheumatoid and microcrystalline inflammation (aseptic bursitis): Fluid is yellow, sometimes turbid, and has 1,000–40,000 WBCs, few RBCs, slightly

increased protein, and variable glucose; microscopic exam for crystals.

Imaging

- Radiographs may demonstrate soft tissue swelling or adjacent chronic arthritic changes or calcium deposits:
 - Especially recommended when trauma is involved to rule out fracture or foreign body
- MRI and US may aid in diagnosis of deep bursitis and in defining the extent of septic bursitis.
- CT scans can also help differentiate septic from nonseptic bursitis.

DIFFERENTIAL DIAGNOSIS

- Arthritides: Septic, inflammatory, rheumatoid and degenerative joint (osteoarthritis)
- Gout and pseudogout
- Tendonitis, fasciitis, epicondylitis
- Fracture, tendon/ligament tear, contusion, sprain
- Osteomyelitis
- Nerve entrapment
- Also in hips: Neuritis, lumbar spine disease, sacroiliitis



TREATMENT

PRE HOSPITAL

May be difficult to distinguish from fractures; suspicious joints should be immobilized, particularly in the setting of trauma.

INITIAL STABILIZATION/THERAPY

- Immobilize joint if pain is severe.
- Shoulders should not be immobilized for >2–3 days because of the risk of adhesive capsulitis.

ED TREATMENT/PROCEDURES

- Nonseptic bursitis:
 - Rest and removal of aggravating factors (e.g., avoid direct pressure and repetitive use; protective padding where necessary)
 - Ice affected areas for 10 min, 4 times a day until improved; may alternate with heat.
 - NSAIDs for at least 7 days; best if continued for 5 days after improvement to help prevent recurrence
 - If fluctuant, then aspirate and place compression dressing
 - If no improvement within 5–7 days and infection has been ruled out (by

culture), injection of lidocaine and corticosteroids may be considered:

- Mix 2 mL of 2% lidocaine with appropriate depo-corticosteroid (see below) and inject 1–3 mL of this mixture into the bursa using sterile technique.
- Steroid injections should not be repeated until 4 wk have passed, and no > 2 injections per bursa should be performed without consultation.
- Septic bursitis:
 - Superficial bursae: Aspiration and antibiotics may be sufficient with close follow-up.
 - Other major bursae: Antibiotics and drainage of bursae (leaving in perforated drainage catheter can reduce period of treatment and avoid eventual bursectomy)
 - Febrile patients may need IV antibiotics.
 - Base antibiotic choice on the Gram stain when available or empiric coverage based on local susceptibilities:
 - Penicillinase-resistant penicillins may be used if Gram stain shows gram-positive cocci in chains but should be broadened for MRSA coverage if cocci in clusters are seen
 - If gram-negative organisms are found, blood cultures should be done and another primary source for the infection should be sought.
- Antibiotics should be continued for 5–7 days beyond resolution of signs of infection (thus may require follow-up)
- Treat associated diseases as needed (e.g., gout).

MEDICATION

- NSAIDs (many choices; a few are listed here):
 - Naprosyn: 500 mg PO q12h
 - Ibuprofen: 600 mg PO q6h (peds: 5–10 mg/kg PO q6h)
 - Ketorolac: 30 mg IV/IM q6h or 10 mg PO q4h–q6h
 - Meloxicam: 7.5 mg PO q12h or 15 mg PO daily
- Corticosteroids for intrabursal injection:
 - Triamcinolone acetonide: 20–40 mg (1st choice)
 - Methylprednisolone acetate: 20–40 mg
 - Dexamethasone acetate/sodium: 8 mg



FOLLOW-UP

DISPOSITION

- Most patients may be treated as outpatients.
- Most patients respond to therapy in 3–4 days and may follow-up within 1 wk or PRN.
- Septic bursitis requires repeated bursal aspiration every 3–5 days until sterile.

Admission Criteria

- Patients with systemic inflammatory response syndrome (SIRS), large surrounding cellulitis, unable to take PO antibiotics, failed outpatient therapy, or immunosuppressed
- Unusual organisms, extrabursal primary site, or deep bursal involvement

Discharge Criteria

- Able to tolerate pain
- Septic bursitis are safe to discharge if appropriately treated and close follow-up is secure

Issues for Referral

Rheumatology or orthopedic referral is recommended for patients who do not respond to intrabursal steroids or recurrent bursitis or need operative management.

FOLLOW-UP RECOMMENDATIONS

- Close follow-up for septic bursitis
- PRN to the emergency department for worsening symptoms but otherwise follow-up with primary care physician.

PEARLS AND PITFALLS

- Exam alone may be unreliable in distinguishing between traumatic and septic bursitis:
 - Aspiration and fluid analysis may be the only method of distinguishing.
- Beware of risk for GI hemorrhage associated with PO NSAIDs and for nephrotoxicity with ketorolac
- If presents with the 4 signs of infection—*humor, dolor, rubor, and calor*—then it is likely septic but still needs an aspiration and culture
- Beware of the potential of seeding organisms to adjacent joints when aspirating septic bursae.

ADDITIONAL READING

- DeLee JC, Drez D, Miller MD, ed. *DeLee & Drez's Orthopaedic Sports Medicine: Principles and Practice*. 3rd ed. Philadelphia, PA: Saunders Elsevier; 2010:889–891, 1209–1212, 1246–1249, 1455–1458, 2030–2041.
- Fayad LM, Carrino JA, Fishman EK. Musculoskeletal infection: Role of CT in the emergency department. *Radiographics*. 2007;27(6):1723–1736.
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for prepatellar and olecranon bursitis in Switzerland. *Europ J Trauma Emerg Surg.* 2013;39(1):65–72.



CODES

ICD9

- 726.10 Disorders of bursae and tendons in shoulder region, unspecified
- 726.33 Olecranon bursitis
- 727.3 Other bursitis

ICD10

- M70.20 Olecranon bursitis, unspecified elbow
- M71.9 Bursopathy, unspecified
- M75.50 Bursitis of unspecified shoulder

CALCIUM CHANNEL BLOCKER POISONING

Christopher S. Lim • Steven E. Aks



BASICS

DESCRIPTION

- 3 classes of calcium channel blockers (CCBs):
 - Phenylalkylamines (verapamil):
 - Vasodilation resulting in a decrease in BP
 - Negative chronotropic and inotropic effects: Reflex tachycardia not seen with a drop in BP.
 - Dihydropyridine (nifedipine):
 - Decreased vascular resistance resulting in a drop in BP
 - Little negative inotropic effect: Reflex tachycardia occurs
 - Benzothiazepine (diltiazem):
 - Decreased peripheral vascular resistance leading to a decrease in BP
 - Heart rate (HR) and cardiac output initially increased
 - Direct negative chronotropic effect, which leads to a fall in HR
- Effects of calcium channel blockade
 - Calcium plays key role in cardiac and smooth muscle contractility
 - CCBs prevent
 - the entry of calcium, resulting in a lack of muscle contraction
 - the normal release of insulin from pancreatic islet cells, resulting in hyperglycemia



DIAGNOSIS

SIGNS AND SYMPTOMS

- Cardiovascular:
 - Hypotension
 - Bradycardia
 - Reflex tachycardia (dihydropyridine)
 - Conduction abnormalities/heart blocks
- Neurologic:
 - CNS depression
 - Coma
 - Seizures
 - Agitation
 - Confusion
- Metabolic:
 - Hyperglycemia

History

- Inquire about risk of medication error.
- Inquire about risk of suicidal ideation with intent.
- Inquire about possible exposure to medications with a pediatric patient.

Physical-Exam

- Hypotension
- Bradycardia
- Skin may be warm instead of cool and clammy.

ESSENTIAL WORKUP

ECG:

- Bradycardia (reflex tachycardia with nifedipine)
- Conduction delays: QRS complex prolongation
- Heart blocks

DIAGNOSIS TESTS & NTERPRETATION

Lab

- Ionized calcium level when administering calcium
- Digoxin level if patient taking digoxin (dictate safety of calcium administration)
- CBC
- Electrolytes, BUN, creatinine, glucose
 - Strongly consider CCB overdose in the setting of bradycardia, hypotension, and hyperglycemia
 - Degree of hyperglycemia may correlate with severity of CCB poisoning in nondiabetics
- Toxicology screen if coingestants suspected

DIFFERENTIAL DIAGNOSIS

- β -Blocker toxicity
- Clonidine toxicity
- Digitalis toxicity
- Acute myocardial infarction with heart block



TREATMENT

PRE HOSPITAL

- Transport pill/pill bottles to ED
- Calcium for bradycardic/unstable patient with confirmed CCB overdose

INITIAL STABILIZATION/THERAPY

- ABCs:

- Airway protection, as indicated
- Supplemental oxygen, as needed
- 0.9% NS IV access
- Hemodynamic monitoring

ED TREATMENT/PROCEDURES

Goals

- HR > 60 beats/min
- Systolic BP > 90 mm Hg
- Adequate urine output
- Improving level of consciousness

GI-Decontamination

- Syrup of ipecac: Contraindicated in the pre-hospital and ED setting
- Activated charcoal:
 - May be helpful, especially in the presence of coingestants

Calcium

- Usually only transiently effective
- Calcium gluconate (10%):
 - Contains 0.45 mEq Ca²⁺/mL
 - Does not cause tissue necrosis as calcium chloride does
 - Calcium gluconate: Preferred agent in an acidemic patient
- Calcium chloride (10%):
 - Contains 1.36 mEq Ca²⁺/mL (3 times more calcium than calcium gluconate)
 - Can cause tissue necrosis and sloughing with extravasation
 - Very irritating to veins
- Follow serum calcium levels if repeated doses of calcium administered.
- Contraindicated in known digoxin toxicity because calcium may cause serious adverse effects in this setting

Bradycardia/Hypotension

- IV fluids:
 - Administer cautiously in the hypotensive patient.
 - Swan-Ganz catheter or central venous pressure (CVP) monitoring to help follow volume status
- Atropine usually ineffective
- High-dose insulin (HDI):
 - CCBs cause myocardial insulin resistance and inhibit insulin release from pancreatic islet cells
 - Results in inefficient fatty acid metabolism

- HDI promotes more efficient myocardial carbohydrate metabolism and has been shown to improve hemodynamic function
- Vasopressor agents:
 - No clear evidence that 1 agent is more effective than another
 - Institute invasive monitoring to help guide treatment.
 - Dopamine:
 - β_1 -Receptor agonist at low doses, which causes a positive inotropic effect on the myocardium
 - α -Receptor agonist at higher doses, which leads to vasoconstriction
 - Epinephrine:
 - Potent α - and β -receptor agonist
- Amrinone:
 - Selective phosphodiesterase inhibitor
 - Indirectly increases cAMP leading to increased inotropy
- Electrical pacing: When other treatment options have failed
- Potential future therapies:
 - Hypertonic sodium bicarbonate
 - IV fat emulsion (20% intralipid)

MEDICATION

- Amrinone: Loading dose 0.75 mg/kg; maintenance drip 2–20 $\mu\text{g}/\text{kg}/\text{min}$; titrate for effect
- Atropine: 0.5 mg (peds: 0.02 mg/kg) IV; repeat 0.5–1 mg IV (peds: 0.04 mg/kg)
- Calcium chloride: 5–10 mL of 10% solution slow IVP (peds: 0.2–0.25 mL/kg; repeat in 10 min if necessary) followed by infusion 20–50 mg/kg/h
- Calcium gluconate: 10–20 mL of 10% solution slow IVP (peds: 1 mL/kg; may repeat in 10 min if necessary)
- Dextrose: 50 mL of 50% solution (peds: 0.25 g/kg of 25% solution)
- Dopamine: 2–20 $\mu\text{g}/\text{kg}/\text{min}$; titrate to effect
- Epinephrine: 1–2 $\mu\text{g}/\text{min}$ (peds: 0.01 mg/kg or 0.1 mL/kg 1:10,000); titrate to effect
- Norepinephrine: Start 2–4 $\mu\text{g}/\text{min}$ IV; titrate up to 1–2 $\mu\text{g}/\text{kg}/\text{min}$ IV
- Potassium: 40 mEq PO or IV

High-dose Insulin Treatment Protocol

- Should be considered if response to fluid resuscitation is inadequate
- Insulin (regular insulin): 1 IU/kg bolus IV followed by 0.5–1 IU/kg/h titrated up to clinical response
- Administer dextrose if blood glucose < 200 mg/dL
- Administer potassium if serum potassium < 2.5 mEq/L
- Monitor serum glucose and potassium concentrations every 30 min for the 1st 4 hr
- Approximate 24-hr insulin requirement: 1,500 U of regular insulin for adult

patient

First Line

- IV fluids
- Calcium
- HDI
- Vasopressor agents

Second Line

- Amrinone
- IV fat emulsion



FOLLOW-UP

DISPOSITION

Admission Criteria

- Admit symptomatic patients to a monitored bed for hemodynamic monitoring.
- Admit all patients who ingested sustained-release CCBs for 24-hr observation and monitoring owing to the potential delay in symptoms.

Discharge Criteria

Discharge asymptomatic patients 8 hr after ingestion of immediate-release preparation.

FOLLOW-UP RECOMMENDATIONS

- Psychiatric evaluation for all suicidal patients
- Poison prevention guidance for parents of pediatric accidental ingestion

PEARLS AND PITFALLS

- Consider CCB toxicity in patients presenting hypotensive and bradycardic.
- Consider suicidal gesture in patients presenting with CCB toxicity.
- Consider HDI with dextrose and potassium if fluid resuscitation not rapidly effective.

ADDITIONAL READING

- Greene SL, Gawarammana I, Wood DM, et al. Relative safety of hyperinsulinaemia/euglycaemia therapy in the management of calcium channel blocker overdose: A prospective observational study. *Intensive Care Med.* 2007;33:2019–2024.
- Levine M, Boyer EW, Pozner CN, et al. Assessment of hyperglycemia after calcium

channel blocker overdoses involving diltiazem or verapamil. *Crit Care Med.* 2007;35:2071–2075.

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See Also (Topic, Algorithm, Electronic Media Element)

β-Blocker, Poisoning



CODES

ICD9

972.9 Poisoning by other and unspecified agents primarily affecting the cardiovascular system

ICD10

- T46.1X1A Poisoning by calcium-channel blockers, accidental, init
- T46.1X2A Poisoning by calcium-channel blockers, self-harm, init
- T46.1X4A Poisoning by calcium-channel blockers, undetermined, init

CANDIDIASIS, ORAL

Derrick D. Fenchel • Deepi G. Goyal



BASICS

DESCRIPTION

- Infection of oral mucosa with any species of *Candida*
- Up to 80% of isolates are *Candida albicans* (most common), *Candida glabrata*, and *Candida tropicalis*.
- *Candida* normally present as oral flora in 60% of the healthy population.
- Variations include:
 - Pseudomembranous (thrush)
 - Chronic and acute atrophic candidiasis
 - Angular cheilitis
 - Hyperplastic candidiasis
- More common in neonates, elderly, and immunosuppressed individuals
- Usually benign course in healthy patients
- In immunocompromised patients, more likely to be recurrent and a non-*albicans* species
- May represent an early manifestation of AIDS in HIV-infected patients
- Typically localized
- Risk factors for systemic infection:
 - AIDS
 - Diabetes
 - Hospitalization
 - Immunosuppressive therapy
 - Malignancy
 - Neutropenia
 - Organ transplantation
 - Prematurity

ETIOLOGY

- Usually overgrowth of *C. albicans* from alterations in intraoral environment
- May be medication induced—commonly antimicrobials, inhaled or systemic steroids, chemotherapy, immunosuppressive agents
- Immunocompromised patients
- Alterations or impairment of salivary flow:
 - Anticholinergic or psychotropic medications
 - Sjögren disease
 - Head or neck radiation
- Presence of dentures or other orthodontics:

- Occurs in up to 50–65% of denture wearers
- Common etiology for chronic atrophic candidiasis
- Interruption of epithelial barrier (cheek biting)
- Endocrinopathies (diabetes, hypothyroidism)

Pediatric Considerations

- Acute pseudomembranous candidiasis (thrush) is common in infancy likely because of immaturity of their immune system and lack of mature oral flora
- Initial presentation may be feeding difficulty secondary to dysphagia
- May have concurrent Candida diaper rash
- Consider maternal treatment if breastfeeding:
 - Maternal breast colonization may be cause for persistent thrush. Query maternal nipple pain, burning, itching, or cracked skin

Geriatric Considerations

- Candida organisms are normally present as oral flora from 65–88% of elderly or those in long-term care facilities
- Dentures can lead to Candida overgrowth
- Angular cheilitis more common in the elderly secondary to facial wrinkling



DIAGNOSIS

SIGNS AND SYMPTOMS

- Pseudomembranous candidiasis (thrush):
 - Painless white mucosal plaques
 - Adherent but removable plaques
 - Erythematous base
 - May become confluent and curdlike
 - Anorexia, dysphagia
- Acute atrophic candidiasis:
 - Also referred to as erythematous candidiasis
 - Burning sensation in mouth or on tongue
 - Erythematous with few, if any white patches usually on the palate or dorsum of tongue
 - Tongue may be bright red in color—similar to nutritional deficiency
- Chronic atrophic candidiasis:
 - Also referred as denture stomatitis
 - Irritation around denture-bearing mucosa
- Angular cheilitis:
 - Cracking or erythema at the corners of mouth
 - Lesion can be asymptomatic, pruritic, or painful
 - Superinfection with Staphylococcus or Streptococcus is common

- Hyperplastic candidiasis:
 - Chronic, invasive ulcers
 - Typically on lateral borders of tongue or buccal mucosa
 - High incidence of malignant degeneration in tobacco users

ESSENTIAL WORKUP

- Minimal workup needed in otherwise healthy infant. Diagnosis can be made clinically.
- Determine whether there is a cause for a breakdown of host factors
- If no reason is found, evaluate for possible HIV infection or diabetes
- Exclude a systemic infection

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Clinical diagnosis often sufficient
- CBC if suspect severe infection
- Glucose testing
- Periodic acid-Schiff stain/KOH/fungal culture:
 - Branching, thread-like hyphae, spores or pseudohyphae may be seen
 - Obtain culture and sensitivity if failed first line treatment or high-risk individuals (HIV/AIDs, neutropenic, AIDs, transplant, etc.)

DIFFERENTIAL DIAGNOSIS

- Hairy leukoplakia
- Lichen planus
- Squamous cell carcinoma
- Adherent food/milk



TREATMENT

ED TREATMENT/PROCEDURES

- IV fluids if dehydration and/or unable to tolerate PO fluids
- Topical analgesia: “Magic mouthwash”:
 - Mixture of equal parts of 2% viscous lidocaine, Maalox, and diphenhydramine elixir
 - Swish for 1–2 min, then expectorate
- Topical antifungal medications:
 - Suspension, troches, lozenges
 - Ointments (angular cheilitis)
- Systemic agents reserved for those with severe disease or resistant to topical therapy

- Provide oral hygiene education:
 - Instruct those using steroid inhalers to rinse mouth immediately after use
 - Denture and orthodontic care

MEDICATION

Pediatric Considerations

- Dissolve troche in bottle nipple
- Mix suspensions with fruit juice and freeze into popsicle
- Apply suspensions to affected areas with a cotton-tipped swab
- Instruct parents to disinfect or replace toothbrushes, pacifiers, bottle nipples

Geriatric Considerations

- Angular cheilitis: Treat with topical nystatin ointment
- Dentures: Remove, brush, and soak nightly. Consider overnight rinse with 2% chlorhexidine

First Line

- Nystatin: Oral suspension; neonates 100,000 U; older infants: 200,000 U; children/adults: 400,000–600,000 U. Swish and swallow QID for 7–14 days
- Nystatin pastilles: 200,000 U PO QID for 7–14 days
- Clotrimazole troches: 10 mg PO dissolved slowly 5 times per day for 7–14 days (children > 3 yr)

Second Line

- Oral fluconazole: Loading dose of 200 mg (peds: 6 mg/kg) on day 1, followed by 100 mg (peds: 3 mg/kg) PO daily for 7–14 days
- Itraconazole solution: 200 mg (peds: > 5 yr, 2.5 mg/kg BID, not FDA approved) PO daily for 7–14 days
- Posaconazole 100 mg (peds: > 13 yr refer to adult dosing) PO BID on day 1, then 100 mg PO daily for 13 days
- Systemic Amphotericin B (0.3 mg/kg) daily is the treatment of choice for candidiasis in pregnant women



FOLLOW-UP

DISPOSITION

Admission Criteria

- Inability to tolerate oral intake
- Newly diagnosed immunocompromised state
- Systemic infection

Discharge Criteria

If the candidiasis does not threaten patient's hydration status, discharge

FOLLOW-UP RECOMMENDATIONS

Additional workup for immunodeficiency is warranted in older children and adults with unexplained candidiasis.

PEARLS AND PITFALLS

- Failure to recognize immunodeficiency
- Failure to recognize other intraoral pathology such as squamous cell carcinoma

ADDITIONAL READING

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- Krol DM, Keels MA. Oral conditions. *Pediatr Rev*. 2007;28:15–22.
- Kuyama, K, Sun Y, Taguchi C, et al. A clinico-pathological and cytological study of oral candidiasis. *Open J Stomatology*. 2011;1:212–217.
- Pappas PG, Kauffman CA, Andes D, et al. Clinical practice guidelines for the management of candidiasis: 2009 update by the Infectious Diseases Society of America. *Clin Infect Dis*. 2009;48:503–535.



CODES

ICD9

- 112.0 Candidiasis of mouth
- 771.7 Neonatal Candida infection

ICD10

- B37.0 Candidal stomatitis
- P37.5 Neonatal candidiasis

CARBAMAZEPINE POISONING

James W. Rhee

BASICS

DESCRIPTION

- Therapeutic uses of carbamazepine:
 - Anticonvulsant
 - Treatment of chronic pain
 - Migraine prophylaxis
 - Mood stabilizer
- Mechanism:
 - Anticholinergic
 - Similarities to phenytoin and tricyclic antidepressants (TCAs)
 - Sodium channel blocker
 - Decreases synaptic transmission

ETIOLOGY

Toxicity may occur from:

- Suicide attempt
- Accidental ingestion
- Supratherapeutic dosing
- Drug–drug interaction

DIAGNOSIS

SIGNS AND SYMPTOMS

- Neurologic manifestations common
- Cardiotoxicity rare, except in massive overdose
- CNS:
 - Ataxia
 - Dizziness
 - Drowsiness
 - Nystagmus
 - Hallucinations
 - Combativeness
 - Coma
 - Seizures
- Respiratory system:
 - Respiratory depression
 - Aspiration pneumonia

- Cardiovascular system:
 - Hypotension
 - Conduction disturbances (mostly in elderly)
 - Supraventricular tachycardia
 - Sinus tachycardia or bradycardia
 - ECG changes:
 - Prolongation of PR, QRS, and QTc intervals
 - T-wave changes

Pediatric Considerations

Higher incidence of neurologic manifestations

History

- Overdose of carbamazepine or extended-release versions
- Time of ingestion
- Is the bottle available
- Accidental or intentional ingestion
- Coingestions

Physical-Exam

- May present with seizures or altered mental status
- May be combative or drowsy
- Sinus tachycardia (massive carbamazepine overdose)
- Bradydysrhythmia (often seen in elderly with mild increase in carbamazepine level)
- Anticholinergic manifestations:
 - Decreased bowel sounds
 - Mydriasis
 - Flushing
 - Urinary retention
- Neuromuscular changes:
 - Tremor
 - Slurred speech
 - Myoclonus
 - Choreiform and choreoathetoid movements

ESSENTIAL WORKUP

- Continuous cardiac monitor
- Serum carbamazepine level:
 - Therapeutic, 6–12 µg/L
 - Levels > 25–40 µg/mL associated with serious toxicity:
 - Coma

- Seizures
- Respiratory failure
- Conduction defects
- Serum levels do not clearly predict clinical toxicity:
 - Active metabolite carbamazepine 10, 11 epoxide not measured
 - Neurologic manifestations depend on CNS (not serum) level
- Serial levels may be needed owing to erratic absorption of carbamazepine.
- ECG:
 - Conduction delays:
 - Increased QRS interval
 - Increased PR interval
 - QTc prolongation
 - Dysrhythmias
- Serum acetaminophen level (to evaluate for coingestion in a suicide attempt)

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC:
 - Leukopenia or leukocytosis
- Electrolytes, BUN/creatinine, glucose:
 - Hyperglycemia
 - Hypokalemia
 - Hyponatremia
- Arterial blood gases (ABGs)
- Urinalysis:
 - Glucosuria
 - Ketonuria
- Pregnancy test
- ALT, AST, bilirubin, alkaline phosphatase:
 - May be mildly elevated
 - Usually not clinically significant

Imaging

CXR:

- Aspiration pneumonia
- Pulmonary edema

DIFFERENTIAL DIAGNOSIS

- Drugs that cause decreased mental status:
 - Alcohol
 - Anticholinergics
 - Barbiturates

- Benzodiazepines
- Lithium
- Opiates
- Phenothiazines
- Drugs that cause seizures:
 - Alcohol withdrawal
 - Anticholinergics
 - Camphor
 - Isoniazid
 - Lithium
 - Phenothiazines
 - Sympathomimetics:
 - Amphetamine
 - Cocaine
 - TCAs
- Drugs that cause abnormal movement:
 - Antihistamines
 - Butyrophenones
 - Caffeine
 - Cocaine
 - Levodopa
 - Meperidine
 - Phencyclidine
 - Phenothiazines
 - Phenytoin
 - TCAs



TREATMENT

PRE HOSPITAL

- Do *not* administer ipecac.
- Intubate if significant respiratory depression or airway compromise.
- Secure IV access.
- Get complete information about all products potentially ingested.

INITIAL STABILIZATION/THERAPY

- ABCs
- IV access and fluid resuscitation if hypotensive
- Oxygen
- Cardiac monitor
- Naloxone, thiamine, D₅₀W (or Accu-Chek) if altered mental status

ED TREATMENT/PROCEDURES

- General management:
 - Activated charcoal:
 - Administer sorbitol with 1st dose (only) of activated charcoal.
 - Administer with caution if GI activity is decreased.
 - Contraindicated if bowel sounds are absent
 - Multidose activated charcoal:
 - Decreases mean half-life of carbamazepine
 - Binds unabsorbed drug in GI tract
 - Interrupts enterohepatic circulation
 - Do not give additional sorbitol
 - Charcoal hemoperfusion/hemodialysis:
 - Removes small to moderate amount of ingested dose
 - Patients usually do well with supportive care without hemoperfusion or dialysis
 - Indicated in cases of clinical deterioration or lack of improvement with good supportive care
- Respiratory depression:
 - Intubation
 - Ventilatory support
- Hypotension:
 - Bolus with IV isotonic crystalloid solution
 - Norepinephrine if unresponsive to IV fluids
- Seizures:
 - Diazepam (drug of choice)
 - Phenobarbital (if diazepam ineffective)
 - Phenytoin not effective in most toxic seizures
- Cardiac conduction delay:
 - QRS widening (> 100 msec):
 - Sodium bicarbonate (to overcome sodium channel blockade)
- Psychiatric consultation if suicide attempt

MEDICATION

First Line

- Activated charcoal (initial bolus): Slurry 1–2 g/kg up to 100 g PO
- Multidose activated charcoal: 25 g (peds: 0.25 g/kg) q2h PO after bolus dose (above); can also use 50 g q6h PO/NG

Second Line

- Dextrose: D₅₀W 1 ampule: 50 mL or 25 g (peds: D₂₅W 2–4 mL/kg) IV
- Diazepam: 5–10 mg (peds: 0.2–0.5 mg/kg) IV

- Naloxone (Narcan): 2 mg (peds: 0.1 mg/kg) IV/IM initial dose
- Norepinephrine: 2–4 µg/min (peds: 0.05–0.1 µg/kg/min) IV titrated to effect
- Sodium bicarbonate: 1 or 2 amps IV push (peds: 1–2 mEq/kg)



FOLLOW-UP

DISPOSITION

Admission Criteria

- Decreased mental status at any time, even if resolving (tends to recur with fluctuating drug levels):
 - _ Observe at least 24 hr for late relapse.
- Seizures
- Cardiac dysrhythmias
- Lack of psychiatric clearance after suicidal ingestion

Discharge Criteria

- Asymptomatic after 6 hr of observation
- Normal mental status
- Normal or baseline ECG
- GI motility present
- Psychiatric clearance (after suicidal ingestion)

Issues for Referral

Suicidal patients need psychiatric evaluation referral.

FOLLOW-UP RECOMMENDATIONS

Supratherapeutic dosing will need ongoing monitoring by physician treating underlying disorder.

PEARLS AND PITFALLS

- Carbamazepine levels commonly rebound to higher levels during treatment. Obtain serial measurements for severe ingestions.
- Monitor closely for arrhythmias.
- Multidose charcoal may be needed for more serious ingestions.
- Paradoxical seizures may occur, use benzodiazepines to treat initially (diazepam is the drug of choice).

ADDITIONAL READING

- Brahmi N, Kouraichi N, Thabet H, et al. Influence of activated charcoal on the pharmacokinetics and the clinical features of carbamazepine poisoning. *Am J*

Emerg Med. 2006;24:440–443.

- Perez A, Wiley JF. Pediatric carbamazepine suspension overdose—Clinical manifestations and toxicokinetics. *Pediatr Emerg Care.* 2005;21(4):252–254.
- Pilapil M, Peterson J. Efficacy of hemodialysis and charcoal hemoperfusion in carbamazepine overdose. *Clin Toxicol (Phila).* 2008;46(4):342–343.
- Schmidt S, Schmitz-Buhl M. Signs and symptoms of carbamazepine overdose. *J Neurol.* 1995;242:169–173.



CODES

ICD9

966.3 Poisoning by other and unspecified anticonvulsants

ICD10

- T42.1X1A Poisoning by iminostilbenes, accidental, init
- T42.1X2A Poisoning by iminostilbenes, intentional self-harm, init
- T42.1X4A Poisoning by iminostilbenes, undetermined, initial encounter

CARBON MONOXIDE POISONING

Trevonne M. Thompson

BASICS

DESCRIPTION

- Carbon monoxide (CO) is a colorless, odorless, nonirritating gas.
- Binds to hemoglobin to form carboxyhemoglobin:
 - Decreases O₂-carrying capacity
- Direct cellular toxin
- Impairs cellular O₂ utilization

ETIOLOGY

- Endogenous:
 - Result of normal metabolism
- Incomplete combustion of carbonaceous fossil fuel:
 - Internal combustion engines
 - Natural gas
 - Heaters
 - Indoor grills
 - Fireplaces
 - Furnaces
 - Accidental fires
 - Tobacco smoke
- Methylene chloride:
 - Found in some solvents for paint removal and furniture stripping
 - Converted in vivo to CO after exposure
 - Peak carboxyhemoglobin levels delayed after exposure
 - Half-life is ~ 2 times that of inhaled CO

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- CNS:
 - Headache
 - Dizziness
 - Ataxia
 - Confusion

- Syncope
- Seizures
- GI:
 - Nausea
 - Vomiting
- Cardiovascular:
 - Chest pain
 - Palpitations
- Respiratory:
 - Dyspnea
- Ophthalmologic:
 - Decreased visual acuity

Physical-Exam

- CNS:
 - Acute encephalopathy
 - Seizures
 - Coma
- Cardiovascular:
 - Tachycardia
 - Premature ventricular contractions
 - Dysrhythmias
 - Myocardial ischemia/infarction
- Respiratory:
 - Tachypnea
 - Noncardiogenic pulmonary edema
- Ophthalmologic:
 - Retinal hemorrhage
- Other:
 - Respiratory alkalosis
 - Rhabdomyolysis
 - Lactic acidosis

ESSENTIAL WORKUP

- History:
 - Maintain a high index of suspicion
 - Symptoms may be mild, nonspecific
 - Inquire about the following:
 - Similar symptoms in other household members
 - Malfunctioning furnaces
 - Use of space heaters, open ovens for supplemental heat
 - Ill pets
- Arterial blood gas:

- Normal PaO₂
- Normal calculated O₂ saturation
- Low measured O₂ saturation
- Metabolic acidosis in severe cases
- Carboxyhemoglobin level:
 - Measure as soon as possible
 - Level may not reflect clinical severity:
 - Patient may be critically ill despite unimpressive carboxyhemoglobin level.
 - May be misleadingly low if significant time has passed since exposure
 - Normal range is 0–3% (up to 10% in smokers).

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Pulse oximetry:
 - Falsely elevated SaO₂ reading
 - Pulse oximeter cannot distinguish oxyhemoglobin from carboxyhemoglobin.
- Electrolytes:
 - Metabolic acidosis and elevated anion gap associated with increased clinical severity
- Cardiac enzymes:
 - When myocardial ischemia/infarction suspected
- Pregnancy test
- ECG:
 - CO may precipitate myocardial ischemia/infarction.
 - Dysrhythmias
 - Nonspecific ST-segment and T-wave abnormalities

Imaging

- Chest radiography:
 - Pulmonary edema
- CT scan of the head:
 - To evaluate for intracranial causes of altered mental status when indicated
 - Bilateral globus pallidus low-density lesions may be clue to CO poisoning in unclear cases.

DIFFERENTIAL DIAGNOSIS

- Viral illness/viral syndrome
- Meningitis/encephalitis
- Intracranial hemorrhage
- Gastroenteritis

- Migraine headache
- Tension headache
- Ethanol intoxication
- Sedative–hypnotic overdose
- Cyanide poisoning
- Salicylate overdose
- Toxic alcohol exposure



TREATMENT

PRE HOSPITAL

Administer 100% O₂

INITIAL STABILIZATION/THERAPY

- ABCs
- Establish IV access
- 100% oxygen
- Cardiac monitor

ED TREATMENT/PROCEDURES

- Oxygen:
 - Administer 100% normobaric O₂:
 - Via face mask or endotracheal tube
 - Continue O₂ therapy until carboxyhemoglobin level <10%.
 - Half-life of carboxyhemoglobin:
 - ~300 min in ambient air
 - ~90 min in 100% normobaric O₂
 - ~20 min at 3 atm (hyperbaric O₂)
- Hyperbaric O₂:
 - Dose:
 - 100% O₂ at 3 atm
 - May be repeated
 - Benefits:
 - May reduce delayed neurologic sequelae
 - Decreases half-life of carboxyhemoglobin
 - Potential adverse effects:
 - Tympanic membrane rupture
 - Pneumothorax
 - Seizure
 - Decompression sickness

- Pulmonary edema
- Use of hyperbaric O₂ remains controversial
- Indications for consulting hyperbaricist:
 - Altered mental status/coma
 - Focal neurologic deficits
 - Seizures
 - Cardiovascular compromise (infarction, persistent dysrhythmia)
 - Persistent metabolic acidosis
 - Carboxyhemoglobin level > 25%
 - Pregnancy with carboxyhemoglobin level > 10%

Pregnancy Considerations

- Fetal hemoglobin has higher affinity for CO than adult hemoglobin.
- Fetal carboxyhemoglobin levels 10–15% higher than maternal levels
- Delayed clearance of fetal carboxyhemoglobin compared with maternal



FOLLOW-UP

DISPOSITION

Admission Criteria

- Persistent symptoms after 4 hr of treatment with 100% oxygen
- Evidence of myocardial ischemia or cardiac instability
- Seizures
- Persistent metabolic acidosis
- Syncope

Discharge Criteria

- Asymptomatic after 4 hr of observation
- Absence of aforementioned admission criteria
- Psychiatric clearance if suicidal exposure

Issues for Referral

Need for hyperbaric oxygen therapy

FOLLOW-UP RECOMMENDATIONS

Contact local fire department in cases of CO home exposures.

PEARLS AND PITFALLS

- Suspect CO poisoning in patients who present with headaches when home heaters are initiated.

- Suspect CO poisoning when family members living in the same enclosed space have similar symptoms.
- Administer 100% O₂ and transfer to hyperbaric facility if the above-described criteria is met.

ADDITIONAL READING

- Buckley NA, Juurlink DN, Isbister G, et al. Hyperbaric oxygen for carbon monoxide poisoning. *Cochrane Database Syst Rev*. 2011;13(4):CD002041.
- Guzman JA. Carbon monoxide poisoning. *Crit Care Clin*. 2012;28(4):537–548.
- Kao LW, Nanagas KA. Carbon monoxide poisoning. *Emerg Med Clin North Am*. 2004;22(4):985–1018.
- Weaver LK. Carbon monoxide poisoning. *N Engl J Med*. 2009;360:1217–1225.



See Also (Topic, Algorithm, Electronic Media Element)

Hyperbaric Oxygen



CODES

ICD9

986 Toxic effect of carbon monoxide

ICD10

- T58.11XA Toxic effect of carb monx from utility gas, acc, init
- T58.91XA Toxic effect of carb monx from unsp source, acc, init
- T58.92XA Toxic effect of carb monx from unsp source, self-harm, init

CARDIAC ARREST

Michael W. Donnino • Brandon Giberson • Michael N. Cocchi



BASICS

ALERT

- NOTE: The following information is based on 2010 Advanced Cardiac Life Support (ACLS) Guidelines. Any revisions made by the American Heart Association since then are not available at time of publication.
- Major ACLS Changes for the 2010 revision include:
 - Change in the BLS sequence of treatment from A–B–C (airway, breathing, circulation) to C–A–B (circulation, airway, breathing) to emphasize early chest compressions
 - Emphasis on postcardiac arrest care, particularly implementation of targeted temperature management
 - Removal of atropine from PEA/asystole ACLS algorithms

DESCRIPTION

- Sudden cardiac arrest is characterized by:
 - Unresponsiveness
 - Pulselessness
 - Little to no respiratory effort
- Factors affecting survival:
 - Initial rhythm
 - Total down time
 - Time to successful defibrillation (as indicated)
 - Time to basic life-support interventions

ETIOLOGY

Contributing factors to cardiac arrest are outlined by the American Heart Association as:

- Hypovolemia
- Hypoxia
- Hydrogen ion (acidosis)
- Hypo-/hyperkalemia
- Hypothermia
- Toxins
- Tamponade, cardiac
- Tension pneumothorax
- Thrombosis
- Trauma

Pediatric Considerations

- Sudden cardiac arrest in children is often of a respiratory rather than cardiac etiology
- Follow current ACLS guidelines for pediatric cardiac arrest. Major differences between adult and pediatric cardiac arrest management include:
 - Depth of compressions for pediatric populations should be ~1/3 to 1/2 the depth of the chest
 - For 2 rescuer CPR, a 15:2 compression to ventilation rate is recommended
 - Drug dosage differences: See “Medications” section

Pregnancy Considerations

Follow current ACLS guidelines for management of the pregnant cardiac arrest patient:

- Awareness that airway may be difficult
- Compressions should be performed at a higher location than conventional CPR, slightly above the center of the sternum
- Follow Adult ACLS guidelines for defibrillation
- Pre- or postcardiac arrest pregnant patients should be placed in the left lateral recumbent position; during arrest, perform manual left uterine displacement
- To ensure a best possible outcome for the fetus, all efforts must be geared toward maternal survival
- In the event of a failed maternal resuscitation, an emergent cesarean delivery may be considered

DIAGNOSIS

SIGNS AND SYMPTOMS

- Unresponsiveness
- Pulselessness
- Shallow, gasping respirations may persist for a few minutes
- Occasionally preceded by:
 - Chest pain
 - Dyspnea
 - Palpitations
 - Seizure activity
- Immediately prior to arrest:
 - Shock or hypotension
 - Impaired mentation

ESSENTIAL WORKUP

- Assess circulation, airway, breathing
- Determine shockable vs. nonshockable rhythm and treat accordingly, per ACLS guidelines

DIAGNOSIS TESTS & INTERPRETATION

Lab

Indicated only when successful return of spontaneous circulation (ROSC) is achieved:

- Electrolytes
- BUN/creatinine
- Creatinine kinase with isoenzymes, cardiac troponin
- ABG
- CBC
- Therapeutic drug levels
- Toxicologic testing
- Lactic acid levels

Imaging

- EKG:
 - _ Evaluate for STEMI or ACS
- CXR:
 - _ Endotracheal tube position
 - _ Pneumothorax
 - _ Pulmonary etiology of arrest
- Echocardiogram:
 - _ Pericardial effusion
 - _ Wall motion abnormality
 - _ Valvular dysfunction
- Head CT scan (postresuscitation):
 - _ Rule out bleed/neurologic source

Diagnostic Procedures/Surgery

- Suspected cardiac etiology:
 - _ Cardiac catheterization lab
 - _ Possible cardiac output augmentation device placement
- EEG (postresuscitation)
 - _ Identify and treat seizures

DIFFERENTIAL DIAGNOSIS

Sudden loss of consciousness with a palpable pulse:

- Syncope
- Seizure
- Acute stroke
- Hypoglycemia
- Acute airway obstruction
- Head trauma

- Toxins



TREATMENT

PRE HOSPITAL

- Prompt initiation of standard CPR
- Confirm underlying rhythm
- Early defibrillation of pulseless ventricular tachycardia (VT) or ventricular fibrillation (VF)
- Secure airway and provide adequate respirations. Advanced airway should be deferred if placement interrupts BLS measures
- Postresuscitation care:
 - _ Identify cause of arrest
 - _ 12-lead EKG
 - _ Monitor vital signs
 - _ Fluid bolus and/or vasopressors for hypotension
- Transport to the closest facility that is capable of handling postarrest patients:
 - _ Consider transport to center equipped for interventional cardiac care and those specializing in postarrest care
 - _ Pediatric critical care center for children

INITIAL STABILIZATION/THERAPY

- Initiate ACLS
- Perform standard CPR as long as no pulse is palpable:
 - _ Stop CPR only briefly to check pulse, cardiac rhythm, or defibrillate
- Secure the airway
- Obtain IV/IO access
- Cardiac monitor
- Therapy is based on the underlying rhythm, according to ACLS protocols

ED TREATMENT/PROCEDURES

- Pulseless VT or VF:
 - _ Immediate defibrillation with 1 countershock:
 - Energy selection based on type of defibrillator for biphasic (if unknown use 200 J) or 360 J monophasic
 - _ If defibrillation is unsuccessful, continue CPR for 2 min and re-evaluate rhythm. When IV/IO access is established, and after second rhythm check then consider:
 - Epinephrine
 - Vasopressin
 - _ If refractory to defibrillation and epinephrine, consider:
 - Amiodarone

- Lidocaine
- Magnesium for *torsade de pointes*
- Asystole:
 - Confirm in ≥ 2 leads
 - Epinephrine
 - May substitute vasopressin to replace 1st or 2nd dose of epinephrine
- Pulseless electrical activity:
 - Epinephrine
 - Treat for reversible cause of pulseless electrical activity/asystole
- Postresuscitation:
 - Treat the underlying cause of the arrest.
 - EKG to establish presence of acute coronary syndrome:
 - Immediate catheterization for STEMI
 - Consider catheterization for suspected cardiac etiology without STEMI
 - Ventilatory support
 - Correct electrolyte abnormalities
 - Initiate volume resuscitation and provide vasopressors/inotropic support as needed
 - Targeted temperature management for eligible patients
 - Continuous EEG to rule out seizures

MEDICATION

Medication administration should never interrupt CPR:

- Amiodarone: 300 mg (peds: 5 mg/kg to max. 15 mg/kg) IVP
- Epinephrine: 1 mg (peds: 0.01 mg/kg) IVP q3–5min
- Lidocaine: 1–1.5 mg/kg 1st dose (peds 1 mg/kg) IVP, then 0.5–0.75 mg/kg (peds: 20–50 $\mu\text{g}/\text{min}$) IV, up to 3 mg/kg
- Magnesium: 1–2 g (peds: 25–50 mg/kg max. of 2 g) slow IV
- Vasopressin: 40 U IVP (as replacement for dose 1 or 2 of epinephrine in adult arrest)
- Sodium bicarbonate: 1 mEq/kg (peds: 1 mEq/kg) slow



FOLLOW-UP

DISPOSITION

Admission Criteria

ROSC:

- Intensive care unit
- Postresuscitation care
- Treatment of underlying cause of arrest

Discharge Criteria

None

Issues for Referral

May consider referral to regional cardiac arrest center

FOLLOW-UP RECOMMENDATIONS

Admission to ICU

PEARLS AND PITFALLS

- Provide targeted temperature management in comatose post arrest patients.
- Expect recurrent cardiac arrest and provide close monitoring and appropriate postresuscitative treatment, which may consist of fluids and vasopressors.
- Get a cardiology consultation to determine if patient is candidate for cardiac catheterization.

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CODES

ICD9

427.5 Cardiac arrest

ICD10

I46.9 Cardiac arrest, cause unspecified

CARDIAC PACEMAKERS

Susan P. Torrey



BASICS

DESCRIPTION

- A device that uses electrical impulses to contract the heart muscles and provide an adequate pulse
- Methods of cardiac pacing:
 - Transcutaneous pacing:
 - 2 pads are placed on the chest in the anterior-lateral or anterior-posterior position.
 - The pacing current is gradually increased until electrical capture occurs with a pulse.
 - Emergency therapy used only until transvenous pacing or another therapy can be applied
 - Temporary transvenous pacing:
 - A pacemaker wire is placed through central venous access into the right atrium (RA) or right ventricle (RV) and connected to an external generator outside of the body.
 - Used as a bridge until a permanent pacemaker can be placed or there is no longer a need for a pacemaker
- Permanent, implanted pacemaker has 3 components:
 - A battery-powered energy source:
 - Lithium batteries last 7–10 yr
 - Generator:
 - A sophisticated computer with many programmable parameters
 - Leads connected to the RV/RA:
 - Typically sense intrinsic electrical activity of the heart and pace the myocardium as needed
- Pacemaker magnet:
 - Placed over pacemaker generator
 - Converts pacer to asynchronous mode
 - Useful if pacer spikes not present on ECG
 - A depleted battery will result in decrease in magnet rate by ~10%.

Pacemaker Terminology

- Fixed mode:
 - The pacemaker is set to fire at a set rate regardless of patient's underlying rhythm.
 - Rarely seen

- Demand mode:
 - The pacemaker fires only when necessary.
 - It senses the underlying rhythm.
 - It will only pace if the intrinsic rhythm is absent or less than a set rate.
- Sensing:
 - Pacemaker's ability to determine whether the heart has an intrinsic rhythm
- All pacemakers have a 5-letter code to describe their function.
- For ED purposes, only the 1st 3 letters of the code are necessary:
 - 1st letter in code indicates chamber being sensed by pacemaker:
 - A: Atria
 - V: Ventricle
 - D: Dual (both chambers)
 - 2nd letter in code indicates chamber that can be paced:
 - A: Atria
 - V: Ventricle
 - D: Dual (both chambers)
 - 3rd letter in code describes pacemaker's response to sensed intrinsic complex:
 - T: Trigger (a sensed beat results in a pacing response as when a sensed atrial beat provokes a subsequent ventricular beat)
 - I: Inhibit (a sensed beat precludes pacemaker function)
 - D: Dual (a pacemaker is capable of both functions)
 - O: No response
 - The most common pacemakers are VVI (single lead) and DDD (two leads).

ETIOLOGY

- Pacemaker-associated infection:
 - Infection of pacemaker components often associated with endocarditis
 - *Staphylococcus epidermidis* and *Staphylococcus aureus* account for > 90% of infections.
 - Transesophageal echo is the preferred diagnostic method.
- Venous thrombosis:
 - Very common (overall incidence 30–50%)
 - Symptomatic, acute obstruction is rare (< 3%).
 - Pulmonary embolism is rare.
- Pacemaker failure to discharge impulse
 - Component failure is rare.
 - Battery depletion is rare with routine checks; it is not abrupt.
 - Lead fracture or disconnection
 - Oversensing of muscular activity or external electrical interference
- Pacemaker failure to capture myocardium:
 - Lead dislodgment is common.
 - Twiddler's syndrome:

- Unintentional manipulation of pacemaker generator causing lead to be dislodged from myocardium
- Elevated myocardial threshold:
 - Hyperkalemia
 - Ischemia
- Change in cardiac (QRS) morphology
- Pacemaker-mediated tachycardia:
 - Occurs with dual-chamber pacemakers
 - A re-entry rhythm using generator and intrinsic conduction system
 - Max. rate typically 140 bpm due to built-in safeguards
- Runaway pacemaker:
 - Rare; triggered by battery depletion or component failure
 - Often rapid rates (> 200 bpm) with hemodynamic compromise

DIAGNOSIS

SIGNS AND SYMPTOMS

- Pacemaker failure:
 - Bradycardia
 - Syncope
 - Hypotension, progressive to shock and hemodynamic collapse
 - Fatigue and weakness
 - Dyspnea on exertion or shortness of breath secondary to CHF
 - Ischemic chest pain
 - Altered level of consciousness
- Pacemaker-induced tachycardia:
 - Dyspnea
 - Ischemic chest pain
 - Lightheadedness
 - Syncope
- Pacemaker syndrome:
 - Symptoms related to asynchronous chamber contractions (typical with VVI pacer)
 - Lightheadedness
 - Dyspnea
 - Palpitation
 - Weakness or exercise intolerance
 - Syncope

History

- Date of placement pacemaker
- Compliance with follow-up (battery checks)

- Type of pacemaker

Physical-Exam

General cardiac exam:

- Heart exam for murmurs
- Lung exam for CHF
- Chest wall exam at generator site

ESSENTIAL WORKUP

- 12-lead EKG to assess whether there is any obvious evidence of pacemaker failure
- Metabolic workup to determine whether an acquired medical condition led to an elevated myocardial threshold
- EKG with pacer magnet:
 - Assess magnet rate.
 - Particularly useful when the baseline EKG does not reveal pacer spikes
 - The magnet activates asynchronous pacing mode.
 - Produces pacer spikes at a preprogrammed rate, regardless of the intrinsic rhythm
 - If the magnet rate equals the preprogrammed rate set at implantation, the pacer is okay.
 - If the magnet rate is > 10% slower than at implantation, the battery is depleted.
 - If there are no pacer spikes, there is significant pacemaker malfunction.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Serum potassium
- ABG
- Serum levels of antidysrhythmic drugs

Imaging

CXR:

- Evaluate integrity of pacer lead(s) and position.
- Fractured lead
- Lead dislodgment:
 - Perforation through septum
 - Tip of lead moved (e.g., in pulmonary artery)



TREATMENT

PRE HOSPITAL

Record rhythm strips for analysis

INITIAL STABILIZATION/THERAPY

- Oxygen administered via 100% nonrebreather
- Intubation as needed
- IV access
- Advanced cardiac life support drugs as per usual protocol (especially for bradycardia)
- Defibrillation: Avoid placing paddles over generator.
- Transcutaneous pacemaker in hemodynamically unstable patients with pacemaker failure

ED TREATMENT/PROCEDURES

- Pacemaker failure:
 - Transcutaneous pacemaker
 - Temporary transvenous pacemaker:
 - Obtain central IV access with a Cordis introducer (right IJ preferred)
 - Perform the procedure under fluoroscopy if possible.
 - Set the pulse generator to asynchronous mode.
 - Turn the output dial all the way up.
 - Advance the catheter through the central venous access Cordis until you see a QRS complex on the monitor.
 - Check the femoral pulse.
 - If you have a pulse and see a QRS complex, the pacer is “capturing.”
 - Slowly turn the output dial down until you lose the QRS complex (capture threshold).
 - Turn the output dial up to 2 or 3 times the capture threshold.
 - Continuous EKG monitoring facilitates correct placement.
- Treat hyperkalemia (see “Hyperkalemia”).
- Runaway pacemaker:
 - AV node blocking or reprogramming
 - In extreme situation, may need to disconnect lead from generator surgically

MEDICATION

Adenosine: 6 mg IV bolus



FOLLOW-UP

DISPOSITION

Admission Criteria

- Permanent pacemaker failure or malfunction
- Suspicion of infection involving pacemaker components

Discharge Criteria

- Asymptomatic pacemaker malfunction
- A cardiologist has interrogated the pacemaker

FOLLOW-UP RECOMMENDATIONS

Refer to cardiologist and/or pacemaker clinic

PEARLS AND PITFALLS

- Always consider pacemaker failure in evaluation of cardiac decompensation, bradycardia, or syncope.
- Utilize pacemaker magnet to evaluate function.

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- Stone KR, McPherson CA. Assessment and management of patients with pacemakers and implantable defibrillators. *Crit Care Med.* 2004;32:155–165.



CODES

ICD9

- V45.01 Cardiac pacemaker in situ
- V53.31 Fitting and adjustment of cardiac pacemaker
- 996.61 Infection and inflammatory reaction due to cardiac device, implant, and graft

ICD10

- T82.7XXA Infect/inflm react d/t oth cardi/vasc dev/implnt/grft, init
- Z45.018 Encounter for adjustment and management of other part of cardiac pacemaker
- Z95.0 Presence of cardiac pacemaker

CARDIAC TESTING

Steve R. Grosse • Shamai A. Grossman



BASICS

DESCRIPTION

- Cardiac testing is indicated for emergency patients at risk for heart failure (HF) or acute coronary syndrome (ACS).
- These pathologies may be thought of as a spectrum: Unstable angina can evolve into MI, which in turn can cause HF:
 - ~20% of ED malpractice claims are due to missed diagnosis of ACS.
 - ~2% of patients with ACS are inappropriately discharged from an ED.
 - History, physical exam, and ECG are the critical elements in working up chest pain and ACS/HF.
 - History, physical, and ECG nevertheless miss 1–4% of all heart attacks.
 - Additional tools include imaging modalities and blood tests (e.g., cardiac biomarkers).

ETIOLOGY

ACS is caused by atherosclerotic narrowing of coronary vessels or by coronary vasospasm.

Pregnancy Considerations

In the pregnant patient with chest pain and ischemic changes on ECG, also consider spontaneous coronary artery dissection.



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Anginal symptoms usually are produced by bodily stresses, including exertional and emotional events, and relieved by rest.
- ACS is less likely when chest pain is sharp, stabbing, pleuritic, or reproducible with palpation.
- Ischemia is still diagnosed in 13% of pleuritic chest pain and in 7% of chest pain reproducible with palpation.
- Nitroglycerin may relieve cardiac ischemia, but can also relieve pain in GI and aortic pathology.
- A “GI cocktail” of lidocaine and Maalox, or a proton-pump inhibitor such as omeprazole, may relieve GI pathology, but can also relieve cardiac ischemia.

- Anginal symptoms often last < 20 min but > 5 min
- AMI and UA should be considered if symptoms last > 20 min.

Physical-Exam

Often unremarkable

ESSENTIAL WORKUP

EKG:

- Per ACC/AHA guidelines, a 12-lead ECG should be performed on a patient with chest pain within 10 min of arrival to the ED:
 - A single ECG will miss ~ 50% AMI.
 - Hyperacute T-waves (tall, broad-based, especially in anterior leads) may be the earliest and only sign of AMI.
 - During an MI, the ECG may evolve. Continuous ECG monitoring can identify an additional 16% of acute MIs not seen on initial ECGs. Absent continuous monitoring, consider a repeat EKG 15–60 min after the initial ECG.
 - New ST-segment changes or T-wave inversions are suspicious for ischemia.
 - ST depressions of 1 mm are characteristic of ischemia; or, could be reciprocal changes, so check other leads.
 - STEMI: ST-elevation of > 1–2 mm in ≥ 2 contiguous leads.
 - New left bundle branch block (LBBB) is suggestive of AMI:
 - Old LBBB makes diagnosing AMI difficult: Apply Sgarbossa criteria: AMI is likely if LBBB and > 1 mm ST-elevation concordant with QRS, or ST depression > 1 mm in leads V1, V2, or V3.
 - Current ACCF/AHA guidelines advise that LBBB “not known to be old” in isolation is not diagnostic of AMI, and should be further evaluated with serum biomarkers and immediate cardiac consultation for consideration of echocardiography and invasive angiography.
- Additional-lead EKGs: Standard 12 leads often miss infarcts in the posterior, right, and high lateral walls.
 - Right-sided EKG:
 - Move lead V4 to the right side of chest, midclavicular line, 5th intercostal space, and repeat EKG, to capture infarct in right ventricle.
 - A right-sided EKG is often performed in the setting of a STEMI in inferior leads (II, III, aVF) to diagnose a right ventricular (RV) infarct.
 - Posterior EKG:
 - Leads V7, V8, V9 are placed posterior thorax along 5th intercostal space: V7 at posterior axillary line, V8 at inferior angle of scapula, V9 paraspinous.
 - Performed in setting of inferior or lateral wall MI; or if ST depression in V1–V3. May identify a lateral or left circumflex infarct.

- EKG may be helpful in diagnosing other etiologies of chest pain:
 - Pericarditis is suggested by diffuse ST-elevations followed by T-wave inversions and P-R depression.
 - Pulmonary embolism is suggested by unexplained tachycardia, signs of right heart strain (RVH, RBBB, “p” pulmonale), new-onset atrial fibrillation, or rarely with S1, Q3, T3 pattern.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Cardiac biomarkers:
 - Indicated if the history is suspicious for ACS.
 - Should not be elevated in stable angina and may be normal in unstable angina.
- Troponin T and I: Starts to rise 2–3 hr after onset of chest pain of ACS and peaks in 8–12 hr. Remains elevated 7–14 days:
 - A single troponin has low sensitivity for ACS (1 study of low-risk chest pain in patients with negative initial troponin: 2.3% rate of AMI and 1% rate of death at 30 days).
 - Timing of biomarker testing is critical: ACEP endorses with “moderate clinical certainty” that a single negative troponin can rule out AMI if drawn 8–12 hr after onset of symptoms. However, uncertainty in time of symptom onset, unreliable history, and possibility of preinfarction angina complicates utilizing single troponin.
 - Newer, more sensitive assays may in the future eliminate the need for a 2nd troponin.
 - Minor troponin elevations may occur with renal failure, structural heart disease, CHF (acute or chronic), cardiac pacing, pulmonary embolism, sepsis, stroke.
 - Lack of standardization between assays (particularly with troponin I) means values from 1 lab cannot always be simply compared to values from another.
- CK/CK-Mb: Less sensitive than troponin, rises more slowly. Little gained by using both CK-Mb and troponin assays. Obtain CK-Mb if
 - Renal failure is present (Tn less accurate)
 - Recent prior infarct
- Myoglobin: Rises faster than standard troponin assays and thus able to detect AMI sooner, but max. sensitivity is 70%.
- B-type natriuretic peptide (BNP):
 - Release and synthesis activated by diastolic ventricular stretch.
 - Useful for detecting HF.
 - A cutoff of > 100 pg/mL diagnosed HF with a sensitivity of 90% and specificity of 76%.

- Unclear significance of elevated BNP in setting of ACS.

Imaging

- CXR:
 - Usually normal
 - May show cardiomegaly
 - May show pulmonary edema
 - May identify other etiologies of chest pain, such as pneumonia or widened mediastinum of aortic dissection.
- Rest echocardiography:
 - May identify ACS or AMI based on wall motion abnormalities; also can detect pump failure and valvular abnormalities.
 - Rest echo has a sensitivity of 70% and specificity of 87% for ACS.
 - Rest echo has a sensitivity of 93% and specificity of 66% for AMI.
- Technetium 99m sestamibi:
 - Radioactive IV dye taken up by myocardium, and detected by single photon emission CT (SPECT) imaging. (Also known as myocardial perfusion imaging.)
 - Can be imaged at rest to detect low- or no-flow areas of myocardium; can also be imaged after exercise or pharmacologic stress.
 - Per 2009 AHA/ACC guidelines, reserve for intermediate- to high-risk patients.
 - Has a sensitivity of 81% and specificity of 73% for ACS.
 - Has a sensitivity of 92% and specificity of 67% for AMI.
- CT coronary angiography (CTCA):
 - Imaging to evaluate degree of coronary artery stenosis and calcium deposits
 - Negative predictive value between 97% and 100%, accuracy comparable to stress testing
 - Recent NEJM article suggests CTCA decreases ED length of stay but leads to further downstream testing, radiation exposure, and no decrease in cost of care.
- Exercise stress testing (ETT):
 - May help establish diagnosis of angina, provide prognostic information.
 - 1-mm depression of the ST-segment in 3 consecutive beats and 2 consecutive leads is characteristic of cardiac ischemia.
 - Early positive (within 3 min) stress tests are worrisome for unstable angina.
 - 6 min of exercise using a standard Bruce protocol suggests an excellent prognosis.
 - Exercise stress testing with EKG alone has a sensitivity of 68% and specificity of 77%.
 - Exercise stress testing with echo has a sensitivity of 85% and specificity of 77%.

- Exercise stress testing with technetium^{99m} sestamibi has a sensitivity of 87% and specificity of 64%.
- Cardiac catheterization:
 - Considered the gold standard for evaluating coronary arteries.
 - A history of a recent negative catheterization does not fully exclude AMI, i.e., in cases of vasospasm or cocaine use.

Diagnostic Procedures/Surgery

EKG, cardiac enzymes, echo, stress testing

DIFFERENTIAL DIAGNOSIS

See ACS chapters.



TREATMENT

PRE HOSPITAL

- Cardiac monitoring
- Out-of-hospital EKG:
 - Alone has a sensitivity of 76% and specificity of 88% for ACS.
 - Alone has a sensitivity of 68% and specificity of 97% for AMI.

INITIAL STABILIZATION/THERAPY

- Cardiac monitoring
- Oxygen saturation

ED TREATMENT/PROCEDURES

- See “Acute Coronary Syndrome: Stable Angina”; “Acute Coronary Syndrome: Unstable Angina”; and “Acute Coronary Syndrome: MI” for more detail.
- Guidelines for cardiac testing
- History suggestive of ACS:
 - Obtain ECG and 1st troponin (or other cardiac biomarkers).
- ECG or 1st troponin abnormal:
 - Admit; consider cardiology consult.
- Ongoing chest pain or pressure:
 - Obtain sestamibi or echo.
 - Consider serial EKGs
- Sestamibi, serial EKG or echo abnormal:
 - Admit or cardiology consult.
- Second troponin (or other cardiac biomarkers) abnormal:
 - Admit; consider cardiology consult.
- Ancillary testing:
 - For low- to moderate-risk patients: standard exercise testing (ETT).

- If low-risk patient with good follow-up, ACC/AHA guidelines allow for outpatient stress testing within 72 hr.
- Per 2007 AHA/ACC guidelines CTCA “reasonable alternative” to stress testing.
 - For abnormal or uninterpretable EKG: Stress echo or sestamibi.
 - For patient unable to exert self: Pharmacologic ETT (i.e., dobutamine stress or dipyridamole sestamibi).
 - Ancillary testing abnormal:
 - Cardiology consult or admit.

MEDICATION

Patient should not be started on new antianginal medication before stress testing in the ED.



FOLLOW-UP

DISPOSITION

Admission Criteria

- History suggestive of cardiac etiology for chest pain and ED observation for serial testing unavailable
- Abnormal or changed EKG and ED observation unavailable
- Positive cardiac biomarkers
- Positive rest imaging
- If the diagnosis is unclear, admission to the hospital or an ED observation unit may be useful for serial cardiac biomarkers, EKGs, and further ancillary testing.
- Early positive stress test:
 - If the patient has a positive stress test, the decision for admission should be made in consultation with the primary care physician or cardiologist.

Discharge Criteria

Patients who meet the following criteria are safe to discharge:

- History not suggestive of cardiac etiology for chest pain
- Normal ECG
- Normal cardiac testing

FOLLOW-UP RECOMMENDATIONS

- Abnormal stress test will require close follow-up with cardiology or PCP.
- Undifferentiated CP should have ED stress testing unless clear follow-up is available.

PEARLS AND PITFALLS

- Normal EKG or enzymes do not rule out CAD.
- Repeat EKG or additional leads improve sensitivity in detecting AMI.
- Most ED patients with undifferentiated chest pain will need some form of additional testing.

ADDITIONAL READING

- Cardiac Radionuclide Imaging Writing Group. Criteria for Cardiac Radionuclide Imaging. ACCF/ASNC/ACR/AHA/ASE/SCCT/SCMR/SNM 2009 Appropriate Use Criteria for Cardiac Radionuclide Imaging. Joint guideline of ACC/AHA.
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- Yiadom MY. Acute coronary syndrome clinical presentations and diagnostic approaches in the emergency department. *Emerg Med Clin North Am*. 2011;29:689–697.



CODES

ICD9

- 89.59 Other nonoperative cardiac and vascular measurements
- 411.1 Intermediate coronary syndrome
- 413.9 Other and unspecified angina pectoris

ICD10

- I20.9 Angina pectoris, unspecified
- I24.9 Acute ischemic heart disease, unspecified

CARDIAC TRANSPLANTATION COMPLICATIONS

Jarrold Mosier • Samuel M. Keim

BASICS

DESCRIPTION

- Cardiac transplant recipients are a unique population with increased risk for cardiac ischemia, heart failure, as well as general risks as an immunocompromised host.
- 1,900–2,300 cardiac transplants per yr in US
- 1-yr survival 85–90%; 5-yr survival ~75%
- Typical immunosuppressive therapy to control rejection is a “triple-drug” regimen often including steroids.
- Frequent biopsies are used initially to evaluate rejection; echocardiography often used in children.
- Complications occur most commonly in the 1st 6 wk after cardiac transplantation

Geriatric Considerations

- The proportion of elderly patients on the transplant list, and receiving transplants are increasing.
- Due to changes in immune system with age, elderly transplant recipients are at increased risk of life-threatening infections, and acute rejection.

Pregnancy Considerations

- Pregnancy after cardiac transplant is becoming more common. Between 1988 and 2010, 63 women received either heart or heart–lung transplants. They have reported 108 pregnancies, all progressing to live births.
- Most common complications include hypertension, pre-eclampsia, and rejection.
- Physiologic changes that occur with pregnancy do not relate to increased rate of heart failure in transplant patients.
- Special attention should be paid to these patients regarding rejection and infection given their immunosuppression.

ETIOLOGY

- Rejection
 - Hyperacute rejection
 - Occurs within minutes of transplantation
 - Rare, due to ABO or other graft/host major incompatibility
 - Aggressive and immediately fatal to graft
 - Acute rejection
 - Lymphocyte infiltration and myocyte destruction

- Most common in 1st 6 wk
- May occur at any time
- 75% prevalence
- Chronic rejection
 - Fibrosis and graft vascular disease
 - Long-term complication
 - Incompletely understood etiology
 - No effective therapy
- Cardiac allograft vasculopathy
 - Analogous to accelerated coronary artery disease in native hearts
 - Limits long-term survival, leading cause of mortality after 1 yr
- Immune-mediated atherosclerosis
 - Form of chronic rejection
- Infections
 - 1st mo
 - Bacterial infections are the most common cause of mortality during this high-risk time period
 - Pneumonia (*Pseudomonas*, *Legionella*, other gram-negative organisms)
 - Mediastinitis
 - Wound infection
 - UTI
 - 1st yr
 - Opportunistic and conventional infections
 - Cytomegalovirus (CMV)
 - Herpes simplex virus (HSV)
 - *Legionella*
 - Fungal infections
 - *Pneumocystis carinii*
- Medication toxicity
 - Cyclosporine, Neoral (2nd-generation cyclosporine), tacrolimus:
 - Nephrotoxicity (30% incidence)
 - Hepatotoxicity
 - Neurotoxicity
 - Hyperlipidemia, diabetogenic
 - Azathioprine, mycophenolate mofetil:
 - Bone marrow suppression
 - Leukopenia
 - Sirolimus:
 - Hyperlipidemia
 - Wound healing
 - Steroids
 - Osteoporosis

- Cushing disease
- Neoplasms
 - Secondary to immunosuppression
 - 10–100 times more common vs. general population
 - Skin and lip cancer
 - Lymphomas
 - Kaposi's sarcoma
 - Solid organ neoplasms

Pediatric Considerations

- If the patient is not on steroids, bacteremia risk is similar to that in the general population.
- High incidence of pneumonia
- Patients on steroids may not show meningeal signs



DIAGNOSIS

SIGNS AND SYMPTOMS

- Acute rejection
 - Nonspecific symptoms predominate because the heart is usually denervated
 - Fatigue
 - Dyspnea
 - Low-grade fever
 - Nausea
 - Vomiting
 - May be difficult to differentiate between infection and acute rejection
- Heart failure
 - Tachypnea
 - Rales
 - Hypoxia
 - S3
 - Murmur
 - Edema
- Allograft vasculopathy
 - As early as 3 months after transplantation (20–50% incidence by 5 yr)
 - Denervated hearts do not present with typical angina.
 - Insidious onset
 - Fatigue
 - Cough
 - Dyspnea
 - Acute onset
 - Heart failure

- Sudden death
- Infarction
- Infection (Opportunistic and conventional)
 - Fever
 - Skin lesions (zoster)
 - CMV
 - Mild (flu-like illness)
 - Fever
 - Nausea
 - Malaise
 - Pneumonitis (13–50% mortality)
 - Hepatitis
 - Gastroenteritis
 - Profound leukopenia

Pediatric Considerations

- Higher risk for post-transplant lymphoproliferative disease with Epstein–Barr virus seroconversion
- Like adults, at risk for allograft vasculopathy and its associated cardiac ischemia

ESSENTIAL WORKUP

- Assess for signs of rejection, cardiac dysfunction, and infarction:
 - ECG
 - Cardiac enzymes
 - Chest radiograph
 - Echocardiography
- Possible rejection requires biopsy, consult transplant team.

Pediatric Considerations

Normal fever workup + chest radiograph and ECG; if on steroids, perform LP

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Electrolytes:
 - Cyclosporine effects:
 - Increased blood urea nitrogen, creatinine
 - Hyperkalemia
 - Metabolic acidosis
 - Hyponatremia
- CBC:
 - Relative eosinophilia may indicate rejection over infection
- Blood and urine culture if febrile

- Lumbar puncture if seizures, altered mental status, or severe headache
- BNP (expect baseline elevation)
- CMV titers
- Urine antigen test
- Cyclosporine trough level

Imaging

- ECG
 - Tachycardia
 - 20% decrease in total voltage (nonsensitive)
 - Note that normal rhythm for denervated heart is sinus 90–110 bpm
 - Depending on transplant surgical technique, may see 2 P-waves (native and donor heart):
 - Native P-waves do not correspond to quasi-random signal
- Chest radiograph
 - Cardiomegaly
 - Pulmonary edema
 - Pleural effusions
 - Compare with previous (healthy donor heart may appear large in small recipient)
- Echocardiography
 - Decreased mitral deceleration time
 - Initial diastolic dysfunction
 - Biventricular enlargement
 - Mitral/tricuspid regurgitation

DIFFERENTIAL DIAGNOSIS

- Rejection
- Infection
- Ischemia
- CMV
- Viral illness
- Malignancy
- Cyclosporine toxicity



TREATMENT

PRE HOSPITAL

Adenosine should not be given to patients who have had a heart transplant as the effects may be prolonged and unpredictable.

INITIAL STABILIZATION/THERAPY

- IV access
- Oxygen
- Monitor
- Intubation
- Defibrillation/pacing
- Vasopressors as required
- Arrhythmias
 - Advanced cardiac life support
 - Bradycardia does not respond to atropine; use isoproterenol

ED TREATMENT/PROCEDURES

- Hemodynamically significant rejection
 - Methylprednisolone
 - May also require OKT3 or other anti-T-cell antibody therapy
- Infarct/vasculopathy
 - Aspirin
 - Heparin
 - Possible angioplasty
 - Likely need retransplantation
- CMV
 - Empiric IV ganciclovir
- HSV
 - Oral or IV acyclovir
- Gastroenteritis
 - Search for CMV infection with culture, serology
- Fever without a source
 - Consult infectious disease or transplantation team
- Headache
 - Threshold for CT scan and lumbar puncture should be low (meningitis, abscess)
- Serious illness/trauma/operation
 - Steroid burst
 - Limit NSAID use because risk for renal insufficiency from cyclosporine and tacrolimus.

MEDICATION

- Acyclovir: 5–10 mg/kg IV q8h calculate dose on IBW; genital herpes: 400 mg PO TID × 7–10 days; varicella: 20 mg/kg up to 800 mg PO QID for 5 days
- Ceftriaxone: 50 mg/kg IV q12–24h
- Cyclosporine, CellCept, tacrolimus, sirolimus, Neoral, azathioprine, mycophenolate mofetil: Per transplantation team
- Ganciclovir: Insert IV; 5 mg/kg BID for 2–3 wk (adjust for renal function)
- Isoproterenol: 1–4 µg/min, titrate to effect; max. 10 µg/min

- Methylprednisolone: 1 g IV; peds: 10–20 mg/kg IV
- OKT3, daclizumab or other antibody therapy: Per transplant team

IN PATIENT CONSIDERATIONS

Admission Criteria

- Hemodynamically significant rejection
- Vasculopathy/ischemia
- New dysrhythmia
- Poorly controlled hypertension
- Congestive heart failure
- Dyspnea
- Hypoxia
- Temperature > 38°C in adult or child on steroids
- Suspected CMV (unexplained fever, gastroenteritis, or interstitial pneumonitis)
- Not tolerating oral medicines
- Syncope

Discharge Criteria

- Mild rejection
- Only in consultation with transplantation team
- Fever in nontoxic child:
 - Do not give children stress-dose steroids

ADDITIONAL READING

- Abecassis M, Bridges ND, Clancy CJ, et al. Solid-organ transplantation in older adults: Current status and future research. *Am J Transplant.* 2012;12:2608–2622.
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CODES

ICD9

996.83 Complications of transplanted heart

ICD10

- T86.20 Unspecified complication of heart transplant
- T86.21 Heart transplant rejection
- T86.23 Heart transplant infection

CARDIOGENIC SHOCK

Nadeem Alduaij



BASICS

DESCRIPTION

- Persistent hypotension and tissue hypoperfusion due to cardiac dysfunction in the presence of adequate intravascular volume and left ventricular (LV) filling pressure
- Most common cause of death in hospitalized patients with acute MI (AMI)
- Underlying mechanisms in AMI:
 - Pump failure:
 - $\geq 40\%$ LV infarct
 - Infarct in pre-existing LV dysfunction
 - Reinfarction
 - Mechanical complications:
 - Acute mitral regurgitation
 - Ventricular septal defect
 - LV rupture
 - Pericardial tamponade
 - Right ventricular (RV) infarction
- 5–8% of patients with STEMI develop cardiogenic shock
- Role for a systemic inflammatory response syndrome via excess nitric oxide in the pathophysiology of cardiogenic shock
- Role of initial treatment with β -blockers, ACEI, and high-dose diuretics in cardiogenic shock development

ETIOLOGY

- AMI
- Sepsis
- Myocarditis
- Myocardial contusion
- Valvular disease
- Cardiomyopathy
- Left atrial myxoma
- Drug toxicity:
 - β -blocker
 - Calcium channel blocker
 - Adriamycin



DIAGNOSIS

SIGNS AND SYMPTOMS

- ABCs and vital signs:
 - Patent airway (early)
 - Labored breathing and tachypnea (early); respiratory failure (late)
 - Diffuse crackles or wheezing
 - Hypoxia
 - Hypotension:
 - Systolic BP < 90 mm Hg
 - Decline by at least 30 mm Hg below baseline level
 - Tachycardia
 - Weak pulses
- General:
 - Cyanosis
 - Pallor
 - Diaphoresis
 - Dulled sensorium
 - Decrease in body temperature
 - Urine flow of < 20 mL/hr
- Neck:
 - Jugular venous distention
- Cardiac:
 - Ischemic chest pain
 - Systolic apical blowing murmur
 - Gallop rhythm:
 - S3 reflects severe myocardial dysfunction.
 - S4 is present in 80% patients in sinus rhythm with AMI.
 - Systolic click:
 - Suggests rupture of the chordae tendineae
- Abdominal:
 - Epigastric pain
 - Nausea and vomiting
- Neurologic:
 - Obtundation

History

- Obtain history from patient, family, or EMS for clues to possible etiology
- Medications history

Physical-Exam

- Perform rapid survey and stabilize ABCs
- Distended neck veins and cool extremities distinguish cardiogenic shock from distributive and hypovolemic shock

- Careful heart and lung exam

ESSENTIAL WORKUP

Ancillary studies further define the type and degree of cardiac injury and determine the indications for emergent catheterization or surgical intervention.

DIAGNOSIS TESTS & INTERPRETATION

ECG:

- Normal ECG does not rule out AMI.
- Findings of AMI (ST-elevations in 2 or more contiguous leads)
- May occur in non-ST-elevation acute coronary syndrome
- Dysrhythmias
- LV hypertrophy

Lab

- B-type natriuretic peptide (BNP):
 - Diagnostic and prognostic value
- Creatine kinase (CK), CK-MB, troponin
- Electrolytes and renal function:
 - Acute renal failure is a strong predictor of mortality.
- CBC:
 - Identify anemia or elevated WBC.
- Drug levels (e.g., digoxin)

Imaging

- CXR:
 - Pulmonary congestion
 - Pleural effusion
 - Cardiomegaly
 - Pneumonia
 - Pneumothorax
 - Pericardial effusion
- Emergent echocardiography:
 - Transthoracic echocardiography (TTE) with color Doppler
 - LV contractility looking for hypokinesis, akinesis, or dyskinesis
 - Acute mitral regurgitation or septal defects
 - RV dilatation, tricuspid insufficiency, high pulmonary artery and RV pressures suggest pulmonary embolism
 - RV hypokinesis or akinesis, RV dilatation, normal pulmonary pressures suggest RV infarction
 - Pericardial effusion, right atrium or RV diastolic collapse suggest cardiac tamponade

DIFFERENTIAL DIAGNOSIS

- Obstructive shock:
 - Tension pneumothorax
 - Cardiac tamponade
 - Pulmonary embolism
 - Spontaneous esophageal rupture
 - Air embolus
- Distributive shock:
 - Sepsis
 - Anaphylaxis
 - Addisonian crisis
 - Neurogenic shock
- Hypovolemic shock:
 - Hemorrhage
 - GI losses
 - Dehydration
 - Burns



TREATMENT

PRE HOSPITAL

- ABCs, IV access, O₂, monitor
- Consider fluid bolus if no crackles
- Aspirin
- Nitroglycerin or morphine sulfate for chest pain in absence of hypotension
- Transport AMI patients to facility with 24-hr cardiac revascularization capability

INITIAL STABILIZATION/THERAPY

- ABCs
- 2 large-bore peripheral IV lines
- Cardiac monitor
- Endotracheal intubation for airway compromise:
 - Consider etomidate for induction (minimal effect on BP)
- Fluid challenge (100–250 mL normal saline) in absence of pulmonary congestion
- Foley catheter to monitor urine output

ED TREATMENT/PROCEDURES

- AMI:
 - Aspirin
 - Heparin
 - Thrombolysis if percutaneous coronary intervention or bypass surgery not available

- GP IIb/IIIa inhibitors prior to percutaneous coronary intervention
- Hypotension:
 - Norepinephrine is 1st-line vasopressor
 - Consider dopamine in absence of NE
- Normotensive patient:
 - Dobutamine may be used with NE or dopamine; combine with nitroprusside in acute mitral regurgitation
 - Milrinone may be considered in conjunction with dobutamine or dopamine
- Pulmonary edema:
 - Nitroglycerin drip or furosemide in the normotensive patient
- Prompt cardiology consultation is crucial for the initiation of the following therapies:
 - IABP independently improves survival in experienced centers
 - Early revascularization is the single most important life-saving measure

MEDICATION

- Dobutamine: 3–5 $\mu\text{g}/\text{kg}/\text{min}$, titrate to 20–50 $\mu\text{g}/\text{kg}/\text{min}$ as needed IV
- Dopamine: 3–5 $\mu\text{g}/\text{kg}/\text{min}$, titrate to 20–50 $\mu\text{g}/\text{kg}/\text{min}$ as needed IV
- Furosemide: 40–80 mg/d (peds: 1 mg/kg IV or IM, not to exceed 6 mg/kg) IV or IM
- Milrinone: 50 $\mu\text{g}/\text{kg}$ loading dose, 0.375–0.75 $\mu\text{g}/\text{kg}/\text{min}$ continuous infusion IV
- Nitroglycerin: 10–20 $\mu\text{g}/\text{min}$ (peds: 0.1–1 $\mu\text{g}/\text{kg}/\text{min}$) IV, USE NON-PVC tubing
- Nitroprusside: 0.3 $\mu\text{g}/\text{kg}/\text{min}$, titrate to a max. of 10 $\mu\text{g}/\text{kg}/\text{min}$ IV
- Norepinephrine: 2 $\mu\text{g}/\text{min}$, titrate up as needed IV



FOLLOW-UP

DISPOSITION

Admission Criteria

All patients in cardiogenic shock require admission to a critical care unit.

PEARLS AND PITFALLS

- Cardiogenic shock is the leading cause of death in inpatient AMI.
- Early recognition of preshock states is essential.
- Early revascularization offers better outcomes.

ADDITIONAL READING

- De Backer D, Biston P, Devriendt J, et al. Comparison of dopamine and norepinephrine in the treatment of shock. *N Engl J Med.* 2010;362:779–789.
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Emergency Medicine: A Comprehensive Study Guide. New York, NY: McGraw-Hill; 2010.

- Reynolds HR, Hochman JS. Cardiogenic shock: Current concepts and improving outcomes. *Circulation*. 2008;117:686–697.
- Topalian S, Ginsberg F, Parrillo JE. Cardiogenic shock. *Crit Care Med*. 2008;36:S66–S74.



See Also (Topic, Algorithm, Electronic Media Element)

Shock; MI



CODES

ICD9

785.51 Cardiogenic shock

ICD10

R57.0 Cardiogenic shock

CARDIOMYOPATHY

David T. Chiu • Edward Ullman



BASICS

DESCRIPTION

Diseases of the myocardium associated with cardiac dysfunction:

- Dilated:
 - Idiopathic in 25% of all cases of heart failure
- Hypertrophic
- Restrictive
- Arrhythmogenic right ventricular (RV)
- Unclassified cardiomyopathy
- Specific cardiomyopathy:
 - Heart muscle disease associated with a systemic disease or condition

Pediatric Considerations

- Genetic: 20–30%
- Acquired
- Idiopathic

ETIOLOGY

- Dilated:
 - Idiopathic
 - Viral
 - Genetic/toxic
 - Immune
 - Familial
- Hypertrophic:
 - Familial disease with autosomal dominance
- Restrictive:
 - Idiopathic
 - Amyloid
- Arrhythmogenic RV:
 - Familial disease with dominant and recessive patterns
- Specific infectious:
 - Lyme disease
 - Viral
 - Chagas disease
 - HIV
- Toxic agents:

- Alcohol
- Chemotherapeutic agents
- Peripartum
- Metabolic:
 - Hyperthyroidism
 - Pheochromocytoma
 - Takotsubo (stress catecholamine)
- General system diseases:
 - Lupus
 - Scleroderma
 - Neuromuscular diseases
 - Amyloidosis

Pediatric Considerations

- Idiopathic
- Genetic:
 - Inborn errors of metabolism
 - Malformation syndromes
 - Neuromuscular disease
 - Familial isolated cardiomyopathy disorders
- Acquired:
 - Vitamin and/or trace mineral deficiencies
 - Electrolyte disturbances
 - Endocrine disorders
 - Toxins
 - Collagen vascular disease
 - Immunologic disease
 - Malignancy
 - Morbid obesity
 - Myocarditis
 - Pulmonary disease
 - Kawasaki disease
 - Infection
 - Radiation
 - Congenital heart disease
 - Asphyxia



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Antecedent illness or exposure:
 - Chemotherapy
 - HIV
 - Lyme disease
 - Viral
- Underlying systemic condition:
 - Hemochromatosis
 - Sarcoidosis
 - Pregnancy
- Substance abuse history
- Family history:
 - Familial sudden death
- Exertional complaints (syncope, dyspnea)
- Dizziness
- Near syncope and syncope
- Palpitations
- Sudden death
- Ventricular arrhythmias
- CHF

Pediatric Considerations

- Irritability
- Hepatomegaly
- Generalized muscle weakness
- Acute biochemical crisis
- Hypoglycemia
- Metabolic acidosis
- Hyperammonemia
- Cyanosis
- Encephalopathy
- Dysmorphic features

Pregnancy Considerations

See Cardiomyopathy, Peripartum.

Physical-Exam

- Vital signs
- Cardiopulmonary exam
- Abdominal organomegaly
- Edema
- Other:
 - Rash

- Goiter
- Systemic illness

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC
- Chemistry panel
- Liver function tests, thyroid function tests
- Cardiac biomarkers
- Brain (B-type) natriuretic peptide: Level > 100 pg/mL
- Serologies: Not useful in the ED

Imaging

- CXR:
 - Dilated cardiomyopathy:
 - Cardiomegaly
 - Pulmonary congestion
 - Pleural effusions
 - Hypertrophic cardiomyopathy (HCM):
 - See Cardiomyopathy, Hypertrophic.
 - Restrictive cardiomyopathy:
 - Normal cardiac silhouette
 - Pulmonary congestion
- Emergency transthoracic 2D echocardiography by emergency physician:
 - Depressed LV ejection fraction (EF)
 - Excludes pericardial tamponade
- Formal echocardiography:
 - Study of choice
 - Identification of underlying disease
- Nuclear scintigraphy:
 - When ECG is indeterminate
 - Determination of the thickness of the septum and free wall
 - Alternatives to echocardiography
- CT and MRI distinguish between constrictive pericarditis and restrictive cardiomyopathy.
- MR comprehensive assessment of heart failure: Assess myocardial anatomy, regional and global function, and viability. Allows assessment of perfusion and acute tissue injury (edema and necrosis), and nonischemic heart failure, fibrosis, infiltration, and iron overload can be detected.

Diagnostic Procedures/Surgery

EKG:

- HCM:
 - Left ventricle (LV) hypertrophy
 - Q-waves in leads II, III, aVF, V5, and V6 in the early teenage years (most specific)
- Dilated, Lyme, Chagas, and toxic cardiomyopathies:
 - Atrial fibrillation
 - Heart block
 - Conduction abnormalities
 - Pseudoinfarct pattern with pathologic Q-waves in anterior and inferior leads without coronary artery disease

ALERT

Takotsubo (stress cardiomyopathy) can mimic STEMI (ST-elevation MI).

- Cardiac catheterization:
 - Suspicion of ischemia
 - Treatable systemic disease
- HCM:
 - Assessment of hemodynamic abnormalities
 - Endomyocardial biopsy
 - Evaluation for myocarditis or define etiology
- Cardiac CT or MRI

Pediatric Considerations

- Electrolytes
- pH
- Glucose
- Ammonia level
- Cardiac output
- Dysmorphic evaluation
- EKG
- Echocardiogram:
 - Genetic workup; see individual causes

DIFFERENTIAL DIAGNOSIS

- Other causes of dyspnea:
 - Chronic obstructive pulmonary disease
 - Anemia
 - Asthma
 - Interstitial lung disease
 - Pulmonary embolism
 - Pericardial tamponade
 - Valvular heart disease

- Ischemic heart disease
- Hypothyroidism
- Constrictive pericarditis, commonly confused with restrictive cardiomyopathy
- Other causes of syncope:
 - Hypovolemia
 - Heat disorder
 - Hypoglycemia
 - Arrhythmia
 - Cardiac ischemia



TREATMENT

PRE HOSPITAL

- Monitor
- Oxygen
- Avoid or use a lower dose of nitroglycerin in suspected HCM
- Decompensated heart failure:
 - Nitroglycerin
 - Noninvasive positive-pressure ventilation

INITIAL STABILIZATION/THERAPY

Airway, breathing, and circulation:

- Control airway as needed.
- Supplemental oxygen
- Noninvasive positive-pressure ventilation

ED TREATMENT/PROCEDURES

- Anticoagulation:
 - Dilated cardiomyopathy
 - Standard treatment of atrial fibrillation
 - Systemic embolization
- Limited ED experience with agents effective in HCM:
 - Disopyramide to reduce obstruction
 - Amiodarone to convert and maintain sinus rhythm
- Standard treatment of CHF
- Standard treatment of dysrhythmias

ALERT

- Keep NPO until inborn errors of metabolism ruled out.
- IV fluids:
 - D10 should be given until defects in the protein or fatty acid metabolism pathways are ruled out.

- IV fluids need to be given slowly and judiciously to avoid rapid fluid shifts to the extravascular space.

ALERT

Do not give any products with lactate to avoid worsening any metabolic acidosis or lactic acidemia:

- Antioxidants and vitamin cofactors
- L-Carnitine to increase mitochondrial energy metabolism
- Standard treatment of CHF
- Sodium dichloroacetate (DCA) acutely lowers acetic acid levels in patients with mitochondrial disorders.

MEDICATION

- Amiodarone: 5 mg/kg over 10 min
- Carnitine: (Peds: 50–300 mg/kg/d PO or IV)
- Digoxin: Start 0.125 mg IV
- Diltiazem IV: 0.25 mg/kg actual body weight over 2 min (average adult dose: 20 mg); repeat bolus dose (may be administered after 15 min if the response is inadequate): 0.35 mg/kg actual body weight over 2 min (average adult dose: 25 mg); continuous infusion 10 mg/hr; rate may be increased in 5 mg/hr increments up to 15 mg/hr as needed
- Disopyramide: 100–200 mg PO q6h
- Esmolol IV: Loading dose: 500 µg/kg over 1 min; follow with a 50 µg/kg/min infusion for 4 min; infusion may be continued at 50 µg/kg/min or, if the response is inadequate, titrated upward in 50 µg/kg/min increments (increased no more frequently than q4min) to a max. of 200 µg/kg/min
- Furosemide: 20–40 mg IV to a max. of 200 mg on subsequent doses (peds: 1 mg/kg IV q12–24h)
- Heparin: Load 80 IU/kg IV; then 18 IU/kg/hr
- Metoprolol IV: 2.5–5 mg q2–5min (max. total dose: 15 mg over a 10–15-min period)
- Milrinone: Bolus 50 µg/kg IV over 10 min, then 0.375–0.75 µg/kg/min IV
- Nesiritide: Bolus 2 µg/kg IV, then 0.01 µg/kg/min IV with a max. of 0.03 µg/kg/min
- Nitroglycerin: 5 µg/min IV titrate to SBP

Geriatric Considerations

Use caution in geriatric dosing.



FOLLOW-UP

DISPOSITION

Admission Criteria

- New or suspected cardiomyopathy
- Syncope in which dysrhythmias or HCM are possible etiologies
- Familial history of premature sudden death
- Cardiogenic shock

Discharge Criteria

- Diagnosed cardiomyopathy with mild CHF that improves with ED therapy
- Restrictive cardiomyopathy or HCM
- Cardiology consultation for discharge planning

Issues for Referral

Patients with EF < 35% may require referral for:

- Single-chamber implantable cardioverter defibrillator
- Atrial-synchronized biventricular pacing
- Ventricular assist devices
- Heart transplant

FOLLOW-UP RECOMMENDATIONS

- Primary care
- Cardiology
- Genetic testing may be indicated.

PEARLS AND PITFALLS

- Emergency physician bedside echocardiography is a useful tool for patients with syncope or exertional symptoms
- Obtaining family history in suspected cardiomyopathy

ADDITIONAL READING

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- Egan DJ, Bisanzo MC, Hutson HR. Emergency department evaluation and management of peripartum cardiomyopathy. *J Emerg Med.* 2009;36(2):141–147.
- Karamitsos TD, Francis JM, Myerson S, et al. The role of cardiovascular magnetic resonance imaging in heart failure. *J Am Coll Cardiol.* 2009;54:1407–1424.



See Also (Topic, Algorithm, Electronic Media Element)

- Cardiomyopathy, Hypertrophic
- Cardiomyopathy, Peripartum



CODES

ICD9

- 425.4 Other primary cardiomyopathies
- 425.18 Other hypertrophic cardiomyopathy
- 674.54 Peripartum cardiomyopathy, postpartum condition or complication

ICD10

- I42.2 Other hypertrophic cardiomyopathy
- I42.9 Cardiomyopathy, unspecified
- O90.3 Peripartum cardiomyopathy

CARDIOMYOPATHY, HYPERTROPHIC

L. Kristian Arnold

BASICS

DESCRIPTION

- Hypertrophic cardiomyopathy (HCM):
 - Genetic disorder affecting the sarcomere:
 - Various mutations
 - Many phenotypic variations
 - Hypertrophied (regionally or globally), nondilated left or, rarely, right ventricle in the absence of another cause of degree of hypertrophy observed, such as hypertension or aortic stenosis
 - 2 general types:
 - Nonobstructive (HCM)—75% of patients. Estimated around 1% annual mortality
 - Obstructive (HOCM)—25% of patients. More severe—estimated 2% annual mortality
 - Manifests at all ages, from neonate to elderly:
 - Most manifest in childhood and adolescence—pubertal growth spurt
 - Usually more severe when diagnosed at younger age
 - Small percent progress to reduced LV function
 - Clinical presentation in mid and late life not uncommon
 - May initially be misdiagnosed as asthma, COPD, deconditioning or sleep apnea
 - Lethal arrhythmias more common in younger patients
 - Most common cause of atraumatic death in young (<35 yo) athletes
 - Supraventricular arrhythmias common with incidence increasing with age
 - Atrial fibrillation both common and poorly tolerated
- Prevalence ~ 1 in 500 adults general population:
 - Based on echocardiographic diagnosis
- Structural pathology:
 - Irregular, marked ventricular wall thickening with disarray of myofibrils in the thickened regions and fibrin deposition:
 - Affects higher-pressure LV more than right and, in obstructive form, if obstruction removed, hypertrophy decreases
 - Some phenotypes have progressive wall thinning with age—usually associated with thicker wall early
 - Thickening usually asymmetric involving the septum to a greater extent than the free ventricular wall
 - Atrial dilatation secondary to diastolic filling stiffness

- Impaired microvascular dilation associated with intimal thickening and perivascular collagen deposition
- Outpatient long-term management
 - Avoidance of volume depletion and elevated cardiac demand—depending on degree and location of hypertrophy
 - Pharmacologic
 - β -Blockers or verapamil to slow and control rate, thus prolonging diastole
 - Implantable cardiac defibrillator
 - In patients with history of syncope, cardiac arrest, family member with sudden death, asymptomatic nonsustained ventricular tachycardia (VT), abnormal BP response to exercise, massive hypertrophy
 - Alcohol ablation of hypertrophic outflow-obstructing septal tissue
 - Surgical septal myectomy—improving statistics with more centers performing

RISK FACTORS

Genetics

- 1st cardiac disorder for which genetic basis identified (1989)
- Autosomal-dominant inheritance:
 - > 10 associated genes found:
 - Most encode proteins for sarcomere
 - >700 distinct mutations recognized
 - High penetrance
 - Variable phenotypic expression
 - Some genotypes significantly more lethal:
 - Routine screening impractical at present
 - Increasing complexity with more understanding of interplay between primary sarcomere abnormalities and other genetic and nongenetic factors
 - Some mutations affect cell wall pumps—thus, association with dysrhythmias.

ETIOLOGY

See “Genetics.”

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Obstructive symptoms correlate with exertion or suddenly assuming upright position—activities that decrease venous return or ventricular filling.
 - Severity depends on the location and degree of ventricular wall thickening.
- Shortness of breath
- Dyspnea on exertion
- Exertional or postprandial angina
- Presyncope
- Syncope
- CHF
- Cardiovascular collapse
- Dysrhythmias:
 - Paroxysmal atrial fibrillation:
 - Often leads to significant, rapid clinical deterioration when present with CHF
 - Elevated CVA risk from clots
 - Supraventricular tachycardia
 - Nonsustained VT
 - Bradydysrhythmia rare
 - VT or ventricular fibrillation may lead to sudden death.
- Prior therapy for known HCM might include surgery or alcohol injection to reduce septal bulk:
 - Potential for conduction blocks
 - Potential septal rupture
- Known cases with higher risk of arrhythmia may have implanted defibrillators.
- Family history of sudden death without apparent cause or known HCM

Pediatric Considerations

Due to potential increasing severity in adolescence, any young child with syncope without clear cause, or in association with exercise, should have more extensive, focused history for familial sudden death (standard is 3 generations) and possible referral for evaluation.

Physical-Exam

- No or subtle physical findings
- Most findings in patients with outflow tract obstruction, variably:
 - Loud, left-sided S4
 - Double apical cardiac impulse at the mid to upper sternum
 - Murmur:
 - Crescendo–decrescendo midsystolic murmur at the left sternal edge
 - Radiation to aortic and mitral areas, not to neck or axilla
 - Increasing in intensity with Valsalva maneuver or standing up
 - Quieter with recumbency, squatting, or handgrips

- Frequent associated mitral regurgitation
- With more severe obstruction, a more apparent murmur with radiation to the left sternal border
- Radiation to the axilla if there is associated mitral insufficiency

DIAGNOSIS TESTS & INTERPRETATION

ECG findings:

- Abnormal in >90% pts with HCM
- T-wave inversion >1 mm 2 or + leads V2-V6, II and aVF, or I and aVL or deep TWI in V4-V6 in non-African-Caribbean descent athletes >16 yo
- ST-segment depression >0.5 mm in 2 or more leads requires further investigation
- Q-waves >3 mm in depth or >40 ms duration in at least 2 leads other than III & aVR
- P-wave >120 ms leads I or II with negative portion ≥ 1 mm and ≥ 40 ms in lead V1
- Nonspecific IVCD >140 ms

Lab

- Clinical lab testing is of no assistance.
- Genetic testing may help in outpatient workup, but not in ED.

Imaging

- CXR:
 - Normal in the vast majority
 - Bulge along left heart border representing hypertrophy of free wall of LV
 - Right or left atrial enlargement
 - Pulmonary vascular redistribution
- Transthoracic cardiac echo/Doppler:
 - LV wall >15 mm, with normal or small LV cavity (13–14 mm with other features; e.g., family history in adults), in children ≥ 2 times standard deviation above mean for age, sex, size
 - Systolic outflow obstructions
 - Diastolic filling abnormalities
- Cardiovascular magnetic resonance (CMR)
 - Supplements ECHO
 - Allows more structural detail for evidence of fibrosis
- Stress thallium and PET evaluate ischemia.

Diagnostic Procedures/Surgery

No ED-based procedures are of diagnostic utility.

DIFFERENTIAL DIAGNOSIS

- Vagal and other causes of syncope and presyncope

- Heatstroke
- Aortic stenosis
- Pulmonic stenosis
- Ventricular septal defect
- Mitral regurgitation
- Mitral valve prolapse
- Arteriosclerotic coronary vascular disease
- Differentiate in patients presenting with CHF or angina:
 - _ More ominous in the setting of HCM



TREATMENT

ALERT

Consider HCM in patients who decompensate during standard treatments for CHF, ischemia, or supra-VT, and in young athletes who collapse during or just after exertion—rule out heat stroke. HCM patients may decompensate with IV fluids due to diastolic stiffness.

INITIAL STABILIZATION/THERAPY

- ABCs
- IV catheterization
- Supplemental oxygen
- Cardiac monitor
- Pulse oximetry

ED TREATMENT/PROCEDURES

- Depends on type of presentation: Dysrhythmia, cardiac failure, chest pain or ischemia
- Underlying principle to understand sensitivity to any situation that may impair cardiac filling.
- Patient may need to remain supine.
- Standard CHF or anginal vasodilator therapy may lead to cardiovascular collapse; if this occurs, treat with fluid bolus.
- Attention to any hypovolemia as small degree may significantly impair cardiac output.
- Control rate and improve diastolic filling (underlying principle in treating HCM-associated CHF and angina):
 - _ β -Blockers:
 - Mainstay of therapy
 - Decrease dysrhythmias and lower elevation of pressure gradient across the LV outflow tract
 - _ Calcium channel blockers:

- Verapamil reduces obstruction by decreasing contractility and improving diastolic relaxation and filling.
- Nifedipine relatively contraindicated due to vasodilatation
- Dysrhythmia management:
 - β -Blockers and calcium channel blockers 1st line for supraventricular dysrhythmias
 - Amiodarone:
 - Drug of choice for ventricular dysrhythmias
 - Used when β -blockers and calcium channel blockers fail
 - Electrical cardioversion:
 - Use early in HCM with new atrial fibrillation and CHF

MEDICATION

All medications must be assessed for effect in face of possible outflow track restriction

- Amiodarone: 150 mg over 10 min, then 360 mg over 6 hr, then 540 mg over next 18 hr (peds: 5 mg/kg IV rapid IV/IO bolus, off-label use per manufacturer, but class IIb for VT with a pulse and class indeterminate for VF and pulseless VT, per American Heart Association. Do not use in infants.)
- Propranolol: 1–3 mg (peds: 0.01–0.1 mg/kg slow IV push over 10 min; not to exceed 1 mg/dose) slow IV bolus
- Verapamil: 2.5 mg (peds: > 1 yr: 0.1–0.2 mg/kg/dose over 2 min; repeat q10–30min as needed; not to exceed 5 mg/dose [1st dose] or 10 mg/dose [2nd dose]) IV bolus over 1–2 min, may repeat as 5 mg in 15–30 min
- Phenylephrine: 0.1–0.2 mg (peds: 1–20 μ g/kg) IV slow bolus for severe hypotension (shock) not responding to fluid bolus. Repeat in 10–15 min as needed or start IV drip to titrate to BP; or other pure vasoconstrictor (i.e., no inotropic effect). Maintenance dose: 0.05 μ g/kg/min (peds: 0.1–0.5 μ g/kg/min) IV

First Line

N/A

Second Line

Diltiazem: 0.25 mg/kg (peds: Contraindicated < 12 yr old) IV over 2 min; may repeat in 15 min at 0.35 mg/kg



FOLLOW-UP

DISPOSITION

Admission Criteria

- Unexplained syncope, especially in younger adults.
- ICU admission:

- Syncopal episodes
- CHF
- Angina
- Hemodynamically significant tachydysrhythmia

Discharge Criteria

When increased myocardial wall thickness is an incidental finding during the ED evaluation for another presentation with:

- No history of familial sudden death (proposed guidance—3 generations to rule out in face of suspicion) or personal history of syncope
- Need urgent follow-up with a cardiologist
- Counsel against any activities that may decrease diastolic filling pending follow-up:
 - Counsel against physical exertion until evaluated by cardiologist

Issues for Referral

See “Discharge Criteria.”

FOLLOW-UP RECOMMENDATIONS

See “Discharge Criteria.”

PEARLS AND PITFALLS

- Increasing awareness of genetic and phenotypic variants with implications in definition of “normal variant”:
 - Some authors advocate cardiac ECHO screening for any youth participation in sports.
 - Some authors advocate AICD at diagnosis.
- If HCM is considered in a patient with syncope, it must be ruled out because it is much more likely to be fatal with repeat episodes.

ADDITIONAL READING

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CODES

ICD9

425.18 Other hypertrophic cardiomyopathy

ICD10

I42.2 Other hypertrophic cardiomyopathy

CARDIOMYOPATHY, PERIPARTUM

David W. Callaway



BASICS

DESCRIPTION

- Dilated cardiomyopathy occurring during the last month of pregnancy up to 5 mo following the delivery
- Diagnostic criteria (all required):
 - Onset of myocardial failure during last month of pregnancy or 1st 5 mo after delivery
 - Absence of a specific cause
 - Absence of prior cardiac disease
- Diagnosis requires strict criteria of echocardiographic dysfunction
- Incidence: 3–5/10,000 live births
- ~50% of cases resolve spontaneously
- Mortality: 18–56%
- Risk factors:
 - Older women (> 30 yr)
 - Multiparous women
 - Multiple gestations
 - Prolonged tocolytic therapy (> 4 wk)
 - Obesity
 - Preeclampsia
 - African American
- Systemic and pulmonary embolism more frequent than with other forms of cardiomyopathy
- Factors indicating a poor prognosis:
 - Lower left ejection fraction at 6 mo postpartum
 - Onset > 2 wk postpartum
 - Age > 30 yr
 - African American descent
 - Multiparity

ETIOLOGY

Various causes are suggested but remain unproved:

- Viral infection leading to myocarditis in presence of immunosuppression during pregnancy (*most likely*)
- Immunologic response to an unknown maternal or fetal antigen
- Maladaptive response to the hemodynamic stresses of pregnancy
- Stress-activated cytokines

- Prolonged tocolysis
- Selenium deficiency



DIAGNOSIS

SIGNS AND SYMPTOMS

- Dyspnea
- Dizziness
- Chest pain
- Orthopnea
- Cough
- Paroxysmal nocturnal dyspnea
- Anorexia
- Fatigue
- Arrhythmias

History

- Onset and duration of symptoms
- Unexplained persistent cough
- Excessive weight gain:
 - >2–4 lb/wk
- Prior cardiac disease
- Prior pregnancies and complications

Physical-Exam

- Palpitations
- Jugular venous distention
- Gallop rhythm
- Mitral regurgitation murmur
- Loud P2
- Pulmonary rales
- Peripheral edema (especially rapid onset)
- Hepatomegaly
- Hepatojugular reflux

ESSENTIAL WORKUP

- CXR views:
 - Pulmonary venous congestion
 - Cardiomegaly (can be difficult to differentiate with pregnancy)
 - Pleural effusions
- EKG:
 - Nonspecific

- Left ventricular hypertrophy
- Left atrial enlargement
- T-wave flattening or inversion
- Arrhythmias
- Ventricular ectopy (40%)
- Atrial fibrillation (20%)

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Electrolytes:
 - Generally normal
- BUN, creatinine
- CBC:
 - Mild postpartum anemia may contribute to fatigue and dyspnea.
- Creatine kinase with muscle and brain fraction
- β -Natriuretic peptide (BNP):
 - Useful for distinguishing between heart failure due to diastolic and/or systolic dysfunction and a pulmonary cause of dyspnea
 - BNP > 100 pg/mL diagnosed heart failure with a sensitivity of 90%, a specificity of 76%, and a predictive accuracy of 83%. BNP of \leq 50 pg/mL has a high negative predictive value.

Imaging

- CXR:
 - Cardiomegaly
 - Effusions (usually right sided)
 - 3 phases of pulmonary findings:
 - Stage I: Pulmonary redistribution to upper lung fields (cephalization)
 - Stage II: Interstitial edema with Kerley B lines
 - Stage III: Alveolar edema
 - Bilateral confluent perihilar infiltrates leading to classic butterfly pattern
 - May be asymmetric and mistaken for pneumonia
- Echo:
 - Demonstrates global dilation, cardiac wall thinning, and decreased ejection fraction
 - Criteria for the diagnosis were established by Hibbard et al.:
 - Ejection fraction < 45% or M-mode fractional shortening of < 30%
 - End-diastolic dimension > 2.72 cm/m²
 - Exclude valvular pathology and cardiac tamponade.

Diagnostic Procedures/Surgery

Endomyocardial biopsy:

- Indicated to assess for myocarditis and steroid therapy

DIFFERENTIAL DIAGNOSIS

- Other causes of dilated cardiomyopathy:

- Ischemia
- Infarction
- Valvular rupture or disease
- Chronic HTN
- Familial
- Toxins:
 - Ethanol, anthracyclines, cocaine, drug allergy
- Metabolic:
 - Thiamine
 - Selenium
 - Hypothyroidism
 - Thyrotoxicosis
 - Hypophosphatemia
- Infectious:
 - Viral
 - Parasitic or rickettsial
 - Bacterial
 - Fungal
- Systemic disorders:
 - Sarcoidosis
 - Scleroderma
 - Systemic lupus erythematosus
- Eosinophilic myocarditis
- Neuromuscular dystrophies
- Mitochondrial cardiomyopathies

- Other causes of shortness of breath or edema:

- Pulmonary embolism
- Pneumonia
- Asthma
- Cardiac ischemia
- Anemia
- Hyperthyroidism
- Constrictive pericarditis
- Pericardial tamponade
- Nephrotic syndrome
- Cirrhosis



TREATMENT

PRE HOSPITAL

Differentiate pulmonary edema from acute reactive airway disease.

INITIAL STABILIZATION/THERAPY

ABCs:

- Prompt evaluation of respiratory and hemodynamic status
- Control airway as needed
- Supplemental oxygen
- Continuous positive airway pressure, as needed
- Preload and afterload reduction

ED TREATMENT/PROCEDURES

- Antepartum therapy:
 - _ Nitrates
 - _ Hydralazine
 - _ IV furosemide
 - _ Amlodipine: A dihydropyridine calcium channel blocker that has been shown to improve survival in nonischemic cardiomyopathy patients
 - _ Digoxin to control rate due to atrial fibrillation
 - _ Carvedilol (antepartum and not in acute decompensated phase)
 - _ LMWH if EF < 35%
 - _ Fetal monitoring
- Invasive cardiac monitoring if unstable
- Postpartum therapy:
 - _ Consider adding ACE inhibitors (enalapril) or ARBs.
 - _ Anticoagulation therapy often recommended:
 - 30% of cases complicated by systemic or pulmonary embolism
 - During pregnancy, use SC or IV heparin rather than warfarin, which causes birth defects.
- For severe symptoms or lack of response to standard therapy:
 - _ Dobutamine
 - _ Dopamine
 - _ Nitroprusside
 - _ Assist devices
 - Intra-aortic balloon pump
 - LV assist device
 - Extracorporeal membrane oxygenation
 - _ Immunosuppressive therapy:
 - Advocated for patients who fail to improve within 2 wk of standard medical therapy

- Prednisone with cyclosporine or azathioprine
- Immunoglobulin therapy remains controversial

MEDICATION

- Amlodipine: 2.5–10 mg/d PO
- Bumetanide: 0.5–2 mg IV
- Digoxin: 0.5 mg IV, then 0.25 mg IV q4h for 2 doses; 0.125–0.375 mg/d PO
- Milrinone: 50 µg/kg over 10 min
- Dobutamine: 2–10 µg/kg/min IV
- Dopamine: 2–20 µg/kg/min IV
- Enalapril: 0.625–1.25 mg IV; 2.5–20 mg/d PO
- Furosemide: 20–100 mg IV
- Metoprolol: 12.5 mg PO BID
- Morphine sulfate: 2–4 mg IV q5min
- Nitroglycerin: 0.4 mg sublingual; 1–2 in of nitroglycerin paste; 5–20 µg/min IV, max. of 100–200 µg/min IV. USE NON-PVC tubing
- Nitroprusside: 0.5–10 µg/kg/min IV



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients with pulmonary edema, cardiogenic shock, or evidence of ischemia should be admitted to the ICU.
- All symptomatic patients with new onset of peripartum cardiomyopathy should be admitted.

Discharge Criteria

- Mild left ventricular dysfunction
- Established history of peripartum cardiomyopathy:
 - Mild fluid overload attributable to excessive salt intake
 - Complete resolution of symptoms following ED treatment
 - No evidence of cardiac ischemia
- Close follow-up arranged

Issues for Referral

Close follow-up with a cardiologist

FOLLOW-UP RECOMMENDATIONS

- Drink 6–8 glasses of liquid each day.
- Limit salt intake.

- Avoid alcohol because it may worsen cardiomyopathy.
- Support socks may help decrease the swelling in legs and prevent clot formation.
- Daily weights:
 - Weight gain can be a sign of extra fluid in the body.
 - Call doctor if gain of >2 lb in a day.
- Return for shortness of breath, feeling faint, palpitations.

PEARLS AND PITFALLS

- Remember high rates of thromboembolism in pregnancy and peripartum cardiomyopathy.
- Utilize multidisciplinary approach with cardiology and obstetrics consultations.

ADDITIONAL READING

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CODES

ICD9

674.54 Peripartum cardiomyopathy, postpartum condition or complication

ICD10

O90.3 Peripartum cardiomyopathy

CARPAL FRACTURES

Mary R. Mulcare • Wallace A. Carter



BASICS

DESCRIPTION

- Most commonly injured region of upper extremity
 - Most commonly fractured carpals are the scaphoid (68%) and triquetrum (18%)
- Carpal bone fractures commonly occur with other wrist injuries:
 - Capitate fractures along with scaphoid (scaphocapitate syndrome) sometimes occur with perilunate dislocations
 - Hamate fractures associated with injuries to 4th and 5th CMC and metacarpals concurrent with distal radius fractures

ETIOLOGY

- Fall on outstretched hand (FOOSH) with a hyperextended or hyperflexed wrist
- Direct blow
- Axial loading
- Chronic use injury



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- FOOSH or direct blow
 - Hyperflexion → dorsal avulsion fracture
 - Hyperextension → volar avulsion fracture
- Hook of hamate fractures:
 - Associated with a forceful swing of a racquet or club

Physical-Exam

- Pain, swelling, decreased range of motion
- Individual palpation of each carpal bone is possible with correct positioning of wrist
- Scaphoid fractures:
 - Snuffbox tenderness is sensitive but not very specific
 - Specificity improved with pronation and ulnar deviation of wrist
 - Scaphoid compression test (axial loading thumb causes pain) is also not very specific

- Tenderness of tubercle on palmar aspect at distal wrist crease with wrist in extension
- More specific than snuffbox tenderness

ESSENTIAL WORKUP

- A complete physical exam of the entire upper extremity and shoulder girdle:
 - Evaluate for associated injuries
- Neurovascular exam is essential
- Hamate fractures may be associated with ulnar nerve or artery injuries

DIAGNOSIS TESTS & INTERPRETATION

Imaging

- Anterior–posterior, lateral, oblique views of the hand and wrist
- Special views (e.g., scaphoid views) may be obtained for most of the carpals if physical exam is suspicious
- CT scan has superior sensitivity for fractures
- MRI can diagnose ligamentous injuries

DIFFERENTIAL DIAGNOSIS

- Metacarpal base fractures
- Distal radius or ulna fractures
- Lunate or perilunate dislocations

Pediatric Considerations

Be wary of epiphyseal injuries of the distal radius: Children rarely get simple sprains or fractures of the wrist.



TREATMENT

PRE HOSPITAL

- Prevent contamination of any lacerations overlying the area
- Patients with swelling or significant pain at the wrist or hand:
 - Elevate extremity and apply ice
 - Remove jewelry, watches
 - Immobilize extremity with padded board splints to reduce further injury

INITIAL STABILIZATION/THERAPY

As in any trauma, assess for other more serious injuries.

ED TREATMENT/PROCEDURES

- Isolated carpal bone fractures can be initially managed with splinting
 - Goal to obtain and maintain normal alignment

- Thumb spica:
 - Scaphoid and trapezium fractures
 - Wrist held in slight extension
- Sugar tong splint:
 - Capitate and lunate fractures
 - Extends from MCP joint on dorsal side of hand, wrapping around the elbow, ending at midpalmar crease
 - Wrist neutral
- Volar splint:
 - Triquetrum, pisiform, trapezoid, hamate fractures
 - Extends from midpalmar crease to below the elbow
 - Wrist in slight extension
 - Splint suspected fractures (especially scaphoid) based on physical exam despite negative radiographs
- Open carpal fractures:
 - Requires extensive, high-pressure irrigation
 - Parenteral antibiotics against *Staphylococcus aureus*, with gram-negative coverage in Grade III (involving significant soft tissue damage) open fractures
 - Monitor neurovascular status
 - Tetanus prophylaxis
 - Immediate orthopedic consultation

MEDICATION

- Mild oral analgesics, oral narcotics, NSAIDs for patient comfort
- Proper splinting will relieve most of the pain for these injuries



FOLLOW-UP

DISPOSITION

Admission Criteria

- Open fractures are admitted for early operative irrigation and débridement
- Patients with injuries requiring surgical management (open reduction, unstable displaced fractures) sometimes are admitted for early intervention

Discharge Criteria

Closed, nondisplaced carpal fractures treated with adequate splinting may be discharged to have orthopedic follow-up in 7–10 days.

FOLLOW-UP RECOMMENDATIONS

- Confirmed fractures are referred to orthopedics for definitive casting and further

management

- Missed fractures or improper splinting can lead to long-term complications and disability:
 - Untreated scaphoid, capitate, and lunate fractures lead to high rates of nonunion and avascular necrosis
 - Splinting is crucial for long-term function and mobility
 - Immobilize any injury with significant pain and refer for repeat radiographs in 7–10 days or more advanced imaging (CT or MRI)

PEARLS AND PITFALLS

- Carpal fractures may not be apparent on initial radiographs and may lead to long-term disability if not treated appropriately
- Splint all suspected fractures and refer for repeat radiographs or consider CT scanning in ED or outpatient setting
- Most (90%) scaphoid fractures are isolated injuries:
 - All other carpal fractures are more often associated with other wrist or hand injuries
- Adequate treatment involves splinting in position of function and referral for definitive casting and management

ADDITIONAL READING

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- Phillips TG, Reibach AM, Slomiany WP. Diagnosis and management of scaphoid fractures. *Am Fam Physician.* 2004;70:879–884.
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CODES

ICD9

- 814.00 Closed fracture of carpal bone, unspecified
- 814.01 Closed fracture of navicular [scaphoid] bone of wrist
- 814.03 Closed fracture of triquetral [cuneiform] bone of wrist

ICD10

- S62.009A Unsp fracture of navicular bone of unsp wrist, init
- S62.109A Fracture of unsp carpal bone, unsp wrist, init for clos fx

- S62.116A Nondisp fx of triquetrum bone, unsp wrist, init for clos fx

CARPAL TUNNEL SYNDROME

Matthew T. Spencer • Linda L. Spillane

BASICS

DESCRIPTION

- Carpal tunnel syndrome is caused by compression of the median nerve as it passes through the carpal tunnel.
- The carpal tunnel is the area bound by the carpal bones and the transverse carpal ligament.
- The median nerve, flexor digitorum profundus, flexor digitorum superficialis (FDS), and flexor pollicis longus are located in the carpal tunnel.
- Carpal tunnel syndrome can be classified as acute or chronic.

ETIOLOGY

- Acute:
 - Trauma
 - Infection
 - Snake bite
 - Hemorrhage
 - High-pressure injection injury
- Chronic:
 - Occupational/overuse syndromes—high impact, repetitive motion
 - Pregnancy, birth control pills
 - Granulomatous disease: Tuberculosis, sarcoidosis
 - Mass lesions with median nerve compression
 - Osteophytes
 - Amyloid
 - Multiple myeloma
 - Rheumatoid arthritis
 - Endocrine disorders: Hypothyroidism, diabetes mellitus, acromegaly
 - Chronic hemodialysis
 - Idiopathic

Pediatric Considerations

Idiopathic causes are rare in children; most cases have a correctable cause including:

- Trauma
- Mucopolysaccharidosis
- Hamartoma of the median nerve
- Anomalous FDS
- Hemophilia with hematoma

SIGNS AND SYMPTOMS

History

- Acute or chronic onset
- Numbness/paresthesia in a median nerve distribution:
 - Thumb, index, middle, and radial aspect of ring finger
- Pain:
 - Location: Wrist or hand, sometimes radiating to elbow, forearm, or shoulder
 - Often worse at night—relieved by “shaking out” the hand
 - Exacerbated by repetitive wrist movement and by activities in which the wrist is flexed (e.g., driving)

Physical-Exam

- Weakness of the abductor pollicis brevis and opponens muscles:
 - Innervated by the recurrent branch of the median nerve
 - Patient may complain of dropping things or having decreased fine motor control.
- Loss of 2-point discrimination:
 - Late finding, highly specific
- Atrophy of thenar muscles:
 - Late finding, highly specific

ESSENTIAL WORKUP

- History of characteristic nocturnal pain and paresthesia in the median nerve distribution.
- Muscle weakness and thenar wasting are later findings.
- Provocative testing:
 - Overall poor sensitivity and specificity
 - Phalen test:
 - Wrist flexion for 60 sec produces numbness or tingling in the median nerve distribution.
 - Tinel sign:
 - Gentle tapping over the median nerve at wrist produces tingling in the fingers in the median nerve distribution.
 - Carpal compression test:
 - Direct pressure applied over the proximal carpal ligament for 30 sec produces tingling in the fingers in the median nerve distribution.
 - Tourniquet test:
 - BP cuff inflated to just above the patient’s systolic BP for 2 min produces paresthesia in the median nerve distribution.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Not indicated in most cases
- Thyroid function studies; rheumatoid factor and immune panel if indicated by history and physical exam

Imaging

- Wrist radiograph if trauma or degenerative arthritis suspected
- CT in select cases (not routine):
 - May show encroachment of carpal tunnel
- MRI displays the soft tissues well but not recommended for routine diagnosis:
 - Findings: Palmar bowing of transcarpal ligament, flattened median nerve, median nerve or synovial swelling, fluid in carpal tunnel, signal abnormality of median nerve
- Ultrasound can be diagnostic:
 - Sensitivity of 44–95%; specificity of 57–100%
 - Findings: Median nerve swelling at proximal canal, median nerve flattening at distal canal, bowing of transcarpal ligament

Diagnostic Procedures/Surgery

Nerve conduction studies and electromyography are criterion standard tests.

DIFFERENTIAL DIAGNOSIS

- Cervical nerve root compression:
 - Origin of median nerve is at the 6th and 7th cervical roots.
 - Symptoms are aggravated by erect posture and neck movement.
- Hand–arm vibration syndrome:
 - Characterized by Raynaud, numbness and tingling in ulnar and median nerve distributions when exposed to cold or vibration, weakened grip, and upper extremity myalgias
 - Associated with prolonged exposure to vibration
- Thoracic outlet obstruction
- Osteoarthritis of the 1st carpometacarpal joint
- Brachial plexitis
- Generalized neuropathy
- Syringomyelia
- Multiple sclerosis



TREATMENT

INITIAL STABILIZATION/THERAPY

None necessary

ED TREATMENT/PROCEDURES

- Acute:
 - Hand surgery consultation for surgical release of transverse carpal ligament using either open or endoscopic technique
- Chronic:
 - Analgesics
 - Oral corticosteroids
 - Local corticosteroid injection
 - Avoidance of repetitive wrist movement
 - Splint wrist in neutral position (0°):
 - Worn at night until follow-up
 - Yoga
 - Referral:
 - Primary care physician
 - Occupational medicine for ergometric testing if caused by repetitive motion, and tendon gliding, nerve gliding, or carpal bone mobilization exercises
 - Hand surgeon for evaluation of surgical intervention

MEDICATION

- Analgesics:
 - There are many choices
 - NSAIDs have not been shown to improve long-term outcome
- Oral corticosteroids—short-term benefit:
 - Prednisone: 20 mg daily × 7 days, 10 mg daily × 7 days
 - Prednisolone: 20–25 mg daily, tapered over 2–4 wk
- Local corticosteroid injection—transient relief in 2/3 of patients (many different regimens):
 - Hydrocortisone: 20 mg
 - Methylprednisolone: 15–40 mg
 - Triamcinolone: 20 mg
 - Usually combined with 0.15–0.5 mL 2% lidocaine



FOLLOW-UP

DISPOSITION

Admission Criteria

Acute carpal tunnel syndrome requiring surgical decompression

Discharge Criteria

Chronic carpal tunnel syndrome after adequate pain control

FOLLOW-UP RECOMMENDATIONS

Primary care physician or directly to a specialist in occupational medicine or hand surgery within 1–2 wk

ADDITIONAL READING

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CODES

ICD9

354.0 Carpal tunnel syndrome

ICD10

- G56.00 Carpal tunnel syndrome, unspecified upper limb
- G56.01 Carpal tunnel syndrome, right upper limb
- G56.02 Carpal tunnel syndrome, left upper limb

CAUDA EQUINA SYNDROME

Daniel F. Morris

BASICS

DESCRIPTION

Compression of lumbar and sacral nerve fibers in cauda equina region:

- Nerve fibers below conus medullaris
- Fibers end at L1–L2 interspace.

RISK FACTORS

- Neoplasm
- IV drug use
- Immunocompromised state
- Trauma

ETIOLOGY

- Herniated disc most common:
 - _ L4–L5 discs > L5–S1 > L3–L4
 - _ Most common in 4th and 5th decades of life
- Mass effect from:
 - _ Myeloma, lymphoma, sarcoma, meningioma, neurofibroma, hematoma
 - _ Spine metastases (breast, lung, prostate, thyroid, renal)
 - _ Epidural abscess (especially in IV drug users)
- Blunt trauma
- Penetrating trauma
- Spinal anesthesia

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Low back pain
- Sciatica/radicular pain (unilateral or bilateral)
- Lower-extremity numbness or weakness
- Difficulty ambulating owing to weakness or pain
- Bladder or rectal dysfunction:
 - _ Retention or incontinence

Physical-Exam

- Lumbosacral (LS) tenderness
- Lower-extremity sensory or motor deficits:
 - May be asymmetric
- Decreased foot dorsiflexion strength
- Decreased quadriceps strength
- Decreased deep tendon reflexes
- Saddle hypalgesia or anesthesia
- Decreased anal sphincter tone

ESSENTIAL WORKUP

- Neurologic exam most essential:
 - Straight-leg raise
 - Lasègue sign:
 - With patient supine, flex hip and dorsiflex foot.
 - Pain or spasm in posterior thigh indicates nerve irritation.
 - Perineal sensation
 - Rectal tone
 - Anal wink: Reflex contraction of external anal sphincter with gentle stroking of skin lateral to anus
- Postvoid residual volume:
 - Estimate by bladder catheterization or using US.
 - >50–100 mL is considered abnormal.
 - Residual increases with age.
 - Diagnosis unlikely if normal

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Based on differential diagnoses
- CBC, urinalysis, ESR, and C-reactive protein (CRP)

Imaging

- Radiographs of LS spine
- MRI of spine is definitive study.
- CT myelogram if MRI unavailable

DIFFERENTIAL DIAGNOSIS

- Osteoarthritis, LS strain, sciatica
- Vertebral fracture (pathologic and nonpathologic)
- Osteomyelitis
- Spinal epidural abscess
- Conus medullaris or higher cord compression
- Ankylosing spondylitis, spinal stenosis

- Abdominal aortic aneurysm dissection
- Vascular claudication
- Hip pathology
- Acute transverse myelitis



TREATMENT

PRE HOSPITAL

- Manage airway and traumatic injuries as indicated.
- If evidence of trauma, patient should be transported with full spine immobilization.

ALERT

Even in nontrauma patient, consider spinal immobilization given possibility of unstable lesion.

INITIAL STABILIZATION/THERAPY

- Spine immobilization if trauma or unstable spine lesion suspected
- Analgesia
- NPO until evaluated by neurosurgery

ED TREATMENT/PROCEDURES

- Repeat neurologic exams to detect progression.
- For acute spinal cord trauma (< 8 hr), begin high-dose methylprednisolone protocol.
- Immediate neurosurgical consultation in all cases
- Initiate antibiotics for epidural abscess in consultation with neurosurgery.
- Controversy exists regarding urgency of decompression:
 - Recommendations range from within 6 hr of onset to within 24 hr.

MEDICATION

- Methylprednisolone (high-dose steroid protocol): 30 mg/kg IV bolus, then 5.4 mg/kg/h infusion over next 23 hr. Should be started within 8 hr of injury.



FOLLOW-UP

DISPOSITION

Admission Criteria

- All patients with acute cauda equina syndrome must be admitted to neurosurgical service.
- Patients have good prognosis with rapid surgical decompression.

- Treatment should not be delayed.
- Patients presenting late (> 48 hr) also benefit from surgical decompression.

Discharge Criteria

Patients with established cauda equina syndrome with prior complete evaluation and no new neurologic deficits may be discharged with close follow-up with their neurosurgeon.

PEARLS AND PITFALLS

Ideally, diagnose patients in early phase before irreversible neurologic dysfunction:

- Back pain out of proportion
- Fever and back pain
- Back pain in high-risk groups; screen with ESR/CRP when infection suspected

ADDITIONAL READING

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- Ma B, Wu H, Jia LS, et al. Cauda equina syndrome: A review of clinical progress. *Chin Med J (Engl).* 2009;122(10):1214–1222.
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CODES

ICD9

- 344.6 Cauda equina syndrome
- 344.60 Cauda equina syndrome without mention of neurogenic bladder
- 344.61 Cauda equina syndrome with neurogenic bladder

ICD10

G83.4 Cauda equina syndrome

CAUSTIC INGESTION

Paul Kolecki



BASICS

DESCRIPTION

- Alkalis:
 - Dissociate in the presence of H_2O to produce hydroxy (OH^-) ions, which leads to liquefaction necrosis
 - Postingestion—mainly damages the esophagus:
 - Gastric damage can occur (see “Acids”).
 - Esophageal damage (in the order of increasing damage) consists of:
 - Superficial hyperemia
 - Mucosal edema
 - Superficial blisters
 - Exudative ulcerations
 - Full-thickness necrosis
 - Perforation
 - Fibrosis with resulting esophageal strictures
 - Do *not* directly produce systemic complications.
- Acids:
 - Dissociate in the presence of H_2O to produce hydrogen (H^+) ions, which leads to a coagulation necrosis with eschar formation
 - Postingestion—damages the stomach because of rapid transit time through esophagus:
 - Esophageal damage can occur (see “Alkalis”).
 - Gastric damage (in the order of increasing damage) consists of:
 - Edema
 - Inflammation
 - Immediate or delayed hemorrhage
 - Full-thickness necrosis
 - Perforation
 - Fibrosis with resulting gastric outlet obstruction
 - Well-absorbed and can cause hemolysis of RBCs and a systemic metabolic acidosis

ETIOLOGY

- Direct chemical injuries
- Injuries occur secondary to acid and alkali exposures.
- Many caustic agents (acids and alkalis) are found in common household and

industrial products.

- Caustic substances:
 - Ammonia hydroxide
- Glass cleaners:
 - Formaldehyde:
 - Embalming agent
 - Hydrochloric acid:
 - Toilet bowl cleaners
 - Hydrofluoric acid:
 - Glass etching industry
 - Microchip industry
 - Rust removers
 - Iodine:
 - Antiseptics
 - Phenol:
 - Antiseptics
 - Sodium hydroxide:
 - Drain cleaners
 - Drain openers
 - Oven cleaners
 - Sodium borates, carbonates, phosphates, and silicates:
 - Detergents
 - Dishwasher preparations
 - Sodium hypochlorite
 - Bleaches
 - Sulfuric acid:
 - Car batteries
 - Button batteries



DIAGNOSIS

SIGNS AND SYMPTOMS

- Oropharyngeal:
 - Pain
 - Erythema
 - Burns
 - Erosions
 - Ulcers
 - Drooling
 - Hoarseness
 - Stridor
 - Aponia

- Absence of visible lesions in the oropharynx does not exclude visceral injuries.
- Pulmonary:
 - Tachypnea
 - Cough
 - Pneumonitis if aspirated
- GI:
 - Pain
 - Emesis or hematemesis
 - Melena, dysphagia
 - Odynophagia
 - Esophageal or gastric perforation
 - Peritonitis owing to perforation
- Cardiovascular:
 - Tachycardia
 - Hypotension
 - Orthostatic changes
- Hematologic:
 - Acid ingestion can cause RBC hemolysis.
- Dermatologic:
 - Pain
 - Erythema
 - 1st-, 2nd-, or 3rd-degree burns
- Ocular:
 - Pain
 - Erythema
 - Injection
 - Corneal burns
 - Full-thickness corneal damage
- Metabolic:
 - Metabolic acidosis

ESSENTIAL WORKUP

- History of or signs and symptoms of an exposure
- Absence of oropharyngeal lesions does *not* exclude visceral injury.

DIAGNOSIS TESTS & NTERPRETATION

Lab

- CBC
- Electrolytes, BUN, creatinine, glucose
- Arterial blood gas
- Blood cultures:

- If mediastinitis or peritonitis suspected
- Type and cross-match

Imaging

Chest and abdominal radiographs for:

- Esophageal or gastric perforation

Diagnostic Procedures/Surgery

- Esophageal and gastric endoscopy:
 - For symptomatic patients to determine the extent of injury
 - Perform within the 1st 12–24 hr after ingestion.
 - Not recommended in the presence of respiratory distress without proper airway management
 - Not recommended in the presence of severe pharyngeal damage
- Radiographic oral contrast imaging not recommended acutely:
 - May be used in follow-up for assessment for strictures

DIFFERENTIAL DIAGNOSIS

- Chemical injuries from corrosives, acids, alkalis, desiccants, vesicants, and oxidizing and reducing agents
- Foreign body ingestion
- Upper airway infection or angioedema



TREATMENT

PRE HOSPITAL

- For oral burns or symptoms: Rinse mouth liberally with water or milk.
- Water or milk can be given to following patients:
 - Able to drink
 - Not complaining of significant abdominal pain
 - Do not have airway compromise or vomiting
- Copious irrigation for ocular or dermal exposure

INITIAL STABILIZATION/THERAPY

- ABCs:
 - Prophylactic intubation if there is any evidence of respiratory compromise
 - Blind nasotracheal intubation contraindicated
- Treat hypotension with 0.9% NS IV fluid resuscitation.

ED TREATMENT/PROCEDURES

- Decontamination:
 - Dermal or ocular exposure:

- Immediate and thorough irrigation with water or 0.9% NS until physiologic pH attained
- Alkalis typically require more irrigation than acids.
- Ipecac, activated charcoal, gastroesophageal lavage (large-bore or an NG tube), and a neutralizing acid or base are all contraindicated with caustic ingestions.
- Dilution:
 - Water or milk in the 1st 30 min of ingestion:
 - Especially useful for solid caustic alkali ingestions
 - Excessive intake may induce vomiting and worsen esophageal damage.
 - If respiratory distress, intubate before dilution.
 - Contraindicated if esophageal or gastric perforation suspected
- Keep patient NPO if oral exposure.
- Broad-spectrum antibiotics if mediastinitis or peritonitis suspected
- Antiemetics for nausea and vomiting
- Treat dermal exposures according to standard burn recommendations.
- Detailed exam for ocular exposures
- IV proton pump inhibitors or H₂ blockers for symptomatic relief
- Gastroenterology and surgical consultation
- Benefit of corticosteroids following esophageal damage is controversial:
 - May prevent the formation of esophageal stricture
 - May promote bacterial invasion, immune suppression, and tissue softening
 - The decision to initiate corticosteroids requires input from entire team caring for patient.
 - Initiate broad-spectrum antibiotics if corticosteroids are given.
- Laparoscopy or laparotomy for perforation and full-thickness necrosis
- Topical hydrofluoric acid exposure (options depend on severity and location):
 - IM injection of 5% calcium gluconate (0.5 mL/cm² of skin with 30G needle)
 - Intra-arterial infusion of 10 mL of 10% calcium gluconate in 40 mL D₅W over 4 hr

MEDICATION

- Methylprednisolone: 40 mg q8h IV (peds: 2 mg/kg/d IV); the course of therapy is 14–21 days followed by a corticosteroid taper.
- Ondansetron: 4 mg (peds: 0.1–0.15 mg/kg) IV
- Pantoprazole: 40 mg IV
- Prochlorperazine (Compazine): 5–10 mg IV (peds: 0.13 mg/kg per dose IM)
- Ranitidine (Zantac): 50 mg IV q6–8h



FOLLOW-UP

DISPOSITION

Admission Criteria

- All symptomatic patients
- Nonaccidental ingestion

Discharge Criteria

- Asymptomatic patients who accidentally ingested and are able to swallow without difficulty
- Minimal oropharyngeal pain with a corresponding visible lesion; no drooling; no respiratory compromise; no deep throat, chest, or abdominal pain; and able to swallow without difficulty

FOLLOW-UP RECOMMENDATIONS

Psychiatric referral for intentional ingestion

PEARLS AND PITFALLS

- Dilute with milk or water at home or in the ED within the 1st 30 min.
- Perform copious irrigation of ocular or dermal exposure:
 - _ Alkalis require more irrigation than acids.

ADDITIONAL READING

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- Riffat F, Cheng A. Pediatric caustic ingestion: 50 consecutive cases and a review of the literature. *Dis Esophagus.* 2009;22:89–94.
- Salzman M, O'Malley RN. Updates on the evaluation and management of caustic exposures. *Emerg Med Clin North Am.* 2007;25(2):459–476.



CODES

ICD9

- 947.0 Burn of mouth and pharynx
- 947.2 Burn of esophagus
- 947.3 Burn of gastrointestinal tract

ICD10

- T28.5XXA Corrosion of mouth and pharynx, initial encounter
- T28.6XXA Corrosion of esophagus, initial encounter
- T28.7XXA Corrosion of other parts of alimentary tract, init encntr

CAVERNOUS SINUS THROMBOSIS

Joanna W. Davidson

BASICS

DESCRIPTION

- Thrombosis of a branch of the major intracerebral venous drainage system
- Most commonly infectious
- Spreads from facial, odontogenic, or sinus infection
- Less frequently occurs with hypercoagulable state

Anatomy

3 primary sites of thrombosis:

- Cavernous sinus—Most common:
 - Drainage from superficial venous system
- Superolateral to the sphenoid sinus and surrounds the sella:
 - Cranial nerves (CN) III, IV, V1, and V2 traverse the lateral wall of the sinus.
 - CN VI and the internal carotid artery occupy the medial portion of the sinus.
- Can also involve transverse sinus and superficial sagittal sinus

PATHOPHYSIOLOGY

- Hematogenous spread of facial, otic, or neck infection into venous drainage system
- Contiguous spread directly from infected sinus cavities (sphenoid, ethmoid > frontal)
- Bacterial overgrowth leads to inflammation and coagulation, resulting in thrombosis.
- Venous engorgement of cavernous sinus can affect adjacent structures:
 - Ophthalmoplegia from inflammation of CN III, IV, or VI
 - Pupillary fixation from CN III
 - Sensory deficits or paresthesia of forehead or cheek from CN V1 and V2

ETIOLOGY

- Septic:
 - *Staphylococcus aureus* accounts for 70%
 - *Streptococcus pneumoniae*, gram-negative bacilli, and anaerobes also seen
 - Fungi less common; include *Aspergillus* and *Rhizopus* species
- Aseptic:
 - Less common
 - Granulomatous conditions (TB)
 - Inflammatory disorders

- From mass effect (tumors at base of skull, aneurysms)
- Hypercoagulable states

Pediatric Considerations

- Children may present with nonspecific symptoms such as decreased energy, vomiting, fever.
- Have high level of suspicion for any child with recent otitis or pharyngitis with worsening symptoms, declining mental status, or signs of increased intracranial pressure (ICP):
 - HTN, bradycardia, lethargy, vomiting, gait instability
- More common in the neonatal period, when diagnosis can be extremely difficult to make

DIAGNOSIS

SIGNS AND SYMPTOMS

- Symptoms:
 - Headache occurs in 90% of patients.
 - Fever
 - Ocular or retrobulbar pain
 - Facial swelling
 - Visual disturbance
 - Facial dysesthesia
 - Lethargy or altered mental status
- Signs:
 - Periorbital edema is earliest sign
 - Chemosis with retinal vein engorgement
 - Ptosis, proptosis
 - Ophthalmoplegia
 - CN palsies:
 - Lateral gaze palsy (CN VI)
 - Hypo/hyperesthesia of V1 and V2 (CN V)
 - Meningismus
 - Altered level of consciousness or coma
 - Seizures
 - Sepsis with cardiovascular instability or collapse

History

High-risk historical factors include:

- A history of trauma
- Previous ear/nose/throat (ENT) or neurosurgical instrumentation
- History of central face furuncle that was manipulated

- Diabetes or immunocompromised state (HIV, steroid use, cancer) may increase risk

ESSENTIAL WORKUP

- Clinical diagnosis: Venous engorgement, ocular symptoms, unilateral symptoms that become bilateral, rapidly progressive
- Labs nonspecific
- Imaging findings can be subtle

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Neither sensitive nor specific
- CBC:
 - _ Leukocytosis
 - _ Bandemia
- PT/PTT/INR
- ESR and D-dimer usually elevated
- Lumbar puncture/CSF: Parameningeal inflammation or frank meningitis

Imaging

- CT scan:
 - _ Can be normal early in disease course
 - _ Noncontrast can show increased density
 - _ Contrast can show filling defect
 - May identify original source of infection (e.g., sinusitis)
 - Dilated superior ophthalmic vein
 - Associated intracranial hemorrhage
 - Signs of increased ICP: Small ventricles, loss of sulci
- MRI with MR angiography (MRA)/MR venography (MRV):
 - _ Diagnostic modality of choice
 - _ Direct visualization of intracranial vessels and sinuses
 - _ Capable of visualizing thrombus at any stage

DIFFERENTIAL DIAGNOSIS

- Meningitis/encephalitis
- Intracranial abscess
- Periorbital and orbital cellulitis
- Internal carotid artery aneurysm or fistula
- Pseudotumor cerebri
- Acute angle-closure glaucoma
- Intracranial hemorrhage
- Tolosa–Hunt syndrome: Rare granulomatous inflammation of cavernous sinus

ALERT

- Extremely difficult diagnosis to make.
- Maintain a high level of suspicion in toxic-appearing patients with recent ENT infections or in patients with refractory headache and risk factors for hypercoagulability or intracranial infection.



TREATMENT

PRE HOSPITAL

ALERT

- Patients can be altered and unstable.
- May require rapid assessment and stabilization of airway, breathing, and circulation (ABCs)

INITIAL STABILIZATION/THERAPY

- Careful assessment of mental status with intubation for airway protection as needed
- Aggressive fluid resuscitation for cardiovascular instability

ED TREATMENT/PROCEDURES

- Broad-spectrum antibiotics with multiple drug regimens:
 - Cover for gram positives, gram negatives, as well as anaerobes.
 - Nafcillin or vancomycin (for methicillin-resistant *S. aureus* [MRSA]) + ceftriaxone:
 - Add metronidazole or clindamycin in significant infections.
- Heparin:
 - Attenuates clot propagation and decreases morbidity/mortality.
 - Controversial in transverse and sagittal thrombosis owing to higher risk of subsequent hemorrhage
 - Administer only after ruling out bleed on CT scan.
 - Questionable superiority of LMWH over IV heparin
 - Endovascular TPA in severe refractory cases
- Systemic steroids:
 - Believed to be of benefit with concomitant pituitary insufficiency, and with infectious or inflammatory etiologies
- Appropriate management of increased ICP as needed
- Surgical consultation for drainage of primary site of infection (e.g., dental abscess or sinusitis)

MEDICATION

- Ceftriaxone: 2 g/d IV (peds: 80–100 mg/kg/d to q12h)
- Clindamycin: 300–900 mg IV q6–12h (neonates: 10–20 mg/kg/24h IV divided q6–

- 12h; peds: 25–40 mg/kg/24h divided q6–8h)
- Metronidazole: 500 mg IV q6h (neonates: 7.5–30 mg/kg/24h IV divided q12–24h; peds: 30 mg/kg/24h IV divided q6h)
- Nafcillin: 1–2 g IV q4h (peds: 50–75 mg/kg/24h IV divided q8–12h depending on age)
- Vancomycin: 1 g IV q12h (peds: 10–20 mg/kg IV q8–12h depending on age)

First Line

- Broad-spectrum antibiotics
- Anticoagulation

Second Line

- Dexamethasone or hydrocortisone IV
- Endovascular thrombolytics in selected cases



FOLLOW-UP

DISPOSITION

Admission Criteria

- All patients with sinus thrombosis warrant admission to a monitored setting.
- Consider ICU admission.

Discharge Criteria

None

FOLLOW-UP RECOMMENDATIONS

Neurologic and neurosurgical consultation

COMPLICATIONS

- Blindness
 - 1/6 left with visual impairment
- CN palsies
- Meningitis or intracranial abscess
- Seizures, especially in superior sagittal sinus thrombosis
- Pituitary necrosis and insufficiency from local invasion
- Septic emboli
- Sepsis and shock
- 30% mortality

PEARLS AND PITFALLS

- Diagnosis is made on clinical evaluation and confirmatory lab evidence. Maintain a high index of suspicion.
- Noncontrast head CT is often negative or nonspecific. MRI/MRV is the diagnostic imaging modality of choice and should be pursued in high-risk individuals.
- Administer IV antibiotics early, especially in any ill-appearing patient with ENT or neurologic complaints.
- Hypercoagulable states result in *both* central and peripheral venous thrombosis. Workup and management decisions must include consideration of systemic thromboembolism.

ADDITIONAL READING

- Cannon ML, Antonio BL, McCloskey JJ, et al. Cavernous sinus thrombosis complicating sinusitis. *Pediatr Crit Care Med*. 2004;5(1):86–88.
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- Laupland KB. Vascular and parameningeal infections of the head and neck. *Infect Dis Clin North Am*. 2007;21(2):577–590, viii.
- Misra UK, Kalita J, Bansal V. D-dimer is useful in the diagnosis of cortical venous sinus thrombosis. *Neurol India*. 2009;57(1):50–54.
- Sztajnkrycer M, Jauch EC. The difficult diagnosis: Unusual headaches. *Emerg Med Clin North Am*. 1998;16(4):741–760.



See Also (Topic, Algorithm, Electronic Media Element)

Headache



CODES

ICD9

- 325 Phlebitis and thrombophlebitis of intracranial venous sinuses
- 437.6 Nonpyogenic thrombosis of intracranial venous sinus

ICD10

- G08 Intracranial and intraspinal phlebitis and thrombophlebitis
- I67.6 Nonpyogenic thrombosis of intracranial venous system

CELLULITIS

John Mahoney • Dolores Gonthier

BASICS

DESCRIPTION

- Acute, spreading erythematous superficial infection of skin and SC tissues:
 - Variety of pathogens
 - Extension into deeper tissues can result in necrotizing soft tissue infection
- Progressive spread of erythema, warmth, pain, and tenderness
- Predisposing factors:
 - Lymphedema
 - Tinea pedis
 - Open wounds
 - Pre-existing skin lesion (furuncle)
 - Prior trauma or surgery
 - Retained foreign body
 - Vascular or immune compromise
 - Injection drug use

ETIOLOGY

- Simple cellulitis:
 - Group A streptococci
 - *Staphylococcus aureus*—including resistant strains such as community-associated methicillin-resistant *S. aureus* (CA-MRSA; see below):
 - CA-MRSA risk factors include: Prior MRSA infection, household contact of CA-MRSA patient, daycare contact of MRSA patients, children, soldiers, incarcerated persons, athletes in contact sports, IV drug users, men who have sex with men
 - Different antibiotic susceptibility than nosocomial MRSA
 - CA-MRSA now sufficiently prevalent to warrant empiric treatment
 - Suspect CA-MRSA in unresponsive infections
- Nosocomial MRSA:
 - Risk factors: Recent hospital or long-term care admission, surgery, injection drug use, vascular catheter, dialysis, recent antibiotic use, unresponsive infection
 - Resistant to most antibiotics (see “Treatment”)
- Extremity cellulitis after lymphatic disruption:
 - Nongroup A β -hemolytic streptococci (groups C, B, G)
- Cellulitis in diabetics:
 - Can be polymicrobial with *S. aureus*, streptococci, gram-negative bacteria,

and anaerobes, especially when associated with skin ulcers

- Periorbital cellulitis:
 - *S. aureus*
 - Streptococcal species
- Buccal cellulitis:
 - *Haemophilus influenzae* type B
 - Anaerobic oral flora, associated with intraoral laceration or dental abscess
- Less common causes:
 - Clostridia
 - Anthrax
 - *Pasteurella multocida*—common after cat and dog bites
 - *Eikenella corrodens*—human bites
 - *Pseudomonas aeruginosa*:
 - Hot-tub folliculitis—self-limited
 - Foot puncture wound
 - Ecthyma gangrenosum in neutropenic patients
 - Erysipelothrix species—raw fish, poultry, meat, or hide handlers
 - *Aeromonas hydrophila*—freshwater swimming
 - *Vibrio* species—seawater or raw seafood

Pediatric Considerations

- Facial cellulitis in children:
 - *Streptococcus pneumoniae*
 - *H. influenzae* type B, although incidence declining since introduction of HIB vaccine
- Perianal cellulitis:
 - Group A streptococci
 - Associated or antecedent pharyngitis or impetigo
- Neonates:
 - Group B streptococci



DIAGNOSIS

SIGNS AND SYMPTOMS

- Common to all syndromes:
 - Pain, tenderness, warmth
 - Erythema
 - Edema or induration
 - Fever/chills
 - Tender regional lymphadenopathy
 - Lymphangitis
 - Accompanying SC abscess possible

- Suspect deep abscess especially if treatment failure on initial antibiotic
- Superficial vesicles
- Buccal cellulitis:
 - Odontogenic cases more serious:
 - Toothache, sore throat, or facial swelling
 - Progressive extension into soft tissues of neck with fever, erythema, neck swelling, and dysphagia

Pediatric Considerations

- Facial cellulitis in children:
 - Erythema and swelling of the cheek and eyelid
 - Rapidly progressive
 - Usually unilateral
 - Upper respiratory tract symptoms
 - Risk for cavernous sinus thrombosis and permanent optic nerve injury
- Perianal cellulitis:
 - Erythema and pruritus extending from the anus several centimeters onto adjacent skin
 - Pain on defecation
 - Blood-streaked stools

History

Patients often incorrectly attribute CA-MRSA infection with spontaneous abscess to a spider bite

Physical-Exam

In simple cellulitis, physical findings can suggest the etiology and help narrow empiric antibiotic coverage:

- Staph etiology: Focal abscess or pustule with: Fluctuance, yellow or white center, central point or “head,” or draining pus, indolent progression
- Strep etiology: Sharply demarcated borders, lymphangitis, pre-existing lymphedema, concomitant nausea from toxin

ESSENTIAL WORKUP

- Cellulitis is a clinical diagnosis.
- Physical exam to reveal infection source

DIAGNOSIS TESTS & INTERPRETATION

Lab

- WBC generally unnecessary
- Gram stain and culture to focus antimicrobial selection and reveal resistant pathogens (MRSA):

- Aspirate point of maximal inflammation or punch biopsy if there is no wound to culture
- Perform in treatment failures and consider in admitted patients
- Blood culture:
 - Usually negative in uncomplicated cellulitis
 - May identify organism in patients with:
 - Lymphedema
 - Buccal or periorbital cellulitis
 - Saltwater or freshwater source
 - Fever or chills

Imaging

- Plain radiographs may reveal abscess formation, SC gas, or foreign bodies:
 - Extension to bone (osteomyelitis) not visualized early on plain radiographs
- Extremity vascular imaging (Doppler US) can help rule out deep venous thrombosis (DVT).
- US useful for diagnosing abscess if physical exam is equivocal or if there is a broad area of cellulitis
 - In cellulitis may see characteristic “cobblestone” appearance and thickening of SC layer, both due to edema
- CT or MRI can help rule out necrotizing fasciitis

DIFFERENTIAL DIAGNOSIS

- Necrotizing fasciitis
- Lymphangitis or lymphadenitis
- Thrombophlebitis or DVT:
 - Differentiation from cellulitis:
 - Absence of initial traumatic or infectious focus
 - No regional lymphadenopathy
 - Presence of risk factors for DVT
- Insect bite
- Allergic reaction
- Acute gout or pseudogout
- Ruptured Baker cyst
- Herpetic whitlow
- Neoplasm
- Phytophotodermatitis
- Erythema chronicum migrans lesion of Lyme disease
- Differential diagnosis of facial cellulitis:
 - Allergic angioedema
 - Conjunctivitis
 - Contusion

Pediatric Considerations

Differential diagnosis of perianal cellulitis:

- **Candida intertrigo**
- **Psoriasis**
- **Pinworm infection**
- **Child abuse**
- **Behavioral problem**
- **Inflammatory bowel disease**



TREATMENT

INITIAL STABILIZATION/THERAPY

Airway compromise possible with deep extension of facial or neck cellulitis

ED TREATMENT/PROCEDURES

- **General principles:**
 - Consider local prevalence of resistant pathogens in addition to usual causes
 - In simple cellulitis, periorbital cellulitis, and diabetic patients, need to include CA-MRSA coverage in empiric therapy
 - Usual outpatient treatment: 7–10 days
 - Cool compresses for comfort
 - Analgesics
 - Extremity elevation
 - Treat predisposing tinea pedis with topical antifungal such as clotrimazole
- **Simple cellulitis:**
 - **Outpatient:**
 - Oral Cephalexin + TMP/SMX (to cover CA-MRSA)
 - Alternatives to cephalexin: Oral dicloxacillin, macrolide, or levofloxacin
 - Alternatives to TMP/SMX: Clindamycin or Doxycycline
 - **Inpatient:**
 - IV nafcillin or equivalent, + IV vancomycin (to cover CA-MRSA)
- **Extremity cellulitis after lymphatic disruption:**
 - Same as simple cellulitis
- **Cellulitis in diabetics:**
 - **Outpatient:**
 - Amoxicillin/clavulanate + TMP/SMX (to cover CA-MRSA), or clindamycin
 - **Inpatient:**
 - IV ampicillin/sulbactam or imipenem cilastatin or equivalent; + IV vancomycin (to cover CA-MRSA)
- **Periorbital cellulitis in adults:**

- Outpatient: Oral dicloxacillin or azithromycin; + TMP/SMX (to cover CA-MRSA)
- Inpatient: IV vancomycin
- Buccal cellulitis in adults:
 - Outpatient: Oral amoxicillin/clavulanate
 - Inpatient: IV ceftriaxone
 - Odontogenic source:
 - Drainage essential
 - Coverage for anaerobes: Clindamycin
- Facial cellulitis in children:
 - IV ceftriaxone
- Perianal cellulitis:
 - Outpatient: Oral penicillin VK
 - Inpatient: IV penicillin G (aqueous)
- Animal or human bite:
 - Oral amoxicillin/clavulanate
- Foot puncture wound:
 - Oral or IV ciprofloxacin or IV ceftazidime
- MRSA:
 - Nosocomial MRSA: IV vancomycin or oral or IV linezolid
 - CA-MRSA:
 - PO: TMP/SMX, clindamycin or doxycycline
 - IV: Vancomycin or clindamycin

MEDICATION

- Amoxicillin/clavulanate: 500–875 mg (peds: 45 mg/kg/24h) PO BID or 250–500 mg (peds: 40 mg/kg/24h) PO TID
- Ampicillin/sulbactam: 1.5–3 g (peds: 100–300 mg/kg/24h up to 40 kg; over 40 kg give adult dose) IV q6h
- Azithromycin: (Adults and peds) 10 mg/kg up to 500 mg PO on day 1, followed by 5 mg/kg up to 250 mg PO daily on days 2–5
- Ceftazidime: 500–1,000 mg (peds: 150 mg/kg/24h; max. 6 g/24h; use sodium formulation in peds) IV q8h
- Ceftriaxone: 1–2 g (peds: 50–75 mg/kg/24h) IV daily
- Cephalexin: 500 mg (peds: 50–100 mg/kg/24h) PO QID
- Ciprofloxacin: (Adult only) 500–750 mg PO BID or 400 mg IV q8–12h
- Clindamycin: 450–900 mg (peds: 20–40 mg/kg/24h) PO or IV q6h
- Dicloxacillin: 125–500 mg (peds: 12.5–25 mg/kg/24h) PO q6h
- Doxycycline: 100 mg PO BID for adults
- Erythromycin base: (Adult) 250–500 mg PO QID
- Imipenem cilastatin: 500–1,000 mg (peds: 15–25 mg/kg) IV q6h; max. 4 g/24h or 50 mg/kg/24h, whichever is less
- Levofloxacin: (Adult only) 500–750 mg PO or IV daily

- Linezolid: 600 mg PO or IV q12h (peds: 30 mg/kg/24h div. q8h)
- Nafcillin: 1–2 g IV q4h (peds: 50–100 mg/kg/24h divided q6h); max. 12 g/24h
- Penicillin VK: 250–500 mg (peds: 25–50 mg/kg/24h) PO q6h
- Penicillin G (aqueous): 4 mU (peds: 100,000–400,000 U/kg/24h) IV q4h
- Trimethoprim/sulfamethoxazole (TMP/SMX): 2 DS tabs PO q12h (peds: 6–10 mg/kg/24h TMP div. q12h)
- Vancomycin: 1 g IV q12h (peds: 10 mg/kg IV q6h; dosing adjustments required younger than age 5 yr); check serum levels



FOLLOW-UP

DISPOSITION

Admission Criteria

- Toxic appearing
- Tissue necrosis
- History of immune suppression
- Concurrent chronic medical illnesses
- Unable to take oral medications
- Unreliable patients

Discharge Criteria

- Mild infection in a nontoxic-appearing patient
- Able to take oral antibiotics
- No history of immune suppression or concurrent medical problems
- No hand or face involvement
- Has adequate follow-up within 24–48 hr

FOLLOW-UP RECOMMENDATIONS

- Follow-up within 24–48 hr
- Sooner if worsening symptoms, including new or worsening lymphangitis, increasing area of redness, worsening fever
- Outline the border of erythema before discharge to aid in assessing response to therapy

PEARLS AND PITFALLS

- Strep and staph are most common causes
- CA-MRSA now significant cause of cellulitis, frequent enough to warrant including coverage in empiric treatment
- Clinicians not accurate at identifying MRSA at the bedside
- A deep abscess may be misclassified as cellulitis

- Use clinical suspicion and ultrasound to avoid missing an abscess

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See Also (Topic, Algorithm, Electronic Media Element)

- Abscess, Skin/Soft Tissue
- Lymphadenitis
- Lymphangitis
- MRSA
- Necrotizing Fasciitis



CODES

ICD9

- 682.3 Cellulitis and abscess of upper arm and forearm
- 682.6 Cellulitis and abscess of leg, except foot
- 682.9 Cellulitis and abscess of unspecified sites

ICD10

- H05.019 Cellulitis of unspecified orbit
- L03.90 Cellulitis, unspecified
- L03.119 Cellulitis of unspecified part of limb

CENTRAL RETINAL ARTERY OCCLUSION

Yasuharu Okuda • Braden Hexom

BASICS

DESCRIPTION

- Obstruction of the central retinal artery associated with sudden painless loss of vision
- Usually occurs in persons 50–70 yr of age
- Ophthalmic artery is 1st branch of carotid.
- Risk factors include HTN, atherosclerotic disease, sickle cell disease, vasculitis, valvular heart disease, lupus, trauma, and coronary artery disease.
- Incidence of 1–10/100,000
- Often described as a “stroke of the eye”

ETIOLOGY

- Embolic:
 - Occlusion by intravascular material from a proximal source:
 - Atherosclerotic disease (majority)
 - Carotid artery stenosis
 - Valvular heart disease (cardiogenic emboli)
 - Atrial myxoma
 - Dissection of the ophthalmic artery
 - Carotid artery dissection
- Thrombotic:
 - Obstruction of flow from the rupture of a pre-existing intravascular atherosclerotic plaque
 - Hypercoagulable states (sickle cell)
- Inflammatory:
 - Due to temporal arteritis, lupus, vasculitis
- Arterial spasm:
 - Associated with migraine headaches
- Decreased perfusion:
 - Low-flow conditions such as in severe hypotension or high-pressure situations seen in acute angle-closure glaucoma or retrobulbar hemorrhage

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Sudden, painless, monocular loss of vision
- Prior episodes of sudden visual loss:
 - May last a few seconds to minutes (amaurosis fugax)
 - Caused by transient embolic phenomena or decreased ocular blood flow

Physical-Exam

- Significantly decreased visual acuity
- Afferent pupillary defect usually present
- Retinal appearance:
 - Emboli visualized within vascular tree of the retina
 - Appears as glinting white or yellow flecks (Hollenhorst plaques) within the vessels
 - Ischemic edema visible within 15–20 min of occlusion
 - “Cherry-red spot” remains over the fovea (only area where there is very thin retina allowing the vascular choroids to show through).
 - Affected arteries empty or showing dark red stationary or barely pulsatile segmented rouleaux (“box-carring”)
 - Within 1–2 hr opacification of the usually transparent infarcting retinal nerve layer occurs.
- Partial field deficits:
 - Occur only if branch of central retinal artery involved

ESSENTIAL WORKUP

- Visual acuity and visual field testing
- Fundoscopic exam
- Intraocular pressure measurements
- Emergent ophthalmologic consultation

DIAGNOSIS TESTS & INTERPRETATION

Lab

Directed toward evaluating underlying etiology of occlusion:

- CBC with differential and platelet count
- PT/PTT
- Electrolytes, BUN/creatinine, glucose
- Electronic spin resonance for giant cell arteritis (in patients > 55 yr old)
- ANA, RF, CRP, ESR
- Rapid plasma reagin (RPR)
- Hemoglobin electrophoresis
- Serum protein electrophoresis

Imaging

Directed toward evaluating underlying etiology of occlusion:

- Carotid artery ultrasound/Doppler
- Possibly echocardiography
- Fluorescein angiography or electroretinography to confirm the diagnosis

DIFFERENTIAL DIAGNOSIS

- Acute angle-closure glaucoma
- Central retinal vein occlusion
- Giant cell arteritis (temporal arteritis)
- Optic neuritis
- Retinal detachment



TREATMENT

ALERT

Initiate treatment immediately because irreversible visual loss occurs at 90 min:

- Only immediate treatment may help to salvage or restore sight to the affected eye.
- Goals of therapy include dislodging or dissolving the embolus, arterial dilation to improve forward flow, and reduction of intraocular pressure to improve the perfusion gradient.

ED TREATMENT/PROCEDURES

- Immediate global massage in an attempt to dislodge the embolus:
 - _ Lay patient flat and apply digital global massage bolus.
 - _ On closed eyelid, apply constant pressure for 15 sec and remove for 15 sec. Repeat for 5 cycles.
- Initiate high-flow oxygen via 100% nonrebreather:
 - _ Consider transfer to a facility capable of providing hyperbaric oxygen (HBO) if < 24 hr from symptom onset
 - _ May use inhaled carbogen (a mixture of carbon dioxide and oxygen gas) if available
- Administer IV acetazolamide to decrease intraocular pressure.
- Apply topical timolol maleate to reduce intraocular pressure.
- Administer aspirin and IV heparin for prevention of clot propagation.
- Obtain emergent ophthalmology consultation for:
 - _ Anterior chamber paracentesis to help reduce intraocular pressure
 - _ Possible intra-arterial fibrinolysis for clot lysis
- Administer high-dose systemic steroids in suspected cases of inflammatory arteritis.

MEDICATION

First Line

- Acetazolamide: 500 mg IV or PO

- Carbogen: Inhalation of 95% oxygen and 5% carbon dioxide mixture
- Heparin: 80 U/kg IV bolus then 18 U/kg/h continuous infusions (rate adjusted based on PTT level)
- Timolol maleate 0.5% solution: 1 drop topically to affected eye

Second Line

- Methylprednisolone: 250 mg IV in suspected cases of inflammatory arteritis
- Aspirin: 325 mg PO
- Mannitol
- Sublingual nitroglycerin



FOLLOW-UP

DISPOSITION

Admission Criteria

Required for workup of proximal cause in acute cases (source of embolism, thrombosis, or inflammatory)

Discharge Criteria

Chronic retinal artery occlusion with no evidence of active disease can be worked up as an outpatient.

Issues for Referral

All suspected cases warrant emergent ophthalmology consultation.

FOLLOW-UP RECOMMENDATIONS

Most cases will require carotid ultrasound to exclude atherosclerotic disease.

PEARLS AND PITFALLS

- Amaurosis fugax (transient, possibly resolved retinal artery occlusion) is a sentinel event and may lead to complete occlusion or stroke. Do not ignore these symptoms and urgent workup is required.
- Retinal artery occlusion is a medical emergency requiring immediate treatment to prevent loss of the eye.
- It is important to document a full eye exam including visual acuity and evaluation of the optic fundus.

ADDITIONAL READING

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with conventional treatment in patients with acute central retinal artery occlusion. *J Neurol Neurosurg Psychiatry*. 2005;76(2):196–199.

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See Also (Topic, Algorithm, Electronic Media Element)

- Central Retinal Venous Occlusion
- Visual Loss



CODES

ICD9

362.31 Central retinal artery occlusion

ICD10

- H34.10 Central retinal artery occlusion, unspecified eye
- H34.11 Central retinal artery occlusion, right eye
- H34.12 Central retinal artery occlusion, left eye

CENTRAL RETINAL VEIN OCCLUSION

Lisa Jacobson • Yasuharu Okuda

BASICS

DESCRIPTION

Disease characterized by decreased visual acuity resulting from venous occlusion of any etiology

ETIOLOGY

- Ischemic CRVO:
 - _ 20–25% of cases
 - _ Blocked venous return leads to backflow in capillaries, hemorrhage, and macular edema.
 - _ Limited space at lamina cribrosa predisposes to thrombosis due to slow flow and vessel wall changes
 - _ Theorize that arteriosclerotic changes in the adjacent artery may impinge upon the vein.
 - _ Blood viscosity also thought to play a role
- Nonischemic CRVO:
 - _ Milder, incomplete occlusion

DIAGNOSIS

SIGNS AND SYMPTOMS

Classic description:

- Acute, unilateral, painless vision loss
- “Blood and thunder” appearance on fundoscopy

History

- Painless, unilateral vision loss
- If nonischemic, may be incomplete and intermittent vision loss

Physical-Exam

- Decreased visual acuity:
 - _ Usually worse than 20/200
- Afferent pupillary defect
- Dilated tortuous veins
- Retinal hemorrhages:
 - _ If central, findings in all 4 quadrants
 - _ Extensive hemorrhages give a dramatic look to fundus classically described

as “blood and thunder appearance.”

- Disk edema
- Cotton wool spots

ESSENTIAL WORKUP

- BP
- Visual acuity:
 - Hand movements typically is all that is seen.
- Visual fields
- Fundoscopy
- Tonometry:
 - Normal pressures are between 10 and 21 mm Hg.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC
- PT/PTT
- ESR
- ANA
- Serum protein electrophoresis

Imaging

Fluorescein angiography:

- Ophthalmologists use this to map areas of nonperfusion.
- Differentiates between ischemic and nonischemic

Diagnostic Procedures/Surgery

Gonioscopy:

- Measure iris or angle neovascularization.

DIFFERENTIAL DIAGNOSIS

- Amaurosis fugax/transient ischemic attack
- Cavernous sinus thrombosis
- DM
- HTN/hypertensive retinopathy
- Hyperviscosity syndromes:
 - Sickle cell, polycythemia, leukemia, multiple myeloma
- Hysterical blindness
- Ocular ischemia syndrome
- Papilledema
- Retinal artery occlusion
- Retinal detachment

- Severe anemia with thrombocytopenia
- Temporal arteritis
- Vitreal hemorrhage



TREATMENT

PRE HOSPITAL

No specific interventions need occur prior to arrival at the hospital in regard to the eye.

INITIAL STABILIZATION/THERAPY

- Initiate steps to lower intraocular pressure (IOP) if it is elevated.
- Treat underlying medical problems.

ED TREATMENT/PROCEDURES

- Recognition and prompt ophthalmologic referral is the cornerstone of ED treatment.
- Though not proven, the following may be tried in consultation with an ophthalmologist:
 - Aspirin
 - Anti-inflammatory agents
 - Systemic steroids
 - Systemic anticoagulation
 - Fibrinolytics (controversial)
 - Laser chorioretinal anastomosis

MEDICATION

There is no proven treatment for CRVO, ophthalmologists may treat with the following:

- Intravitreal triamcinolone
- Antivascular endothelial growth factor:
 - Bevacizumab

Considerations in Prescribing

Use of oral contraceptives can increase the risk of CRVO.



FOLLOW-UP

DISPOSITION

Admission Criteria

Patients may be admitted for surgical intervention, depending upon the ophthalmologist.

Discharge Criteria

Patients can be discharged from the ED as long as they have immediate follow-up with an ophthalmologist.

Issues for Referral

- If no ophthalmologist is available, treatment should be initiated for concomitant conditions and patient transferred to nearest hospital with ophthalmologic consultation.
- Ophthalmologists often perform panretinal photocoagulation if neovascularization is found.

FOLLOW-UP RECOMMENDATIONS

- Patients with ischemic CRVO need prolonged follow-up to catch neovascularization and glaucoma that typically develop.
- Patients with CRVO likely have other vascular diseases and need complete medical workups.
- Patients should also follow with an internist to manage comorbidities and risk factors.

PEARLS AND PITFALLS

- Increased IOP resulting from neovascularization and edema can cause vascular insufficiency and with delayed treatment vision loss can be permanent.
- When patients present with bilateral CRVOs or CRVO at a young age, workup must search for hyperviscosity syndromes.

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See Also (Topic, Algorithm, Electronic Media Element)

- Central Retinal Artery Occlusion
- Visual Loss



CODES

ICD9

362.35 Central retinal vein occlusion

ICD10

- H34.811 Central retinal vein occlusion, right eye
- H34.812 Central retinal vein occlusion, left eye
- H34.819 Central retinal vein occlusion, unspecified eye

CEREBRAL ANEURYSM

Veronique Au • Rebecca Smith-Coggins

BASICS

DESCRIPTION

- Abnormal, localized dilation or outpouching of cerebral artery wall:
 - Occurs in 5–10% of population
- Rupture of saccular aneurysms account for 5–15% of strokes
- Of those that rupture:
 - 40% occur at anterior communicating artery (ACA)
 - 30% at internal carotid (IC)
 - 20% in middle cerebral artery (MCA)
 - 5–10% in vertebrobasilar artery (VBA) system

ETIOLOGY

- Asymptomatic in 3.2% of population
- “Congenital,” saccular, or berry aneurysms most common (90%):
 - Develop at weak points in arterial wall and bifurcations of major cerebral arteries
 - Incidence increases with age
 - Multiple in 20–30%
 - Increased incidence:
 - Polycystic kidney disease
 - Cerebral arteriovenous malformation
 - Type III collagen deficiency
 - Fibromuscular dysplasia
 - Ehlers–Danlos syndrome
 - Marfan syndrome
 - Pseudoxanthoma elasticum
 - Neurofibromatosis
 - Moyamoya syndrome
 - Coarctation of the aorta
 - Tuberous sclerosis
 - Sickle cell disease
 - Osler–Weber–Rendu syndrome
 - α 1-Antitrypsin deficiency
 - Systemic lupus erythematosus
 - Glucocorticoid remediable hyperaldosteronism
- Arteriosclerotic, fusiform, or dolichoectatic (7%):
 - More common in peripheral arteries

- Inflammatory (mycotic):
 - 10% of patients with bacterial endocarditis
- Traumatic, associated with severe closed head injury
- Neoplastic, embolized tumor fragments
- Familial correlation: 1st-degree relative with history of aneurysm essentially doubles lifetime risk

Pediatric Considerations

- Although rare in children, more likely to be giant (> 25 mm)
- Occur in the posterior circulation

DIAGNOSIS

SIGNS AND SYMPTOMS

- Commonly asymptomatic before rupture
- Sentinel headaches occur in 30–60% of patients before rupture:
 - Can be unilateral
- Seizures, syncope, or altered level of consciousness

History

- Onset of headache
- Family history
- Altered mental status
- Focal neurologic deficits
- Rupture results in subarachnoid hemorrhage:
 - Headache: Severe (“worst headache ever”) with sudden onset (“thunderclap”)
 - Different from prior headaches
 - Classically without focal deficits
 - Nuchal rigidity (most common sign) secondary to blood in CSF

Physical-Exam

Compression of adjacent structures may cause neurologic symptoms:

- ACA aneurysms:
 - Optic tract: Altitudinal field cut or homonymous hemianopsia
 - Optic chiasm: Bitemporal hemianopsia
 - Optic nerve: Unilateral amblyopia
- Aneurysms at IC–posterior communicating artery junction:
 - Oculomotor nerve: Fixed and dilated pupil, ptosis, diplopia, and temporal deviation of eye with inability to turn eye upward, inward, or downward
- Aneurysms in cerebral cortex may produce focal deficits including:
 - Hemiparesis

- Hemisensory loss
- Visual disturbances
- Aphasia
- Seizures

ESSENTIAL WORKUP

- Complete neurologic examination
- Emergent noncontrast head CT scan will diagnose 90–95% of subarachnoid hemorrhages
- Lumbar puncture with CSF analysis if CT scan is negative

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Coagulation studies
- Baseline CBC with platelets and differential
- Electrolytes
- Renal and liver function tests
- Arterial blood gas

Imaging

- CXR for pulmonary edema
- 4-vessel cerebral angiography remains gold standard
- MRA
- Helical CT scanning may be useful in detecting aneurysms > 3 mm
- Transcranial Doppler US may be useful in detecting vasospasm.

Diagnostic Procedures/Surgery

Lumbar puncture if suspect aneurysmal leak or rupture with normal head CT

DIFFERENTIAL DIAGNOSIS

- Neoplasm
- Arteriovenous malformation
- Optic neuritis
- Migraine
- Meningitis
- Encephalitis
- Hypertensive encephalopathy
- Hyperglycemia or hypoglycemia
- Temporal arteritis
- Acute glaucoma
- Subdural hematoma
- Epidural hematoma

- Intracerebral hemorrhage
- Thromboembolic stroke
- Air embolism
- Sinusitis



TREATMENT

PRE HOSPITAL

- Cautions:
 - _ Neurologic examination in the field can be extremely helpful
 - _ Assess:
 - Level of consciousness
 - Glasgow coma scale score
 - Gross motor deficits
 - Speech abnormalities
 - Gait disturbance
 - Facial asymmetry
 - Other focal deficits
- Patients with subarachnoid hemorrhage may need emergent intubation for rapidly deteriorating level of consciousness
- Patients must be transported to a hospital with emergent CT scanning and intensive care unit (ICU)-level treatment

INITIAL STABILIZATION/THERAPY

- ABCs:
 - _ Supplemental oxygen
 - _ Rapid-sequence intubation may be required for airway protection or for controlled ventilation
 - _ Continuous cardiac monitoring and pulse oximetry
- For altered mental status:
 - _ Check blood glucose immediately, give D₅₀ (if glucose is low)
 - _ Naloxone
 - _ Thiamine
- Reversal of anticoagulation
- Prevention of acute increases in intracranial pressure from vomiting should be accomplished with antiemetics
- Seizures should be managed acutely with IV benzodiazepines and fosphenytoin/phenytoin
- Seizure prophylaxis is controversial and not recommended

ED TREATMENT/PROCEDURES

Following initial stabilization, the major goals of early treatment of ruptured or leaking

aneurysms are to prevent re-rupture, cerebral vasospasm, and hydrocephalus (see “Subarachnoid Hemorrhage”).

SURGERY/OTHER PROCEDURES

- Optimal timing for angiography and surgery remain controversial, but trend is toward early surgery to decrease incidence of rebleeding and cerebral vasospasm
- Early placement of ventriculostomy in appropriate patients may allow for direct intracranial pressure monitoring and often decreases systemic hypertension

Pediatric Considerations

Aneurysms in children have a high rate of hemorrhage and should be repaired early

MEDICATION

First Line

- Labetalol: 20–30 mg/min IV bolus, then 40–80 mg q10min max. 300 mg; follow with continuous infusion 0.5–2 mg/min
- Nimodipine: 60 mg PO/nasogastric q4h
- Ondansetron: 4 mg PO/SL/IV q4h PRN (peds: 0.1 mg/kg IV; max. 4 mg/dose)
- Prochlorperazine: 5–10 g IV/IM q6–8h (peds: 0.2 mg/kg/d IM in 3 or 4 div. doses); max. 40 mg/d

Second Line

- Diazepam: 5–10 mg IV q10–15min max., 30 mg (peds: 0.2–0.3 mg/kg q5–10min max. 10 mg)
- Docusate sodium: 100 mg PO BID
- Fosphenytoin: 15–20 mg/kg phenytoin equivalents (PE) at rate of 100–150 mg/min IV/IM
- Hydralazine: 10–20 mg IV q30min
- Lorazepam: 2–4 mg IV q15min PRN (peds: 0.03–0.05 mg/kg/dose; max. 4 mg/dose)
- Nicardipine: 5 mg/h IV infusion, increase by 2.5 mg/h q5–15min max. 15 mg/h (peds: Dosing unavailable)
- Phenytoin: 15–20 mg/kg IV load at max. 50 mg/min; max. 1.5 g (adult and peds); maintenance 4–6 mg/kg/d IV/IM



FOLLOW-UP

DISPOSITION

Admission Criteria

- Any patient with acute aneurysmal subarachnoid hemorrhage should be admitted,

preferably to ICU

- Any patient with symptomatic unruptured aneurysm should receive admission and urgent neurosurgical consultation, given high rate of rupture

Discharge Criteria

- Patients with incidentally discovered asymptomatic intracranial aneurysms may be discharged with close neurosurgical follow-up
- Note that overall risk of rupture is 1–2%/yr and that critical size at which risk for rupture outweighs risk for surgery is controversial (classically 10 mm, but probably in the 4–8-mm range).

FOLLOW-UP RECOMMENDATIONS

- Neurosurgery
- Neurology
- Primary care

PEARLS AND PITFALLS

- CT scan alone is not sufficient to exclude subarachnoid hemorrhage
- Vasospasm is typically seen on day 3 after bleed or surgery
- Nimodipine can prevent or treat vasospasm but should never be administered IV
- Nitroprusside and nitroglycerine should be avoided due to tendency to increase cerebral blood volume and thereby intracranial pressure

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Subarachnoid Hemorrhage



CODES

ICD9

- 430 Subarachnoid hemorrhage
- 437.3 Cerebral aneurysm, nonruptured
- 747.81 Anomalies of cerebrovascular system

ICD10

- I60.7 Nontraumatic subarachnoid hemorrhage from unspecified intracranial artery
- I67.1 Cerebral aneurysm, nonruptured
- Q28.3 Other malformations of cerebral vessels

CEREBRAL VASCULAR ACCIDENT

Veronique Au • Rebecca Smith-Coggins

BASICS

DESCRIPTION

Interruption of blood flow to a specific brain region:

- Neurologic findings are determined by specific area affected
- Onset may be sudden and complete, or stuttering and intermittent
- Responsible for 1 in 18 deaths in US
- 610,000 new strokes every year in US

RISK FACTORS

- Diabetes
- Smoking
- HTN
- Coronary artery disease, dysrhythmias
- Peripheral vascular disease
- Oral contraceptive use
- Polycythemia vera
- Sickle cell anemia
- Deficiencies of antithrombin III, protein C or S

ETIOLOGY

- May be ischemic (thrombotic, embolic, or secondary to dissection/hypoperfusion) or hemorrhagic
- Thrombotic stroke is caused by occlusion of blood vessels:
 - Clot formation at an ulcerated atherosclerotic plaque is most common
 - Sludging (sickle cell anemia, polycythemia vera, protein C deficiency)
- Embolic stroke is caused by acute blockage of a cerebral artery by a piece of foreign material from outside the brain, including:
 - Cardiac mural thrombi associated with mitral stenosis, atrial fibrillation, cardiomyopathy, CHF, or MI
 - Prosthetic or abnormal native valves
 - Atherosclerotic plaques in the aortic arch or carotid arteries
 - Atrial myxoma
 - Ventricular aneurysms with thrombi
- Arterial dissection:
 - Carotid artery dissection
 - Arteritis (giant cell, Takayasu)
 - Fibromuscular dysplasia
- Global ischemic or hypotensive stroke is caused by an overall decrease in systemic

BP: Sepsis, hemorrhage, shock

- Hemorrhagic stroke:
 - Intracranial hemorrhage
 - Subarachnoid hemorrhage

Pediatric Considerations

- Usually attributable to an underlying disease process, such as sickle cell anemia, leukemia, infection, or a blood dyscrasia
- Younger children often present with seizures and/or altered mental status



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Time of onset (or time last seen at baseline)
- Trauma/surgery
- Medications
- Altered mentation/confusion
- Headache
- Vertigo/dizzy
- Focal neurologic deficits

Physical-Exam

- General:
 - Cheyne–Stokes breathing, apnea
 - HTN
 - Cardiac dysrhythmias, murmurs
- Anterior cerebral artery:
 - Contralateral hemiplegia (lower/upper)
 - Hemisensory loss
 - Apraxia
 - Confusion
 - Impaired judgment
- Middle cerebral artery:
 - Contralateral hemiplegia (upper/lower)
 - Hemisensory deficits
 - Homonymous hemianopsia
 - Dysphasia
 - Dysarthria
 - Agnosia
- Posterior cerebral artery:

- Cortical blindness in half the visual field
- Visual agnosia
- Altered mental status
- Impaired memory
- 3rd-nerve palsy
- Hemiballismus
- Vertebrbasilar system:
 - Impaired vision, visual field defects, nystagmus, diplopia
 - Vertigo, dizziness
 - Crossed deficits: Ipsilateral cranial nerve deficits with contralateral motor and sensory deficits
- Basilar system:
 - Quadriplegia
 - Locked-in syndrome
 - Coma
- Watershed area (boundary zone between anterior, middle, and posterior circulation):
 - Cortical blindness
 - Weakness of proximal upper and lower extremities with sparing of face, hands, and feet

ESSENTIAL WORKUP

- Detailed neurologic exam; consider calculating National Institutes of Health stroke scale (NIHSS).
- Emergent noncontrast head CT scan to distinguish ischemic from hemorrhagic events:
 - May be normal in 1st 24–48 hr
 - GOALS:
 - CT completed within 25 min of arrival
 - CT read by a radiologist within 45 min
 - Thrombolytics administered within 1 hr of presentation
- If CT is normal and subarachnoid hemorrhage is suspected, emergent lumbar puncture is indicated
- EKG to evaluate for dysrhythmias or presence of MI
- Oxygen saturation measurement
- Rapid glucose determination

DIAGNOSIS TESTS & NTERPRETATION

Lab

- Baseline CBC, electrolytes, renal function tests, liver function test, prothrombin time, partial thromboplastin time
- Urinalysis:

- Hematuria can be seen in subacute bacterial endocarditis with embolic stroke.
- Sedimentation rate:
 - Elevated in subacute bacterial endocarditis, vasculitis, hyperviscosity syndromes
- Consider additional tests: Cardiac enzymes, urine pregnancy test, drug screen, alcohol level, ABG, and blood cultures.

Imaging

- Noncontrast head CT
- MRI can detect ischemia < 2 hr after onset
- CXR
- Carotid US

Diagnostic Procedures/Surgery

- EKG to evaluate for arrhythmia
- Lumbar puncture if subarachnoid hemorrhage is suspected and head CT nondiagnostic

DIFFERENTIAL DIAGNOSIS

- Intracranial bleeding
- Hypoglycemia
- Seizure disorder; Todd paralysis
- Panic attacks, depression, conversion reaction
- Transient global amnesia
- Meningoencephalitis
- Peripheral neuropathy
- Intracranial abscess
- Migraine
- Air embolism
- Transient ischemic attack
- Encephalopathy
- Neoplasm
- Giant cell/Takayasu arteritis
- Multiple sclerosis
- Compressive myelopathy
- Vestibulitis
- Medication effect/toxidrome



TREATMENT

PRE HOSPITAL

- Patients may have difficulty moving or communicating after cerebral vascular accident
- Neurologic exam in field is helpful:
 - Should include assessment of consciousness level, Glasgow coma scale score, gross motor deficits, speech abnormalities, gait disturbance, facial asymmetry, and other focal deficits
- Check fingerstick glucose

INITIAL STABILIZATION/THERAPY

- Manage airway:
 - Supplemental oxygen 2–4 L
 - Rapid-sequence intubation may be required for airway protection or controlled ventilation to decrease intracranial pressure
- For altered mental status, give naloxone and thiamine and check blood glucose

ED TREATMENT/PROCEDURES

- Treat elevated BP with labetalol, nicardipine, nitroprusside, or hydralazine:
 - Systolic BP >220 mm Hg or diastolic BP >120 mm Hg on repeated measurements
 - If indicated for other concurrent problems (MI, aortic dissection, CHF, hypertensive encephalopathy)
 - Initial goal is systolic BP <180 mm Hg, diastolic <110 mm Hg
- Control seizures with benzodiazepines, then fosphenytoin/phenytoin
- Maintain euvolemia and normothermia.
- Thrombolytics:
 - Ischemic stroke only; administer within 4.5 hr of symptom onset
 - Contraindications:
 - Any history of intracranial hemorrhage
 - Recent stroke or head trauma <3 mo ago
 - Major surgery <14 days ago
 - Systolic BP >185 mm Hg; diastolic BP >110 mm Hg
 - Bleeding diathesis
 - Noncompressible arterial puncture <7 days ago
 - MI <3 mo ago
 - Anticoagulation: INR >1.7, PT >15 sec, or prolonged PTT; use of heparin within 48 hr
 - Platelets <100,000
 - Intracranial neoplasm
 - Seizure at stroke onset
 - Minor or rapidly improving symptoms
 - Pregnancy
 - Internal bleed (GI/GU) <3 wk ago
 - Blood glucose <50

- Age < 18 yr
 - Avoid anticoagulants and antiplatelet drugs for 24 hr
- Treat increased intracranial pressure and cerebral edema:
 - Elevate head of bed 30°
 - Controlled ventilation to keep partial pressure of carbon dioxide 35–40 mm Hg
 - Mannitol
- Urgent neurosurgical decompression may be required with brainstem compression in cases of vertebrobasilar stroke or hemorrhage.
- In patients with completed or minor strokes, aspirin may prevent recurrence.
- For focal embolic/thrombotic strokes:
 - Recannulation
 - US-enhanced thrombolysis
 - Intra-arterial thrombolysis or clot retrieval

ALERT

For patients presenting between 3 and 4.5 hr of onset, there are additional exclusion criteria:

- Age > 80 yr
- Oral anticoagulant use (regardless of INR)
- NIH-SS > 25 or > 1/3 MCA territory involved
- History of previous stroke *and* diabetes

MEDICATION

First Line

- Alteplase (tPA): 0.9 mg/kg IV; max. 90 mg, with 10% of dose given as bolus and remainder infused over 60 min
- Aspirin: 81–325 mg PO/PR
- Labetalol: 10–20 mg IV bolus, repeat q10min max. 300 mg; follow with continuous infusion 0.5–2 mg/min

Second Line

- Clopidogrel: 75 mg PO daily
- Diazepam: 5 mg IV q5–10min max. 20 mg
- Enalapril: 0.675–1.25 mg IV
- Hydralazine: 10–20 mg IV q30min
- Mannitol (15–25% solution): 0.5–2 g/kg IV over 5–10 min, then 0.5–1 g/kg q4–q6h
- Nicardipine: 5 mg/h IV, increase by 2.5 mg/h q5–15min max. 15 mg/h
- Nitroprusside: 0.25–10 µg/kg/min IV
- Trimetaphan: 1–4 mg/min IV

Pediatric Considerations

- Heparin or low-molecular-weight heparin is often used in children with ischemic stroke
- May call 1-800-NOCLOTS for pediatric stroke consultation and guidance



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients with acute cerebral vascular accident should be admitted to hospital
- Indications for ICU:
 - _ Severely decreased level of consciousness
 - _ Hemodynamic instability
 - _ Life-threatening cardiac dysrhythmias
 - _ Significantly increased intracranial pressure
 - _ Administration of alteplase

Discharge Criteria

- Patients who present with completed strokes that are days to weeks old may be discharged if they are able to function independently or have adequate social support
- Patients with multiple prior strokes who experience relatively minor new episodes may also be treated on outpatient basis if similar criteria are met and stroke is completed

FOLLOW-UP RECOMMENDATIONS

- Neurology
- Primary care
- Speech therapy/occupational therapy

PEARLS AND PITFALLS

- Always note pre-hospital observations
- Onset of symptoms is crucial to determining treatment with tPA
 - _ Include additional exclusion criteria between 3 and 4.5 hr
- Avoid aggressive BP correction due to risk of hypoperfusion and extension of stroke
- Door to needle goal is <60 min

ADDITIONAL READING

- Clinical Policy: Use of intravenous tPA for the management of acute ischemic

stroke in the emergency department. *Ann Emerg Med.* 2013;61:225–243.

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- www.ninds.nih.gov/doctors



See Also (Topic, Algorithm, Electronic Media Element)

- Transient Ischemic Attack
- Intracranial Hemorrhage



CODES

ICD9

- 434.01 Cerebral thrombosis with cerebral infarction
- 434.11 Cerebral embolism with cerebral infarction
- 434.91 Cerebral artery occlusion, unspecified with cerebral infarction

ICD10

- I63.59 Cereb infrc due to unsp occls or stenosis of cerebral artery
- I63.8 Other cerebral infarction
- I63.9 Cerebral infarction, unspecified

CERVICAL ADENITIS

Julie Zeller



BASICS

DESCRIPTION

- Acute bacterial infection of a cervical lymph node
 - Often arising after a prior bacterial infection of the head or neck area
- Primarily a pediatric disease:
 - Becoming more common in adults owing to immunocompromised disease states (HIV, cancer, transplant patients)
- Any cervical node can become infected:
 - >80% of childhood cervical lymphadenitis involves the submandibular or deep cervical nodes
 - Jugulodigastric node located just below the angle of the mandible is common site
 - Cervical nodes act as the final common pathway for lymphatic drainage of all areas of the head and neck
 - Initial lymphadenopathy results after bacterial invasion of regional areas of the head and neck
 - Local lymph nodes swell secondary to hyperplasia of sinusoidal cells and infiltration of lymphocytes
 - If the infection is not contained, the bacteria enter the lymph system and proliferate (lymphadenitis)
 - Pus forms when neutrophils are incited, and an abscess develops when host defenses are unable to clear infection
 - Clinically manifests as warm, tender, swollen, erythematous node

ETIOLOGY

- ~70% of cases are a result of group A β -hemolytic Streptococcal infection
 - 20% Staphylococcal infection
 - 10% related to viral infection or other bacteria
- Infections secondary to community-acquired MRSA (CA-MRSA) have increased in frequency
- Children have one of the highest rate of CA-MRSA colonization and invasive disease
- Mycobacteria TB:
 - Scrofula or tuberculous lymphadenitis
 - Rarely seen
 - Usually a chronic lymphadenitis in the posterior cervical nodes
 - Purified protein derivative (PPD) is usually strongly reactive

- Treatment is nonsurgical
- Atypical mycobacteria (nontuberculous) *Mycobacterium avium* complex:
 - More commonly seen
 - Usually a chronic lymphadenitis in the submandibular or anterior cervical nodes
 - PPD test results are unreliable
 - Treatment is primarily surgical
- *Bartonella henselae* (cat scratch disease):
 - Subacute lymphadenitis
 - Fever and mild systemic symptoms occur in only ~ 3% of patients
 - Has indolent course but usually spontaneously resolves after 4–6 wk
- Anaerobes:
 - Consider when associated with infections of the teeth or gingiva
- Rare organisms:
 - Gram-negative bacilli
 - *Yersinia pestis*
 - Group B streptococcus
 - *Francisella tularensis*
 - *Alpha-streptococcus*
 - Anthrax

Pediatric Considerations

- One of the most common causes of a neck mass in a child
- Overall, group A Streptococcus and *Staphylococcus aureus* most common causes
- In neonates, group B Streptococcus and *S. aureus* most common
- Group B *Streptococcal cellulitis*–adenitis syndrome:
 - Infants are usually 3–7 wk of age, male, febrile, with submandibular or facial cellulitis, and an ipsilateral otitis media
 - 94% incidence of concurrent bacteremia
- *S. aureus* associated with more indolent course and higher frequency of suppuration
- Viral infections generally result in bilateral lymphadenopathy

Geriatric Considerations

- Consider malignancy over infection in this population, especially in the absence of fever, leukocytosis, etc.
- Fixed, nontender, hard node most likely not cervical adenitis

DIAGNOSIS

SIGNS AND SYMPTOMS

- Enlarged, tender cervical lymph node

- Usually unilateral and solitary
- Warmth and erythema of overlying skin
- Early in course, node is firm but may become fluctuant later
- With or without fever
- Malaise
- Irritability in infants and children
- Usually a concurrent head and neck infection:
 - Pharyngitis, tonsillitis, peritonsillar abscess
 - Otitis media, otitis externa
 - Dental infection
 - Impetigo, scalp infection

History

- Time of onset of symptoms
- Associated symptoms: Fever, weight loss, rash
- Exposures/travel history
- Comorbidities/birth history for infants

Physical-Exam

Complete evaluation of head and neck with attention to airway and patient's clinical appearance

ESSENTIAL WORKUP

- Cervical adenitis is a clinical diagnosis
- Identify primary source of infection in head and neck area (e.g., otitis media, tonsillitis)
- If no primary inflammatory source of infection in head and neck:
 - Address possible TB exposure with PPD
 - Look for signs of systemic disease and viral illness

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Unnecessary if a treatable primary source of infection confirmed
- Blood cultures for toxic-appearing patients
- Sepsis workup in neonates
- If cause unclear, the following lab tests may help to discern a nonbacterial cause (see “Differential Diagnosis”):
 - Leukocyte count with differential
 - Monospot
 - Throat cultures
 - Antibody titers (Epstein–Barr virus, CMV, toxoplasmosis)

Imaging

- CXR study, lateral neck, or Panorex:
 - Helpful if source of infection unclear or to rule out a deep space infection
 - Chest radiograph study to screen for TB
- CT or MRI of neck:
 - Helpful to exclude deep space infections or delineating embryonic developmental masses
- US:
 - Can differentiate cystic from solid structures, but other findings nonspecific
 - Can identify deep-cavity abscess if not palpable on exam
- Excisional biopsy

Diagnostic Procedures/Surgery

- Needle aspiration:
 - All fluctuant nodes should be aspirated
 - Send for Gram stain and acid-fast stains, aerobic and anaerobic cultures, mycobacteria, and fungi
 - If any suspicion of tuberculous lymphadenitis, the node should not be aspirated owing to risk for sinus development and chronic drainage
- Intradermal skin testing:
 - Mycobacteria, catscratch disease

DIFFERENTIAL DIAGNOSIS

- Lymphadenopathy (inflammation of node but no bacterial infection) can be a sign of many systemic diseases; usually these nodes are multiple and bilateral
- Viral infections are a common cause:
 - Respiratory viruses (adenoviruses, rhinoviruses, enteroviruses)
 - Epstein–Barr virus, herpes simplex virus, varicella-zoster virus, CMV
 - Mumps, rubella, rubeola
- Specific pediatric diseases with cervical adenitis in their diagnostic criteria:
 - Kawasaki disease
 - Kikuchi disease
 - Periodic fever, aphthous stomatitis, pharyngitis, and cervical adenitis known by mnemonic PFAPA (seen in preschool-aged children)
- Toxoplasmosis
- Congenital cysts:
 - Brachial cleft cysts, thyroglossal duct cysts, cystic hygromas
- Malignancies:
 - Leukemia, lymphoma, rhabdomyosarcoma, thyroid carcinoma
 - Rare cause of a nonspecific lump in children (< 2% overall)
- Other systemic diseases:
 - Lupus, sarcoidosis



TREATMENT

INITIAL STABILIZATION/THERAPY

- Oxygen, monitor airway for any signs of compromise
- Universal precautions

ED TREATMENT/PROCEDURES

- Treatment directed toward the primary source of infection in the head and neck:
 - If unsure of cause, treat for group A Streptococcus and *S. aureus*
 - Consider MRSA if symptoms not improving on standard antibiotic therapy
- Aspirate all fluctuant nodes
- Many oral antibiotics are effective:
 - Cephalexin
 - Cefadroxil
 - Amoxicillin/clavulanic acid
- Patients with suspected dental, periodontal, or anaerobic causes of illness:
 - Clindamycin
 - Amoxicillin/clavulanic acid
- CA-MRSA:
 - Clindamycin (many isolates are now resistant)
 - Bactrim
 - Vancomycin or Linezolid if toxic and requiring inpatient care
- Treatment should be for at least 10 days, even if symptoms resolve sooner
- Warm, moist compresses
- Analgesics, as needed

MEDICATION

First Line

- Cefadroxil: 500 mg (peds: 30 mg/kg/24 h) PO q12h
- Cephalexin: 250–500 mg (peds: 25–50 mg/kg/24 h) PO q6h
- Amoxicillin/clavulanic acid: 250–500 mg (peds: 20–40 mg/kg/24 h) PO q8h
- Clindamycin: 300 mg (peds: 8–25 mg/kg/24 h) PO q6h
- TMP-SMX (Bactrim): DS (160/800) 2 tabs PO BID (peds: 40 mg/200 mg/10 kg/PO BID)

Second Line

- Cefazolin: 1–2 g (peds: 25–50 mg/kg/24 h) IV q8h
- Nafcillin: 1–2 g (peds: 50–200 mg/kg/24 h) IV q4–6h
- Clindamycin: 600–900 mg (peds: 20–40 mg/kg/24 h) IV q8h
- Ampicillin–sulbactam: 1.5–3 g (peds: 200 mg/kg/d) q6h
- Vancomycin: 10–15 mg/kg IV Q12h (peds: 40–60 mg/kg/d div q8h)

- Linezolid (alternative to Vancomycin): 600 mg IV BID for children >12 or 30 mg/kg/8 h with max. dose of 1,200 mg for children <12 yr



FOLLOW-UP

DISPOSITION

Admission Criteria

- Neonates
- Airway compromise
- Patient appears ill
- Immunocompromised
- Inability to take PO
- Not improving on oral antibiotics

Discharge Criteria

- Most patients can be discharged on PO antibiotics
- Close follow-up with a recheck in 2–3 days
- Ability to take PO antibiotics and fluids
- Return to the ED if:
 - Symptoms worsen
 - Abscess develops
 - Voice changes
 - Dyspnea develops
 - Systemic symptoms develop

Issues for Referral

Clinical exam concerning for malignancy or congenital abnormality (brachial cleft/thyroglossal duct cyst)

FOLLOW-UP RECOMMENDATIONS

- Mandatory recheck in 48 hr to ensure improvement
- Referral to dentist or ENT depending on source of infection

PEARLS AND PITFALLS

- Cervical adenitis is a clinical diagnosis
- Unilateral warm, tender, swollen, erythematous lymph node
- Most common bacteria responsible for infection are group A *Strep* and *S. aureus*.
- Consider group B *Strep* in infants and MRSA for infections not improving on standard antibiotics
- Disposition should be influenced by patient's clinical status

ADDITIONAL READING

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- Healy CM. Diagnostic approach to and initial treatment of cervical lymphadenitis in children. UpToDate.com/online
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- Swanson D. Etiology and clinical manifestations of cervical lymphadenitis in children. UpToDate.com/online



See Also (Topic, Algorithm, Electronic Media Element)

- [Kawasaki Disease](#)
- [Lymphadenitis](#)



CODES

ICD9

683 Acute lymphadenitis

ICD10

[L04.0 Acute lymphadenitis of face, head and neck](#)

CESAREAN SECTION, EMERGENCY

Jonathan B. Walker • James S. Walker

BASICS

ALERT

- The sole indication for ED physician to perform emergency perimortem cesarean section is a gravid female (>24 wk gestation) in cardiopulmonary arrest who has not responded to initial resuscitative measures, regardless of cause
- The most important predictor of fetal survival is length of time between maternal cardiac arrest and cesarean delivery:
 - _ Cesarean section should begin within 4 min of maternal arrest
 - _ Goal is delivering fetus within 1 min
- Obtain immediate consultations from obstetrics, pediatrics (and surgery, if trauma related):
 - _ Do not defer or delay performing procedure until arrival of consultants
- Do not perform emergent cesarean section if patient is <24 wk gestation

ETIOLOGY

- Trauma (penetrating or blunt):
 - _ Major cause of maternal mortality
- Pulmonary embolus:
 - _ Thromboembolism is most common cause of nontraumatic maternal mortality
- Cerebral vascular accident
- Amniotic fluid embolism
- DIC
- Placenta previa
- Eclampsia
- Miscellaneous medical disorders:
 - _ Asthma
 - _ CHF
 - _ MI
 - _ Drug overdose

DIAGNOSIS

SIGNS AND SYMPTOMS

History

Gravid female (>24 wk gestation determined by uterine fundal height) who is in

cardiopulmonary arrest

Physical-Exam

Patient is determined to be >24 wk gestation if uterus is at least 4 finger breadths above umbilicus

ESSENTIAL WORKUP

- Physical exam for apnea and pulselessness in obviously gravid female:
 - Quickly evaluate for reversible causes of cardiopulmonary arrest:
 - Hypoxia
 - Hypovolemia
 - Hydrogen ion (acidosis)
 - Hypokalemia/hyperkalemia
 - Hypoglycemia
 - Hypothermia
 - Trauma
 - Thromboembolism
 - Toxins/poisons
 - Tension pneumothorax
 - Tamponade (pericardial)
 - Supine hypotension syndrome (compression of inferior vena cava by enlarged uterus)
- Assess gestational age by uterine fundal height
 - Distance from pubis to fundus in centimeters is roughly equivalent to gestational age in weeks, i.e., 24 cm = 24 wk
- US is beneficial if *immediately* available to assess fetus.

DIAGNOSIS TESTS & INTERPRETATION

Imaging

- None necessary to establish cardiopulmonary arrest
- Do *not* use valuable time attempting to determine fetal heart tones

DIFFERENTIAL DIAGNOSIS

Cardiopulmonary arrest is final common pathway:

- Evaluate for underlying cause



TREATMENT

PRE HOSPITAL

Cautions:

- Minimal scene time, “scoop and run”

- Place the patient in the left lateral decubitus position to avoid compression of inferior vena cava (supine hypotension syndrome)
- Trauma patient requiring spinal immobilization:
 - Uterus can be manually displaced to left
 - Backboard can be wedged to keep right hip elevated 45°

INITIAL STABILIZATION/THERAPY

- Standard resuscitation measures:
 - Emergency intubation
 - Use a smaller endotracheal tube (0.5–1 mm less in internal diameter compared to that used for nonpregnant women)
 - High-flow oxygen
 - Cardiac and BP monitoring
 - 2 large-bore peripheral IV lines:
 - Fluid resuscitation
 - O-negative blood if indicated
- Fetal survival correlates with maternal survival and adequacy of initial maternal resuscitation
- If patient is at <24 wk gestation, use advanced cardiac life support (ACLS) and advanced trauma life support protocols directed at maternal resuscitation
 - Do *not* perform emergent cesarean section
- If patient is >24 wk gestation, use 4-min rule:
 - Perform ACLS or advanced trauma life support for 4 min
 - If no response, proceed to immediate emergency cesarean section
 - Goal is to deliver fetus within 1 min
 - If it is obvious there is no chance for maternal survival, begin perimortem cesarean section immediately

ED TREATMENT/PROCEDURES

- Call for immediate obstetric, surgical, and pediatric consultations:
 - Do *not* delay performing procedure while waiting for consultants
- Ensure a Foley catheter has been inserted to decompress bladder, but do not delay procedure
- Perform cesarean section:
 - Use linea nigra as landmark for vertical midline incision
 - Incise abdominal wall from pubic hairline to 5 cm above umbilicus.
 - This incision should pass through fascial and peritoneal layers
 - Retract urinary bladder inferiorly against pubic symphysis
 - Make small vertical incision in lower uterine segment, just cephalad to urinary bladder
 - Extend incision cephalad with scissors:
 - Insert your free hand into uterus
 - Lift uterine wall away from fetus to avoid fetal injury

- Deliver fetus
- Clamp umbilical cord in 2 places and cut between the 2 clamps
- Manually deliver placenta
- Perform neonatal resuscitation, as indicated
- Immediately reassess maternal vital signs because occasionally spontaneous circulation may return
- Continue maternal resuscitation as appropriate
- Suture uterus with running lock stitch using no. 0 polyglactin suture
- Suture fascia and peritoneum with running stitch using no. 0 polyglactin suture
- Close the skin with staples or suture
- Administer broad-spectrum antibiotics
- If maternal return of circulation is obtained, consider starting therapeutic hypothermia protocol

MEDICATION

First Line

Resuscitative measures/ACLS medications directed at mother:

- Treatment of underlying cause

Second Line

Neonatal resuscitation should be anticipated:

- Oral tracheal intubation



FOLLOW-UP

DISPOSITION

Admission Criteria

- The infant should be admitted to NICU
- If maternal resuscitation is successful, patient should be admitted to appropriate ICU

Discharge Criteria

Neither infant nor mother should be discharged from ED

PEARLS AND PITFALLS

- Only females > 24 wk pregnant in cardiopulmonary arrest qualify for the procedure.
- Decision to perform perimortem cesarean section must be made quickly (within 4

- min of maternal cardiopulmonary arrest)
- Procedure must be done quickly (<1 min).

ADDITIONAL READING

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CODES

ICD9

- 427.5 Cardiac arrest
- 659.83 Other specified indications for care or intervention related to labor and delivery, antepartum condition or complication

ICD10

- I46.9 Cardiac arrest, cause unspecified
- O99.419 Diseases of the circ sys comp pregnancy, unsp trimester

CHANCROID

Norbert Elsner



BASICS

DESCRIPTION

- Sexually transmitted genital ulcerative disease:
 - Increased risk for HIV infection
- A common cause of genital ulceration in Africa, southeast Asia, and Latin America:
 - Uncommon in US where herpes simplex virus (HSV) > syphilis > > chancroid, but likely underreported

ETIOLOGY

Causative agent: *Haemophilus ducreyi*

- Highly infectious bacterium



DIAGNOSIS

SIGNS AND SYMPTOMS

- Begins as a single erythematous papule or pustule:
 - Quickly erodes into painful chancres (1–20 mm)
 - Soft and friable with ragged, irregular borders
- Primary ulcer usually excavated
- Moist, granulation tissue at base
- Purulent or hemorrhagic exudate
- Location:
 - Male:
 - Penile shaft, glans, internal surface of foreskin, anus
 - Female:
 - Cervix, vagina, vulva, perineum, anus
- Occurs 4–7 days (median) after exposure
- Incubation period 3–10 days (range 1–35 days)
- Inguinal adenopathy:
 - In ~50% of men; less common in women
 - Appears 3–14 days after initial ulcer
 - Unilateral (usually)
 - Painful
 - Suppurative large nodes (buboes)
 - May rupture and form chronic draining sinuses
- Dysuria, dyspareunia secondary to contact with lesions
- Variants:

- Phagedenic:
 - Secondary superinfection (especially fusospirochetal) and rapid extensive tissue destruction
- Giant chancroid:
 - Very large single ulcer
- Serpiginous ulcer:
 - Rapidly spreading, indolent, shallow ulcers in groin or thigh
- Follicular:
 - Multiple small ulcers with perifollicular distribution

ESSENTIAL WORKUP

Clinical diagnosis based on appearance is often inaccurate, and lab tests difficult or unavailable, so consider:

- CDC case definitions:
 - Definite: Positive culture of *H. ducreyi*
 - Probable: Typical signs, symptoms of chancroid + negative dark-field exam for *Treponema pallidum* + negative syphilis serology + negative culture for HSV (or clinical exam atypical for herpes)

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Gram stain unreliable (positive in 50–80%):
 - Gram-negative coccobacilli
 - Linear or “school-of-fish” pattern
- Culture extremely difficult (positive in 0–80%); requires complex media:
 - Obtain specimen from:
 - Base of ulcer
 - Needle aspiration of inguinal node by placing needle through normal skin (to avoid formation of fistula)
- Polymerase chain reaction (PCR) assay:
 - Sensitive and specific, but not widely available
- RPR:
 - Coinfection with syphilis is common
 - Part of CDC guidelines for probable clinical diagnosis of chancroid
- HSV culture:
 - Part of CDC guidelines for probable clinical diagnosis of chancroid
- HIV testing

DIFFERENTIAL DIAGNOSIS

- Infectious:
 - Syphilis (*T. pallidum*):
 - Chancre usually painless, indurated, clean

- Herpes genitalis (*HSV*):
 - Vesicular, multiple, recurrent
- Granuloma inguinale (donovanosis) (*Klebsiella granulomatis*):
 - Ulcer margins elevated; + induration
- Lymphogranuloma venereum (*Chlamydia trachomatis*):
 - Often single lesion; tender, fluctuant, unilateral lymphadenopathy
- Noninfectious:
 - Drug eruption
 - Less common:
 - Pyoderma gangrenosum
 - Behçet disease



TREATMENT

INITIAL STABILIZATION/THERAPY

Usual precautions for patient exam and handling of specimens

ED TREATMENT/PROCEDURES

Antibiotics:

- Azithromycin: Single PO dose
- Ceftriaxone: Single IM dose (pregnancy: 1st line)
- Ciprofloxacin: PO × 3 days:
 - NOT for pregnant/lactating patients
- Erythromycin base: PO × 7 days:
 - Pregnancy: 2nd line
- Needle aspiration of suppurative nodes (> 5 cm diameter):
 - To prevent chronic sinus drainage from spontaneous rupture
 - Use 18G needle through lateral intact skin.
 - May require repetition
- Recommend concurrent HIV, syphilis, HSV testing, *and* follow-up testing in 3 mo if initially negative

MEDICATION

First Line

- Azithromycin: 1 g PO × 1
- Ceftriaxone: 250 mg IM × 1

Second Line

- Ciprofloxacin: 500 mg PO BID for 3 days
- Erythromycin base: 500 mg PO QID for 7 days



FOLLOW-UP

DISPOSITION

Admission Criteria

- Sexual abstinence or condom use until lesions healed
- Clinical course:
 - Symptoms improve within 2 days of treatment
 - Ulcers improve within 3–7 days
 - Possible delayed resolution in those HIV-positive or uncircumcised

FOLLOW-UP RECOMMENDATIONS

- Examine and treat sexual partners (regardless of presence/absence of symptoms) if contact within 10 days of symptom onset
- HIV-positive patients require assured follow-up if using single-dose therapy (higher treatment failure rate)

PEARLS AND PITFALLS

- Initiate treatment if probable CDC case guidelines met; do not wait for culture results
- Higher risk of treatment failure in HIV-infected patients
- Presumptive treatment of sexual contacts
- Treatment failure: Consider drug resistance, medication noncompliance, coinfection (syphilis).

ADDITIONAL READING

- Centers for Disease Control and Prevention. Sexually Transmitted Diseases Treatment Guidelines 2010. Available at www.cdc.gov/std/treatment. Accessed March 1, 2013.
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- Marx JA, Hockberger RS, Walls RM, eds. *Rosen's Emergency Medicine: Concepts and Clinical Practice*. 7th ed. St. Louis, MO: Mosby; 2010.



CODES

ICD9

099.0 Chancroid

ICD10

A57 Chancroid

CHEMICAL WEAPONS POISONING

Patrick M. Whiteley



BASICS

DESCRIPTION

Chemical agents that affect CNS, pulmonary, cardiovascular, dermal, ocular, or GI systems when exposed to victims

ETIOLOGY

- **Blood agents: Cyanide:**
 - Inhibition of cellular respiration by binding to ferric ion in cytochrome oxidase a-a3 and uncoupling oxidative phosphorylation
- **Blister agents: Sulfur mustard, nitrogen mustard, lewisite, phosgene oxime:**
 - Alkylation and cross-linking of purine bases of DNA and amino acids resulting in change in structure of nucleic acid, proteins, and cellular membranes
- **Lacrimators and riot control agents: 1-chloroacetophenone (CN; Mace), o-chlorobenzylidene malononitrile (CS), oleoresin capsaicin-pepper spray (OC), chloropicrin, adamsite (DM):**
 - Mucous membrane irritators
- **Pulmonary irritants (choking agents):**
 - High water solubility: Ammonia:
 - Mucous membrane irritation of eyes and upper airway
 - Intermediate water solubility: Chlorine:
 - Forms hydrochloric acid, hydrochlorous acids, which form free radicals causing upper airway and pulmonary irritation
 - Low water solubility: Phosgene:
 - Mild irritant effects initially, then delayed pulmonary edema as late as 24 hr
 - Direct pulmonary damage after hydrolysis in lungs to hydrochloric acid
- **Nerve agents:**
 - Anticholinesterase inhibitors—causes cholinergic overstimulation at muscarinic, nicotinic, and CNS sites
- **Incapacitating agents: 3-quinuclidinyl benzilate (BZ):**
 - Anticholinergic (antimuscarinic)



DIAGNOSIS

SIGNS AND SYMPTOMS

History

Multiple victims, house fire, known exposure (agent determines history findings)

Physical-Exam

- Blood agents (cyanide and cyanogens):
 - Vital signs:
 - Tachypnea and hyperpnea (early); respiratory depression (late)
 - Hypertension and tachycardia (early); hypotension and bradycardia (late)
 - Death within seconds to minutes
 - CNS:
 - Headache
 - Mental status changes
 - Seizures
 - Pulmonary:
 - Dyspnea
 - Noncardiogenic pulmonary edema
 - Cyanosis uncommon
 - GI:
 - Odor of bitter almonds (sometimes)
 - Burning in mouth and throat
 - Nausea, vomiting
- Blister agents (mustards, lewisite):
 - General:
 - Mortality, 2–4%
 - Dermatologic:
 - Skin erythema, edema, pruritus can appear 2–24 hr after exposure.
 - Necrosis and vesiculation appear 2–18 hr after exposure.
 - Head, eyes, ears, nose, and throat (HEENT):
 - Airway occlusion from sloughing of debris
 - Laryngospasm, sore throat, sinusitis
 - Eye pain, photophobia, lacrimation, blurred vision, blepharospasm, periorbital edema, conjunctival edema, corneal ulceration
 - Pulmonary:
 - Bronchospasm, tracheobronchitis
 - Respiratory failure
 - Hacking cough
 - GI:
 - Nausea, vomiting
 - Hematologic:
 - Leukopenia
- Lacrimators and riot control agents (tear gases):
 - HEENT:

- Eye pain
- Lacrimation
- Blepharospasm
- Temporary blindness
- Dermatologic:
 - Skin irritation
 - Papulovesicular dermatitis (tear gas)
 - Superficial burns
- Pulmonary:
 - Cough
 - Chest tightness
 - Dry throat
 - Sensation of suffocation
 - Pulmonary edema when exposed to high concentrations without ventilation
- Pulmonary irritants (choking agents):
 - HEENT:
 - Eye pain, lacrimation, blepharospasm
 - Temporary blindness
 - Dermatologic:
 - Skin irritation, dry throat, nasal irritation
 - Pulmonary:
 - Shortness of breath, cough, bronchospasm
 - Chest pain
 - Pulmonary edema as late as 24 hr from exposure (phosgene)
- Nerve agents (sarin, tabun, soman, VX):
 - *SLUDGE* syndrome:
 - Salivation
 - Lacrimation
 - Urination
 - Defecation
 - GI cramps
 - Emesis
 - *Bronchorrhea, bronchoconstriction, bradycardia (most life threatening)*
 - Abdominal upset
 - Miosis
 - HEENT:
 - Miosis
 - Hypersecretion by salivary, sweat, lacrimal, and bronchial glands
 - CNS:
 - Irritability, nervousness
 - Giddiness
 - Fatigue, lethargy, depression

- Ataxia, convulsions, coma
- Pulmonary:
 - Bronchoconstriction
 - Bronchorrhea
- GI:
 - Nausea, vomiting, diarrhea
 - Crampy abdominal pains
 - Urinary and fecal incontinence
- Musculoskeletal:
 - Fasciculations, skeletal muscle twitching
 - Weakness
 - Flaccid paralysis
- Incapacitating agents (BZ):
 - Anticholinergic (antimuscarinic) toxidrome:
 - Hot as a hare
 - Dry as a bone
 - Red as a beet
 - Blind as a bat
 - Mad as a hatter
 - Hypertension
 - Tachycardia
 - Hyperpyrexia
 - Urinary retention
 - Decreased bowel sounds

ESSENTIAL WORKUP

- History and symptoms key to type of agent exposure
- Physical exam:
 - Cyanide (bitter almonds, comatose, hypotensive, metabolic acidosis)
 - Mustard (faint, sweet odor of mustard or garlic, blisters, sloughing of skin, dyspnea)
 - Check for SLUDGEABAM syndrome.
 - Lacrimators (eye irritation, lacrimation, blepharospasm)
 - Choking agents (dyspnea, bronchospasm)

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Arterial blood gases:
 - Cyanide:
 - Decreased atrioventricular (AV) oxygen saturation gap
 - Lactic acidemia with high anion gap metabolic acidosis
 - Arterialization of venous blood

- Cyanide levels cannot be performed in clinically relevant timeframe.
- CBC:
 - Leukopenia, thrombocytopenia, anemia with significant mustard exposure
- Electrolytes, BUN, creatinine, glucose
- Urinalysis
- Creatine phosphokinase (CPK)
- Lactate for cyanide
- Erythrocyte cholinesterase activity for nerve agents

Imaging

CXR for pulmonary edema

DIFFERENTIAL DIAGNOSIS

- Asthma/COPD
- Stevens–Johnson syndrome
- Toxic epidermal necrolysis
- Pemphigus vulgaris
- Scalded skin syndrome
- Organophosphate or carbamate pesticide poisoning
- Botulism
- Radiation poisoning
- CHF
- Anaphylactoid reaction



TREATMENT

PRE-HOSPITAL

- Avoid contamination of environment and clinicians:
 - Use level A or level B personal protective equipment.
 - Decontamination:
 - Dermal wet decontamination primarily for nerve and blistering agents
 - Dry decontamination (removal of clothing and jewelry) for other agents
- Administer atropine even if patient is tachycardic because condition may result from hypoxia.

INITIAL STABILIZATION/THERAPY

- ABCs
- Patient decontamination:
 - Brush off powder from chemical.
 - Irrigate skin and eyes with copious amounts of water or saline.

- Remove and dispose of clothing in double bags.
- Protection for health care workers:
 - Level A or B personal protective suit
 - Chemical-resistant suit
 - Heavy rubber gloves and boots, neoprene gloves
- Administer oxygen, place on cardiac monitor, and measure pulse oximetry.
- Establish IV access with 0.9% NS.

ED TREATMENT/PROCEDURES

- Decontamination: Reduce secondary exposure
- Blood agents:
 - High flow 100% NRB oxygen
 - Benzodiazepines for seizures
 - Hydroxocobalamin (1st line)
 - Cyanide antidote kit (2nd line), may be repeated.
- Blister agents:
 - Supportive care
 - Standard burn management
 - Atropine to relieve eye pain
 - Monitor fluids, electrolytes, complete blood chemistry.
 - Monitor CBC for nadir.
 - Supportive care for sepsis, anemia, hemorrhage
 - Granulocyte colony-stimulating factor (G-CSF) for neutropenia
- Choking agents, lacrimators, riot control agents:
 - Supportive care, bronchodilators
 - Eye irrigation
 - CXR and careful monitoring for respiratory complications
 - Phosgenes require monitoring for delayed pulmonary edema for 24 hr
- Nerve agents:
 - Supportive care:
 - 100% oxygen
 - Frequent airway suctioning
 - Atropine 2 mg IV q5min until reversal of bronchorrhea, bronchoconstriction, and hypoxemia:
 - Antagonizes muscarinic effects and some CNS but no effect on skeletal muscle weakness or respiratory failure
 - Pupillary response and heart rate are not useful measures of adequate atropinization.
 - Stop atropine after patient regains consciousness and spontaneous ventilation (may need for periodic relapses); give as much as it takes to reverse respiratory compromise.
 - Pralidoxime chloride (2-PAM or Protopam):
 - Regenerates cholinesterase by reversing phosphorylation (unless

- aging has occurred)
- Reduces abnormal skeletal muscle movements, improves skeletal muscle weakness, and reverses flaccid paralysis
- May repeat 1st dose or start on continuous infusion
- If improvement from 1st dose, repeat 60–90 min later.
- Diazepam: Administer for seizures.
- Incapacitating agents (BZ):
 - Supportive care
 - Aggressive IV fluid hydration
 - Benzodiazepines for agitation and increased muscular activity
 - Consider physostigmine in consultation with a poison center.

MEDICATION

- Albuterol using nebulizer: 2.5 mg in 2.5 mL NS (peds: 0.1–0.15 mg/kg/dose)
- Atropine: 2 mg IM or IV (5–6 mg in severely poisoned adults; peds: 0.02–0.08 mg/kg), then q5–10min titrate to clinical effect
- Cyanide antidote kit:
 - Inhale amyl nitrite ampule for 30 sec qmin until sodium nitrite given.
 - Sodium nitrite: 10 mL of 3% solution or 300 mg IV over 3–5 min (peds: 0.15–0.33 mL/kg):
 - Monitor methemoglobin levels to keep < 30%.
 - Sodium thiosulfate: 50 mL IV of 25% solution or 12.5 g (peds: 1.65 mL/kg)
- Diazepam: 5–10 mg IV over 3–5 min (peds: 0.2–0.4 mg/kg up to 10 mg over 2–3 min)
- Hydroxocobalamin: 5 g IV
- Pralidoxime chloride (2-PAM, Protopam): 1–2 g IV over 20–30 min or 600 mg IM (diluted with water or saline to concentration of 300 mg/mL) given with 1st 3 atropine doses (peds: 25–50 mg/kg/dose IV), repeat in 2 hr if muscle weakness has not been relieved, and in 4–6-hr intervals if necessary. Continuous infusion of 500 mg/h has been used for organophosphate poisoning



FOLLOW-UP

DISPOSITION

Admission Criteria

- ICU admission for symptomatic patients with significant exposure
- Hospital admission to monitor for developing complications for blister, choking, lacrimating agents, incapacitating agents

Discharge Criteria

Riot control exposures:

- Observe in ED for 6 hr and discharge if symptoms resolve.

PEARLS AND PITFALLS

Must perform adequate decontamination

ADDITIONAL READING

- Davis K, Aspera G. Exposure to liquid sulfur mustard. *Ann Emerg Med.* 2001;37:653–656.
- Keyes DC. Chemical warfare agents. In: Dart RC, Caravati EM, McGuigan MA, et al., eds. *Medical Toxicology*. 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2004:1777–1794.



CODES

ICD9

- 987.5 Toxic effect of lacrimogenic gas
- 987.7 Toxic effect of hydrocyanic acid gas
- 987.9 Toxic effect of unspecified gas, fume, or vapor

ICD10

- T57.3X4A Toxic effect of hydrogen cyanide, undetermined, init encntr
- T59.3X4A Toxic effect of lacrimogenic gas, undetermined, initial encounter
- T59.94XA Toxic effect of unsp gases, fumes and vapors, undet, init

CHEST PAIN

Josh W. Joseph • Edward Ullman



BASICS

DESCRIPTION

- One of the most frequent chief complaints in the ED
- Often the presenting symptom of a high-risk etiology:
 - Acute coronary syndrome
 - Pulmonary embolism
 - Aortic dissection
- Assume life threatening until proven otherwise.
- Categorization may suggest the underlying etiology, but the presentation of chest pain can be extremely variable and vague.
- Thoracic pain:
 - May involve the myocardium, pericardium, the ascending aorta, pulmonary artery, mediastinum, and esophagus
 - Pain is deep, visceral, and poorly localized.
 - Characteristics vary from severe and crushing to mild, burning, or indigestion.
- Epigastric pain:
 - May involve the descending aorta, diaphragmatic muscles, gallbladder, pancreas, duodenum, and stomach
 - Pain is generally referred to the xiphoid region and in the back.
- Pleuritic pain:
 - Inflammation or trauma to the ribs, cartilage, muscles, nerves, pleural or pericardial surface
 - Pain increased by breathing, laughing, coughing, sneezing
 - Tenderness to palpation may be present.
 - Diaphragmatic pleurisy:
 - Sharp shooting pains in the epigastrium, lower retrosternal area, or shoulder intensified by thoracic movement
- Chest wall pain:
 - Inflammation of skin and SC structures of the chest wall
 - Pain is reproduced by:
 - Palpation
 - Horizontal flexion of the arms
 - Extension of the neck
 - Vertical pressure on the head

ETIOLOGY

- Thoracic:
 - Acute coronary syndrome
 - Pericarditis
 - Myocarditis
 - Stress-induced cardiomyopathy
 - Cardiac syndrome X
 - Stimulant use
 - Thoracic aortic dissection
 - Esophagitis
 - Esophageal spasm
 - GERD
 - Esophageal hyperalgesia
 - Abnormal motility patterns and achalasia
 - Esophageal rupture and mediastinitis
- Epigastric:
 - Dissection of the descending aorta
 - Peptic ulcer disease
 - Pancreatitis
 - Cholecystitis
 - Splenic rupture
 - Hepatic injury
 - Subdiaphragmatic abscess
- Pleuritic pain:
 - Pulmonary embolism
 - Pneumothorax
 - Pneumonia
 - Costochondritis
- Diaphragmatic pleurisy:
 - Splenic rupture
 - Hepatic injury
 - Subdiaphragmatic abscess
- Esophageal rupture
- Intercostal myositis
- Intercostal neuralgia
- Pectoralis minor strain
- Pericarditis
- Pleuritis
- Pneumonitis
- Rib fractures
- Acute chest syndrome of sickle cell
- Chest wall twinge syndrome:
 - Brief episodes of sharp anterior chest pain lasting 30 sec–3 min, aggravated by deep breathing and relieved by shallow respirations

- Chest wall pain:
 - Chest wall hematoma
 - Chest wall laceration
 - Herpes zoster
 - Thrombophlebitis of the thoracoepigastric vein
 - Xiphisternal arthritis
 - Adiposis dolorosa
 - Breast abscess, fibroadenosis, carcinoma

DIAGNOSIS

SIGNS AND SYMPTOMS

- Coronary artery disease:
 - Pressure
 - Squeezing pain
 - Radiation to arm, jaw
 - Shortness of breath
 - Diaphoresis
 - Nausea
 - Vomiting
 - Weakness
 - Fatigue especially in women or elderly
 - Signs of CHF
 - Anxiety
- Aortic dissection:
 - Sudden onset of pain with maximal intensity early
 - Tearing pain
 - Radiation to back and/or flank
 - HTN
 - Diastolic murmur of aortic insufficiency
 - Difference in upper-extremity pulses
 - Syncope
 - Nausea
 - Vomiting
 - Associated neurologic changes (i.e., visual changes)
- Pulmonary embolism:
 - Pleuritic pain
 - Shortness of breath
 - Anxiety
 - Diaphoresis
 - Tachypnea
 - Tachycardia

- Low-grade fever
- Syncope
- Localized rales
- Wheezing
- Acute pericarditis:
 - Substernal pain
 - Varies with respiration
 - Increased with recumbency
 - Relieved by leaning forward
 - Anxiety
 - Anorexia
 - Fever
 - Pericardial friction rub
- Pneumothorax:
 - Pleuritic pain
 - Shortness of breath
 - Anxiety
 - Tachypnea
 - Decreased unilateral breath sounds
 - Can be spontaneous (young), or associated with very minor trauma (elderly)

History

- The history is the most important tool to distinguish between the various etiologies.
- Have the patient define the key features:
 - Duration
 - Location:
 - Retrosternal
 - Subxiphoid
 - Diffuse
 - Frequency:
 - Constant
 - Intermittent
 - Sudden vs. delayed onset
 - Precipitating factors:
 - Exertion
 - Stress
 - Food
 - Respiration
 - Movement
 - Timing:
 - Context of onset of pain (i.e., at rest, exertional)

- Duration of pain
- Quality:
 - Burning
 - Squeezing
 - Dull
 - Sharp
 - Tearing
 - Heavy
- Associated symptoms:
 - Shortness of breath
 - Diaphoresis
 - Nausea
 - Vomiting
 - Jaw pain
 - Back pain
 - Radiation
 - Palpitations
 - Syncope
 - Fever
 - Weakness: Generalized vs. focal
 - Fatigue

Physical-Exam

- Cardiac exam for murmurs, rub, decreased heart sounds, or extra heart sounds
- Chest exam for decreased breath sounds, rales, wheezing
- Extremity exam for decreased pulses, pulsus paradoxus
- Skin exam for lesions of herpes zoster
- Abdominal exam for tenderness, rebound, guarding

DIAGNOSIS TESTS & INTERPRETATION

EKG:

- Inexpensive and available
- Obtain and interpret within 10 min of arrival
- Serial EKG can be useful in patients with high concern for ACS and a negative initial EKG.
- See specific etiologies.

Lab

- Lab testing should be individualized to the patient and the presentation, based on the risk of potential life threats.
- See “Cardiac Testing.”
- D-Dimer:

- Sensitive but poor specificity for physical exam
- Indicated for low-risk patient if there is an indication to rule out pulmonary embolus
- Controversial use as a screening test for aortic dissection

Imaging

- CXR:
 - Pneumothorax
 - Pneumonia
 - CHF
 - Aortic dissection:
 - Widened mediastinum seen in ~ 55–62% of patients
 - A pleural effusion is found in ~ 20% of patients.
 - Apical capping
 - Aortic knob obliteration
 - A normal chest radiograph is found in 12–15% of patients.
 - Acute pericarditis:
 - Usually normal unless massive effusion enlarges cardiac silhouette
 - Esophageal rupture:
 - Usually will show mediastinal air
 - May have left pleural effusion
- Helical CT scan:
 - Pulmonary embolism
 - Sensitive for aortic dissection
- Ventilation/perfusion scan:
 - Useful in pulmonary embolus
 - Must have normal CXR
- Angiography:
 - Pulmonary embolism; although rarely done
 - Useful in dissection, especially in stable patients
- US:
 - Test of choice for pericardial and valvular disease
 - Transesophageal Echo can be used in diagnosis of aortic dissection, especially in unstable patients and those unable to tolerate contrast.
 - Right ventricular dilation and hypokinesia is suggestive for pulmonary embolus and can be used to guide therapy
 - Bedside transthoracic Echo can be used to quickly discover significant pericardial effusion, pneumothorax, and pleural effusion

DIFFERENTIAL DIAGNOSIS

See “Etiology.”



TREATMENT

PRE HOSPITAL

- Therapeutic interventions should be guided by the patient's presentation, risk factors, and past history.
- If a cardiac life threat is suspected:
 - IV access
 - Cardiac monitoring
 - EKG
 - Oxygen
 - Baby aspirin/Full aspirin
 - Pain control:
 - Nitrates
 - Morphine

INITIAL STABILIZATION/THERAPY

As guided by the patient's presentation:

- ABCs
- IV
- Oxygen
- Cardiac monitoring

ED TREATMENT/PROCEDURES

- IV, oxygen, and monitoring
- EKG
- Treatment varies based on suspected etiologies.

MEDICATION

Dependant on etiology



FOLLOW-UP

DISPOSITION

Admission Criteria

Dependent on the risk for life-threatening cardiopulmonary etiologies

Discharge Criteria

Safe if patient is deemed to have low-risk etiology of chest pain

Issues for Referral

Follow-up with primary care physician on low-risk chest pain for outpatient assessment

FOLLOW-UP RECOMMENDATIONS

Patient should be instructed to return if:

- Chest discomfort lasts > 5 min
- Chest discomfort gets worse in any way
- History of angina, and discomfort not relieved by usual medicines
- Shortness of breath, sweats, dizziness, vomiting, or nausea with chest pain or chest discomfort
- Chest discomfort moves into your arm, neck, back, jaw, or stomach

PEARLS AND PITFALLS

- Caution in only ordering a single biomarker
- Using response to medications as a diagnostic tool
- Not using serial EKG in patients with suspected ACS or repeating EKGs when patients have recurrent chest pain

ADDITIONAL READING

- Anderson JL, Adams CD, Antman EM, et al. 2011 ACCF/AHA Focused Update Incorporated Into the ACC/AHA 2007 Guidelines for the Management of Patients With Unstable Angina/Non–ST-Elevation Myocardial Infarction A Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. *Circulation*. 2011;123(18): e426–e579.
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CODES

ICD9

- 786.50 Chest pain, unspecified
- 786.51 Precordial pain
- 786.59 Other chest pain

ICD10

- R07.2 Precordial pain
- R07.9 Chest pain, unspecified
- R07.89 Other chest pain

CHEST TRAUMA, BLUNT

Lisa G. Lowe Hiller



BASICS

DESCRIPTION

- Significant source of morbidity and mortality in US
- ~12 thoracic trauma victims per million population per day
- ~33% of these injuries require hospital admission.
- Directly responsible for 20–25% of all deaths attributed to trauma
- Contributing cause of death in 25% of patients who die from other traumatic injuries

ETIOLOGY

- Common mechanisms of injury include:
 - Motor vehicle collisions (70–80%)
 - Motorcycle collisions
 - Pedestrians struck by a motor vehicle
 - Falls from great heights
 - Assaults
 - Blast injuries
 - Sports-related injuries
- Injuries can result from direct blunt force to the chest or from forces related to rapid deceleration.



DIAGNOSIS

SIGNS AND SYMPTOMS

- Obvious contusion, wound, or other defect of the chest wall
- Paradoxical chest wall movement suggests flail chest segment.
- Usually occurs in combination with other injuries
- Hypotension
- Some patients with severe intrathoracic injuries, such as traumatic aortic disruption, may have *no* visible external signs of trauma.

History

- Time of injury
- Mechanism of injury
- Estimates of motor vehicle accident (MVA) velocity and deceleration
- Loss of consciousness
- Chest pain

- Pain with deep inspiration or cough
- Dyspnea

Physical-Exam

- Unilaterally absent breath sounds
- Crepitus or subcutaneous air in the chest wall
- Decreased or absent breath sounds
- Tenderness to palpation on the chest wall
- Jugular venous distention
- Tracheal deviation away from midline
- Hyper-resonance to percussion on involved side

ESSENTIAL WORKUP

- Check airway, breathing, and circulation (ABCs) to determine the patient's stability
- Focused exam of the chest:
 - Respiratory effort and rate
 - Chest wall excursion
 - Crepitus
 - Subcutaneous air
 - Breath sounds and heart sounds
 - Presence of jugular venous distention
- Obtain a supine CXR immediately:
 - Avoid an upright CXR because of potential for other injuries (especially spinal injuries)
- ECG and monitor to detect myocardial ischemia or dysrhythmias
- Consider use of US for detecting small pneumothoraces, especially given the poor sensitivity of supine CXR in detecting such injuries.

DIAGNOSIS TESTS & NTERPRETATION

Lab

- Baseline hemoglobin
- Pulse oximetry
- ABG
- Serum lactate
- Type and cross-match
- Coagulation profile
- Cardiac enzymes when indicated
- Periodic chemistry panel for patients receiving significant fluid resuscitation

Imaging

- CXR is the initial radiologic study of choice:

- If CXR reveals widened mediastinum and patient hemodynamically stable, repeat film in upright position.
- Chest CT is more specific for pneumothoraces and pulmonary contusions/occult injuries.
- Thoracic US can be efficiently used for detecting pneumothoraces and pericardial injuries. The sensitivity, specificity, and overall accuracy in the ED setting for such injuries is >90%.
- Chest CT with contrast, or aortic angiogram, is useful in identifying aortic and large-vessel injuries.
- Esophagoscopy for direct endoscopic visualization if esophageal injury suspected
- Contrast esophagogram (with water and then barium) for possible esophageal injuries if esophagoscopy negative, but patient at risk for esophageal injury (e.g., pneumomediastinum)
 - Combination of these 2 tests in sequence (each 80–90% sensitive individually) reaches close to 100% sensitivity
- ECG if sternal tenderness is present or abnormalities on cardiac monitor

Diagnostic Procedures/Surgery

- If patient's condition is unstable, emergency thoracotomy may be necessary to repair a traumatic aortic disruption.
- If there are signs of cardiac tamponade, and patient is stable, perform an echocardiogram urgently:
 - Pericardial effusions, wall motion defects, aortic injuries, valvular or other intracardiac pathology may also be identified.
- If there are signs of cardiac tamponade and the patient is unstable, consider emergent pericardiocentesis, followed by immediate transport to the OR for a pericardial window.
- Bronchoscopy often indicated for possible upper airway injuries (e.g., a large persistent air leak after chest tube)

Pregnancy Considerations

- In pregnant patients, remember to use the least amount of radiation available and to shield the uterus during imaging when possible.
- Take note of the differences in anatomy of the thoracic cavity in pregnant patients, as well as differences in lab values, intravascular volume, and cardiovascular physiology.
- See “Pregnancy,” “Trauma in,” for details.

DIFFERENTIAL DIAGNOSIS

- Simple pneumothorax
- Tension pneumothorax
- Open pneumothorax
- Hemothorax

- Rib fractures
- Flail chest
- Sternoclavicular fractures/dislocations
- Pulmonary contusion
- Myocardial contusion
- Myocardial rupture
- Cardiac (pericardial) tamponade
- Traumatic aortic disruption
- Esophageal injury
- Large vascular injury (subclavian artery, pulmonary artery)
- Tracheobronchial injury
- Diaphragmatic injury

Pediatric Considerations

The rib cage is highly elastic in children and can withstand significant forces without overt signs of external trauma and can underestimate even major intrathoracic injuries.

Geriatric Considerations

Elderly patients have been shown to have greater respiratory complications, including ARDS and pneumonia, than younger patients in the setting of blunt chest trauma. This is especially true in those >85 yr of age.



TREATMENT

PRE HOSPITAL

- All patients with any signs of life in the field should be transported to a trauma center.
- Full spinal precautions should be employed.
- Needle decompression is indicated for tension pneumothorax:
 - Unilaterally absent breath sounds
 - Hypotension
 - Jugular venous distention
 - Hyper-resonance to percussion
- If large, open pneumothorax exists, tape an occlusive dressing on 3 sides only to prevent causing a tension pneumothorax.
- Do not delay transport to hospital for IV access.

INITIAL STABILIZATION/THERAPY

- ABCs management; intubate patient early if signs of respiratory insufficiency, shock, or altered mental status exist.
- Resuscitation attempts should be initiated only in patients who arrive in the ED with vital signs.

- Any patient who presents in blunt traumatic arrest is not likely to survive a thoracotomy in the ED, and it is therefore generally not indicated in this group.
- If the patient's condition is unstable and clinically shows signs of a tension pneumothorax, perform needle thoracostomy and place a chest tube immediately afterward.
- Do not wait to obtain a CXR.
- Place chest tube on the affected side or bilaterally if injury site is unclear.
- Deliver oxygen by nonrebreather face mask for stable patients.
- Obtain vascular access, preferably 2 large-bore IV lines (> 18G).
- Maintain spinal immobilization.

ED TREATMENT/PROCEDURES

- Tube thoracostomy if pneumothorax or hemothorax is identified:
 - 36F chest tube in an adult
 - In children, use the largest tube that the intercostal space will accommodate.
- Provide resuscitation with isotonic fluids and blood products as necessary:
 - Aggressive fluid resuscitation may be harmful if severe pulmonary contusions exist (consider permissive hypotension).
- Workup for associated intra-abdominal injuries (e.g., with abdominal US, abdominal CT scan, less commonly diagnostic peritoneal lavage):
 - Patients with chest trauma frequently have concomitant intra-abdominal injuries.

MEDICATION

- Tetanus booster if indicated
- Consider methylprednisolone (for signs of spinal cord injury): 30 mg/kg IVI over 1 hr, followed by a continuous drip of 5.4 mg/kg/h for next 23 hr

ALERT

- This practice is under debate and becoming less utilized, so know your hospital's protocol.
- Judicious doses of short-acting analgesics (fentanyl 1–2 µg/kg IV, morphine 0.1 mg/kg IV) as needed for pain control.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients with conduction blocks, frequent ectopy, or ischemic changes visible on EKG should be admitted to a monitored setting for possible myocardial contusion.

- Hemodynamically unstable patients should go to the OR on an emergency basis for thoracotomy or laparotomy.
- >1,000–1,500 mL of blood drawn out of the chest tube on initial insertion indicates need for thoracotomy/operative management.
- >200 mL of blood per hour from chest tube for several hours suggests the need for operative intervention.
- Patients with significant rib fractures should be admitted for pain control:
 - Consider epidural catheter for analgesia.
- Patients who lose vital signs in the ED should undergo rapid open thoracotomy.

Discharge Criteria

Patients with clinically insignificant chest wall contusions and an initial negative upright CXR may be observed for 6 hr in the ED and often be discharged if a repeat radiograph at that time reveals no pneumothorax, hemothorax, or pulmonary contusion, the patient is able to breathe deeply and to cough, remains clinically stable, and has no other significant injuries.

Issues for Referral

- Notify trauma surgeon promptly about patients with significant injuries requiring surgical intervention or admission.
- Indications for emergent surgical referral:
 - Traumatic thoracotomy with loss of chest wall integrity
 - Blunt diaphragmatic injuries
 - Massive air leak following chest tube insertion
 - Massive hemothorax or continued high rate of blood loss via the chest tube (i.e., 1,500 mL on insertion of tube or continued loss of 200–300 mL/hr)
 - Radiographically or endoscopically confirmed tracheal, major bronchial, or esophageal injury
 - GI tract contents recovered on chest tube placement
 - Cardiac tamponade
 - Radiographic confirmation of a great-vessel injury
 - Embolism or missile into pulmonary artery, great vessel, or heart

FOLLOW-UP RECOMMENDATIONS

- Patients should be closely followed by trauma or cardiothoracic surgeons after hospital discharge, as indicated, depending upon the injuries discovered and treatment rendered.
- Patients with thoracostomy tubes should have a CXR and routine wound care follow-up within 48 hr to remove the dressing and reassess clinical status.

PEARLS AND PITFALLS

- Blunt chest trauma is responsible for up to 1/4 of all trauma-related deaths.

- Trauma patients arriving at a nontrauma center should be stabilized and transferred to facilities that can provide definitive care as soon as possible.
- Open thoracotomy in the ED has not been shown to improve survival in patients found to be in cardiopulmonary arrest after blunt trauma and is generally only indicated if the patient arrives in the ED with vital signs present.
- The extent of injury is not always clinically obvious upon initial presentation. This is particularly true in pediatric patients.

ADDITIONAL READING

- American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular. *Circulation*. 2010;122(18 suppl 3)
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- Karmy-Jones R, Jurkovich GJ. Blunt chest trauma. *Curr Probl Surg*. 2004;41(3):211–380.
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CODES

ICD9

- 807.4 Flail chest
- 922.1 Contusion of chest wall
- 959.11 Other injury of chest wall

ICD10

- S20.20XA Contusion of thorax, unspecified, initial encounter
- S22.5XXA Flail chest, initial encounter for closed fracture
- S29.9XXA Unspecified injury of thorax, initial encounter

CHEST TRAUMA, PENETRATING

Jean C.Y. Lo



BASICS

ETIOLOGY

- Gunshot wounds or stab wounds most common
- Impalement on a sharp object from a fall can occur.



DIAGNOSIS

SIGNS AND SYMPTOMS

- Object impaled in the chest wall
- Obvious wound in the chest wall with or without bleeding
- Chest pain
- Dyspnea
- Respiratory distress
- Altered mental status from hypoxemia
- Absent or altered breath sounds on 1 or both sides
- Hypotension
- Jugular venous distention

ESSENTIAL WORKUP

- Perform routine assessment of airway, breathing, and circulation.
- Rapid exam:
 - Respiratory effort and rate
 - Chest excursion
 - Crepitus
 - Subcutaneous air
 - Breath sounds and heart sounds
- Upright CXR is preferred for identifying a pneumothorax:
 - Supine CXR should be taken 1st if spinal precautions must be maintained.
- Baseline hemoglobin
- Pulse oximetry
- ABG
- Serum lactate
- Type and screen

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Perform echocardiogram if signs of tamponade present or if wound is close to the heart:
 - In stab wound to precordium and pericardial sac, hemopericardium may decompress into hemothorax, thus not apparent on initial echo:
 - Repeat pericardial US is recommended after tube thoracostomy decompression of the hemothorax.
 - Residual hemothorax represents pericardial injury or cardiac laceration.
- ECG

Imaging

- With gunshot wounds, other areas (abdomen, pelvis) should be imaged:
 - Total number of wounds and bullets must be the same.
- Arteriogram of aortic arch, carotid arteries, or subclavian artery if great vessel injury is suspected
- Esophageal Gastrografin swallow or endoscopy to identify esophageal perforation
- Bronchoscopy to identify tracheobronchial injuries

DIFFERENTIAL DIAGNOSIS

- Simple pneumothorax
- Tension pneumothorax
- Open pneumothorax
- Hemothorax
- Rib fractures
- Flail chest
- Pulmonary contusion
- Myocardial contusion
- Myocardial rupture
- Pericardial tamponade
- Traumatic aortic disruption
- Esophageal injury
- Large vessel injury
- Tracheobronchial injury
- Diaphragmatic injury
- Intra-abdominal injury
- Spinal cord injury



TREATMENT

PRE HOSPITAL

- Cautions:
 - All patients with signs of life in the field according to reports from EMS

- personnel should be transported to a trauma center.
- Full spinal immobilization if spinal injury suspected
- Never remove objects impaled in the chest because exsanguination may follow.
- Needle decompression may be necessary if tension pneumothorax suspected:
 - Unilaterally absent breath sounds, hypotension, jugular venous distention
- If large open pneumothorax exists, occlusive dressing taped on 3 sides:
 - A totally occlusive dressing may produce a tension pneumothorax.
- Controversies:
 - Do not delay transport to hospital to obtain IV access:
 - IV access may be established en route.
 - Do not delay transport to hospital by applying full spinal immobilization to patients who do not have clear clinical signs of spinal injury.

INITIAL STABILIZATION/THERAPY

- Airway, breathing, and circulation management:
 - Intubate for signs of serious chest injury, obvious respiratory distress, or hypotension.
- Oxygen by nonrebreather face mask for patients in stable condition
- Obtain vascular access, 2 peripheral large-bore IV lines (> 18G), and fluid resuscitation as needed:
 - Restrictive fluid resuscitation is associated with shorter hospital length of stay and lower overall mortality.
 - In penetrating aortic trauma, permissive hypotension at systolic BP 90 mm Hg until definitive surgical control prevents further hemorrhage.
- For tension pneumothorax, perform a needle thoracostomy and place a chest tube immediately.
- Do not wait to get a CXR.
- Sonogram has demonstrated higher sensitivity than CXR in diagnosing pneumothorax.
- For pericardial tamponade, perform an emergency pericardiocentesis:
 - Follow by rapid transport to the operating room for a pericardial window
- Maintain spinal immobilization if indicated.

ED TREATMENT/PROCEDURES

- Notify trauma surgeon about patient's arrival.
- Tube thoracostomy if a pneumothorax or hemothorax is identified:
 - 36G chest tube in an adult
 - In children, use largest tube the intercostal space will accommodate.
- Fluid resuscitation as necessary:
 - Contused lung parenchyma will have leaky capillary beds, and aggressive crystalloid resuscitation may aggravate pulmonary dysfunction.

- Any wound with an entry or exit site below the nipple or the posterior tip of the scapula is concerning for an intra-abdominal injury:
 - Workup with a diagnostic peritoneal lavage (DPL), US, CT scan, exploratory laparotomy, or laparoscopy
 - DPL positive with 5,000 RBC
- Describe the nature of wounds accurately:
 - Retain any bullet fragments, clothes, or tissue removed from the wound.
- Probing a chest wound is contraindicated because it can create a pneumothorax or worsen hemorrhage.
- Impaled objects should be removed only in the operating room.
- Tetanus booster if indicated

MEDICATION

- Methylprednisolone (for spinal cord injury): 30 mg/kg IV over 1 hr, followed by a continuous drip of 5.4 mg/kg/h for 23 hr
- Small doses of short-acting analgesics (fentanyl, 1–2 µg/kg IV, morphine 0.1 mg/kg IV) or sedatives (midazolam, 0.05 mg/kg IV) as needed for pain control and sedation
- Treat with IV antibiotics if wound grossly contaminated (e.g., cephalexin 1 g IV).



FOLLOW-UP

DISPOSITION

Admission Criteria

- All patients with penetrating chest trauma should be admitted.
- In penetrating torso trauma, resuscitative thoracotomy in the ED demonstrates survival when pre-hospital CPR does not exceed 15 min.
- A patient who has signs of life in the field but no BP on arrival in the ED should have an emergency thoracotomy performed by the most experienced person present:
 - If the source of bleeding is controlled and there are signs of cardiac activity, the patient should go to the operating room for formal operative repair.
- Hemodynamically unstable patients should go immediately to the operating room.
- Any patient with intrathoracic penetration should have a chest tube placed and should be admitted to a monitored setting.
- > 1,000–1,500 mL of blood drawn out of the chest tube on initial insertion indicates the need for thoracotomy.
- > 200 mL/hr of blood from a chest tube for several hours suggests the need for surgical intervention.
- Patients with large, persistent air leaks usually require surgery.
- Patients with significant rib fractures should be admitted and have an epidural

catheter placed for pain control and pulmonary toilet.

Discharge Criteria

Patients with isolated minor chest wounds and a normal CXR can be observed for 3 hr in the ED and have a repeat radiographic study; if no intrathoracic penetration is suspected, the patient can be discharged:

- CT chest may be an alternative to CXR, if no intrathoracic penetration is suspected; patient can be discharged without repeat radiograph.

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CODES

ICD9

- 862.9 Injury to multiple and unspecified intrathoracic organs, with open wound into cavity
- 875.0 Open wound of chest (wall), without mention of complication
- 875.1 Open wound of chest (wall), complicated

ICD10

- S21.90XA Unsp open wound of unspecified part of thorax, init encntr
- S21.93XA Puncture wound w/o foreign body of unsp part of thorax, init
- S21.94XA Puncture wound w foreign body of unsp part of thorax, init

CHOLANGITIS

Robert G. Buckley



BASICS

DESCRIPTION

- Partial or complete obstruction of the common bile duct owing to gallstones, tumor, cyst, or stricture
- Increased intraluminal pressure in biliary tree
- Bacterial multiplication results in bacteremia and sepsis.
- Purulent infection of biliary tree, which may involve the liver and gallbladder
- Mirizzi syndrome is defined as common bile duct obstruction owing to extrinsic compression from gallbladder or cystic duct edema or stones.

ETIOLOGY

- Bacterial sources of infection include:
 - Ascending duodenal source
 - Gallbladder infection
 - Portal venous seeding
 - Hematogenous spread with hepatic secretion
 - Lymphatic spread
- Bacterial organisms include:
 - Anaerobes (*Bacteroides* and *Clostridium* species)
 - Intestinal coliform (*Escherichia coli*)
 - Enterococcus
- AIDS sclerosing cholangitis characterized by:
 - Papillary stenosis
 - Sclerosing cholangitis
 - Extrahepatic biliary obstruction
 - Cytomegalovirus (CMV), *Cryptosporidium*, and microsporidia isolated, but causal role not established



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Charcot triad:
 - Classic presentation of fever and chills; right upper quadrant (RUQ) pain and jaundice found in only 50–70%
- Addition of shock and altered mental status denotes a more advanced form of

biliary sepsis known as *Reynolds pentad*.

- Abdominal pain present in >70%—localizing to RUQ.
- AIDS sclerosing cholangitis presents with similar symptoms but with more chronic indolent course and near-normal serum bilirubin levels.

Physical-Exam

- Fever found in >90%
- Peritoneal findings found in 30%
- Clinically apparent jaundice may be absent in up to 40%.

ESSENTIAL WORKUP

- ECG in patients at risk for coronary artery disease
- CBC
- LFT
- Amylase, lipase
- Urinalysis
- Blood cultures
- Gallbladder US or hepatiminodiacetic acid (HIDA) scan

DIAGNOSIS TESTS & NTERPRETATION

Lab

- CBC:
 - Leukocytosis with left shift unless immunocompromised or severe sepsis
- LFTs consistent with cholestasis:
 - Elevated direct bilirubin and alkaline phosphatase
- Minimal elevation of transaminases (<200 IU/mL)
- Changes may lag symptom onset by 24–48 hr.
- Amylase and lipase normal or mildly elevated
- Urinalysis positive for bilirubin

Imaging

- US detects the level of ductal obstruction and the presence of gallstone etiology.
- Radionuclide scanning (HIDA):
 - Indicates obstruction when tracer not found in duodenum within 1 hr
 - More sensitive than US in detecting obstruction in the 1st 24–48 hr before ductal dilation occurs
- CT scan and CRX:
 - Useful to rule out intestinal obstruction, perforation, or pneumonia
 - 20% gallstones radiopaque
- Magnetic resonance cholangiopancreatography (MRCP) is highly accurate for biliary obstruction but unnecessary if endoscopic retrograde cholangiopancreatography (ERCP) will be performed.

Diagnostic Procedures/Surgery

Emergency invasive biliary imaging and drainage by ERCP (or surgical/percutaneous if not available), if no response to medical treatment in 12–24 hr

DIFFERENTIAL DIAGNOSIS

- Acute cholecystitis
- Hepatitis or hepatic abscess
- Acute pancreatitis
- Right pyelonephritis
- Right lower lobe pneumonia or pulmonary embolism
- Perforated duodenal ulcer
- Appendicitis
- Sepsis with nonspecific elevation of LFTs
- Fitz-Hugh and Curtis syndrome



TREATMENT

PRE HOSPITAL

Stabilize septic shock.

INITIAL STABILIZATION/THERAPY

- Immediate IV fluid resuscitation for dehydration, hemodynamic compromise, and sepsis
- 80% respond to IV antibiotics within 1st 24 hr
- Vasopressors (dopamine) for hypotension refractory to volume replacement

ED TREATMENT/PROCEDURES

- Broad-spectrum antibiotics for coliforms, anaerobes, and enterococcus such as:
 - Ampicillin/sulbactam + aminoglycoside (e.g., gentamicin)
 - Imipenem–cilastatin
 - Piperacillin/tazobactam + aminoglycoside (e.g., gentamicin)
 - For penicillin allergy:
 - Adults—use levofloxacin (Levaquin) and metronidazole
 - Pediatrics—use clindamycin and metronidazole
- Substitute aztreonam for aminoglycoside in renal insufficiency.
- NPO
- Nasogastric (NG) suctioning if protracted vomiting or ileus
- IV fluid (0.9% NS) replacement and maintenance
- Narcotic analgesia if hemodynamically stable and diagnosis reasonably established
- Immediate surgical and GI consultation
- Emergency invasive biliary drainage procedure (surgical, percutaneous, or ERCP) if no response to medical treatment in 12–24 hr

MEDICATION

- Ampicillin/sulbactam: 3 g (peds: 200 mg/kg/24 h) IV piggyback (IVPB) q6h
- Aztreonam: 2 g (peds: 120 mg/kg/24 h) IVPB q6h
- Clindamycin: 600–900 mg (peds: 25–40 mg/kg/24 h) IVPB q6–8h
- Dopamine: 2–20 µg/min IVPB; titrate to maintain BP
- Gentamicin: 1.5–2 mg/kg (peds: 6–7 mg/kg/24 h) IVPB q8h; follow levels
- Imipenem–cilastatin: 500 mg (Peds 60–100 mg/kg/24 h) q6h
- Levaquin: 500 mg IVPB q24h; contraindicated in peds
- Hydromorphone: 0.5–2 mg IV (0.01–0.02 mg/kg), titrated to pain relief.
- Metronidazole: 500 mg (peds: 30 mg/kg/24 h) IVPB q6h
- Piperacillin/tazobactam: 3.375 mg (peds: 300 mg/kg/24 h) IVPB q6h
- Ondansetron: 4–8 mg IV, (0.15–0.3 mg/kg) IV (not to exceed 8 mg/dose IV), q4h PRN vomiting



FOLLOW-UP

DISPOSITION

Admission Criteria

- All patients with acute cholangitis should be admitted with immediate surgical and gastroenterologic consultation.
- Admit patients with signs of septic shock to the ICU.

Discharge Criteria

None

Issues for Referral

Surgery/GI consultation

FOLLOW-UP RECOMMENDATIONS

Admission to hospital for IV antibiotic and possible biliary drainage procedure.

PEARLS AND PITFALLS

- Aggressively fluid resuscitate patients.
- Administer antibiotics.
- Obtain GI and surgical consultations.

ADDITIONAL READING

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- Yusuf TE, Baron TH, AIDS Cholangiopathy. *Curr Treat Options Gastroenterol*. 2004;7:111–117.



See Also (Topic, Algorithm, Electronic Media Element)

- Cholecystitis
- Cholelithiasis



CODES

ICD9

576.1 Cholangitis

ICD10

K83.0 Cholangitis

CHOLECYSTITIS

Robert G. Buckley

BASICS

DESCRIPTION

Cholecystitis is defined as inflammation of the gallbladder.

ETIOLOGY

- **Acute calculous cholecystitis:**
 - Owing to bile stasis secondary to prolonged obstruction by a gallstone (see “Cholelithiasis”) in the gallbladder neck, cystic duct, or common bile duct
 - Leads to increased intraluminal pressure and mucosal damage
 - Release of inflammatory mediators results in distention, edema, and increased vascularity.
 - Coliforms and anaerobes lead to infection—primary causal role is controversial.
- **Acalculous cholecystitis:**
 - 10% of cases
 - Underlying critical illness leads to biliary stasis and mucosal ischemia.
 - Subsequent mucosal inflammation and infection

Pediatric Considerations

- *Acute calculous cholecystitis* extremely rare in childhood (see “Cholelithiasis”)
- *Acalculous cholecystitis* more common than calculous form in children:
 - Associated with systemic bacterial infections, scarlet fever, Kawasaki disease, and parasitic infections

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- **Acute calculous cholecystitis:**
 - Dull, aching, epigastric, or right upper quadrant (RUQ) pain
 - Radiation to tip of right scapula, acromion, or thoracic spine
 - Duration > 6 hr more suggestive of cholecystitis than uncomplicated biliary colic
 - As inflammation progresses, parietal peritoneal irritation leads to sharp, localized pain.
 - Nausea, vomiting, fever, and chills often reported, but absent in most cases

- Jaundice in 20%
- History of prior attacks of biliary colic or known gallstones favors diagnosis.
- Acalculous cholecystitis:
 - Occurs in critically ill patients (burns, sepsis, trauma, or postoperative)
 - Localized pain and tenderness frequently absent
 - Often presents with symptoms of generalized sepsis of unknown source

Physical-Exam

- Localized parietal peritoneal signs:
 - Percussion tenderness
 - Rebound
 - Found as the disease progresses
- Murphy sign:
 - Inspiratory arrest with gentle palpation of RUQ owing to increased pain
 - Found in most cases

ESSENTIAL WORKUP

- ECG in patients at risk for coronary artery disease
- CBC
- LFT
- Amylase, lipase
- Urinalysis
- Human chorionic gonadotropin (hCG)
- Gallbladder US or HIDA scan

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC:
 - WBC $> 12,000$ cells/mm³ supports diagnosis, but may be normal in more than half of cases
- LFTs:
 - Transaminases, bilirubin, amylase, and lipase may be minimally elevated, but are generally normal.
 - Disproportionate elevation of direct bilirubin and alkaline phosphatase compared with transaminases suspicious for common duct obstruction or cholangitis

Imaging

- US:
 - Generally the 1st-line imaging procedure

- Positive findings include gallbladder wall thickening (>5 mm) or pericolic fluid—sensitivity, 90%; specificity, 80%.
- Optimal if patient NPO >8 hr
- Radionuclide scanning (HIDA):
 - Most useful when clinical suspicion remains high despite equivocal findings on US or when acalculous cholecystitis suspected
 - Positive when tracer seen in small bowel but inflamed gallbladder fails to visualize
 - Sensitivity, >95%; specificity, 90%
 - False-positive results increase in nonfasting state.
 - Addition of IV morphine induces Sphincter of Oddi contraction which improves gallbladder filling and reduces false-positive scan results.
- CT scanning:
 - Exclude intestinal perforation or obstruction
 - Air in the gallbladder wall consistent with emphysematous cholecystitis
 - Gallstones radiopaque in up to 20%

DIFFERENTIAL DIAGNOSIS

- Biliary colic
- Hepatitis or hepatic abscess
- Cholangitis
- AIDS sclerosing cholangitis
- Pancreatitis
- Intestinal perforation
- Peptic ulcer disease
- Gastritis
- Duodenal perforation
- Right lower lobe pneumonia, pleurisy, or pulmonary infarction
- MI
- Abdominal aortic aneurysm
- Appendicitis
- Fitz-Hugh and Curtis syndrome
- Pyelonephritis



TREATMENT

PRE HOSPITAL

Establish IV access for patients with vomiting or severe pain.

INITIAL STABILIZATION/THERAPY

- IV, oxygen, cardiac monitoring until myocardial ischemic cause excluded
- Initiate IV fluid therapy for dehydration, hemodynamic compromise, or sepsis.

ED TREATMENT/PROCEDURES

- Broad-spectrum antibiotics for coliforms, anaerobes, and enterococcus:
 - Ampicillin/sulbactam
 - Piperacillin/tazobactam
 - Add aminoglycoside if sepsis or cholangitis suspected (see “Cholangitis”).
- Alternative antibiotics for penicillin allergic:
 - Adults: Levofloxacin (Levaquin) and metronidazole
 - Peds: Clindamycin with aminoglycoside
- NPO
- IV fluid replacement and maintenance
- Antiemetics (ondansetron) if vomiting
- Nasogastric (NG) suctioning if refractory vomiting or ileus
- Narcotic analgesics (hydromorphone) with antiemetic (ondansetron):
 - Administer for refractory pain once diagnosis is reasonably established.
 - Morphine sulfate may lead to spasm at sphincter of Oddi (clinical significance not well established).
- Anticholinergics (glycopyrrolate) of no proven benefit for acute biliary pain.
- Surgical consultation

MEDICATION

- Ampicillin/sulbactam: 3 g (peds: 200 mg/kg/24h) IV piggyback (IVPB) q6h
- Clindamycin: 600–900 mg (peds: 25–40 mg/kg/24h) IVPB q6–q8h
- Gentamicin: 1.5–2 mg/kg (peds: 6–7 mg/kg/24h) IVPB q8h; follow levels
- Levaquin: 500 mg IVPB q24h; contraindicated in peds
- Hydromorphone: 0.5–2 mg IV (0.01–0.02 mg/kg), titrated to pain relief.
- Metronidazole: 500 mg (peds: 30 mg/kg/24h) IVPB q6h
- Piperacillin/tazobactam: 3.375 mg (peds: 300 mg/kg/24h) IVPB q6h
- Ondansetron: 4–8 mg IV (peds: 0.15–0.3 mg/kg) IV (not to exceed 8 mg/dose IV), q4h PRN vomiting



FOLLOW-UP

DISPOSITION

Admission Criteria

- All cases of cholecystitis should be admitted for parenteral antibiotics, analgesia, fluid replacement, and cholecystectomy in 24–72 hr.
- Unstable patients (gallbladder perforation or sepsis) require immediate surgery.

Discharge Criteria

None

Issues for Referral

General surgery consult for patients with cholecystitis. GI consult if choledocholithiasis or cholangitis suspected.

FOLLOW-UP RECOMMENDATIONS

Inpatient admission for antibiotics and surgical evaluation.

PEARLS AND PITFALLS

- US is the 1st-line imaging procedure.
- Perform a radionuclide scanning (HIDA) when clinical suspicion is high with equivocal US or when acalculous cholecystitis suspected.

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See Also (Topic, Algorithm, Electronic Media Element)

- Cholangitis
- Cholelithiasis



CODES

ICD9

- 574.00 Calculus of gallbladder with acute cholecystitis, without mention of obstruction
- 575.0 Acute cholecystitis
- 575.10 Cholecystitis, unspecified

ICD10

- K80.00 Calculus of gallbladder w acute cholecyst w/o obstruction
- K81.0 Acute cholecystitis

- K81.9 Cholecystitis, unspecified

CHOLELITHIASIS

Robert G. Buckley



BASICS

DESCRIPTION

- Symptoms arise when gallstones pass through the cystic or common bile ducts leading to impedance of normal bile flow and gallbladder spasm.
- Biliary dyskinesia produces symptoms identical to biliary colic in the absence of stones.
- Choledocholithiasis (common bile duct stones), may lead to prolonged pain, elevated LFTs and bilirubin, and to more complications like cholangitis or pancreatitis.

ETIOLOGY

- Cholesterol stones:
 - Most common type of gallstone
 - Form when solubility exceeded
- Pigment stones:
 - 20%
 - Composed of calcium bilirubinate
 - Associated with clinical conditions such as hemolytic anemias that lead to increased concentration of unconjugated bilirubin
- Incidence increases with age and favors females to males 2:1. Other risk factors include Hispanic ethnicity, obesity, pregnancy, rapid weight loss, and drugs that induce biliary stasis (e.g., ceftriaxone and oral contraceptives).
- Gallstones are exceedingly rare in childhood and are most commonly associated with sickle cell disease, hereditary spherocytosis, or other hemolytic anemias that result in pigment stone formation.
- Biliary sludge:
 - Nonstone, crystalline, granular matrix
 - Associated with rapid weight loss, pregnancy, ceftriaxone or octreotide therapy, and organ transplantation
 - May develop symptoms identical to cholelithiasis and its complications
- “Porcelain gallbladder” from mucosal precipitation of calcium salts owing to recurrent obstruction of cystic duct.



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Dull, aching epigastric or right upper quadrant (RUQ) pain:
 - Arising over 2–3 min, continuous (rather than colicky), and lasting from 30 min–6 hr before dissipating
 - May radiate to the tip of right scapula, acromion, or thoracic spine
 - Often correlated with ingestion of large, fatty meal
- Anorexia
- Nausea and vomiting
- Afebrile:
 - Fever and chills suggest cholecystitis or cholangitis

Physical-Exam

- Tenderness to deep palpation but without rebound
- Murphy sign (inspiratory arrest during deep palpation of the RUQ) may be present during the episode of colic, but should resolve when symptoms pass.

ESSENTIAL WORKUP

- Obtain ECG on those whose pain may be owing to myocardial ischemia.
- CBC
- LFTs
- Amylase, lipase
- Urinalysis
- Human chorionic gonadotropin (hCG)

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC:
 - WBC count usually normal, but may elevate after vomiting
 - Leukocytosis suggestive of cholecystitis or cholangitis
- LFTs:
 - Usually normal
 - Elevation suggests common duct obstruction, cholangitis, cholecystitis, or hepatitis.
- Amylase/lipase
 - Normal or minimally elevated with passage of gallstone
 - Elevation in context of severe persistent epigastric pain suggests pancreatitis.
- Urinalysis:
 - Exclude nephrolithiasis or pyelonephritis.
 - Bilirubinuria suggests common duct obstruction or hepatitis.

Imaging

- US:
 - Detects gallstones with sensitivity and specificity > 90%
 - Dilation of common bile duct > 10 mm indicates obstruction, but no dilation may be present with acute obstruction.
 - Gallbladder wall thickening > 5 mm or pericolic fluid 90% sensitive and 80% specific for cholecystitis
 - Accuracy enhanced in fasting patient (> 6 hr) with noncontracted gallbladder
- Radionuclide scanning (HIDA):
 - Cannot detect gallstones
 - Passage of tracer into small intestine without visualization of gallbladder highly diagnostic of cystic duct obstruction and cholecystitis:
 - Sensitivity and specificity roughly 95%
 - Failure of tracer to pass into duodenum suggests common bile duct obstruction. Accuracy enhanced by morphine injection during scan causing sphincter of Oddi spasm and improving gallbladder filling.
- CT scanning:
 - Less sensitive than US to detect gallstones:
 - Only 20% radiopaque.
 - Most useful to exclude other causes of upper abdominal pain such as aortic aneurysm, perihepatic abscess, or pancreatic pseudocyst
 - Detects rare complications such as air in gallbladder wall in emphysematous cholecystitis, air-filled gallbladder in biliary-enteric fistula or a “Porcelain gallbladder.”
- Plain radiographs:
 - Most useful for diagnosis of intestinal obstruction or rare abnormalities such as air in gallbladder wall in emphysematous cholecystitis, air-filled gallbladder in biliary-enteric fistula or a “Porcelain gallbladder.”

DIFFERENTIAL DIAGNOSIS

- MI
- Abdominal aortic aneurysm
- Acute cholecystitis, cholangitis, or choledocholithiasis
- Renal colic or pyelonephritis
- Duodenal ulcer perforation
- Acute pancreatitis
- Intestinal obstruction
- Peptic ulcer disease, gastritis, or GERD
- Right lower lobe pneumonia, pleurisy, or pulmonary infarction
- Hepatitis or hepatic abscess
- Fitz-Hugh and Curtis syndrome



TREATMENT

PRE HOSPITAL

Initiate IV access for patients with nausea or vomiting.

INITIAL STABILIZATION/THERAPY

IV fluid bolus if vomiting or hypotensive

ED TREATMENT/PROCEDURES

- IV hydration with 0.9% NS if vomiting
- NPO
- Parenteral NSAIDs (ketorolac) may lessen biliary spasm, but may exacerbate peptic causes of pain.
- Narcotic analgesics (hydromorphone) with antiemetic (ondansetron):
 - Administer for refractory pain once diagnosis is reasonably established.
 - Morphine sulfate may lead to spasm at sphincter of Oddi (clinical significance not well established).
- Anticholinergics (glycopyrrolate) have no proven benefit in the treatment of acute biliary pain.

MEDICATION

- Ketorolac: 60 mg IM or 30 mg (peds: Start 0.5 mg/kg for 1st dose up to 1 mg/kg/24h) IV q6h. In elderly: 30 mg IM or 15 mg IV
- Hydromorphone: 0.5–2 mg IV (0.01–0.02 mg/kg), titrated to pain relief.
- Ondansetron: 4–8 mg IV (0.15–0.3 mg/kg) IV (not to exceed 8 mg/dose IV), q4h PRN vomiting.



FOLLOW-UP

DISPOSITION

Admission Criteria

Admission and surgical or gastroenterologic consultation for evidence of:

- Acute cholecystitis
- Acute cholangitis
- Common duct obstruction
- Gallstone pancreatitis

Discharge Criteria

- Lack of clinical, lab, or radiographic evidence of cholecystitis, cholangitis, common duct obstruction, or pancreatitis
- Resolution of all pain and tenderness

- Ability to tolerate oral fluids

Issues for Referral

- General surgery referral for all cases of biliary colic with documented cholelithiasis or for radiographic finding of a “Porcelain gallbladder” (due to increased risk of gallbladder carcinoma).
- GI referral for choledocholithiasis.

FOLLOW-UP RECOMMENDATIONS

Surgical follow-up for patients with symptomatic gallstones

PEARLS AND PITFALLS

- Alternative causes of upper abdominal pain may be falsely attributed to incidental finding of gallstones.
- An ultrasound is more sensitive and specific for cholelithiasis.
- Radionuclide scanning (HIDA) is highly diagnostic of cystic duct obstruction and cholecystitis.
- CT scans may miss gallstones if the stones are not radiopaque.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Cholangitis
- Cholelithiasis



CODES

ICD9

- 574.20 Calculus of gallbladder without mention of cholecystitis, without mention of obstruction
- 574.21 Calculus of gallbladder without mention of cholecystitis, with obstruction

- 574.90 Calculus of gallbladder and bile duct without cholecystitis, without mention of obstruction

ICD10

- K80.20 Calculus of gallbladder w/o cholecystitis w/o obstruction
- K80.21 Calculus of gallbladder w/o cholecystitis with obstruction
- K80.70 Calculus of GB and bile duct w/o cholecyst w/o obstruction

CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Adam Z. Barkin

BASICS

DESCRIPTION

- 3rd leading cause of death in US
- A disease characterized by airflow obstruction due to several processes:
 - Emphysema: Irreversible alveolar destruction with loss of airway elastic recoil. Represents accelerated aging of the lung
 - Chronic bronchitis: Airway inflammation without alveolar destruction
 - Reactive airway disease: Reversible bronchospasm, mucous plugging, and mucosal edema
- COPD affects ~10% of the population and 50% of smokers.
- Increased incidence of hypertension, diabetes, heart failure, and cardiovascular disease in those with COPD
- Frequent exacerbations lead to:
 - Greater mortality
 - Faster decline in lung function
 - Worse quality of life
 - Increased risk of hospitalization
- Medical Research Council (mMRC) dyspnea scale
 - Grade 0: Only breathless with strenuous exercise
 - Grade 1: Short of breath when hurrying or walking up a slight hill
 - Grade 2: Walk slower than people of same age due to dyspnea or have to stop for breath when walking on level ground
 - Grade 3: Stop for breath after 100 m on level ground
 - Grade 4: Too breathless to leave the house or breathless when dressing/undressing
- GOLD guidelines
 - Group A
 - No more than 1 exacerbation/yr
 - FEV1 > 80% predicted
 - mMRC of 0 or 1
 - Group B
 - mMRC of 2 or more
 - FEV1 50–80% of predicted
 - Group C
 - mMRC < 2
 - ≥ 2 exacerbations/yr
 - FEV1 30–49% of predicted

- Group D
 - High symptom burden
 - mMRC \geq 2
 - High risk for exacerbations
 - FEV1 < 30% of predicted

RISK FACTORS

Genetics

α_1 -Antitrypsin deficiency

ETIOLOGY

- Smoking is the overwhelming cause:
 - COPD develops in 15% of smokers.
- Air pollution
- Airway hyper-responsiveness
- α_1 -Antitrypsin deficiency
- Autoimmunity may play a role
- Acute exacerbations:
 - Viral infections
 - >50% of exacerbations associated with recent cold symptoms
 - Decreased immunity may make the host more susceptible to a COPD exacerbation
 - Rhinovirus
 - Respiratory syncytial virus (RSV)
 - Bacterial infections
 - Bacteria isolated in 40–60% of sputum during acute exacerbation
 - Most common:
 - *Haemophilus influenzae*
 - *Moraxella catarrhalis*
 - *Streptococcus pneumoniae*
 - More likely if:
 - Increased dyspnea
 - Increased sputum volume
 - Purulent sputum
 - Pollutants
 - Changes to immunity
 - Increased airway inflammation
 - Seasonal variations
 - More common and more severe in winter

SIGNS AND SYMPTOMS

History

- Dyspnea on exertion
- Cough
- Sputum production
- Fatigue
- Wheezing
- Orthopnea
- Altered mental status

Physical-Exam

- Wheezing
- Retractions
- Decreased air movement
- Cyanosis
- Prolonged expiratory phase
- Barrel chest
- Lower-extremity edema
- Jugular venous distension
- S3 and S4 gallops
- Altered mental status secondary to carbon dioxide narcosis

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC:
 - _ Elevated hematocrit may indicate chronic hypoxemia.
 - _ Increased neutrophils and elevated WBC may indicate infection.
- Arterial blood gas:
 - _ Retaining carbon dioxide
 - _ Acidosis
 - _ Oxygenation
- β -Natriuretic peptide:
 - _ Differentiate between COPD and CHF
- Sputum sample
- Theophylline level as needed

Imaging

- CXR:
 - _ Pneumothorax
 - _ Pneumonia

- CHF
- Lobar collapse
- Chest CAT scan:
 - When needed to evaluate for pulmonary embolus or further characterize disease

Diagnostic Procedures/Surgery

- Pulse oximetry
- ECG
- Pulmonary function tests
- Echocardiography:
 - To diagnose left or right ventricular failure or strain

DIFFERENTIAL DIAGNOSIS

- Pneumothorax
- CHF
- Pneumonia
- Pulmonary embolus
- Upper airway obstruction
- Asthma
- Restrictive lung disease
- ARDS
- Pleural effusions
- Acute coronary syndrome
- Pericardial effusion
- Metabolic derangement



TREATMENT

PRE HOSPITAL

Supplemental oxygenation:

- 100% via nonrebreather
- Do not withhold for fear of CO₂ retention.
- Initiate nebulized bronchodilator therapy.

INITIAL STABILIZATION/THERAPY

- Oxygen therapy:
 - Maintain oxygen saturation > 90–92%.
 - Patients at risk for CO₂ narcosis are those with slow respiratory rate.
 - Monitor closely for ventilation suppression.
- Noninvasive ventilation:
 - Treatment of choice in hypercapneic respiratory failure if ventilatory

support required

- May prevent intubation
- May help resolve hypercarbia
- Intubation for airway control:
 - Clinical tiring
 - Altered mental status
 - Inability to comply with emergent therapy
 - Ineffective ventilation
 - CO₂ narcosis

ED TREATMENT/PROCEDURES

- Continuous ECG and pulse oximetry monitoring
- Bronchodilator therapy
- β -Agonists:
 - Albuterol
- Anticholinergics:
 - Ipratropium bromide
- Corticosteroids:
 - Anti-inflammatory effects
 - Reduce relapses
 - Methylprednisolone or prednisone
- Antibiotics:
 - Fever, increased sputum production, and/or dyspnea
 - Macrolides also may have anti-inflammatory effects unrelated to their antibacterial role
- Methylxanthines
 - Theophylline
- Ventilator settings:
 - Allow sufficient expiratory time to minimize air trapping and subsequent barotrauma.
 - Permissive hypercapnia

MEDICATION

- Albuterol: 2.5 mg nebulized q10–30min
- Azithromycin: 500 mg PO/IV once, then 250 mg/d PO for 4 days
- Ceftriaxone: 1 g IV q24h
- Ipratropium bromide: 0.5 mg nebulized q6h
- Levofloxacin: 500 mg PO/IV q24h
- Methylprednisolone: 125 mg IV q6h
- Prednisone: 40–60 (1–2 mg/kg) mg/d PO for 5 days
- Terbutaline: 0.25 mg SC q30min

First Line

- Albuterol
- Ipratropium bromide
- Prednisone or methylprednisolone



FOLLOW-UP

DISPOSITION

Admission Criteria

- ICU admission:
 - Intubated patients
 - CO₂ narcosis with oxygen saturation < 90%
 - Clinical tiring in the ED
 - Severe acidosis
 - Concomitant cardiac or pulmonary disease
 - Acute coronary syndrome
 - Arrhythmia
 - CHF
 - Pulmonary embolism
- Regular hospital bed:
 - COPD patients with an additional pulmonary insult:
 - Pneumonia
 - Lobar collapse
 - Increased work of breathing
- Exercise intolerance
- Failure to improve in ED
- Failed outpatient treatment
- 3 criteria can predict mortality at admission:
 - Age > 70 yr
 - Number of clinical signs of severity:
 - Cyanosis, accessory muscle use, etc.
 - Dyspnea at baseline

Discharge Criteria

- Mild flare
- Resolution in ED
- Ambulatory oxygen saturation > 92%

FOLLOW-UP RECOMMENDATIONS

- Smoking cessation
- Ensure vaccinations are up-to-date (influenza annually, pneumococcal at least once).

- Identify and avoid triggers (e.g., cold air, perfumes)
- Possible referral for lung volume reduction surgery

PEARLS AND PITFALLS

- Noninvasive positive pressure ventilation is the therapy of choice when optimal medical therapy is insufficient
- Nebulized steroids may be used more for acute exacerbation of COPD in the future.
- Patients with COPD are at increased risk for diabetes, hypertension, and cardiovascular disease.
- Consider routine influenza and pneumococcal vaccinations for those with COPD.

ADDITIONAL READING

- Agusti A, Barnes PJ. Update in chronic obstructive pulmonary disease 2011. *Am J Respir Crit Care Med.* 2012;185:1171–1176.
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- Cosio MG, Saeta M, Agusti A. Immunologic aspects of chronic obstructive pulmonary disease. *N Engl J Med.* 2009;360:2445–2454.
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- Sutherland ER, Cherniack RM. Management of chronic obstructive pulmonary disease. *NEJM.* 2004;350:2689–2697.



See Also (Topic, Algorithm, Electronic Media Element)

- Asthma
- Congestive Heart Failure
- Dyspnea
- Pulmonary Embolism



CODES

ICD9

- 491.9 Unspecified chronic bronchitis
- 492.8 Other emphysema
- 496 Chronic airway obstruction, not elsewhere classified

ICD10

- J42 Unspecified chronic bronchitis
- J43.9 Emphysema, unspecified

- J44.9 Chronic obstructive pulmonary disease, unspecified

CIRRHOSIS

Ahmed Nadeem • Paul J. Allegretti

BASICS

DESCRIPTION

- Progressive process of inflammation, cellular injury and necrosis, diffuse fibrosis, and formation of regenerative nodules
- Loss of lobular and vascular architecture
- Irreversible in advanced stages
- Intrahepatic portal hypertension owing to increased resistance at the sinusoid, compression of the central veins, and anastomosis between the arterial and portal systems
- 10th leading cause of death in US

ETIOLOGY

- Chronic alcohol abuse (most common cause in US)
- Chronic viral hepatitis, B or C (2nd most common cause in US)
- Autoimmune hepatitis
- Biliary cirrhosis, primary (PBC) or secondary (sclerosing cholangitis)
- Metabolic:
 - Hereditary hemochromatosis
 - Wilson disease
 - Porphyrria
- Drugs:
 - Acetaminophen
 - Methotrexate
 - Amiodarone
 - Methyl dopa
- Hepatic congestion:
 - Right-sided heart failure
 - Pericarditis
 - Budd–Chiari syndrome (hepatic venous outflow obstruction)
- Infiltrative:
 - Sarcoidosis
 - Amyloidosis
 - Nonalcoholic steatohepatitis (NASH)
 - Hepatocellular carcinoma, diffusely infiltrating
- Infections:
 - Brucellosis
 - Echinococcosis

- Tertiary syphilis
- Schistosomiasis

Pediatric Considerations

- Congenital
- Arteriohepatic dysplasia, biliary atresia, cystic fibrosis, α_1 -antitrypsin deficiency
- Metabolic
- Fructosemia, tyrosinemia, galactosemia, glycogen storage diseases
- Infectious
- Congenital hepatitis B



DIAGNOSIS

SIGNS AND SYMPTOMS

- May be silent
- Insidious onset with nonspecific findings:
 - Malaise
 - Fatigue
 - Anorexia
 - Nausea and vomiting
 - Weight loss
 - Pruritus
 - Hyperpigmentation
- Jaundice
- Abdominal collateral circulation including caput medusae
- Hepatomegaly
- Splenomegaly
- Abdominal discomfort or tenderness
- Fever
- Feter hepaticus
- Asterixis
- Hypotension
- Cruveilhier–Baumgarten murmur
- Renal insufficiency
- Spider telangiectasias
- Palmar erythema
- Dupuytren contractures
- Parotid and lacrimal gland enlargement
- Terry nails
- Muehrcke lines
- Clubbing
- Feminization:

- Testicular atrophy
- Impotence
- Loss of libido
- Gynecomastia
- Amenorrhea
- Complications:
 - When complications develop, patient is considered to have decompensated disease.
 - Ascites
 - Spontaneous bacterial peritonitis (SBP)
 - Hepatic encephalopathy (HE)—may be precipitated by:
 - GI bleed
 - Infections
 - Increased dietary protein
 - Hypokalemia
 - Sedatives
 - Constipation
 - Azotemia
 - Alkalosis
 - Variceal hemorrhage:
 - 1/3 of patients with variceal bleed.
 - Each bleeding episode carries a 33% mortality rate.
 - Hepatic venous pressure gradient > 12 mm Hg increases risk of bleed.
 - Portal hypertensive gastropathy or peptic ulcer disease
 - Hepatorenal failure:
 - Caused by decreased renal perfusion during severe decompensated cirrhosis
 - May be iatrogenic: Secondary to diuretics, NSAIDs, IV contrast, aminoglycosides, large-volume paracentesis
 - High mortality rate
 - Hepatopulmonary syndrome:
 - Intrapulmonary vascular dilation and hypoxia
 - Results in increased alveolar–arterial gradient

ESSENTIAL WORKUP

Detailed history and physical exam to search for clues to liver disease

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC:
 - Anemia
 - Macrocytosis

- Leukopenia and neutropenia
- Thrombocytopenia
- Impaired liver function:
 - High bilirubin
 - Low albumin
 - High globulins
 - Prolonged PT
 - Varying degrees of DIC
 - Hypoglycemia
- Increased liver enzymes:
 - Aspartate alanine aminotransferase (AST, SGOT), alanine aminotransferase (ALT, SGPT)—reflect injury
 - Ratio of AST:ALT ≥ 2 in alcoholic liver disease
 - Alkaline phosphatase and 5'-nucleotidase reflect cholestasis.
 - γ -Glutamyltranspeptidase (GGT)
 - May be normal in inactive cirrhosis
- Electrolytes, BUN, and creatinine
- Hyponatremia:
 - Renal dysfunction and hepatorenal syndrome
- Arterial blood gases or pulse oximeter for:
 - Suspected pneumonia
 - CHF
 - Hepatopulmonary syndrome
- Search for cause:
 - Hepatitis B surface antigen
 - Hepatitis C antibody
 - Antinuclear antibody (ANA) and antismooth muscle antibody (autoimmune hepatitis)
 - Antimitochondrial antibody (PBC)
 - Serum iron, transferrin saturation, and ferritin (hemochromatosis)
 - Ceruloplasmin (Wilson disease)
 - α_1 -Antitrypsin deficiency
 - Serum immune electrophoresis (high IgM in PBC)
 - Cholesterol (chronic cholestasis)
 - α -Fetoprotein (hepatocellular cancer)

Imaging

- US for liver architecture, biliary obstruction, ascites, portal vein thrombosis, splenomegaly
- CT scan to explore abnormal finding on ultrasound
- CXR for pleural effusion, cardiomegaly, and CHF

Diagnostic Procedures/Surgery

- Esophagogastroduodenoscopy (EGD) indicated for upper GI bleeding or variceal surveillance
- Variceal ligation or endoscopic sclerotherapy
- Paracentesis for significant ascites or SBP

DIFFERENTIAL DIAGNOSIS

- Ascites:
 - Increased right heart pressure
 - Hepatic vein thrombosis
 - Peritoneal malignancy/infection
 - Pancreatic disease
 - Thyroid disease
 - Lymphatic obstruction
- Upper GI bleeding:
 - Peptic ulcer disease
 - Gastritis
- Encephalopathy:
 - Metabolic
 - Toxic
 - Intracranial process



TREATMENT

PRE HOSPITAL

- Naloxone, dextrose (or Accu-Chekk), and thiamine for altered mental status
- Reverse hypotension with IV fluids to prevent acute ischemic hepatic injury.

INITIAL STABILIZATION/THERAPY

Treat complications such as GI bleeding or HE.

ED TREATMENT/PROCEDURES

- For suspected variceal bleed:
 - IV proton pump inhibitors
 - IV octreotide-splanchnic vasoconstrictor
 - Reverse coagulopathy:
 - Fresh-frozen plasma 1 IU/hr until bleeding is controlled
 - Desmopressin (DDAVP)—improves bleeding time and prolonged PTT
 - Balloon tamponade with Sengstaken–Blakemore tube or a variant for variceal compression (rarely used anymore, prophylactic intubation recommended)
 - Emergent endoscopic sclerotherapy
- Initiate broad-spectrum antibiotics in suspected sepsis or SBP:

- Cefotaxime
- Ticarcillin–clavulanate
- Piperacillin–tazobactam
- Ampicillin–sulbactam
- Treat complicating conditions such as ascites, HE, SBP.
- Treat pruritus with:
 - Diphenhydramine 25–50 mg IM/IV q4h
 - Cholestyramine, ursodeoxycholic acid, or rifampin
 - Naloxone infusion 0.2 µg/kg/min for temporary relief for extreme cases
- β-Blocker (propranolol) for esophageal varices:
 - Titrated to pulse rate of 60 or 25% reduction of resting pulse
 - With or without isosorbide dinitrate
 - Decreases rebleeding rate
 - May delay or prevent occurrence of 1st bleed
- Relieve biliary obstruction (e.g., stricture) by endoscopic, radiologic, or surgical means.
- Provide nutritious diet, high in calories and adequate in protein (1 g/kg), unless there is complicating HE
- Consult transplantation coordinator whenever postliver transplantation patient presents to the ED with liver dysfunction, suspected sepsis, or possible treatment-related complication.

SPECIAL THERAPY

- Hemochromatosis: Phlebotomy or deferoxamine (iron-chelating agent)
- Autoimmune hepatitis: Prednisone with or without azathioprine
- Chronic hepatitis B or C: α-Interferon (avoid in decompensated cirrhosis)
- PBC: Ursodeoxycholic acid
- Wilson disease: Penicillamine
- The only cure for most advanced cirrhosis is liver transplantation.

MEDICATION

- Azathioprine: 1–2 mg/kg PO daily
- Cefotaxime: 1–2 g q6–8h (peds: 50–180 mg/kg/d q6h) IV
- Cholestyramine: 4 g PO 1–6 times per day
- Desmopressin (DDAVP): 0.3 µg/kg in 50 mL saline infused over 15–30 min
- Dextrose: D₅₀W 1 amp (50 mL or 25 g; peds: D₂₅W 2–4 mL/kg) IV
- Naloxone: 0.2–2 mg (peds: 0.1 mg/kg) IV or IM initial dose
- Lactulose: 15–30 mL TID—goal is 2–3 stools per day
- Octreotide: 25–50 µg IV bolus followed by 50 µg/hr IV infusion
- Piperacillin–tazobactam: 3.375 g IV q6h (peds: 100–400 mg/kg/d div. q6–8h; renal dosing required)
- Prednisone: 40 mg (peds: 1–2 mg/kg) PO daily
- Propranolol: 40 (initial) to 240 mg (peds: 1–5 mg/kg/d) PO TID

- Rifampin: 600 mg (peds: 10–20 mg/kg) PO daily
- Thiamine: 100 mg (peds: 50 mg) IV or IM
- Ursodeoxycholic acid: 8–10 mg/kg/d TID



FOLLOW-UP

DISPOSITION

Admission Criteria

- Acute decompensation or complicating conditions
- 1st presentation with clinically evident cirrhosis, unless close outpatient workup is possible
- Advanced grades HE, sepsis, active GI bleed, and hepatorenal and hepatopulmonary syndromes require ICU.
- Advanced stages of hepatocellular carcinoma

Discharge Criteria

Most patients with compensated cirrhosis can be treated as outpatients.

FOLLOW-UP RECOMMENDATIONS

GI for all new cases

PEARLS AND PITFALLS

- Prognosis is highly variable.
- Patients present with a wide variety of signs and symptoms related to end-stage liver disease.
- New cases need full workup and GI consultation for management.
- Any complication puts patient in decompensated state.
- SBP symptoms are frequently vague:
 - Must have a high suspicion and low threshold for paracentesis when considering SBP

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Ascites
- Hepatic Encephalopathy
- Hepatitis
- Spontaneous Bacterial Peritonitis
- Varices



CODES

ICD9

- 571.2 Alcoholic cirrhosis of liver
- 571.5 Cirrhosis of liver without mention of alcohol
- 571.6 Biliary cirrhosis

ICD10

- K70.30 Alcoholic cirrhosis of liver without ascites
- K74.5 Biliary cirrhosis, unspecified
- K74.60 Unspecified cirrhosis of liver

CLAVICLE FRACTURE

Sean Patrick Nordt

BASICS

DESCRIPTION

- Clavicular fractures account for 5% of all fractures in all age groups.
- 80% of clavicular fractures involve the middle 3rd.
- 15% occur in the distal 3rd.
- 5% occur in the medial 3rd.

Classification

- Group I: Middle-3rd fractures
- Group II: Distal-3rd fractures:
 - Type I: Coracoclavicular ligaments are intact (nondisplaced).
 - Type II: Severing of the coracoclavicular ligaments (conoid)
 - Type III: Articular surface involvement of the acromioclavicular joint
- Group III: Medial (proximal)-3rd fractures

ETIOLOGY

Mechanism:

- Direct trauma to the clavicle
- Fall on the lateral shoulder
- Fall on the outstretched hand

Pediatric Considerations

- Most common of all pediatric fractures
- May occur in newborns secondary to birth trauma

Geriatric Considerations

Geriatric patients who sustain a clavicular fracture may have difficulty performing activities of daily living. The patient's social and living situations should be assessed to determine a safe discharge plan that may require additional assistance at home.

Pregnancy Considerations

Clavicular fractures are the result of direct trauma. Patients who are pregnant should be appropriately worked up for other injuries but also should receive fetal monitoring to ensure the health of the fetus. Even minor injuries can result in trauma or harm to the fetus.

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Local pain, tenderness, and swelling over the fracture site
- Crepitus is often present owing to the clavicle's SC position
- Arm held in adduction against the chest wall with resistance to motion
- Shoulder displaced anteriorly and inferiorly

Physical-Exam

- Palpate the clavicle for tenderness, crepitus, and swelling.
- Examine the humerus and shoulder joint for other fractures, dislocations, or subluxations.
- Determine whether the fracture is open or closed.
- Evaluate for associated injuries (often serious and life threatening) that must be excluded:
 - Skeletal injuries:
 - 1st rib fracture with underlying aortic injury
 - Sternoclavicular joint separation/fracture-dislocation
 - Acromioclavicular joint separation/fracture-dislocation
 - Cervical spine injuries

DIAGNOSIS TESTS & INTERPRETATION

Imaging

- AP radiographs of both clavicles are mandatory and must include:
 - Upper 3rd of the humerus
 - Shoulder girdle (rule out other fractures)
 - Upper lung fields (rule out pneumothorax)
- Oblique and apical lordotic views:
 - May be helpful, especially for medial and distal clavicle fractures that are not easily visualized on the AP view
 - Stress views (weight bearing) for distal clavicle fractures are no longer routinely recommended.
- Angiography:
 - Should be performed if there is any evidence or suspicion of vascular injuries (most commonly subclavian vessels)

DIFFERENTIAL DIAGNOSIS

- Distal fractures: Consider acromioclavicular separation.
- Medial fractures: Consider sternoclavicular separation.
- Shoulder fracture–dislocation



TREATMENT

PRE HOSPITAL

- Ice packs to affected area
- Pain management using either narcotics or NSAIDs
- Immobilize affected side in a sling.

INITIAL STABILIZATION/THERAPY

Airway management and resuscitate as indicated

ED TREATMENT/PROCEDURES

- Open fracture: Uncommon occurrence, but usually requires open débridement and internal fixation (obtain immediate orthopedic referral)
- Closed fracture: If severely displaced, attempt closed reduction and immobilize depending on type of fracture:
 - _ Middle 3rd:
 - If nondisplaced, a sling or shoulder immobilizer is enough to provide support.
 - Controversy exists as to whether closed reduction is necessary because the alignment is rarely maintained regardless of splinting technique.
 - To perform a closed reduction, 1% lidocaine should be injected into the fracture hematoma. The shoulders are pulled upward, outward, and backward, and the fracture is then manipulated into place.
 - Sedation may be given to alleviate pain or anxiety.
 - A figure-of-eight splint or shoulder immobilizer is then applied.
 - Ice should be applied for the 1st 24 hr.
 - Analgesia (narcotics or NSAIDs) for pain
 - _ Distal 3rd type I:
 - Ice for the 1st 24 hr.
 - Immobilization with a sling or shoulder immobilizer
 - Orthopedic referral
 - Analgesia (narcotics or NSAIDs) for pain
 - Early range of motion
 - _ Distal 3rd type II:
 - Ice for the 1st 24 hr.
 - Immobilization with a sling or shoulder immobilizer
 - Orthopedic referral (may require operative repair)
 - Analgesia (narcotics or NSAIDs) for pain
 - _ Distal 3rd type III: Same as type II
 - _ Medial (proximal) 3rd:
 - Ice for the 1st 24 hr.
 - Immobilization in a sling or shoulder immobilizer for support

- Analgesia (narcotics or NSAIDs) for pain
- Orthopedic follow-up
- Reassess neurovascular status after all splints are applied.

Pediatric Considerations

- Children who do not cooperate with the figure-of-eight splint should be referred to an orthopedic surgeon for possible shoulder spica placement.
- Most children will tolerate a shoulder immobilizer best.

MEDICATION

- Acetaminophen: 650 mg to 1000 mg (peds: 10--15 mg/kg) PO q6h prn. Do not exceed 3 g/24 hr
- Ibuprofen: 600–800 mg PO q6h PRN with meals (peds: 10 mg/kg PO q6h PRN)
- Adults: Hydrocodone/Acetaminophen 5 mg/325 mg one to two tablets PO q6h prn. Do not exceed 3 g/24 hr of acetaminophen. Avoid concomitant use of acetaminophen-containing medications
- Hydrocodone, oxycodone, and codeine-containing medications should be avoided in pediatric patients



FOLLOW-UP

DISPOSITION

Admission Criteria

- Open fracture
- Associated injuries that are potentially life threatening

Discharge Criteria

- Isolated closed clavicle fracture without other injuries
- Appropriate support services at home (especially for elderly patients)
- Orthopedic follow-up
- Adequate pain management

Issues for Referral

Open fracture, complex injury, signs of neurovascular injury require immediate orthopedic referral.

FOLLOW-UP RECOMMENDATIONS

Follow-up with an orthopedic surgeon:

- Seek medical care immediately with any changes in neurologic function, sensation, or motor strength.

PEARLS AND PITFALLS

- Always be wary of associated injuries that can be life threatening including cervical spine injury, aortic injury, and other cardiopulmonary injuries:
- Always assess for any neurologic deficits associated with the fracture.

ADDITIONAL READING

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- Toogood P, Horst P, Samagh S, et al. Clavicle fractures: A review of the literature and update on treatment. *Phys Sportsmed.* 2011;39:142–150.
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CODES

ICD9

- 810.00 Closed fracture of clavicle, unspecified part
- 810.02 Closed fracture of shaft of clavicle
- 810.10 Open fracture of clavicle, unspecified part

ICD10

- S42.009A Fracture of unsp part of unsp clavicle, init for clos fx
- S42.026A Nondisp fx of shaft of unsp clavicle, init for clos fx
- S42.009B Fracture of unsp part of unsp clavicle, init for opn fx

COAGULOPATHY REVERSAL (NONWARFARIN AGENTS)

Susanne M. Hardy • John P. Lemos

BASICS

DESCRIPTION

- Patient on anticoagulant medications with minor, major, or clinically significant bleeding needing close monitoring +/- anticoagulant reversal
- Anticoagulant medication
 - _ Indirect inhibitors of thrombin
 - Unfractionated heparin (UFH)
 - Low-molecular-weight heparin (LMWH)
 - Enoxaparin
 - Dalteparin
 - Tinzaparin
 - _ Anti-platelet agents
 - Aspirin
 - Clopidogrel hydrogen sulfate (Plavix)
 - _ Factor Xa inhibitors (FXa inhibitors)
 - Fondaparinux (Arixtra)
 - Rivaroxaban (Xarelto)
 - _ Direct thrombin inhibitors (DTIs)
 - Argatroban
 - Bivalirudin (Angiomax)
 - Dabigatran (Pradaxa)
 - Hirudin derivatives
 - Desirudin
 - Lepiruden (Refludan)

Pediatric Considerations

- Heparin and LMWH are the most commonly utilized anticoagulants beyond warfarin
- Routine use of DTIs is being studied

Geriatric Considerations

Excretion primarily renal with FXa inhibitors, Dabigatran, and Hirudin derivatives necessitating caution with impaired renal function

EPIDEMIOLOGY

Incidence and Prevalence Estimates

- Indirect inhibitors of thrombin
 - Up to 1/3 patients develop bleeding complication
 - 2–6% of bleeding is major
- Anti-platelet agents
 - > 300 over-the-counter medications contain aspirin
 - Conflicting studies regarding increased hematoma expansion and mortality
- FXa Inhibitors
 - Unknown
- DTIs
 - Unknown

ETIOLOGY

- Indirect inhibitors of thrombin
 - Combines with antithrombin III to inactivate activated FXa and also inhibits thrombin
 - LMWH has a reduced ability to inactivate thrombin
 - Half-life is dose dependent (30–150 min), can be up to 8 hr with LMWH
- Anti-platelet agents
 - Inactivates cyclooxygenase-1 (COX-1) preventing formation of thromboxane A₂, which inactivates platelets
 - Single dose suppresses for 1 wk
 - New platelet production recovers 10%/day
 - Patients may manifest normal hemostasis with as few as 20% platelets with normal COX1 activity
 - Aspirin half-life 15–30 min
 - Clopidogrel half-life 8 hr
- FXa inhibitors
 - Binds to antithrombin III, catalyzing FXa inhibition
 - No direct inhibitory effect on thrombin
 - Half-life 12–21 hr in normal renal function
- DTIs
 - Competitively targets active site of thrombin +/- exosite (substrate binding site)
 - Half-life long with dabigatran (14–17 hr) and short with others (20–45 min)



DIAGNOSIS

- Patient on anticoagulants with active bleeding
- Indications for reversal
 - Serious or life-threatening bleeding
 - Trauma
 - GI bleeding
 - Intracerebral hemorrhage (ICH)

- Procedural

SIGNS AND SYMPTOMS

History

- Type of anticoagulant
- Last anticoagulant use
- Length of anticoagulant
- Recent injury or trauma
- Bleeding location
- Symptoms (fatigue, lightheadedness, headache, abdominal pain)

Physical-Exam

- VS +/- orthostatics
- Search for hemorrhage locations/signs of trauma
- Comprehensive neurologic exam
- Rectal with stool guaiac test

ESSENTIAL WORKUP

- CBC
- PT/INR
- PTT
- Stool guaiac test
- +/- Fibrinogen/DIC panel

DIAGNOSIS TESTS & INTERPRETATION

- Indirect inhibitors of thrombin
 - PTT
 - Anti-FXa
 - High is > 0.8 U/mL
- Anti-platelet agents
 - Bleeding time
- FXa inhibitors
 - Anti-FXa
 - PT, PTT minimally helpful
 - Fondaparinux level (institution specific)
- DTIs
 - PTT minimally helpful
 - Dabigatran level aka dilute thrombin time (institution specific)

DIFFERENTIAL DIAGNOSIS

- Disseminated intravascular coagulopathy
- Inherited coagulation disorders

- Platelet dysfunction:
 - _ TTP/HUS
 - _ HIT
 - _ ITP



TREATMENT

PRE HOSPITAL

- Pressure to hemorrhage (if possible)
- 2 large-bore IVs
- IV fluids

INITIAL STABILIZATION/THERAPY

- Same as pre-hospital
- Hold anticoagulants

ED TREATMENT/PROCEDURES

- Indirect inhibitors of thrombin
 - _ Level bleeding
 - Minor: Observe PTT, anti-FXa
 - Major: Protamine (Class II for UFH and Class III for LMWH)
 - _ Protamine
 - 1 mg IV neutralizes 100 U UFH administered in prior 3–4 hr
 - If < 30 min since UFH, use 1 mg/100 U UFH
 - If 30–120 min, use 0.5 mg/100 U UFH
 - If > 120 min, use 0.25 mg/100 U UFH
 - Give slowly IV over 1–3 min not to exceed 50 mg in any 10-min period
 - Short half-life, may need to re-dose
 - Protamine reversal effectiveness is compound specific for LMWH (does not reverse enoxaparin completely)
 - 1 mg for each 1 mg/100 IU LMWH given in last 8 hr
 - If 8–12 hr since LMWH, use 0.5 mg for each 1 mg/100 IU LMWH
 - If > 12 hr since LMWH, no protamine suggested
 - For LMWH, if PTT remains prolonged, may repeat with half of the 1st dose
 - High or excessive dosing can have a paradoxical anticoagulant effect
 - Rapid administration can cause hypotension, bradycardia, and anaphylaxis
 - Anaphylaxis is more likely with a fish allergy or prior exposure to protamine and if concerned, can premedicate with corticosteroids and antihistamines

- Anti-platelet agents
 - Level bleeding
 - Minor: Observe bleeding
 - Major: DDAVP +/- platelet transfusion(s) (class III)
 - Desmopressin (DDAVP)
 - Induces the release of von Willebrand factor and factor VIII
 - 0.3 µg/kg IV over 15 min
 - Effect is immediate
 - Multiple doses associated with tachyphylaxis, hyponatremia, and seizures
 - Platelets
 - Transfuse to increase count by 50,000/µL (on average, 1 U increases platelet count by 10k)
 - May need to repeat transfusions daily
 - Risks include infection transmission, acute lung injury, and allergic reactions
- FXa inhibitors
 - Level bleeding
 - Minor: Observe bleeding
 - Major: PCC or rFVIIa (Class III), consider hemodialysis (HD) for fondaparinux, consider charcoal if rivaroxaban and ingested in previous 2 hr
 - Prothrombin complex concentrates (PCCs)
 - 3 factor: Contains factors II, IX, X and low concentrations of nonactivated factor VII + anticoagulant protein C, protein S, antithrombin III
 - 4 factor: Contains II, IX, X, activated VII
 - Factor 4 is now available widely in the US
 - FDA approved for bleeding episodes in patients with hemophilia B
 - Dose 25–50 U/kg not to exceed 2 mL/min
 - Give 1–2 U FFP for factor VIIa component
 - Effect in < 30 min
 - Limited data to support use in trauma
 - Vary widely in composition
 - Several contain heparin
 - Long-term safety has not been assessed
 - Associated with risk of thrombosis
 - Allergic reactions may occur
 - Recombinant activated factor VII (rFVIIa)
 - FDA approved for bleeding episodes in patients with hemophilia A and B
 - Off-label use for life-threatening bleeding
 - Dose 15–90 µg/kg (suggested 40 µg/kg) IV over 3–5 min

- Effect in < 30 min
- May repeat in 2 hr if continued bleeding
- Associated with risk of thrombosis
- Ultrafiltration/HD
 - For fondaparinux, may remove 20%
- Activated charcoal
 - If ingestion within 1–2 hr of rivaroxaban
- DTIs
 - Level bleeding
 - Minor: Observe bleeding (DTIs have short half-life except dabigatran, which is 14–17 hr), IV fluids to improve renal clearance
 - Major: PCC or rFVIIa (no strong evidence for either), consider DDAVP, activated charcoal if within 1–2 hr ingestion, consider HD (especially if dabigatran)
 - PCC
 - Dose 25–50 U/kg not to exceed 2 mL/min
 - Give 1–2 U FFP if using 3 factor
 - rFVIIa
 - Dose 100 µg/kg IV over 3–5 min
 - May repeat in 2 hr if continued bleeding
 - DDAVP
 - Dose 0.3 µg/kg IV over 15 min
 - Demonstrated effectiveness with hirudin
 - Ultrafiltration/HD
 - Consider early in course for dabigatran and major bleeding
 - Activated charcoal
 - If ingestion within 1–2 hr



FOLLOW-UP

DISPOSITION

Admission Criteria

- Clinically significant bleeding
- Utilization of reversal agents

Discharge Criteria

- Insignificant bleeding that is controlled without the use of anticoagulant reversal
- Discussion with outpatient hematologist or primary care physician (PCP) is ideal for follow-up

Issues for Referral

- Blood bank reversal medication availability
- Surgical/Interventional Radiology specialty availability to control hemorrhage

FOLLOW-UP RECOMMENDATIONS

Close follow-up and monitoring is paramount

PEARLS AND PITFALLS

- Prophylactic heparin dosing does not typically confer an increased risk of major bleeding
- LMWH is not always reversed with protamine—it is compound specific
- If > 12 hr have elapsed since LMWH administration, protamine may not be necessary
- Single-dose aspirin suppresses COX1 for 1 wk
- Caution is needed with renal impairment if utilizing FXa inhibitors, dabigatran, or hirudin derivatives
- FFP as 1st-line replacement has to be weighed against extensive volume expansion

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- Tawil I, Seder D, Duprey J. Emergency management of coagulopathy in acute intracranial hemorrhage. *EB Medicine. EM Crit Car*. 2012;2:2.
- van Ryn J, Stangier J, Haertter S, et al. Dabigatran etexilate—a novel, reversible, oral direct thrombin inhibitor: Interpretation of coagulation assays and reversal of anticoagulant activity. *Thromb Haemost*. 2010;103:1116–1127.



CODES

ICD9

- 286.6 Defibrination syndrome
- 286.9 Other and unspecified coagulation defects
- V58.61 Long-term (current) use of anticoagulants

ICD10

- D65 Disseminated intravascular coagulation
- D68.9 Coagulation defect, unspecified
- Z79.01 Long term (current) use of anticoagulants

COCAINE POISONING

Steven E. Aks

BASICS

DESCRIPTION

- Sympathomimetic
- Inhibits neurotransmitter reuptake at the nerve terminal
- Metabolism:
 - Hepatic degradation
 - Nonenzymatic hydrolysis
 - Cholinesterase metabolism

ETIOLOGY

- IV, nasal, oral administration of cocaine
- Oral ingestion:
 - Body stuffers:
 - Ingest hastily wrapped packets in attempt to evade police.
 - Body packers:
 - Ingest cocaine packets to smuggle the drug using couriers' oral, rectal, and vaginal cavities.
 - Cocaine is wrapped carefully in packets containing large amounts of drug.

DIAGNOSIS

SIGNS AND SYMPTOMS

- Sympathomimetic toxidrome
- Cardiovascular:
 - HTN
 - Tachycardia
 - Chest pain (angina)
- Respiratory:
 - Tachypnea
 - Pleuritic chest pain:
 - Pneumomediastinum
 - Pneumothorax
 - Bronchitis
 - Pulmonary infarction
 - Cough
- CNS:

- Agitation
- Tremulousness
- Coma
- Seizures
- Stroke
- Miscellaneous:
 - Hyperthermia (poor prognosis)
 - Limb ischemia (inadvertent intra-arterial injection)
 - Corneal ulcerations (heavy crack smokers):
 - Owing to local chemical and thermal irritation that disrupts corneal epithelium
 - Rhabdomyolysis

History

For body packers and stuffers:

- Time since ingestion
- Route of ingestion (oral, rectal, vaginal)
- Number of packets ingested
- Material and method of packing

Physical-Exam

Sympathomimetic toxidrome:

- HTN
- Tachycardia
- Tachypnea
- Hyperthermia
- Diaphoresis
- Mydriasis
- Neuromuscular hyperactivity

ESSENTIAL WORKUP

- Recognition of the sympathomimetic toxidrome caused by cocaine:
 - Distinguish from anticholinergic toxidrome.
- Toxidrome recognition:
 - Sympathomimetic:
 - Heart rate (tachycardia)
 - BP (increased)
 - Moist skin
 - Bowel sounds present
 - Temperature (increased)
 - No urinary retention
 - Anticholinergic:
 - Heart rate (tachycardia)

- BP (increased)
- Dry skin
- Bowel sounds diminished
- Temperature (increased)
- Urinary retention present

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC
- Electrolytes, BUN, creatinine, glucose
- Urinalysis dip for myoglobin
- Cardiac enzymes (troponin, creatine phosphokinase [CPK]) for:
 - Anginal chest pain
 - Abnormal results on ECG
- CPK for evidence of myoglobinuria
- Toxicology screen

Imaging

- ECG:
 - For anginal chest pain
 - Consider possibility of myocardial infarction with cocaine-related chest pain.
- CXR:
 - For chest pain or shortness of breath
 - Check for pneumomediastinum, pneumothorax, aortic rupture.
- Abdominal radiograph:
 - For body packers/stuffers
 - Usually produces negative result for stuffers because drug is loosely packed in cellophane
 - Positive for packers because drug is densely packed and usually radiopaque
- CT of the abdomen with contrast:
 - When unreliable history of body packers/stuffers and nothing visualized on abdominal frontal radiograph
- CT brain scan:
 - For altered mental status or severe headache
 - Detects cerebral ischemia or hemorrhage

DIFFERENTIAL DIAGNOSIS

Other agents with sympathomimetic effects

- Theophylline
- Caffeine
- Amphetamines

- Albuterol
- Tricyclic antidepressants
- Antihistamines
- Phencyclidine (PCP)
- Thyrotoxicosis
- Neuroleptic malignant syndrome
- Hallucinogens



TREATMENT

PRE HOSPITAL

- Establish IV access
- Cardiac monitor:
 - Chest pain may be ischemic.
 - Benzodiazepine therapy to control agitation
- When drug is used as a “speedball” (combination of heroin and cocaine), administer naloxone in increments to reverse coma.

INITIAL STABILIZATION/THERAPY

- ABCs
- Establish IV access.
- Establish cardiac monitor.
- Provide therapy with naloxone (Narcan), thiamine, dextrose (or Accu-Check) for altered mental status

ED TREATMENT/PROCEDURES

- Supportive care for mildly symptomatic patients
- Benzodiazepines:
 - For agitation and tremor
 - Initial agents for hypertension and tachycardia
- Cooling measures for hyperthermia:
 - Evaporative–convective method
- Treat rhabdomyolysis:
 - Hydrate with 0.9% NS
 - Alkalinization with IV bicarbonate in severe cases
- Cardiac chest pain:
 - Aspirin
 - Nitrates
 - Oxygen
 - Opiates
 - Avoid β -blockers because of unopposed α -stimulation
 - Angiography/angioplasty/thrombolysis for acute myocardial infarction

- HTN/tachycardia:
 - Benzodiazepine initial agent
 - Use α -blocking agent (phentolamine) as sole agent or combine with β -blocker (propranolol, esmolol) if unresponsive to benzodiazepine.
 - Use labetalol cautiously (does not have equal α - and β -blocking properties).
 - IV nitroglycerin/nitroprusside for severe unresponsive hypertension
- Body packer/stuffers:
 - Treat asymptomatic or minimally symptomatic body packers and body stuffers:
 - Single-dose activated charcoal is appropriate for asymptomatic or minimally symptomatic body stuffers
 - Whole-bowel irrigation with polyethylene glycol electrolyte lavage solution (efficacy unknown)
 - Consult with surgeons for symptomatic body packers and stuffers.
 - If toxicity is not easily managed with previously suggested pharmacologic therapy, remove the drug packets intraoperatively.

MEDICATION

First Line

- Diazepam: 5 mg incremental doses IV
- Lorazepam: 2 mg incremental doses IV

Second Line

- Activated charcoal slurry: 1–2 g/kg up to 90 g PO
- Dextrose: D₅₀W 1 ampule (50 mL or 25 g) (peds: D₂₅W 2–4 mL/kg) IV
- Esmolol: 50–200 μ g/kg/min IV infusion titrated to effect
- Naloxone (Narcan): 2 mg (peds: 0.1 mg/kg up to 2 mg) IV or IM initial dose
- Nitroglycerin: 10–100 μ g/min IV infusion
- Nitroprusside: 0.3 μ g/kg/min IV (titrate to effect up to 10 μ g/kg/min)
- Phentolamine: 5 mg IV q15–24min (titrate to clinical effect)
- Polyethylene glycol (GoLYTELY): 1–2 L PO/hr until packet passage (efficacy controversial)



FOLLOW-UP

DISPOSITION

Admission Criteria

- Altered mental status
- Abnormal vital signs: Heart rate > 100 bpm, diastolic BP > 120 mm Hg, or hypotension

- Hyperthermia
- Cocaine-induced myocardial ischemia
- Body stuffers and body packers
- ICU admission for moderate to severe toxicity

Discharge Criteria

- Mental status and vital signs normal after 6 hr of observation
- Body packers or stuffers with confirmed expulsion of packets and no clinical signs of toxicity
- Stuffers may be discharged if uncomplicated packets were ingested and if asymptomatic for 12–24 hr.

PEARLS AND PITFALLS

- Benzodiazepines are the 1st-line treatment for the sympathomimetic toxidrome from cocaine.
- Avoid β -blockers in the hyperdynamic cocaine intoxicated patient.
- Consider a broad differential in cocaine-associated chest pain.
- An abdominal flat plate radiograph will be of some value in a body packer, but of no value in imaging packets in a body stuffer.

ADDITIONAL READING

- Hoffman RS. Cocaine. In: Goldfrank LR, ed. *Goldfrank's Toxicologic Emergencies*. 9th ed. Stamford, CT: Appleton & Lange; 2010:1091–1102.
- Jones JH, Weir WB. Cocaine-associated chest pain. *Med Clin North Am*. 2005;89:1323–1342.
- June R, Aks SE, Keys N, et al. Medical outcome of cocaine bodystuffers. *J Emerg Med*. 2000;18:221–224.
- Kalimullah EA, Bryant SM. Case files of the medical toxicology fellowship at the Toxikon Consortium in Chicago: Cocaine-associated wide-complex dysrhythmias and cardiac arrest-treatment nuances and controversies. *J Med Toxicol*. 2008;4:277–283.



CODES

ICD9

970.81 Poisoning by cocaine

ICD10

- T40.5X1A Poisoning by cocaine, accidental (unintentional), init
- T40.5X4A Poisoning by cocaine, undetermined, initial encounter

- T40.5X2D Poisoning by cocaine, intentional self-harm, subs encntr

COLON TRAUMA

Stephen R. Hayden

BASICS

DESCRIPTION

- Trauma that perforates the colon inflames the cavity in which it lies.
- Peritoneal inflammation from hollow viscus perforation often requires hours to develop.
- Mesenteric tears from blunt trauma cause hemorrhage and bowel ischemia.
- Delayed perforation from ischemic or necrotic bowel may occur.
- Peritonitis and sepsis may develop from the extravasated intraluminal flora.
- Ascending and descending colon segments are retroperitoneal.
- The left colon has a higher bacterial load than the right.
- Morbidity and mortality increase if the diagnosis of colon injury is delayed.

ETIOLOGY

- Penetrating abdominal trauma:
 - The colon is the 2nd most commonly injured organ in penetrating trauma.
 - Gunshot wounds have the highest incidence.
 - Transverse colon is most commonly injured.
 - Often presents with peritonitis
- Blunt abdominal trauma:
 - Colon rarely injured in blunt trauma
 - Burst injury occurs from compression of a closed loop of bowel.
 - Intestine may be squeezed between a blunt object (lap belt) and vertebral column or bony pelvis.
 - Sudden deceleration may produce bowel–mesenteric disruption and consequent devascularization.
 - With deceleration, the sigmoid and transverse colon are most vulnerable.
- Transanal injury:
 - Iatrogenic endoscopic or barium enema injury
 - Foreign bodies used during sexual activities may reach and injure the colon.
 - Compressed air under high pressure such as at automobile repair facilities can perforate the colon even if the compressor nozzle is not fully inserted anally.
 - Swallowed sharp foreign bodies (toothpick) may penetrate the colon, particularly the cecum, appendix, and sigmoid:
 - Most foreign bodies pass without complications.

Pediatric Considerations

Unlike adults, children have an equal frequency of blunt and penetrating colon injuries.

DIAGNOSIS

SIGNS AND SYMPTOMS

- Colon trauma is generally associated with other intra-abdominal and extra-abdominal injuries, commonly to the small intestine.
- Injuries of significant severity may have minimal early findings.
- It is uncommon to determine specific organ injury on physical exam.
- Assess on exam:
 - Abdomen for peritoneal signs
 - Ecchymosis or hematoma on lower abdomen from lap-belt compression
 - Ecchymosis on epigastric region from steering-wheel compression
 - Grey Turner sign (flank hematomas) resulting from retroperitoneal bleeding.
 - Foreign bodies or blood on digital rectal exam (be careful if sharp object suspected)
 - Note: Abdominal wall ecchymosis or hematoma is not always present despite existing injury.
 - Note: Bowel sounds are not helpful.

ESSENTIAL WORKUP

- Serial abdominal exam because inflammation takes time to develop
- Abdominal CT with contrast is the best diagnostic study in stable patients.
- US and diagnostic peritoneal lavage (DPL) are helpful in the potentially unstable patient.

DIAGNOSIS TESTS & INTERPRETATION

- No individual test or combination of currently available diagnostic modalities is adequate to exclude blunt colonic injury.
- Signs of peritoneal irritation owing to intestinal injury typically develop hours after the event.

Lab

- Electrolytes
- Calcium, magnesium

Imaging

- CT is more useful for detecting penetrating vs. blunt colon injury.
- CT with triple contrast allows intraperitoneal and retroperitoneal visualization.
- Oral contrast is not essential in blunt abdominal trauma CT evaluation.
- Although CT may miss colon injuries, abnormal findings are typical.
- CT is only moderately sensitive at identifying hollow viscus injury.

- Hollow viscus injury—associated CT findings include extraluminal gas or contrast, mesenteric fat streaking, and free fluid without solid organ injury.
- Water-soluble enema with fluoroscopy is useful if other test results are inconclusive.
- Plain abdominal radiographs can show indirect signs such as intraperitoneal and retroperitoneal free air.
- FAST US exam does not evaluate for enteric injury and retroperitoneal hemorrhage.
- See “Abdominal Trauma, Blunt”; “Abdominal Trauma, Imaging.”

Diagnostic Procedures/Surgery

- DPL or ultrasound in addition to CT will increase sensitivity.
- In blunt trauma, DPL will often not detect retroperitoneal injuries and enteric injury as intra-abdominal bleeding is limited.
- Fecal or vegetable material on DPL analysis indicates hollow viscus injury.
- Lavage white cell response may be negative secondary to delayed peritoneal inflammation.
- In hollow viscus injury, lavage WBC count: RBC ratio is higher than that seen with solid organ injuries.

DIFFERENTIAL DIAGNOSIS

- Other intra-abdominal injuries
- A fractured pelvis may present similarly to intraperitoneal injuries in children.



TREATMENT

PRE HOSPITAL

- Cautions:
 - _ Follow standard pre-hospital guidelines for trauma management (ABCs).
 - _ Do not remove penetrating foreign bodies.
 - _ Do not attempt to replace eviscerated bowel; cover with moist saline dressings.
 - _ Obtain history regarding mechanism of injury, vehicular damage, and seat belt use.
- Controversies:
 - _ Use of intravenous crystalloid resuscitation is still considered the standard of care.

INITIAL STABILIZATION/THERAPY

- Refer to topic on abdominal trauma.
- ABCs should precede abdominal evaluation.
- Aggressive management with IV crystalloid resuscitation and blood replacement as

needed.

ED TREATMENT/PROCEDURES

- Early surgical consultation; surgery is definitive treatment.
- Cover eviscerated bowel in moist saline gauze, in a nondependent position.
- Administer broad-spectrum antibiotics to cover gram-negative aerobic and anaerobic bacteria.
- The efficacy of multiple-agent and single-agent antibiotic regimens is similar.
- Ensure tetanus prophylaxis.

MEDICATION

- Ampicillin: 2 g (peds: 50 mg/kg) IV q6h + gentamicin 2 mg/kg (peds: 2.5 mg/kg) IV q8h + metronidazole 500 mg IV q6h (peds: Use clindamycin 25–40 mg/kg IV q24h div. q6–q8h)
- Aztreonam: 2 g IV q8h (peds: 90–120 mg/kg IV q24h div. q6–q8h) + clindamycin 900 mg IV q8h (peds: Use clindamycin 25–40 mg/kg IV q24h div. q6–q8h)
- Cefoxitin: 2 g IV q8h (peds: 40 mg/kg IV q6h)
- Piperacillin/tazobactam: 4.5 g (peds: 75 mg/kg) IV q8h



FOLLOW-UP

DISPOSITION

Admission Criteria

- Colon injuries require admission for surgical repair or monitoring.
- All penetrating foreign bodies must be removed to prevent sepsis.
- Patients with abdominal ecchymosis require hospital admission and observation because of potential for undiagnosed hollow viscus injury.

Discharge Criteria

- Patients in whom serious abdominal injury is not suspected and with completely normal abdominal exam, normal hemodynamic status, and no other injury may be considered for discharge with appropriate precautions.
- If there is any doubt about the possibility of colon injury, the patient should be admitted and observed.

PEARLS AND PITFALLS

Patients may initially present with paucity of symptoms:

- Observation and serial exams are indicated if mechanism suggests significant blunt abdominal trauma.

ADDITIONAL READING

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- Greer LT, Gillern SM, Vertrees AE. Evolving colon injury management: A review. *Am Surg*. 2013;79(2):119–127.
- Steele SR, Maykel JA, Johnson EK. Traumatic injury of the colon and rectum: The evidence vs dogma. *Dis Colon Rectum*. 2011;54(9):1184–1201.
- Williams MD, Watts D, Fakhry S. Colon injury after blunt abdominal trauma: Results of the EAST Multi-Institutional Hollow Viscus Injury Study. *J Trauma*. 2003;55(5):906–912.



CODES

ICD9

- 863.40 Injury to colon, unspecified site, without mention of open wound into cavity
- 863.42 Injury to transverse colon, without mention of open wound into cavity
- 863.50 Injury to colon, unspecified site, with open wound into cavity

ICD10

- S36.501A Unspecified injury of transverse colon, initial encounter
- S36.509A Unspecified injury of unspecified part of colon, initial encounter
- S36.539A Laceration of unspecified part of colon, initial encounter

COMA

Gregory D. Jay • Linda C. Cowell

BASICS

DESCRIPTION

- Light coma:
 - _ Responds to noxious stimuli
- Deep coma:
 - _ Does not respond to pain
- Unresponsiveness:
 - _ Loss of either arousability or cognition:
 - Loss of arousal
 - Arousal is primarily a brainstem function.
 - Impairment of the reticular activating system
 - Loss of cognition
 - Requires dysfunction of both cerebral hemispheres
 - _ Stupor:
 - Deep sleep, although not unconsciousness
 - Exhibits little or no spontaneous activity
 - Awaken with stimuli
 - Little motor or verbal activity once aroused
- Obtundation:
 - _ Mental blunting with mild or moderate reduction in alertness
- Delirium:
 - _ Floridly abnormal mental status
 - _ Irritability
 - _ Motor restlessness
 - _ Transient hallucinations
 - _ Disorientation
 - _ Delusions
- Clouding of consciousness:
 - _ Disturbance of consciousness
 - _ Impaired capacity to think clearly or perceive, respond to, and remember current stimuli

ETIOLOGY

- Diffuse brain dysfunction (69%):
 - _ Lack of nutrients:
 - Hypoglycemia
 - Hypoxia

- Poisoning:
 - Ethanol
 - Isopropyl alcohol
 - Ethylene glycol
 - Methanol
 - Salicylates
 - Sedative-hypnotics
 - Narcotics
 - Anticonvulsants
 - Isoniazid
 - Heavy metals
 - Opiates
 - Benzodiazepines
 - Anticholinergics
 - Lithium
 - Phencyclidine
 - Cyanide
 - Carbon monoxide
 - Isoniazid
- Infection:
 - Bacterial/tuberculous/syphilitic meningitis
 - Encephalitis
 - Falciparum meningitis
 - Typhoid fever
 - Rabies
- Endocrine disorders:
 - Myxedema coma
 - Thyrotoxicosis
 - Addison disease
 - Cushing disease
 - Pheochromocytoma
- Metabolic disorders:
 - Hepatic encephalopathy
 - Uremia
 - Porphyria
 - Wernicke encephalopathy
 - Aminoacidemia
 - Reye syndrome
 - Hypercapnia
- Electrolyte disorders:
 - Hypernatremia, hyponatremia
 - Hypercalcemia, hypocalcemia
 - Hypermagnesemia, hypomagnesemia

- Hypophosphatemia
- Acidosis, alkalosis
- Temperature regulation:
 - Hypothermia
 - Heat stroke
 - Neuroleptic malignant syndrome
 - Malignant hyperthermia
- Uremia
- Postictal state, status epilepticus
- Psychiatric
- Shock
- Fat embolism
- Hypertensive encephalopathy
- Supratentorial lesions (19%):
 - Hemorrhage (15%):
 - Intraparenchymal hemorrhage
 - Epidural hematoma
 - Subdural hematoma
 - Subarachnoid hemorrhage
 - Infarction (2%):
 - Thrombotic arterial occlusion
 - Embolic arterial occlusion
 - Venous occlusion
 - Tumor or abscess (2%):
 - Hydrocephalus
 - Herniation
 - Hemorrhage from erosion into adjacent blood vessels
- Subtentorial lesions (12%):
 - Infarction
 - Hemorrhage
 - Tumor
 - Basilar migraine
 - Brainstem demyelination

Pregnancy Considerations

Eclampsia

DIAGNOSIS

SIGNS AND SYMPTOMS

History

Ongoing disturbance of consciousness

Physical-Exam

- No spontaneous eye opening
- Lack of response to painful stimuli
- No motor activity
- Regular cardiorespiratory function
- Glasgow Coma Scale (GCS) scoring:
 - Eye opening (E):
 - Spontaneously: 4
 - To verbal command: 3
 - To pain: 2
 - No response: 1
 - Best motor response (M) to verbal command:
 - Obeys: 6
 - Best motor response to painful stimulus:
 - Localizes to pain: 5
 - Withdraws to pain: 4
 - Flexion—abnormal: 3
 - Extension—abnormal: 2
 - No response: 1
 - Best verbal response (V):
 - Oriented and converses: 5
 - Disoriented and converses: 4
 - Verbalizes: 3
 - Vocalizes: 2
 - No response: 1
 - $GCS = E + M + V$
- Hypothermia:
 - Infection, hypoglycemia, myxedema coma, alcohol and sedative-hypnotic poisoning
- Fever:
 - Infection, thyrotoxicosis, anticholinergics, sympathomimetics, neuroleptic malignant syndrome, hypothalamic hemorrhage
- HTN
- Structural lesion, hypertensive encephalopathy
- Hypotension
- Mydriasis:
 - Organophosphates
- Miosis:
 - Narcotics
 - Anticholinergics
 - Pontine lesion

- Loss of pupillary reflexes or unequal pupils:
 - Structural lesions
- Evidence of head trauma
- Nuchal rigidity:
 - Meningitis
 - Subarachnoid hemorrhage
- Decorticate posturing:
 - Flexion of elbows and wrists
 - Adduction and internal rotation of shoulders
 - Supination of the forearms
 - Suggests severe damage above the midbrain
- Decerebrate posturing:
 - Extension of elbows and wrists
 - Adduction and internal rotation of shoulders
 - Pronation of the forearms
 - Suggests damage at the midbrain or diencephalon
- Asymmetric movements:
 - Structural lesions
 - Persistent twitching of an extremity:
 - Status epilepticus

ESSENTIAL WORKUP

- Detect and treat reversible causes.
- Immediate exclusion of comalike states:
 - Noting resistance to passive opening of eyelids, fluttering of eyelids when stroked, abrupt eyelid closure, eye movements by saccadic jerks (rather than roving), or finding the eyes rolled back
 - Provocation of nystagmus with ice-water caloric testing
 - Before paralyzing a patient for intubation, an attempt should be made to detect a locked-in syndrome.
 - Demonstrating that the patient is able to blink on verbal command will establish this diagnosis.
 - Intubation is still indicated to prevent aspiration.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Dextrostix
- CBC
- Electrolytes
- Blood and urine toxicologic screen

Imaging

Head CT:

- Diagnosis of hemorrhage and midline shift
- CT angiography for suspected cerebrovascular accident

Diagnostic Procedures/Surgery

- Lumbar puncture:
 - _ All patients with coma of unknown etiology, particularly if fever is present
 - _ Antibiotics may be administered for as long as 48 hr before lumbar puncture.
 - _ CT should be performed before lumbar puncture if there is evidence of increased intracranial pressure, a mass lesion, pre-existing trauma, or focal findings.
- Risk of tonsillar herniation in patients with a mass lesion is very small.
- EEG:
 - _ Performed to rule out suspected seizure activities
 - _ Little use in the emergency evaluation
 - _ Unlike EEG studies performed in a lab, lighting will cause artifacts.

DIFFERENTIAL DIAGNOSIS

- Locked-in syndrome
- Psychogenic unresponsiveness
- Stupor
- Catatonia
- Akinetic mutism



TREATMENT

PRE HOSPITAL

- Airway management if loss of airway patency
- Endotracheal intubation if no response to coma cocktail
- IV access
- Dextrose or Dextrostix
- Narcan
- Monitor
- Look for signs of an underlying cause:
 - _ Medical alert bracelets
 - _ GCS
 - _ Pupils
 - _ Extremity movements

INITIAL STABILIZATION/THERAPY

- Airway management

- Empiric use of naloxone
- Empiric dextrose:
 - Administer if serum glucose cannot be measured at the bedside
 - Can safely be administered before thiamine
 - Does not worsen outcome in patients with stroke

ED TREATMENT/PROCEDURES

- Specific therapy directed at underlying cause once identified
- Consider empiric use of antibiotics for coma of undetermined etiology:
 - Broad-spectrum with good cerebrospinal fluid penetration such as ceftriaxone
- Stop seizure activity with benzodiazepines, phenytoin, and phenobarbital.
- Empiric treatment for a toxic ingestion:
 - Activated charcoal
- Correct body temperature:
 - Aggressive rewarming for patients with core temperature between 32°C and 35°C and invasive rewarming for <32°C
 - Ice packs and forced air movement over exposed wetted skin if severe hyperthermia

MEDICATION

- Ceftriaxone: 100 mg/kg IV
- Dextrose: 1–2 mL/kg of D₅₀W IV; neonate 10 mL/kg D₁₀W IV; peds 4 mL/kg D₂₅W IV
- Diazepam: 0.1–0.3 mg/kg slow IV (max. 10 mg/dose) q10–15min × 3 doses
- Flumazenil: 0.20 mg IV qmin × 1–5 doses
- Fomepizole: 15 mg/kg IV
- Lorazepam: 0.05–0.1 mg/kg IV (max. 4 mg/dose q10–15min)
- Mannitol: 0.25–1 g/kg IV over 20 min
- Naloxone: 0.01 mg/kg to 0.01–0.1 mg/kg
- Phenobarbital: 10–20 mg/kg IV, monitor for respiratory depression
- Phenytoin: Infuse at <50 mg/min; 18–20 mg/kg IV/IO or fosphenytoin 15–20 mg/kg IV/IO
- Physostigmine: 0.5–2 mg IV
- Thiamine: 100 mg IM or 100 mg thiamine in 1,000 mL of IV fluid wide open
- Pyridoxine: 70 mg/kg IV (Max. 5 g on a 1:1 basis with INH overdose)



FOLLOW-UP

DISPOSITION

Admission Criteria

Patients who do not have a readily identifiable and completely reversible cause of coma should be admitted.

Discharge Criteria

Comatose patients with correctable hypoglycemia and opiate toxicity who respond completely to aggressive ED treatment can be discharged.

Issues for Referral

Further delineation or prevention of possible adverse medication reaction

FOLLOW-UP RECOMMENDATIONS

- If discharged, urgent PCP F/U is needed.
- Consideration of adverse medication reaction
- Supervision for 24 hr postdischarge

PEARLS AND PITFALLS

- Rapid medical stabilization
- Neuroimaging for structural lesions
- Metabolic and toxicologic assessment
- Identification of unusual causes of coma
- Dischargeable patients require period of ED observation.

ADDITIONAL READING

- Glauser J. Coma: A systematic approach to patient evaluation and management 2008. Available at <http://www.thefreelibrary.com/Coma+A+Systematic+Approach+to+Patient+Ea0206595218>
- Plum F, Posner J. *The Diagnosis of Stupor and Coma*. 4th ed. Philadelphia, PA: FA Davis; 2007.
- *The Martin A. Samuels neurology review for primary care physicians*. Available at http://www.cmeinfo.com/store_temp/The_Martin_A._Samuels_Neurology_Review_for
- Weiner W, Shulman L. *Emergent and Urgent Neurology*. 2nd ed. Philadelphia, PA: Lippincott, Williams & Williams; 1999.

CODES

ICD9

780.01 Coma

ICD10

- R40.20 Unspecified coma
- R40.244 Oth coma,w/o Glasgow coma scale score,or w/part score report
- R40.2110 Coma scale, eyes open, never, unspecified time

COMPARTMENT SYNDROME

Chester D. Shermer



BASICS

DESCRIPTION

- Elevated tissue pressure in closed spaces that compromises blood flow through capillaries
- Normal tissue pressure is < 10 mm Hg.
- Capillary blood flow in a compartment is compromised at pressures > 20 mm Hg.
- Muscles and nerves can develop ischemic necrosis at pressures > 30 mm Hg.
- When distal pulses are diminished on exam, muscle necrosis is probably present.
- The 4 compartments of the leg are most frequently involved, but compartment syndrome can occur in the arm, forearm, hand, foot, shoulder, buttocks, and thigh.

ETIOLOGY

- Decreased compartment size: Circumferential cast, burn eschar, or military antishock trousers (MAST)
- Increased compartment contents: Compression of the compartment from edema or hematoma caused by direct trauma, fracture, overexertion of muscles, contrast extravasation, injection of recreational drugs, postischemic time, or limb compression during prolonged recumbency



DIAGNOSIS

ALERT

- Keep the extremity at the level of the heart to promote arterial flow but not diminish venous return.
- Do not use ice if compartment syndrome is suspected—it may compromise microcirculation.

SIGNS AND SYMPTOMS

- Severe, constant pain over the compartment that is disproportionate to extent of injury
- Pain increases with active contraction and passive stretching.
- Muscle weakness
- Hypesthesia
- 6 P's: Pain, pressure, paresis, paresthesia, and pulses present

History

- Elicit above symptoms in proper clinical setting.

- 6 P's

Physical-Exam

- Tenderness of muscle compartment
- Assess motor and neurologic function.

DIAGNOSIS TESTS & INTERPRETATION

Imaging

Radiographs should be performed if fracture is suspected.

Diagnostic Procedures/Surgery

- Measurement of compartment pressures with a system such as the Stryker IC pressure monitor system (Stryker Surgical, 2825 Airview Boulevard, Kalamazoo, MI 49002. www.stryker.com), using an 18G needle or continuous pressure monitoring with the attachment for an indwelling catheter
- Technique is as follows:
 - Prep overlying skin with antiseptic solution.
 - Local anesthetic can be infiltrated into the *SC tissue only*, taking care not to inject intramuscularly.
 - The needle used for pressure measurements is advanced through the skin until a popping sensation is felt when the fascia is pierced.
 - 0.2 mL of saline is injected to clear the lumen of the needle, and the intracompartmental pressure measurement is then read.
 - To ascertain correct placement of the needle within the compartment, external pressure may be applied over the muscle compartment, or the muscles can be passively stretched to increase the intracompartmental pressure transiently; once these maneuvers are discontinued, the pressure should drop to baseline and stabilize.

DIFFERENTIAL DIAGNOSIS

- Chronic compartment syndrome
- Fascial hernia
- Stress fracture
- Arterial occlusion
- Neurapraxia
- Deep venous thrombosis
- Cellulitis
- Osteomyelitis
- Tenosynovitis
- Synovitis



TREATMENT

INITIAL STABILIZATION/THERAPY

- Acutely injured extremities that are casted should have the cast univalved and spread and underlying cast padding should be cut.
- Keep the extremity at the level of the heart.

ED TREATMENT/PROCEDURES

- Acute compartment syndrome is a surgical emergency.
- Mainstay of treatment is fasciotomy, particularly for compartment pressures $> 30\text{--}40$ mm Hg.

MEDICATION

- Medications are not helpful, including steroids or vasodilators, in the treatment of compartment syndrome.
- Pain medication is essential after diagnosis is made or consultant evaluation is begun.

First Line

IV narcotic analgesics may provide some relief, although the pain is frequently so severe that only decompression in the OR can provide relief.

Second Line

Oral narcotic analgesics and nonsteroidal agents are of very little benefit acutely.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Emergent orthopedic or surgical consultation for compartment pressures > 30 mm Hg
- For compartment pressures > 20 mm Hg but < 30 mm Hg, surgical consultation should be sought and the patient admitted.
- For compartment pressures between 15 and 20 mm Hg, serial measurement of pressures should be taken; if the patient cannot be relied on to return for repeat measurements, the patient should be admitted.

Discharge Criteria

Compartment pressure $< 10\text{--}15$ mm Hg: Patients should be given symptomatic treatment and instructed to return for increased pain, swelling, development of

paresthesia.

Issues for Referral

If the clinician suspects chronic compartment syndrome, then prompt referral to orthopedic surgeon is necessary. Direct communication is best to express your concerns.

PEARLS AND PITFALLS

- Must measure compartment pressures or arrange transfer to higher level of care if capability is lacking.
- Care must be taken when measuring compartment pressures to avoid injury to tendons, nerves, and blood vessels.
- Must consider concomitant rhabdomyolysis in crush-type injuries.

ADDITIONAL READING

- Farr D, Selesnick H. Chronic exertional compartment syndrome in a collegiate soccer player: A case report and literature review. *Am J Orthop*. 2008;37(7):374–377.
- Mabee JR. Compartment syndrome: A complication of acute extremity trauma. *J Emerg Med*. 1994;12(5):651–656.
- Reis ND, Better OS. Mechanical muscle-crush injury and acute muscle-crush compartment syndrome. *J Bone Joint Surg Br*. 2005;87(4):450–453.
- Sahni V, Garg D, Garg S, et al. Unusual complications of heroin abuse: transverse myelitis, rhabdomyolysis, compartment syndrome, and ARF. *Clin Toxicol (Phila)*. 2008;46:153.



See Also (Topic, Algorithm, Electronic Media Element)

- Acute Compartment Syndrome <http://emedicine.medscape.com/article/307668-overview>
- Compartment Syndromes of Extremities [https://online.epocrates.com/u/2911502/Compartment + syndrome + of + extremity](https://online.epocrates.com/u/2911502/Compartment+syndrome+of+extremity)
ICID = EMN0909



CODES

ICD9

- 958.90 Compartment syndrome, unspecified
- 958.91 Traumatic compartment syndrome of upper extremity
- 958.92 Traumatic compartment syndrome of lower extremity

ICD10

- T79.A0XA Compartment syndrome, unspecified, initial encounter
- T79.A19A Traumatic compartment syndrome of unsp upper extremity, init
- T79.A29A Traumatic compartment syndrome of unsp lower extremity, init

CONGENITAL HEART DISEASE, ACYANOTIC

Lynne M. Palmisciano • William J. Lewander

BASICS

DESCRIPTION

Abnormality in the cardiocirculatory system that is present at birth but does not cause mixing of deoxygenated and oxygenated blood:

- L→R shunting lesions:
 - Ventricular septal defect (VSD)
 - Atrial septal defect (ASD)
 - Patent ductus arteriosus (PDA)
 - Endocardial cushion defects (AV canal)
- Ventricular outflow obstructions:
 - Coarctation of aorta (LV)
 - Aortic stenosis (LV)
 - Pulmonic stenosis (RV)
 - Hypoplastic left-heart syndrome (HLHS)
- Ductal dependent: Symptoms as DA closes:
 - Coarctation of aorta
 - Critical aortic stenosis
 - Critical pulmonic stenosis
 - HLHS

ETIOLOGY

For most forms, cause is unknown:

- Genetic: Down (AV canal), Turner (coarct)
- Environmental: Congenital rubella (PDA, AS)

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Many asymptomatic
- Lethargy, poor feeding, and failure to thrive
- Dyspnea on exertion
- Recurrent respiratory infections

Physical-Exam

- VSD and AV canal:

- Dusky color, hepatomegaly
- Holosystolic and diastolic murmurs + thrill
- Hyperdynamic precordium, displaced PMI
- ASD:
 - Fixed, split S2
 - Systolic ejection and diastolic murmurs
- PDA:
 - “Machine-like” murmur and bounding pulses
- Coarctation:
 - Differential cyanosis (pink only upper 1/2)
 - BP upper extremities > BP lower extremities
 - ↓ or absent lower-extremity pulses
- AS:
 - Harsh systolic murmur, thrill, aortic click
- PS:
 - Systolic ejection murmur, thrill, pulmonic click
 - Widely split S2
 - Jugular venous A-waves
- HLHS:
 - Dusky, listless, tachypneic, ↓ pulses
 - Single heart sound, systolic ejection murmur

ESSENTIAL WORKUP

- Oxygen saturation (pre- and postductal)
- ABG, CBC, basic chemistries, and glucose
- Sepsis evaluation
- CXR to assess pulmonary blood flow
- EKG (axis, hypertrophy, conduction delays)
- 4-extremity BPs
- Cardiology consult with ECG

DIAGNOSIS TESTS & INTERPRETATION

Imaging

- CXR:
 - L→R shunting lesions all show cardiomegaly (specific chambers) and ↑ pulmonary markings
 - ASD (RA, RV), VSD (RV, LA), PDA (LA, LV)
 - AV canal (globular; all chambers enlarged)
- Obstructive lesions: Normal to cardiomegaly

Diagnostic Procedures/Surgery

EKG:

- ASD: Right axis deviation:
 - RVH or right bundle branch block (RBBB)
- VSD–LAH, LVH (if large, also RVH):
 - Notched or peaked P-waves (large VSD)
- PDA: Biventricular hypertrophy (large PDA)
- AV canal: Superior axis, LVH, RVH:
 - RBBB and prolonged PR interval
- AS: Normal to LVH (severe cases)
- PS: Normal to RVH, RAE (severe cases):
 - RBBB
- Coarctation of aorta: RVH or RBBB
- HLHS: RAE, RVH, peaked P-waves

DIFFERENTIAL DIAGNOSIS

- CHF
- Hypertrophic cardiomyopathy
- Cardiogenic shock
- Aortic dissection
- Myocarditis
- Bronchopulmonary dysplasia
- Pulmonary HTN
- Pneumonia/bronchiolitis
- Hypoglycemia
- Adrenal insufficiency, CAH
- Glycogen storage diseases
- Sepsis
- Shock



TREATMENT

INITIAL STABILIZATION/THERAPY

- Maintain warmth and oxygenation.
- Treat hypoglycemia and acidosis.
- Establish IV access.
- Prepare for endotracheal intubation.

ALERT

High oxygen tensions promote ductal closure.

ED TREATMENT/PROCEDURES

- Administer prostaglandin E₁ (PGE₁) to dilate or reopen the ductus arteriosus:
 - Continuous IV infusion 0.05–0.1 µg/kg/min

- Complications include apnea, bradycardia, hypotension, and seizures.
- Evaluate and treat alternate causes:
 - Septic workup and empiric antibiotics
 - Maintain normoglycemia
- Circulatory collapse from CHD:
 - Fluid resuscitation (increments of 10 mL/kg)
 - Inotropes
 - Aggressive treatment of acidosis
- CHF:
 - Digoxin and diuretics

MEDICATION

- Ampicillin 50 mg/kg IV
- Digoxin dosing requires extreme caution:
 - Range 25–40 µg/kg IV
- Dobutamine: 5–20 µg/kg/min IV
- Dopamine: 2–20 µg/kg/min IV
- Epinephrine: 0.1–2 µg/kg/min IV
- Furosemide: 1 mg/kg IV
- Gentamicin: 4 mg/kg/d IV or 2.5 mg/kg/dose
- Milrinone 0.25–1 µg/kg/min
- PGE₁: 0.05–0.1 µg/kg/min
- Sodium bicarbonate: 1–2 mEq/kg IV



FOLLOW-UP

DISPOSITION

Admission Criteria

- All newborns with suspected CHD:
 - Admit to pediatric ICU.
- CHD with acute worsening of cyanosis or CHF
- CHD with pneumonia or bronchiolitis

Discharge Criteria

Determine in consult with cardiologist

Issues for Referral

Primary care physician to coordinate care with cardiologist and cardiothoracic surgery

FOLLOW-UP RECOMMENDATIONS

Plan for follow-up should be made in consult with the pediatric cardiologist.

PEARLS AND PITFALLS

- Acyanotic lesions presenting at 2–12 wk:
 - Coarctation as DA closes
 - Septal defects as pulmonary vascular resistance drops
- Classic ECG in AV canal: Superior QRS axis
- Classic CXR in coarctation: Rib notching (late)

ADDITIONAL READING

- Bonow RO, Mann DL, Zipes DP, et al., eds. Congenital heart disease. *Braunwald's Heart Disease*. 98th ed. Philadelphia, PA: Saunders Elsevier; 2012:1411–1467.
- Dolbec K, Mick N. Congenital heart disease. *Emerg Med Clin North Am*. 2011;29:811–827.
- Yee L. Cardiac emergencies in the first year of life. *Emerg Med Clin North Am*. 2007;25:981–1008.



See Also (Topic, Algorithm, Electronic Media Element)

- Congestive Heart Failure
- Failure to Thrive
- Neonatal Sepsis



CODES

ICD9

- 745.4 Ventricular septal defect
- 745.5 Ostium secundum type atrial septal defect
- 746.89 Other specified congenital anomalies of heart

ICD10

- Q21.0 Ventricular septal defect
- Q21.1 Atrial septal defect
- Q24.8 Other specified congenital malformations of heart

CONGENITAL HEART DISEASE, CYANOTIC

Lynne M. Palmisciano • William J. Lewander



BASICS

DESCRIPTION

- Aberrant embryonic development results in mixing of deoxygenated and oxygenated blood returning to systemic circulation by 2 mechanisms:
 - Right-to-left intracardiac shunt
 - Anatomic defects of the aortic root
- Subtypes: 5 T's, 2 E's, single ventricle:
 - Tetralogy of Fallot (TOF):
 - Ventricular septal defect (VSD)
 - Right ventricular (RV) outflow obstruction
 - Overriding aorta
 - RV hypertrophy (RVH)
 - Transposition of the great arteries (TGA):
 - Aorta arises from RV and pulmonary artery from left ventricle (LV)
 - Tricuspid atresia:
 - No outlet from right atrium to RV
 - Obligatory atrial level connection
 - Truncus arteriosus:
 - Single arterial trunk for systemic, pulmonic, and carotid circulations
 - Total anomalous pulmonary venous return (TAPVR):
 - Pulmonary veins drain into systemic venous circulation
 - Supracardiac, cardiac, infracardiac, or mixed
 - Ebstein anomaly of tricuspid valve:
 - Abnormal and displaced tricuspid valve divides RV resulting in poor RV function
 - Eisenmenger syndrome:
 - Complication in longstanding acyanotic heart disease with L→R shunts
 - Pulmonary vascular resistance reaches suprasystemic levels; R→L shunt
 - Single ventricle physiology:
 - Total mixing of systemic and venous return

ETIOLOGY

For most forms, cause is unknown



DIAGNOSIS

- Most common initial ED presentations of cyanotic congenital heart disease (CHD):
 - Cyanosis
 - CHF
 - Circulatory collapse
- Physiologic stress triggers cyanosis in older patients with CHD:
 - Cardiac shunt obstruction
 - Pulmonary disease
 - Decreased systemic vascular resistance
 - Fever
 - Dehydration

SIGNS AND SYMPTOMS

- Central cyanosis:
 - Visible in lips, nail beds, mucosa
 - Increases with cry or agitation
 - Minimal change with 100% O₂
- CHF:
 - Rales, gallop, hepatomegaly, scalp edema
- Hypercyanotic spells or “Tet spells”:
 - Restless and hyperpneic then increased cyanosis then syncope
 - Follows reductions in already compromised pulmonary blood flow:
 - Wakening, feeding, vigorous cry, exercise
- Older child may compensate by squatting.
- Temporary reduction or absence of systolic ejection murmur during spell

History

- Family history of CHD:
 - If parent or sibling: Increased risk of CHD
 - If 2 relatives: Risk of CHD triples
- Prenatal history:
 - Exposure to teratogens
 - Abnormal fetal ultrasound
- TOF:
 - Often asymptomatic at birth; symptoms develop as RV infundibulum hypertrophies
 - Severe RV outflow obstruction; neonatal cyanosis (duct-dependent lesion)
 - Cyanosis during crying or feeding (Tet spells) in toddlers
 - Older, uncorrected patients:
 - Dyspnea on exertion and growth delay
- Tricuspid atresia:
 - Usually cyanotic from birth
 - Feeding difficulties

- Older patients show dyspnea on exertion and easy fatigability
- Ebstein anomaly:
 - Teens may present with dysrhythmias.
- TGA:
 - Presents in 1st hr to days of life
- TAPVR:
 - Neonatal presentation; severely ill:
 - Cyanosis
 - Does not improve with mechanical ventilation
 - Infantile presentation; heart failure:
 - Mild cyanosis
 - If no obstruction to pulmonary venous return:
 - Asymptomatic or mild cyanosis
 - Frequent pneumonias
 - Growth problems
- Truncus arteriosus:
 - Mild cyanosis in newborn
 - Heart failure in older infants

Physical-Exam

- TOF:
 - Loud systolic murmur left sternal border (LSB)
 - Systolic thrill in 50%
 - +/- Continuous murmur of PDA
 - Loud, single 2nd heart sound (S2)
 - RV prominence/bulge
 - Older, uncorrected patients:
 - Dusky, blue skin
 - Clubbing of digits
 - Retinal engorgement
- Tricuspid atresia:
 - Tachypnea
 - Regurgitant murmur from VSD at LSB
 - +/- continuous PDA murmur
 - Single S2
 - Prominent LV impulse
- Ebstein anomaly:
 - Holosystolic murmur of tricuspid regurgitation
 - Many have diastolic murmur
 - Gallop
- TGA:
 - Single, loud S2
 - Severe hypoxemia

- TAPVR:
 - Neonatal; severe tachypnea and cyanosis
 - Infantile; heart failure:
 - Tachycardia
 - Systolic ejection murmur at LSB
 - Mid-diastolic murmur at lower LSB
 - Gallop
 - Fixed, split S2
 - Hepatomegaly
- Truncus arteriosus:
 - Newborn:
 - Mild cyanosis
 - Regurgitant systolic murmur at LSB
 - Single S2
- Older infants; heart failure:
 - Hyperdynamic precordium
 - Bounding pulses
 - Wide pulse pressure
 - Loud, single S2
 - Systolic ejection murmur and thrill
 - Mid-diastolic murmur

ESSENTIAL WORKUP

- Oxygen saturation
- ABG
- CBC, glucose
- Sepsis evaluation
- CXR to assess pulmonary blood flow
- EKG to assess for hypertrophy and QRS axis
- Cardiology consult and ECG

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Decreased room air oxygen saturation
- Hyperoxia test:
 - ABG in room air and after several minutes 100% oxygen:
 - $\text{PaO}_2 > 150$ in O_2 ; no intracardiac shunt
 - $\text{PaO}_2 < 100$ in O_2 ; highly suspicious of cyanotic CHD
- CBC: Erythrocytosis with chronic cyanosis

Imaging

- CXR:
 - Decreased pulmonary blood flow:
 - TOF (enlarged RV)
 - Tricuspid atresia (enlarged LV)
 - Single ventricle physiology
 - Increased pulmonary flow:
 - Transposition of the great vessels (large RV)
 - TAPVR (large RV)
 - Truncus arteriosus (large LV and RV)
 - Classic CXR descriptions:
 - Boot-shaped heart: TOF; large and upturned RV looks like toe of boot
 - Egg on a string: TGA; narrow mediastinum, great vessels anterior/posterior position
 - Snowman sign: Supracardiac TAPVR; upper portion formed by pulmonary veins

Diagnostic Procedures/Surgery

EKG:

- TOF:
 - Right axis deviation (RAD)
 - RVH
- TAPVR:
 - RAD
 - RVH and right atrial enlargement (RAE)
- Transposition of the great vessels:
 - RAD
 - RVH
- Tricuspid atresia:
 - Superior axis
 - LVH, RAE, LAE
- Truncus arteriosus:
 - RVH, LVH
- Ebstein anomaly:
 - Right bundle branch block
 - Often Wolff–Parkinson–White

DIFFERENTIAL DIAGNOSIS

- Pulmonary:
 - Pneumothorax/hemothorax
 - Bronchopulmonary dysplasia
 - Congenital lung hypoplasia/dysplasia
 - Pulmonary hemorrhage
 - Pulmonary embolus

- Pulmonary HTN
- Diaphragmatic hernia
- Foreign body/anatomic obstruction
- Cardiac:
 - Cyanotic CHD
 - CHF
 - Cardiogenic shock
- Infectious:
 - Pneumonia, bronchiolitis
 - Sepsis
- Neurologic:
 - Seizure
 - Neuromuscular disease
 - Drug-induced respiratory depression
- Other:
 - Polycythemia
 - Methemoglobinemia
 - Dehydration
 - Hypoglycemia



TREATMENT

INITIAL STABILIZATION/THERAPY

- Maintain warmth (cold \uparrow O_2 consumption).
- Treat hypoglycemia and acidosis.
- Maintain oxygenation.
- Establish IV access.
- Prepare for endotracheal intubation.

ALERT

- High oxygen tensions promote ductal closure.
- Place air filters on all IV lines to avoid paradoxical emboli through R \rightarrow L shunt.

ED TREATMENT/PROCEDURES

- Administer prostaglandin E_1 (PGE_1) to dilate or reopen the ductus arteriosus:
 - Continuous IV infusion 0.05–0.1 $\mu\text{g}/\text{kg}/\text{min}$
 - Complications include apnea, bradycardia, hypotension, and seizures:
 - Generally intubate prior to transport
 - Not effective for obstructed TAPVR:
 - May require ECMO awaiting surgery
 - Overall benefits far outweigh potential risks
- Evaluate and treat alternate causes of cyanosis:

- Septic workup and empiric antibiotics
- Fluid resuscitate (increments of 10 mL/kg)
- Maintain normoglycemia
- Patients with Tet spells:
 - Provide a calming environment.
 - Place child in knee-chest position.
 - Supplemental O₂ if not agitating to patient
 - IV or IM morphine
 - For severe cases not responding to above:
 - IV bicarbonate to treat severe acidosis
 - IV phenylephrine to ↑systemic vascular resistance and reduce R→L shunt
 - IV propranolol for β-adrenergic blockade
- Cyanosis in the older patient with known CHD:
 - 10–20 mL/kg NS IV if dehydration likely
 - Supplemental O₂ if suspicious for pulmonary diseases
 - Antipyretics for fever
 - Antibiotics for pneumonia/infectious process
- Circulatory collapse from CHD:
 - Fluid resuscitation
 - Inotropes: Dobutamine, dopamine, milrinone
 - Aggressive treatment of acidosis

MEDICATION

- Acetaminophen: 15 mg/kg PO or PR
- Ampicillin: 50 mg/kg IV
- Dobutamine: 5–20 µg/kg/min IV
- Dopamine: 5–20 µg/kg/min IV
- Gentamicin: 4 mg/kg/d IV or 2.5 mg/kg/dose
- Ibuprofen: 10 mg/kg PO (>6 mo)
- Milrinone: 0.25–1 µg/kg/min
- Morphine sulfate: 0.1 mg/kg SC, IM, or IV
- Phenylephrine: 0.5–5 µg/kg/min IV
- Propranolol: 0.1 mg/kg IV
- PGE₁: 0.05–0.1 µg/kg/min
- Sodium bicarbonate: 1–2 mEq/kg IV



FOLLOW-UP

DISPOSITION

Admission Criteria

- All newborns with suspected CHD:
 - Admit to pediatric ICU.
- CHD with acute worsening of cyanosis or CHF
- CHD with symptomatic pneumonia or respiratory syncytial virus

Discharge Criteria

- Determine in consult with cardiologist
- Patients who respond to minimal intervention (i.e., TOF patients treated noninvasively)
- Ensure close follow-up.

Issues for Referral

- Primary care physician to coordinate care
- Cardiologist for diagnosis, medical management, and ongoing monitoring
- Cardiothoracic evaluation for surgery

FOLLOW-UP RECOMMENDATIONS

- Plan for follow-up should be determined in consult with the pediatric cardiologist.
- Clear instructions for return visits, as any physiologic stress may worsen condition.

PEARLS AND PITFALLS

- Visual appearance of cyanosis requires $> 3\text{--}5$ mg/dL deoxygenated hemoglobin.
- Duct-dependent lesions:
 - Present at 2–3 wk of age
 - Sudden cyanosis or cardiovascular collapse
 - Treat with PGE1:
 - Beware apnea and hypotension

ADDITIONAL READING

- Apitz C, Webb GD, Redington AN. Tetralogy of Fallot. *Lancet*. 2009;374:1462–1471.
- Bonow RO, Mann DL, Zipes DP, et al., eds. Congenital heart disease. *Braunwald's Heart Disease*. 98th ed. Philadelphia, PA: Saunders Elsevier; 2012:1411–1467.
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- Fleisher GR, Ludwig S, Bachur RG, et al., eds. Cardiac emergencies. *Textbook of Pediatric Emergency Medicine*. 6th ed. Philadelphia, PA: Lippincott Williams, & Wilkins, 2010:690–701.
- Yee L. Cardiac emergencies in the first year of life. *Emerg Med Clin North Am*. 2007;25:981–1008.



CODES

ICD9

- 745.2 Tetralogy of fallot
- 745.4 Ventricular septal defect
- 746.89 Other specified congenital anomalies of heart

ICD10

- Q21.0 Ventricular septal defect
- Q21.3 Tetralogy of Fallot
- Q24.8 Other specified congenital malformations of heart

CONGESTIVE HEART FAILURE

Naomi George • Robert A. Partridge

BASICS

DESCRIPTION

- A clinical syndrome in which the heart fails to maintain adequate circulation for metabolic needs, characterized by chronic debility, acute decompensation, and high mortality.
- Acute Decompensated Heart Failure (ADHF) is a rapidly progressive failure state (hr–days)
 - Common reason for presentation to the ED
 - Usually caused by a precipitating event in which the heart does not have the reserve to compensate for the added burden
- Chronic HF is a progressive failure state (mo–yr) characterized by cardiac remodeling and neurohormonal changes, with multiple subclasses:
 - Systolic heart failure
 - Impaired contractile or pump function causing decreased ejection fraction
 - Diastolic heart failure
 - Impaired ventricular relaxation resulting in decreased cardiac filling
 - Low-output failure
 - Decreased cardiac output
 - High-output failure:
 - Normal or increased cardiac output, but insufficient to meet metabolic demands
 - Left-sided failure
 - Systolic or diastolic (or both) dysfunction of the left ventricle
 - Resultant pulmonary congestion
 - Right-sided heart failure
 - Due to either intrinsic dysfunction or secondary to left heart failure or pulmonary hypertension (cor pulmonale)
 - Hepatic enlargement, JVD, and dependent edema can occur
- CHF affects ~5.8 million Americans.
- Estimated 2012 cost of CHF is \$40 billion
- ADHF is the leading Medicare diagnosis for hospitalized patients ≥ 65 yr old.

ETIOLOGY

Underlying causes and acute precipitants

- Decreased myocardial contractility:
 - Myocardial ischemia/infarction

- Cardiomyopathy (including, alcoholic and pregnancy-related)
- Myocarditis
- Dysrhythmias
- Decreased contractile efficiency:
 - Drug related (negative inotropes)
 - Metabolic disorders
- Pressure overload states:
 - HTN
 - Valvular abnormalities
 - Arrhythmia
 - Congenital heart disease
 - Pulmonary embolism
 - Primary pulmonary hypertension, sleep apnea syndromes (right heart failure)
- Restricted cardiac output:
 - Myocardial infiltrative disease
- Volume overload:
 - Dietary indiscretion (sodium overload)
 - Drugs leading to sodium retention (glucocorticoids, NSAIDs)
 - Overload due to transfusion or IV fluid
- High demand states:
 - Hyperthyroidism, thyrotoxicosis
 - Pregnancy
 - A-V fistula
 - Beriberi (thiamine deficiency)
 - Paget disease
 - Severe anemia
 - Aortic insufficiency
- Pediatric etiologies: Volume/pressure overload lesions vs. acquired HD:
 - 1st 6 mo: VSD and PDA
 - Older children: Subvalvular aortic stenosis, coarctation
 - Acquired dysfunction: Nonspecific age of onset, including myocarditis, valvular disease, and cardiomyopathies; cocaine/stimulant abuse in adolescents



DIAGNOSIS

SIGNS AND SYMPTOMS

- Poor perfusion:
 - Fatigue, somnolence, lightheadedness
 - Palpitations, or irregular pulse
 - Shortness of breath

- Cool extremities
- Worsening renal function
- Congestion
 - Dyspnea, cough
 - Orthopnea
 - Paroxysmal nocturnal dyspnea
 - Evidence of sleep disordered breathing
 - Decreased exercise tolerance
 - Elevated JVD or abdominojugular reflex
 - Dependent edema (poor sensitivity and specificity)
 - Rales and/or wheezing, (absent in 80% with chronically elevated filling pressure due to compensatory lymphatic drainage)
 - Pleural effusion, dullness at lung bases
 - S3 gallop and/or S4.
 - Laterally displaced apical impulse
 - Hepatic enlargement/tenderness
 - Nausea
 - Ascites
- ADHF with hemodynamic instability:
 - Confusion, anxiety, syncope
 - Tachypnea
 - Tachycardia
 - Hypotension
 - Cool, pale or cyanotic extremities
 - Narrow pulse pressure or pulsus alternans
 - Cheyne–Stokes respirations

ESSENTIAL WORKUP

- The CXR is important in confirming the diagnosis and assessing severity.
- 12-hr radiographic lag from onset of symptoms may occur.
- Radiographic findings may persist for several days despite clinical improvement.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Chemistry/electrolytes:
 - Establish baseline renal function when initiating diuretics, or ACE inhibitors
 - Hyperkalemia possible with low output
 - Hyponatremia associated with poor prognosis
- CBC:
 - Anemia can cause or exacerbate failure
 - Infection can cause or exacerbate failure
- Liver function tests:

- Increase suggests hepatic congestion, or ischemia.
- Thyroid function tests:
 - Specifically in patients >65 yr old or in a-fib
- Cardiac enzymes:
 - Evaluate for ischemia or infarction
- ANA and rheumatoid factor: Suspected lupus
- Viral panel: Suspected myocarditis
- BNP:
 - Useful for distinguishing cardiac vs. pulmonary cause of dyspnea
 - BNP >500 pg/mL, HF likely (ppv 90%)
 - BNP <100 pg/mL, HF unlikely, (npv 90%)
 - BNP 100–500 pg/mL, consider PE, cor pulmonale, renal failure, or stable underlying HF.
 - REDHOT II Study: BNP levels are better than physicians at predicting which patients are more likely to have bad outcomes
 - EPs were blinded to BNP values. 78% of patients discharged from ED had BNP >400.
 - Of those discharged with a BNP >400, 90-day mortality was 9%
 - BNP levels rise with age and are affected by gender, comorbidity, and drug therapy and should not be used in isolation
 - BNP levels may be low in acute pulmonary edema (<1–2 hr) and obesity (BMI >30).
- NT-proBNP: Cleavage product of prohormone.
 - NT-proBNP >1,000 pg/mL predictive of HF
 - NT-proBNP <300 pg/mL unlikely to be HF

Imaging

- CXR:
 - Cardiomegaly (sensitive)
 - Specific signs of CHF:
 - Cephalization (vascular prominence in the upper lungs due to fluid overload)
 - Interstitial edema/Kerley B lines
 - Alveolar edema
 - Effusions (usually right sided)
 - Bilateral confluent perihilar infiltrates leading to classic butterfly pattern:
 - May be asymmetric and mistaken for pneumonia
- EKG:
 - Underlying cardiac ischemia
 - Presence of dysrhythmias
 - Left-ventricular hypertrophy
 - Heart block
 - Normal EKG has high negative predictive value for systolic dysfunction.

- 2-D Cardiac Echo:
 - Ejection fraction
 - Acute valvular pathology
 - Pericardial tamponade
 - Pericardial thickening in constrictive pericarditis
 - Ventricle dilation, or hypertrophy
 - Regional wall motion abnormalities

DIFFERENTIAL DIAGNOSIS

- Left-sided CHF:
 - Acute exacerbation of COPD
 - Asthma exacerbation
 - Acute respiratory distress syndrome
 - Pneumonia, bronchitis
 - Constrictive pericarditis
 - Anemia, malnutrition
 - Pericardial tamponade
 - Coarctation of aorta
- Right-sided HF:
 - Nephrotic syndrome, chronic renal failure
 - Cirrhosis
 - Left-side heart failure
 - Pulmonary embolism
 - Sleep disordered breathing
 - Venous stasis



TREATMENT

PRE HOSPITAL

- IV access
- Supplemental oxygen
- Cardiac monitor and pulse oximetry
- EKG
- Sublingual nitrates for active chest pain without hypotension
- Furosemide
- Endotracheal intubation may be required.

INITIAL STABILIZATION/THERAPY

- IV access
- Supplemental oxygen
- Cardiac monitor and pulse oximetry
- EKG

- Elevate head of bed to reduce venous return.
- Control airway as needed:
 - Noninvasive positive pressure ventilation
 - CPAP vs. BiPAP
 - Reduce work of breathing, improve oxygenation, decrease need for intubation, possible mortality benefit
 - Some studies report higher incidence of MI with BiPAP over CPAP in acute CHF; studies not conclusive
 - Intubation for impending respiratory failure

ED TREATMENT/PROCEDURES

- General: Oxygenate, ventilate, treat underlying condition when possible
- Congestion with adequate perfusion: Reduce preload, consider fluid restriction
 - Rapidly reduce preload in acute pulmonary edema:
 - Sublingual or IV nitroglycerin
 - Nitro paste
 - IV diuretics (less rapid/effective in patients with poor renal perfusion)
 - Avoid preload reduction in ADHF when suspected etiology is aortic stenosis, HOCM, or pulmonary hypertension.
 - Cautious afterload reduction in ADHF: Avoid ACEi and ARBs in cases of hypotension, acute renal failure, and hyperkalemia.
 - Nesiritide
 - Limited benefit, may cause hypotension
- Poor perfusion with hypotension:
 - Agents that increase contractility:
 - Dobutamine
 - Dopamine
 - Milrinone
 - Avoid vasodilators (nitrates, morphine)
 - Initiate diuretics after inotropes.
- Initiate venous thromboembolism prophylaxis in those with ADHF without contraindications

Pediatric Considerations

- Neonates (1st weeks of life):
 - Suspect ductal-dependent cardiac lesions if clinical CHF and no improvement with O₂:
 - PGE1 to maintain patent ductus
- Children:
 - IV furosemide, and dopamine or milrinone
 - IV nitroglycerin for pulmonary edema

MEDICATION

- Aspirin: 325 mg PO/PR if AMI is suspected
- Bumetanide (Bumex): 1–3 mg IV, max. 10 mg/day
- Dobutamine: 2–10 µg/kg/min IV, max. of 40 µg/kg/min
- Dopamine: 2–20 µg/kg/min IV, max. of 50 µg/kg/min
- Enalapril: 0.625–1.25 mg IV; 2.5–20 mg/d PO
- Furosemide (Lasix): No prior use: 40 mg IVP; prior use: Double 24-hr dose (80–180 mg IV); no effect in 30 min: Redouble dose
- Milrinone: 50 µg/kg IV load; 0.375–0.75 µg/kg/min IV
- Nesiritide: 2 µg/kg bolus, then infusion of 0.01 µg/kg/min
- Nitroglycerin: 0.4 mg sublingual; 1–2 in of nitro paste; 5–20 µg/min IV, max. of 100–200 µg/min IV. USE NON-PVC tubing.
- Nitroprusside: 0.3–10 µg/kg/min IV (starting dose), max. of 10 µg/kg/min

Pregnancy Considerations

ACEi and ARBs are associated with multiple fetal abnormalities and should be held

- Oxygen
- Nitroglycerin
- Furosemide



FOLLOW-UP

DISPOSITION

Admission Criteria

- ICU:
 - Pulmonary edema
 - Cardiogenic shock
 - Concomitant MI or ischemia
- Medical wards:
 - New-onset CHF
 - Symptoms not relieved by ED therapy

Discharge Criteria

- Mild exacerbation of chronic CHF:
 - Responds to ED treatment
 - No other cardiac and pulmonary findings
- Close follow-up should be arranged with continuation of diuretic, vasodilator, or ACE inhibitor therapy and patient lifestyle education.

Issues for Referral

Consider ICD and/or BV pacer in advanced HF

- Shown to decrease mortality and hospitalization rates in select patient groups

FOLLOW-UP RECOMMENDATIONS

- Close follow-up within 1 wk of discharge
- Medication and dietary compliance
- Frequent home monitoring of body weight
- Monitor electrolytes and renal function during chronic diuretic therapy

PEARLS AND PITFALLS

- BNP may be useful if CHF diagnosis uncertain.
- In severe CHF, NIPPV can improve impending respiratory compromise.
- Be vigilant in searching for and treating the underlying cause of the heart failure exacerbation (e.g., MI, PE, valvular pathology).

ADDITIONAL READING

- Heart Failure Society of America. Executive summary: HFSA 2010 Comprehensive Heart Failure Practice Guideline. *J Card Fail.* 2010;16(6):475–535.
- Singer AJ, Birkhahn RH, Guss D, et al. Rapid Emergency Department Heart Failure Outpatients Trial (REDHOTII): A randomized controlled trial of the effect of serial B-type natriuretic peptide testing on patient management. *Circ Heart Failure.* 2009;2:287–293.
- Silvers SM, Howell JM, Kosowsky JM, et al. ACEP Clinical policy: Critical issues in the evaluation and management of adult patients presenting to the emergency department with acute heart failure syndromes. *Ann Emerg Med.* 2007;49(5):627–669.



CODES

ICD9

- 428.0 Congestive heart failure, unspecified
- 428.20 Systolic heart failure, unspecified
- 428.30 Diastolic heart failure, unspecified

ICD10

- I50.9 Heart failure, unspecified
- I50.20 Unspecified systolic (congestive) heart failure
- I50.30 Unspecified diastolic (congestive) heart failure

CONJUNCTIVITIS

Jessica Freedman

BASICS

DESCRIPTION

Inflammation of the conjunctiva arising from a broad group of etiologies. Commonly referred to as “pink eye.”

ETIOLOGY

- Bacterial:
 - *Staphylococcus aureus*
 - *Streptococcus pneumoniae*
 - *Haemophilus influenzae*
 - Gonococcal:
 - Ophthalmic emergency
 - Chlamydia:
 - Transmission occurs via autoinoculation from genital secretions.
 - Often occurs in newborns
- Viral:
 - Adenovirus most common
 - Epidemic keratoconjunctivitis (EKC) is caused by adenovirus subtypes.
 - Frequently associated with upper respiratory infections or exposure to someone with a red eye
 - Most commonly referred to as “pink eye”
 - Herpes simplex virus (HSV)
 - Recurrent ocular infection occurs in 25% patients within 2 yr.
 - Use of steroids is contraindicated:
 - Allergic
 - Frequent history of allergy, atopy, nasal symptoms
 - Contact related
 - May be due to chemical irritation, hypersensitivity from preservatives, medications, shampoo, chlorine, dust, smoke
 - Pseudomonas commonly implicated organism:
 - May be found in patients using saliva to clean contact lenses

DIAGNOSIS

SIGNS AND SYMPTOMS

- General:
 - Red eye (conjunctival irritation)

- Gritty, foreign body sensation
- Sensation of eyes burning
- Discharge
- Eyelid sticking (worse upon awakening)
- Conjunctival edema (chemosis) and eyelid edema
- Itchy eyes
- Increased tearing
- Bacterial:
 - Mucopurulent or purulent discharge
- Gonococcal:
 - Hyperacute, copious purulent discharge:
 - Discharge starts 12 hr after inoculation.
 - Severe chemosis
 - Eyelid swelling
 - Preauricular lymphadenopathy typically absent
 - Invades intact conjunctiva and cornea within 24 hr and causes ulcerations, scarring, and perforations leading to blindness
- Chlamydia:
 - Lacrimation
 - Mucopurulent discharge
 - With or without photophobia
 - Concomitant genital infection (> 50%)
 - Transmission occurs via autoinoculation from genital secretions
- Viral—general:
 - Preauricular adenopathy
- Viral syndrome:
 - Watery, mucous discharge, lacrimation
 - Gritty feeling or foreign body sensation in eye
 - Spreads to other eye in 24–48 hr
 - Pinpoint subconjunctival hemorrhages:
 - Tarsal conjunctiva may have a bumpy appearance.
- EKC:
 - Conjunctival hyperemia
 - Chemosis
 - Corneal infiltrates
 - Decreased vision
- HSV:
 - Acute follicular conjunctival reaction
 - Skin lesions or vesicles along eyelid margin or periocular skin
 - Corneal involvement—dendritic lesion
- Herpes zoster virus (HZV):
 - Associated with pain or paresthesia of the skin
 - Rash or vesicles involving the distribution of cranial nerve V1

- Dendritic characters on cornea
- Rarely vesicles or ulcers form on the conjunctiva.
- Allergic:
 - Hallmark: Itching
 - Red conjunctiva
 - Watery discharge
 - Papillary hypertrophy
 - Frequent history of allergy, atopy, nasal symptoms
- Contact related:
 - Acute symptoms result of corneal ulceration
 - Normal visual acuity and intraocular pressures

ESSENTIAL WORKUP

- History for:
 - Onset of inflammation
 - Environmental or work-related exposure
 - Ill contacts
 - Sexual activity, discharge, rash
 - Use of over-the-counter medicines or cosmetics
 - Systemic diseases
- Careful physical exam including slit-lamp exam including fluorescein staining

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Bacteriologic studies:
 - Not indicated in routine cases
 - Indications:
 - Ophthalmia neonatorum (except chemical)
 - Suspected gonococcal ophthalmia
 - Compromised host
 - Signs and symptoms of systemic disease
 - Refractory to treatment within 48–72 hr (with good compliance)
- Positive Gram stain for gram-negative intracellular diplococci:
 - Sufficient to initiate systemic and topical treatment for gonococcal disease
- Rapid plasma reagent (RPR):
 - For suspected cases of sexually transmitted disease

DIFFERENTIAL DIAGNOSIS

- Acute angle-closure glaucoma (most serious cause)
- Allergies or hypersensitivity
- Anterior uveitis
- Corneal abrasion

- Dry eye
- Foreign body
- Keratitis
- Nasolacrimal obstruction
- Scleritis or episcleritis
- Subconjunctival hemorrhage



TREATMENT

INITIAL STABILIZATION/THERAPY

- Initiate empiric antibiotic therapy with broad-spectrum topical agent.
- Systemic therapy for gonococcal, chlamydial, and meningococcal conjunctivitis, ophthalmia neonatorum, and all severe infections regardless of cause
- Manage herpetic eye infections in consultation with an ophthalmologist.

ED TREATMENT/PROCEDURES

- Remove discharge from the eye(s):
 - _ Contact lens wearers should discontinue use and throw away affected contact lenses.
 - _ Contact lens wearers should discontinue use until:
 - Eye is white.
 - Antibiotic therapy is completed.
 - No discharge for 24 hr
 - _ Frequent handwashing
 - _ No sharing of towels, tissues, cosmetics, linens
 - _ Frequent warm soaks until lashes and eyes free of debris
- Bacterial conjunctivitis:
 - _ Antibiotics—topical:
 - _ Can use ointment or drops
 - _ Continue therapy for 48 hr after clearing of symptoms.
 - _ Discontinue therapy and obtain cultures if no improvement in 48–72 hr (with good compliance).
- Antibiotics—systemic:
 - _ Parenteral therapy mandatory for gonococcal infection
 - _ Chlamydia requires systemic treatment of sexual partners and parents of neonates.
- Viral conjunctivitis:
 - _ No specific antiviral therapy
 - _ Limited use of topical antihistamine or decongestant
- EKC may require steroids and should be prescribed in consult with ophthalmology.
- Allergic conjunctivitis (there may be a lag time of up to 2 wk for improvement with these agents):

- Antihistamine or decongestant drops (naphazoline [Naphcon-A])
- Mast cell stabilizer/antihistamine or NSAID ophthalmic drops as 2nd line
- Artificial tears
- Noninfectious:
 - Eye lubricant drops or ointment
- Empiric treatment:
 - Topical antibiotic ointment or drops

MEDICATION

- General:
 - All contact lens wearers require pseudomonal coverage.
 - Bacterial:
 - Bacitracin ophthalmologic ointment (no pseudomonal coverage)
 - Ciprofloxacin: 0.35% 1 drop q1–6h (has antipseudomonal properties; may be used in children)
 - Erythromycin: 0.5% ointment
 - Gentamicin: 0.3% ointment q3–4h or drops q1–4h (has antipseudomonal coverage)
 - Sulfacetamide: 10% 1 drop q1–6h (lacks pseudomonal coverage)
 - Tobramycin ointment
- Chlamydia:
 - Doxycycline: 100 mg PO BID for 3 wk
 - Erythromycin: 250–500 mg PO QID for 3 wk (peds: 50 mg/kg/d PO in 4 div. doses for 14 days)
 - Sulfisoxazole 500–1,000 mg QID for 3 wk
- Gonococcal:
 - Adults:
 - Ceftriaxone: 1 g IV or IM daily for 3–5 days or PRN
 - Erythromycin: 500 mg PO QID for 2–3 wk or doxycycline 100 mg PO BID for 2–3 wk
 - + topical antibiotics as above
 - Neonates:
 - Penicillin G 100,000 U/kg/d in 4 div. doses for 7 days or ceftriaxone 25–50 mg/kg/d IV for 7 days
- Viral:
 - Artificial tears
 - Naphcon-A or Visine AC 1 or 2 drops QID PRN for no more than 1 wk
- HSV or HZV:
 - Trifluorothymidine: 1% 5 times per day
 - Vidarabine: 3% ointment 5 times per day
- Allergic:
 - Naphazoline (Naphcon-A): 1 drop BID–QID or Visine AC
 - Acular: 1 or 2 drops BID

- Cromolyn sodium 4% (Crolom): 1 drop QID
- Noninfectious and nonallergic:
 - Eye lubricant drops or ointment: Artificial tears or Lacri-Lube
- Empiric treatment:
 - Erythromycin ointment 0.5% (half in QID)
 - Sulfacetamide 10% ophthalmic drops (1 or 2 drops QID) for 5–7 days

Pediatric Considerations

- Often a manifestation of systemic disease in infants
- Conjunctivitis in the 1st 36 hr of life usually chemically induced caused by silver nitrate applied at birth.
- Neonates become infected during passage through the birth canal.
- Gonococcal, herpetic, chlamydial organisms most common
- Ophthalmia neonatorum is conjunctivitis within the 1st 4 wk of life.
- Chlamydia trachomatis is not eradicated by silver nitrate.
- Some newborns treated with erythromycin still develop conjunctivitis.
- Ointment is preferred over drops because of difficulty with administration of drops.



FOLLOW-UP

DISPOSITION

Admission Criteria

Known or suspected gonococcal infection (any age group)

Discharge Criteria

Close follow-up for all cases

Issues for Referral

Diagnosis of EKC and bacterial conjunctivitis requires ophthalmology referral.

FOLLOW-UP RECOMMENDATIONS

All patients with bacterial conjunctivitis require ophthalmology follow-up.

PEARLS AND PITFALLS

- Be sure to disinfect slit lamp and chair used for patients to avoid contamination.
- Conjunctivitis is extremely contagious.
- Viral conjunctivitis contagious for up to 2 wk.
- EKC is especially contagious.
- Extreme caution should be taken when using corticosteroids, as they may worsen

an underlying HSV infection.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Red eye



CODES

ICD9

- 077.99 Unspecified diseases of conjunctiva due to viruses
- 372.03 Other mucopurulent conjunctivitis
- 372.30 Conjunctivitis, unspecified

ICD10

- B30.9 Viral conjunctivitis, unspecified
- H10.029 Other mucopurulent conjunctivitis, unspecified eye
- H10.9 Unspecified conjunctivitis

CONSTIPATION

Julia H. Sone

BASICS

DESCRIPTION

Rome Criteria for the diagnosis of constipation requires 2 or more of the following for at least 3 mo:

- Straining > 25% of the time
- Hard stools > 25% of the time
- Incomplete evacuation > 25% of the time
- 2 or fewer bowel movements per wk

Pediatric Considerations

- 3% of pediatric outpatient visits are because of defecation disorders.
- Children with cerebral palsy often develop functional constipation.
- Can be classified into subgroups:
 - Constipation with anatomical origins (anal stenosis/strictures, ectopic anus, imperforate anus, sacrococcygeal teratomas)
 - Colonic neuromuscular disease (Hirschsprung disease)
 - Defecation disorders (functional constipation and nonretentive fecal soiling)
 - Function fecal retention
- Most common cause of fecal retention and soiling in children is functional fecal retention:
 - Caused by fears associated with defecation
 - Associated with irritability, abdominal cramps, decreased appetite, early satiety

ETIOLOGY

- Metabolic and endocrine:
 - Diabetes
 - Uremia
 - Porphyria
 - Hypothyroidism
 - Hypercalcemia
 - Pheochromocytoma
 - Panhypopituitarism
 - Pregnancy
- Functional and idiopathic:
 - Colonic irritable bowel syndrome
 - Diverticular disease

- Colonic inertia
- Megacolon/megarectum
- Pelvic intussusception
- Nonrelaxing puborectalis
- Rectocele/sigmoidocele
- Posthysterectomy syndrome
- Descending perineum
- Pharmacologic:
 - Analgesics
 - Anesthetics
 - Antacids
 - Anticholinergics
 - Anticonvulsants
 - Antidepressants
 - Antihypertensives
 - Calcium channel blockers
 - Diuretics
 - Ferrous compounds
 - Laxative abuse
 - MAOIs
 - Opiates
 - Paralytic agents
 - Parasympatholytics
 - Phenothiazines
 - Psychotropics
- Neurologic:
 - Central Parkinson disease
 - Multiple sclerosis
 - Cerebrovascular accidents
 - Spinal cord lesions/injury
 - Peripheral Hirschsprung disease
 - Chagas disease
 - Neurofibromatosis
 - Autonomic neuropathy
- Mechanical obstruction:
 - Neoplasm
 - Stricture
 - Hernia
 - Volvulus



SIGNS AND SYMPTOMS

- Constipation is a symptom, not a disease.
- Passage of hard stool
- Straining/difficulty passing stool
- Infrequent bowel movements
- Abdominal distention/bloating
- Firm/hard stool on digital rectal exam:
 - May have empty rectal vault
- Diarrhea (liquid stool passes around firm feces)

History

- Age of onset of symptoms
- Diet and exercise regimen
- Stool size, caliber, consistency, frequency, ease of defecation
- Medical and surgical history:
 - Medications that can slow colonic transit like β -blockers, high-dose calcium channel blockers, narcotics
- Use of enemas, laxatives, and digital manipulation to facilitate defecation
- Associated pelvic floor dysfunction:
 - Urinary symptoms
 - Rectocele

Physical-Exam

- Abdominal exam may reveal a mass due to stool
- Rectal exam to assess for outlet obstruction:
 - Ability to squeeze and relax the sphincter
 - Is there a rectocele or cystocele?
 - Assess firmness of stool

ESSENTIAL WORKUP

Thorough history and physical exam:

- Medical, surgical, and psychiatric investigation and date of onset
- Note abdominal distention, hernias, tenderness, or masses
- Complete anorectal exam for anal stenosis, fissure, neoplasm, sphincter tone, perineal descent, tenderness, spasm

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Only necessary when considering metabolic/endocrine disorders
- CBC if inflammatory or neoplastic origin
- Electrolytes and calcium indicated if at risk of:
 - Hypokalemia

- Hypocalcemia
- Thyroid function test if patient appears to be hypothyroid

Imaging

- Rarely indicated unless an underlying process suspected
- Abdominal radiograph:
 - Large amount of feces in colon
 - Dilated colon that needs decompression
- CT scan of abdomen/pelvis to r/o perforation in elderly, constipated patient with abdominal pain/fever
- Barium/Gastrografin enema study:
 - Diverticulosis
 - Megarectum
 - Megacolon
 - Hirschsprung disease
 - Stricture from inflammation or tumor

DIFFERENTIAL DIAGNOSIS

- See “Etiology.”
- Bowel obstruction



TREATMENT

PRE HOSPITAL

Establish IV access for patients with significant abdominal pain.

INITIAL STABILIZATION/THERAPY

IV fluids for dehydrated/hypotensive patients

ED TREATMENT/PROCEDURES

- Clean out colon:
 - Enemas, suppositories
 - Manual disimpaction of hard stool
 - Laxatives
- Maintain bowel regimen:
 - Increase noncaffeinated fluids (8–10 cups per day).
 - Increase dietary fiber intake (20 g/day).
 - Stool softeners
 - Exercise
 - Change medications causing constipation.

MEDICATION

- Enemas:

- Fleet: 120 mL (peds: 60–120 mL) per rectum (PR)
- Mineral oil: 60–150 mL (peds: 5–11 yr old, 30–60 mL; older than 12 yr, 60–150 mL) PR daily
- Tap water: 100–500 mL PR
- Fiber supplements:
 - Methylcellulose: 1 tbs in cup water PO daily to TID
 - Psyllium: 1–2 tsp in cup of water/juice (peds: Younger than 6 yr, 1/4–1/2 tsp in 2 oz water or juice; 6–11 yr, 1/2–1 tsp in 4 oz water or juice; older than 12 yr, 1–2 tsp in cup water or juice) PO daily to TID
- Laxatives (osmotic):
 - Lactulose: 15–30 mL (peds: 1 mL/kg) PO daily to BID
 - Polyethylene glycol: 17 g (peds: 0.8 g/kg/d dissolved in 4–8 oz of liquid) PO daily dissolved in liquid
 - Milk of magnesia: 2400–4800 mg Mg hydroxide po (peds 6 mo–1 yr: 40 mg/kg Mg hydroxide; 2–5 yr: 400–1200 mg Mg hydroxide; 6–11 yr: 1200–2400 mg Mghydroxide; over 12 yrs: 2400–4800 mg Mg hydroxide) QD or divided bid–qid prn
- Laxatives (stimulant):
 - Bisacodyl: 10–15 mg PO daily (peds: Younger than 3 yr, 5 mg PR daily; 3–12 yr, 5–10 mg PO/PR daily; older than 12 yr, 5–15 mg PO daily or 10 mg PR daily)
 - Senna: 2 tabs PO daily to BID (peds: 2–6 yr, 1/2–1 tab PO daily to BID; 6–12 yr, 1–2 tabs PO daily to BID; older than 12 yr, 2–4 tabs PO daily to BID)
- Stool softeners:
 - Docusate sodium: 100 mg (peds: 3–5 mg/kg/d in div. doses) PO daily to BID
 - Mineral oil: 15–45 mL (peds: 5–15 mL) PO daily
- Suppositories:
 - Glycerin: 1 adult (peds: Infant, 1 infant suppository) PR PRN



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients with severe abdominal pain, nausea, and emesis
- Neurologically impaired, elderly, morbidly obese who cannot be cleaned out in the ED or home
- Bowel obstruction/peritonitis

Discharge Criteria

- No co-morbid illness requiring admission
- Pain free

- Adequately cleaned out

Issues for Referral

GI follow-up for further evaluation and treatment

FOLLOW-UP RECOMMENDATIONS

Primary care or GI follow-up for patients with longstanding constipation

PEARLS AND PITFALLS

- Advise patients regarding appropriate dietary and lifestyle changes to decrease incidence of constipation.
- Perform thorough history and physical exam to exclude significant medical or surgical etiologies for constipation.

ADDITIONAL READING

- Doody DP, Flores A, Rodriguez LA. Evaluation and management of intractable constipation in children. *Semin Colon Rectal Surg.* 2006;17(1):29–37.
- Ford AC, Talley NJ. Laxatives for chronic constipation in adults. *BMJ.* 2012;345:e6168.
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- Talley N. Differentiating functional constipation from constipation-predominant irritable bowel syndrome: Management implications. *Rev Gastroenterol Disord.* 2005;5(1):1–9.
- Wexner SD, Pemberton JH, Beck DE, et al., eds. *The ASCRS Textbook of Colon and Rectal Surgery.* Springer; 2007.



See Also (Topic, Algorithm, Electronic Media Element)

- Abdominal Pain
- Bowel Obstruction



CODES

ICD9

- 564.00 Constipation, unspecified
- 564.09 Other constipation
- 564.8 Other specified functional disorders of intestine

ICD10

- K59.00 Constipation, unspecified

- K59.09 Other constipation
- K59.8 Other specified functional intestinal disorders

CONTACT DERMATITIS

Erik Adler • Jeffrey Druck

BASICS

DESCRIPTION

- Irritant:
 - Immediate eczematous eruption (superficial inflammatory process primarily in epidermis)
 - Most common type of dermatitis
 - Trigger substance itself directly damages the skin resulting in nonimmunologic inflammatory reaction with erythema, dryness, cracking, or fissuring
 - Usually owing to repeated exposure to mild irritant (e.g., water, soaps, heat, friction)
 - Lesions itch or burn:
 - Usually gradual onset with indistinct borders
 - Most often seen on hands
 - May see vesicles or fissures
 - Dry, red, and rough skin
 - Common irritants include cement, hair dyes, wet diapers, rubber gloves, shampoos, frequent hand washing
- Allergic:
 - Delayed (type IV) hypersensitivity reaction (requires prior sensitization)
 - Allergen-induced immune response
 - Local edema, vesicles, erythema, pruritus, or burning
 - Usually corresponds to exact distribution of contact (e.g., watchband)
 - Onset usually within 12–48 hr with prior sensitization; may take 14–21 days for primary exposure
 - Common sources: Nickel, gold, neomycin, bacitracin, preservatives, fragrances, dyes, poison ivy
- Photocontact:
 - Interaction between an otherwise harmless substance on the skin and UV light
 - Common sources: Shaving lotions, sunscreens, sulfa ointments, perfumes.

Pediatric Considerations

- Allergic contact dermatitis is less frequent in children, especially infants, than in adults
- Major sources of pediatric contact allergy:
 - Metals, shoes, preservatives, or fragrances in cosmetics, topical medications,

and plants

- Diaper dermatitis: Prototype for irritant contact dermatitis in children
- Circumoral dermatitis: Seen in infants and small children; may result from certain foods (irritant or allergic reaction)

ETIOLOGY

- Irritant (80% of contact dermatitis), e.g.:
 - Soaps, solvents
 - Chemicals
 - Certain foods
 - Urine, feces
 - Diapers
 - Continuous or repeated exposure to moisture (hand washing)
 - Course paper, glass, and wool fibers
 - Shoe dermatitis: Common; identify by lesions limited to distal dorsal surface of foot usually sparing the interdigital spaces
- Allergic:
 - Plants, poison ivy, oak, sumac (rhus dermatitis):
 - Most common form of allergic contact dermatitis in North America
 - Direct: Reaction to oleoresin urushiol from plant
 - Indirect: Contact with pet or clothes with oleoresin on surface or fur or in smoke from burning leaves
 - Lesions may appear up to 3 days after exposure with prior sensitization (12–21 days after primary exposure) and may persist up to 3 wk
 - Fluid from vesicles is not contagious and does not produce new lesions
 - Oleoresin on pets or clothes remains contagious until removed
 - Cement (prolonged exposure may result in severe alkali burn)
 - Metals (especially nickel)
 - Solvents, epoxy
 - Chemicals in rubber (e.g., elastic waistbands) or leather
 - Lotions, cosmetics
 - Topical medications (e.g., neomycin, hydrocortisone, benzocaine, paraben)
 - Some foods
 - Ability to respond to certain antigens is probably genetically determined
- Photodermatitis:
 - Inflammatory reaction from exposure to irritant (frequently plant sap) and sunlight
 - Typically no response in absence of sunlight



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Date of onset
- Time course
- Pattern of lesions
- Relationship to work
- Pruritic or not
- Mucosal involvement
- Exposure to new products (e.g., lotions, soaps, and cosmetics), foods, medications, and jewelry

Physical-Exam

- Special attention to character and distribution of rash
- *Acute lesions*: Skin erythema and pruritus:
 - May see edema, papules, vesicles, bullae, serous discharge, or crusting
- *Subacute*: Vesiculation less pronounced
- *Chronic lesions*: May see scaling, lichenification, pigmentation, or fissuring with little to no vesiculation; may have characteristic distribution pattern

DIAGNOSIS TESTS & INTERPRETATION

Lab

No specific tests in ED are helpful.

Imaging

No specific tests in ED are helpful

Diagnostic Procedures/Surgery

- Patch testing:
 - Generally not done in ED; refer to allergist/immunologist
- When tinea is suspected, may use Wood lamp for fluorescence

DIFFERENTIAL DIAGNOSIS

- Atopic dermatitis: Associated with family history of atopy
- Seborrheic dermatitis: Scaly or crusting “greasy” lesions
- Nummular dermatitis: Coin-like lesions
- Intertrigo: Dermatitis in which skin is in apposition (axillae, groin area)
- Infectious eczematous dermatitis: Dermatitis with secondary bacterial infection, usually *Staphylococcus aureus*
- Cellulitis: Warm, blanching, painful lesion
- Impetigo: Yellow crusting

- Scabies: Intensely pruritic, frequently interdigital with tracks
- Psoriasis: Silvery adherent, scaling, lesions well delineated, affecting extensor surfaces, scalp, and genital region
- Herpes simplex: Groups of vesicles, painful, burning
- Herpes zoster: Painful, follows dermatomal pattern
- Bullous pemphigoid: Diffuse bullous lesions
- Tinea: Maximal involvement at margins, fluoresces under Wood lamp
- Pityriasis alba: Discrete, asymptomatic, hypopigmented lesions
- Urticaria: Pruritic raised lesions (wheal) frequently with surrounding erythema (flare)
- Acrodermatitis enteropathica: Vesiculobullous lesion of hands and feet, associated with failure to thrive, diarrhea, and alopecia
 - Due to zinc deficiency
- Dyshidrotic dermatitis (eczema)
 - Drug rash
 - Stevens–Johnson syndrome (SJS)
 - Toxic epidermal necrolysis (TEN)
 - Erythema nodosum (EN)



TREATMENT

INITIAL STABILIZATION/THERAPY

Rarely required in absence of concomitant pathology

ED TREATMENT/PROCEDURES

General:

- Primarily symptomatic
- Wash area with mild soap and water
- Remove or avoid offending agent (including washing clothes)
- Cool, wet compresses; especially effective during acute blistering phase
- Antipruritic agents:
 - Topical:
 - Calamine lotion, corticosteroids (do not penetrate blisters); avoid benzocaine or hydrocortisone-containing products, which may further sensitize skin
 - Systemic: Antihistamines, corticosteroids
- Aluminum acetate (Burrows) solution: Weeping surfaces

Irritant dermatitis:

- Remove offending agent
- Wash well with soap and warm water
- Decrease wet/dry cycles (hand washing)
 - Alcohol-based cleansers decrease repetitive trauma

- Bland emollient
- Topical steroids for severe cases (ointment preferred), medium to high potency (hands), BID for several weeks

Allergic dermatitis:

- Topical steroids (ointment preferred) BID for 2–3 wk:
 - Face: Low potency
 - Arms, legs, and trunk: Medium potency
 - Hands and feet: High potency
- Oral steroids for severe cases

Rhus dermatitis:

- Follow general measures plus:
 - Wash all clothes and pets that have come in contact with the plant; oil persists and is contagious
 - Oatmeal baths can provide soothing relief
 - Aseptic aspiration of bullae may relieve discomfort
 - Severe reaction (>10% TBSA): Systemic corticosteroids for 2–3 wk with gradual taper:
 - Premature termination of corticosteroid therapy may result in rapid rebound of symptoms

Shoe dermatitis:

- Follow general measures plus:
 - Wear open-toe, canvas, or vinyl shoes.
 - Control perspiration: Change socks, use absorbent powder.

Diaper dermatitis:

- Follow general measures plus:
 - Topical zinc oxide, petrolatum ointment, or aquaphor
 - Change diapers after each soiling

MEDICATION

Systemic:

- Antihistamine (H₁-receptor antagonist, 1st and 2nd generation):
 - Cetirizine: Adults and children >6 yr, 5–10 mg PO daily (peds: Age 2–6 yr, 2.5 mg PO daily BID)
 - Diphenhydramine hydrochloride: 25–50 mg IV/IM/PO q6h PRN (peds: 5 mg/kg/24h div. q6h PRN)
 - Fexofenadine: 60 mg PO BID or 180 mg PO daily (peds: Age 6–12 yr, 30 mg PO BID)
 - Hydroxyzine hydrochloride: 25–50 mg PO IM up to QID PRN (peds: 2 mg/kg/24h PO div. q6h or 0.5 mg/kg IM q4–6h PRN)
 - Loratadine: 10 mg PO BID
 - For refractory pruritus: Doxepin: 75 mg PO daily may be effective.
- Corticosteroid:
 - Prednisone: 40–60 mg PO daily (peds: 1–2 mg/kg/24h, max. 80 mg/24h)

div. daily/BID

- For refractory pruritis:
 - Doxepin: 75 mg PO daily may be effective.

Topical:

- Aluminum acetate (Burrows) solution: Apply topically for 20 min TID until skin is dry.
- Calamine lotion: q6h PRN
- Topical corticosteroid: Triamcinolone ointment 0.025, 0.1%; cream 0.025, 0.1%; lotion 0.025, 0.1% TID or QID daily
 - Caution: Do not apply to face or eyelids

First Line

- Topical steroids
- Oral antihistamines

Second Line

Oral steroids



FOLLOW-UP

DISPOSITION

Admission Criteria

Rarely indicated unless severe systemic reaction or significant secondary infection

Discharge Criteria

- Symptomatic relief
- Adequate follow-up with primary care physician or dermatologic specialist

FOLLOW-UP RECOMMENDATIONS

- Follow up with primary care physician in 2–3 days for recheck
- Return to ED for: Facial swelling, difficulty breathing, mucosal involvement causing decreased PO intake

PEARLS AND PITFALLS

- Remove offending agent
- Beware of progression to systemic anaphylaxis (e.g., latex allergy)
- Watch out for concurrent bacterial infections
- Rhus dermatitis wounds are no longer contagious after washed with soap and water:
 - Be sure to wash all clothes and animals that have come in contact with

plant as oil remains contagious.

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CODES

ICD9

- 692.2 Contact dermatitis and other eczema due to solvents
- 692.9 Contact dermatitis and other eczema, unspecified cause
- 692.81 Dermatitis due to cosmetics

ICD10

- L25.0 Unspecified contact dermatitis due to cosmetics
- L25.2 Unspecified contact dermatitis due to dyes
- L25.9 Unspecified contact dermatitis, unspecified cause

COR PULMONALE

Richard E. Wolfe

BASICS

DESCRIPTION

Right ventricular hypertrophy (RVH) or dilation caused by elevated pulmonary artery pressure. RVH due to a *systemic* defect or congenital heart disease is not classified as cor pulmonale.

- Acute cor pulmonale:
 - Right ventricle is dilated and muscle wall stretched thin
 - Overload due to acute pulmonary hypertension (HTN)
 - Most often caused by massive pulmonary embolism
- Chronic cor pulmonale:
 - RVH with eventual dilation and right-sided heart failure
 - Caused by an adaptive response to chronic pulmonary HTN
 - Predominately occurs as a result of alveolar hypoxia
- The pulmonary circulation is a low-resistance, low-pressure system:
 - The pulmonary arteries are thin walled and distensible
 - Mean pulmonary arterial pressure is usually 12–15 mm Hg
 - Normal left arterial pressure is 6–10 mm Hg
 - The resulting pressure difference driving the pulmonary circulation is only 6–9 mm Hg
- 3 factors affect pulmonary arterial pressure:
 - Cardiac output
 - Pulmonary venous pressure
 - Pulmonary vascular resistance
- Pulmonary HTN can arise through a number of mechanisms:
 - A marked increase in cardiac output
 - Left-to-right shunt secondary to congenital heart disease
 - Hypoxia:
 - The most common cause of increased pulmonary vascular resistance
 - Hypoxic pulmonary vasoconstriction is an adaptive vasomotor response to alveolar hypoxia
 - A compensatory rise in pressure is seen in the pulmonary arterial system, so flow is maintained across the pulmonary vascular bed.
 - Pulmonary embolus causes a similar change by increasing resistance to pulmonary blood flow
 - Dramatic rises in blood viscosity or intrathoracic pressure also impede blood flow
- Pulmonary HTN is classified into 5 groups

- Group 1: Pulmonary arterial HTN
- Group 2: Pulmonary HTN owing to left heart disease
 - o RV dysfunction in this category is not considered cor pulmonale
- Group 3: Pulmonary HTN owing to lung diseases and/or hypoxia
- Group 4: Chronic thromboembolic pulmonary HTN
- Group 5: Pulmonary HTN with unclear multifactorial mechanisms

EPIDEMIOLOGY

Incidence

- ~86,000 patients die from COPD each yr:
 - Associated RV failure is a significant factor in many of these cases, and accounts for 10–30% of heart failure admissions in US.
- In patients >50 yr with COPD, 50% develop pulmonary HTN and are at risk of developing cor pulmonale.
- The course of cor pulmonale is generally related to the progression of the underlying disease process.
- Once cor pulmonale develops, patients have a 30% chance of surviving 5 yr.

ETIOLOGY

- Chronic hypoxia
 - COPD
 - High-altitude dwellers
 - Sleep apnea
 - Chest deformities
 - o Kyphoscoliosis
- Pulmonary embolism
- Interstitial lung disease
 - Scleroderma
 - Systemic lupus erythematosus
 - Mixed connective tissue disease
 - Sarcoidosis
 - Pulmonary Langerhans cell histiocytosis
 - Neurofibromatosis type
 - Lymphangioleiomyomatosis
- Cystic fibrosis
- Severe anemia
- Obesity
- Pulmonary veno-occlusive disease
- Pulmonary vascular obstruction secondary to tumors or adenopathy
- Increased blood viscosity:
 - Polycythemia vera
 - Leukemia

- Increased intrathoracic pressure:
 - Mechanical ventilation with positive end-expiratory pressure
- Idiopathic primary pulmonary HTN



DIAGNOSIS

SIGNS AND SYMPTOMS

- Exertional dyspnea
- Easy fatigability
- Weakness
- Exertional syncope
- Cough
- Hemoptysis
- Exertional angina even in the absence of coronary disease
- Anorexia
- Right upper quadrant discomfort
- Wheezing
- Hoarseness
- Weight gain
- Hepatomegaly
- Ascites
- Peripheral edema

End-stage cor pulmonale

- Cardiogenic shock
- Oliguria
- Cool extremities
- Pulmonary edema secondary to intraventricular septum impairing left ventricular diastolic function

History

- Exercise intolerance
- Palpitations
- Chest pain
- Lightheadedness
- Syncope
- Swelling of the lower extremities

Physical-Exam

- Jugular venous distention:
 - Prominent A- and V-waves
- Increase in chest diameter
- Crackles and/or wheezes

- Left parasternal heave on cardiac palpation
- Splitting of the 2nd heart sound or murmurs of the pulmonary vasculature may be heard.
- Hepatojugular reflex and pulsatile liver
- Pitting edema of the lower extremities

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Pulse oximetry or ABG:
 - Resting PO₂ 40–60 mm Hg
 - Resting PCO₂ often 40–70 mm Hg
- Hematocrit:
 - Frequently elevated
- B-natriuretic peptide:
 - When elevated, is sensitive for moderate to severe pulmonary HTN, and may be an independent predictor of mortality
 - Elevated level alone is not enough to establish diagnosis of cor pulmonale.
- Other lab tests are not generally useful.

Imaging

- CXR:
 - Signs of pulmonary HTN:
 - Large pulmonary arteries (> 16–18 mm)
 - An enlarged RV silhouette
 - Shows abnormalities in > 90% of patients in the detection of cor pulmonale, but does not indicate the severity of disease
 - Pleural effusions do not occur in the setting of cor pulmonale alone.
- EKG:
 - Right-axis deviation
 - Right bundle branch block
 - RVH
 - Dominant R-wave in V1 and V2
 - Prominent S-wave in V5 and V6
 - Small R-waves and deep S-waves across the precordium
 - Right atrial enlargement
 - Tall, peaked P-waves (P pulmonale)
 - S1Q3 pattern with acute cor pulmonale
 - Transient changes due to hypoxia
 - Right precordial T-wave flattening
- Echocardiography
 - The noninvasive diagnostic method of choice

- RV dilation or RVH
- Assessment of tricuspid regurgitation
- Doppler quantization of pulmonary artery pressure, RV ejection fraction
- Chest CT, ventilation/perfusion scans, or pulmonary angiography:
 - Useful in the setting of acute cor pulmonale
- Magnetic resonance imaging
 - Superior to echocardiography for assessment of right ventricular size and function
- Pulmonary function tests
 - Impaired diffusion capacity due to pulmonary HTN
- Right-heart catheterization:
 - The most precise estimate of pulmonary vascular hemodynamics
 - Gives accurate measurements of pulmonary arterial pressure and pulmonary capillary wedge pressure

DIFFERENTIAL DIAGNOSIS

- Primary disease of the left side of the heart
 - Mitral stenosis
- Congenital heart disease
 - Eisenmenger syndrome
 - Left to right shunt caused by a congenital heart defect in the fetal heart causes increased flow through the pulmonary vasculature, causing pulmonary HTN
- Hypothyroidism
- Cirrhosis



TREATMENT

PRE HOSPITAL

- Supportive therapy:
 - Supplemental oxygen
 - To an endpoint of 90% arterial saturation
- - IV access
 - Cardiac monitoring
 - Pulse oximetry
- Treat bronchospasm from associated respiratory disease:
 - β -Agonist nebulizers
- Caution:
 - Vasodilators and diuretics do not have a role in the field.
 - Severely hypoxic patients may require endotracheal intubation.

INITIAL STABILIZATION/THERAPY

ED therapy is directed at the underlying disease process and reducing pulmonary HTN.

ED TREATMENT/PROCEDURES

- Supplemental oxygen sufficient to raise arterial saturation to 90%:
 - Improving oxygenation reduces pulmonary arterial vasoconstriction and RV afterload.
 - The improved cardiac output enhances diuresis of excess body water.
 - Care must be taken to monitor the patient's ventilatory status and PCO_2 , as hypercapnia may reduce respiratory drive and cause acidosis.
- Diuretics, such as furosemide, may be added cautiously to reduce pulmonary artery pressure by contributing to the reduction of circulating blood volume:
 - Be wary of volume depletion and hypokalemia
- Patients should be maintained on salt and fluid restriction.
- There is no role for digoxin in the treatment of cor pulmonale.
- Bronchodilators:
 - Bronchodilator therapy is particularly helpful for those patients with COPD
 - Selective β -adrenergic agents such as terbutaline 0.25 mg SC may be useful.
 - Bronchodilator affects and reduces ventricular afterload.
 - Theophylline may play a role to improve diaphragmatic contractility and reduce muscle fatigue.
 - Anticoagulation may be considered for those at high risk for thromboembolic disease.
- Acutely decompensated COPD patients:
 - Early steroid therapy
 - Antibiotic administration
- In general, improvement in the underlying respiratory disease results in improved RV function.

MEDICATION

- Furosemide: 20–60 mg IV (peds: 1 mg/kg may increase by 1 mg/kg/q2h not to exceed 6 mg/kg)
- Terbutaline: 0.25 mg SC



FOLLOW-UP

DISPOSITION

Admission Criteria

- New-onset hypoxia
- Anasarca
- Severe respiratory failure

- Admission criteria for the underlying disease process

Discharge Criteria

Patients without hypoxia or a stable oxygen requirement

Issues for Referral

- Close follow-up as long as the underlying etiology has responded to acute management
- The need for a sleep study to assess for sleep apnea should be coordinated by the patient's physician.

FOLLOW-UP RECOMMENDATIONS

Ensure home oxygenation in patients with chronic hypoxia

PEARLS AND PITFALLS

- The physical exam is unreliable for detecting cor pulmonale in patients with COPD, as hyperinflation of the chest obscures the classic findings.
- Vasodilator therapy should only be considered after conventional therapy and oxygenation have failed.

ADDITIONAL READING

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CODES

ICD9

- 415.0 Acute cor pulmonale
- 416.9 Chronic pulmonary heart disease, unspecified

ICD10

- I26.09 Other pulmonary embolism with acute cor pulmonale
- I27.81 Cor pulmonale (chronic)

CORNEAL ABRASION

Denise S. Lawe

BASICS

DESCRIPTION

- Any tear or defect in the corneal epithelium
- May be traumatic, spontaneous, due to foreign body, or contact lens related

ETIOLOGY

- Traumatic:
 - Human fingernail
 - Branches
 - Hairbrushes/combs
 - Sand/stones
 - Snow
 - Pens/pencils
 - Toys
 - Chemical burn
 - Airbag deployment
 - Pepper spray
 - Paper/cardboard
 - Make-up applicator
 - Animal paws
- Foreign body related:
 - Wood
 - Glass
 - Metal
 - Rust
 - Plastic
 - Fiberglass
 - Vegetable matter
 - Eyelid foreign body
- Contact lens related:
 - Over-worn
 - Improperly fitting or cleaned
- Spontaneous:
 - Usually previous traumatic corneal abrasion or an underlying defect in the corneal epithelium

DIAGNOSIS

SIGNS AND SYMPTOMS

- Severe ocular pain
- Gritty (scratchy) discomfort
- Tearing
- Blepharospasm
- Foreign body sensation
- Photophobia (particularly if secondary traumatic iritis present)
- Conjunctival injection
- Diminished or blurred vision
- Headache

History

- Any direct trauma to the globe
- Any known or potential foreign body
- Contact lens use
- Any history of previous corneal abrasion
- Ocular/periocular surgery
- Pre-existing visual impairment
- Time of onset
- Associated symptoms or concomitant injury
- Treatment before visit
- Use of safety glasses (pounding, drilling, grinding metal) or eyeglasses
- Systemic disease (diabetes, autoimmune disorders)
- Tetanus status

Pediatric Considerations

- Signs and symptoms may differ:
 - Excessive crying
- Younger than 12 mo:
 - Frequently no history of eye trauma
 - Might present as the crying inconsolable infant
 - In 1–12 wk old may be an incidental finding and not the cause of their irritability or crying
- Older than 12 mo:
 - More often will have history of minor eye trauma
 - Positive eye signs

Physical-Exam

- If indicated, evaluate for other life-threatening injuries with attention to the primary survey.
- Complete eye exam:
 - Focus is to evaluate for evidence of penetrating injury and/or infection

- Gross visual inspection
- Visual acuity
- Penlight exam to evaluate for conjunctival injection, the pupil shape/reactivity, and for any evidence of corneal infiltrate or opacity
- Evert upper lids to check for retained foreign body
- Slit-lamp exam to evaluate for anterior chamber reaction, infiltrate, corneal laceration, and penetrating trauma
- Fluorescein dye to identify size and location of corneal epithelium defect

DIAGNOSIS TESTS & INTERPRETATION

Pediatric Considerations

Handheld slit-lamp and Wood lamp: Helpful in exam of pediatric eye

DIFFERENTIAL DIAGNOSIS

- Conjunctivitis, viral, or bacterial
- Corneal ulcer
- Glaucoma
- Herpes zoster
- Keratitis, viral or bacterial, or ultraviolet induced
- Recurrent corneal erosion syndrome
- Uveitis
- More extensive pathology than corneal abrasion:
 - Laceration of cornea
 - Perforation of cornea
 - Hyphema
 - Iris prolapse
 - Lens disruption



TREATMENT

INITIAL STABILIZATION/THERAPY

Instill topical anesthetic (proparacaine/tetracaine).

ED TREATMENT/PROCEDURES

- Removal of superficial foreign body:
 - A residual rust ring does not need emergent removal. It can be removed at 24–48 hr
- Oral pain control:
 - Oral narcotics or NSAID or acetaminophen
- Topical pain control:
 - Studies have demonstrated efficacy; however, there are scattered reports of

- adverse effects
 - Avoid in patients with other ocular surface disease and in postoperative patient
 - Topical diclofenac or ketorolac
- Cycloplegic (optional):
 - Cyclopentolate (mydriasis 1–2 days)
 - Tropicamide (mydriasis 6 hr)
 - Homatropine 5%
- Topical antibiotic:
 - This practice has not been rigorously studied.
 - Concern is for superinfection
 - Ointment better than drops because also a lubricant
 - Discontinue antibiotics once symptom free for 24 hr
 - Contact lens wearers must have anti-Pseudomonal coverage:
 - Ciprofloxacin
 - Erythromycin
 - Gentamicin
 - Sulfacetamide
 - Tobramycin/Tobradex
 - Polytrim
- Eye patch:
 - Does not appear to improve healing or reduce pain particularly in the 1st 24 hr
 - Not recommended for small abrasions
 - Never patch the patient who wears contact lens
 - Never patch infection-prone injury (organic matter is at high risk)
 - More research needed to evaluate efficacy of patching in abrasions > 10 mm
- Contact lens
 - No contact lens wear till abrasion healed and eye feels normal for a wk without medication
 - Might consider bandage contact lens in severe pain. Be certain no infection and will need daily follow-up
- Tetanus prophylaxis:
 - Routine tetanus not necessary
 - Update tetanus if abrasion caused by or contaminated with organic matter or dirt
- Emergent ophthalmologic consultation required for retained intraocular foreign body, penetrating injury to globe (or other more serious injury) and any patient with a corneal infiltrate, white spot, or opacity

MEDICATION

- Ciprofloxacin: 0.35% 1 drop QID

- Cyclopentolate: 0.5%, 1%, or 2% drops (mydriasis 1 or 2 drops TID)
- Diclofenac: 0.1% drops 1 drop QID
- Erythromycin: 0.5% ointment QID
- Gentamicin: 0.3% ointment QID
- Gentamicin: 0.3% 2 drops q6h
- Homatropine: 5% solution 2 drops BID
- Ketorolac: 0.5% drops 1 drop QID
- Proparacaine: 0.5% 1 drop once
- Sulfacetamide: 10% drops 2 drops QID
- Sulfacetamide: 10% ointment QID
- Tobradex: Suspension 0.1%/0.3% 2 drops q4–6h
- Tobramycin: 0.3% drops 2 drops q6h
- Tobramycin: 0.3% ointment q6h
- Tropicamide: 0.5%, 1% drops (mydriasis 6 hr) 1 drop q4h



FOLLOW-UP

DISPOSITION

Admission Criteria

Associated injuries requiring admission

Discharge Criteria

All simple corneal abrasions

Issues for Referral

No studies on optimal follow-up. Practice recommendations however dictate all corneal abrasions require follow-up to ensure healing without infection or scarring.

FOLLOW-UP RECOMMENDATIONS

- Follow-up with ophthalmologist for re-exam and ongoing care in 24 hr if in contact lens wearer, the eye has been patched or bandage contact lens applied
- Follow-up with ophthalmologist if central or large abrasion in 24 hr; otherwise follow-up can be in 48–72 hr

PEARLS AND PITFALLS

- Always diligently evaluate for penetrating trauma to the globe.
- Always diligently evaluate for evidence of infection.
- Do not discharge the patient with any topical anesthetic. It is felt to be toxic to the epithelium and retards healing, although a recent small study indicated it might be safe to discharge with dilute proparacaine.

- Do not use a mydriatic agent on a patient with a history of glaucoma.
- Do not recommend return to contact use until followed up and cleared by ophthalmology.

ADDITIONAL READING

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- Van Niel CW. (2010). Corneal abrasions in crying infants: A red herring. Retrieved from Journal Watch Specialties (online).
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See Also (Topic, Algorithm, Electronic Media Element)

- Conjunctivitis
- Corneal Burn
- Corneal Foreign Body
- Red Eye
- Ultraviolet Keratitis



CODES

ICD9

918.1 Superficial injury of cornea

ICD10

- S05.00XA Inj conjunctiva and corneal abrasion w/o fb, unsp eye, init
- S05.01XA Inj conjunctiva and corneal abrasion w/o fb, right eye, init
- S05.02XA Inj conjunctiva and corneal abrasion w/o fb, left eye, init

CORNEAL BURN

Matthew A. Wheatley • Ryan A. Stroder



BASICS

DESCRIPTION

- Inappropriate exposure of cornea to chemicals, heat, cold, electrical, or radiant energy causing damage to the cornea and often extending to adjacent structures
- Severity of injury related to duration of exposure, type of agent, anion concentration, pH level of solution
- Alkalis:
 - Cause immediate rise in pH level
 - Highly soluble in lipids, so rapidly penetrate the eye, causing severe corneal injury and continue to penetrate over time if no intervention undertaken
 - Penetration can occur in < 1 min.
 - Exception: Calcium alkalis penetrate relatively poorly secondary to soap formation; can cause corneal opacification, so may appear worse but actually have better prognosis than other alkali burns.
- Acids:
 - Immediately coagulate proteins of corneal epithelium
 - Cause opacification
 - Coagulation produces barrier to deeper penetration
 - Exception: Lipophilicity of hydrofluoric (HF) acid causes it to act similar to a base with more rapid penetration
- Thermal burns:
 - Affect eyelids more than globe due to reflex blinking and Bell phenomenon (eyes roll up and outward)
 - Cause direct injury to cornea
 - Damage primarily depends on duration and intensity of heat
- Electrical injury:
 - Occurs with current flow through head, with input at or near eye
- Radiation injury:
 - Due to ultraviolet light exposure to cornea

ETIOLOGY

- Alkalis:
 - Ammonia:
 - Fertilizer, refrigerant, household ammonia, cleansing agents
 - Potassium hydroxide:
 - Caustic potash
 - Magnesium hydroxide:

- Sparklers, flares, fireworks
- Lye: NaOH:
 - Caustic soda, drain cleaners
- Lime: CaOH₂ or MgOH₂:
 - Fresh lime, quicklime, calcium hydrate, slaked lime, hydrated lime, plaster, mortar, cement, whitewash
- Nonspecific alkali:
 - Motor vehicle airbag on inflation releases alkali.
- Acids:
 - Sulfuric acid: H₂SO₄:
 - Car battery acid, toilet cleaner
 - Sulfurous acid: H₂SO₃:
 - Preservatives (fruit and vegetable)
 - Acetic acid: CH₃CO₂H:
 - Vinegar
- Bleach
- Refrigerants:
 - HF acid:
 - Etching silicon/glass
 - Cleaning brick
 - Electropolishing metals
 - Control of fermentation in breweries
 - Commercial/household rust removal
- Thermal:
 - Hot liquids, molten metal
 - Flames
 - Hot smoke/gases
 - Flash burn
 - Steam
 - Cigarette burns
- Radiation:
 - Sun lamps
 - Tanning booths
 - High-altitude sunlight
 - Reflection off snow/water
 - Arc welding

Pediatric Considerations

Consider child abuse or neglect.

SIGNS AND SYMPTOMS

- Severe ocular pain
- Photophobia
- Lacrimation
- Foreign body sensation
- Conjunctival injection
- Corneal edema
- Corneal opacification
- Impaired visual acuity
- Limbal blanching
- Lens opacification
- Vesicles clear fluid (hypothermal injury)
- Vesicles hemorrhagic fluid
- Necrosis of iris, ciliary body

History

- Type of exposure:
 - _ Inspect any bottles accompanying the patient for active and inactive ingredients
- Vehicle of exposure:
 - _ Aerosol: Common
 - _ Propellant: May result in intraocular foreign body/perforation
- Duration of exposure
- Time of onset
- Time irrigation initiated
- Pre-existing visual impairment
- Protective eyewear
- Contact lens use
- Treatment before arrival

Physical-Exam

Complete eye exam (after irrigation):

- Visual acuity
- Bright white light for visual inspection of cornea/conjunctivae/limbus
- Slit-lamp to evaluate anterior segment inflammation
- Fluorescein stain:
 - _ Corneal epithelial damage:
 - Punctate corneal lesions with discrete lower border from inferior lid seen in UV radiation burns
 - _ Perforation (Seidel test)
- Check for lenticular clarity
- Fundus exam

- Measure intraocular pressure (especially in delayed presentation)
- Lid/eyelash exam
- Check pH with acid/alkali burns with litmus paper or pH indicator on urine dipstick

DIAGNOSIS TESTS & INTERPRETATION

Diagnostic Procedures/Surgery

- Fluorescein stain
- Check pH

DIFFERENTIAL DIAGNOSIS

- Infection:
 - Viral keratitis
 - Corneal ulcer
- Corneal erosion syndrome:
 - Corneal foreign body
 - Corneal abrasion
 - Hypothermal injury

Pediatric Considerations

Handheld slit-lamp and Wood lamp helpful in exam of child's eye



TREATMENT

PRE HOSPITAL

- Irrigate at scene 15–30 min unless other coexisting life-threatening conditions require immediate transfer
- Bring bottle of substance to hospital
- Continuous irrigation en route to hospital with NS or water

INITIAL STABILIZATION/THERAPY

- Chemical exposure:
 - Suspect acid or alkali in all exposures to unknown substances
 - Irrigate with any available diluting substance but preferably water or NS
- Thermal exposure:
 - Cool-moist dressing with overlying ice packs

ED TREATMENT/PROCEDURES

- Chemical exposure: Alkalis/acids/mace:
 - Continuous irrigation to achieve pH 7.3–7.5 (1–2 L via a Morgan lens > 30–60 min):
 - Measure pH every 30 min

- Dip pH paper in inferior conjunctival fornix
 - Topical anesthetic (proparacaine) may be necessary during irrigation
 - pH should be evaluated at 5 and 30 min after irrigation to ensure normalization of pH
 - Evaluate fornices in detail and eye in full range of motion to ensure removal of all particulate chemical substance
 - Antibiotic prophylaxis for Staphylococcus/Pseudomonas until epithelialization is complete:
 - Gentamicin ointment + erythromycin *or*
 - Bacitracin
 - Cycloplegics to minimize posterior synechiae formation:
 - Cyclopentolate 1%
 - Atropine 1%
 - Oral analgesics
 - If increased intraocular pressure:
 - Immediate ophthalmologic consultation
 - Administer acetazolamide 125 mg PO QID and timolol 0.5% drops BID
- Topical steroids to control anterior uveitis (consult ophthalmology)
- Eye patch (consult ophthalmology)
- May require surgical intervention if frank corneal penetration
- Ophthalmologic consultation by phone in mild injuries
- Immediate ophthalmologic consultation in all moderate to severe injuries; if unavailable at your hospital, arrange transfer to closest eye center
- HF acid:
 - Treat as above, + 1% calcium gluconate eyedrops
 - Systemic analgesia for 24 hr
- Thermal exposure:
 - Frequent moist dressing changes
 - Antibiotics drop QID
 - Generous lubricant application
 - Moisture chamber when extensive injury to eyelid
 - Steroids (consult ophthalmologist; do not use for > 1 wk)
 - Ophthalmology consultation for any 2nd- or 3rd-degree burn to eyelids
 - Cigarette ash and hot liquid splashes usually result in corneal epithelial injury:
 - Treat as corneal abrasion
- Electrical injury:
 - Irrigation
 - Wound care
 - Antibiotic ointment
 - Cycloplegic (if anterior uveitis)
 - Analgesia

- Radiation injury:
 - Topical anesthetic
 - Short-acting cycloplegic
 - Antibiotic ointment
 - Consider oral opioids for pain control

Pediatric Considerations

- Patching poorly tolerated
- May require systemic analgesia for complete exam

MEDICATION

- Artificial tears
- Atropine: 0.5%, 1%, 2% drops (cycloplegia 5–10 days, mydriasis 7–14 days) 1 drop TID
- Bacitracin ointment: QID
- Ciprofloxacin: 0.35% 1 drop QID
- Cyclopentolate: 0.5%, 1%, 2% drops (cycloplegia 1–2 days, mydriasis 1–2 days) 1 drop TID
- Erythromycin: 0.5% ointment QID
- Gentamicin: 0.3% ointment QID
- Gentamicin: 0.3% drops 1 drop q6h
- Homatropine: 5% drops 1–2 drop BID–TID
- Proparacaine: 0.5% drops 1 drop
- Sulfacetamide: 10% ointment QID
- Sulfacetamide: 10% drops QID
- Tetracaine: 0.5% drops 1–2 drops
- Tobramycin: 0.3% ointment q6h
- Tobramycin: 0.3% drops q6h
- Tropicamide: 0.5%, 1% drops (cycloplegia none; mydriasis 6 hr) 1 drop



FOLLOW-UP

DISPOSITION

Admission Criteria

- Intractable pain
- Increased intraocular pressure
- Corneal penetration requiring immediate surgical intervention
- HF acid burn; admit for 24 hr of systemic analgesia
- Suspected child abuse

Discharge Criteria

All mild corneal burns

FOLLOW-UP RECOMMENDATIONS

Mandatory follow-up with ophthalmologist in 12–24 hr; arrange before patient discharge

PEARLS AND PITFALLS

- In chemical exposures, delay exam until eye has been irrigated
- All patients with epithelial defects need 12–24 hr ophthalmology follow-up
- Do not prescribe topical anesthetics for discharged patients

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Corneal Abrasion
- Red Eye



CODES

ICD9

- 940.2 Alkaline chemical burn of cornea and conjunctival sac
- 940.3 Acid chemical burn of cornea and conjunctival sac
- 940.4 Other burn of cornea and conjunctival sac

ICD10

- H16.139 Photokeratitis, unspecified eye
- T26.10XA Burn of cornea and conjunctival sac, unsp eye, init encntr
- T26.60XA Corrosion of cornea and conjunctival sac, unsp eye, init

CORNEAL FOREIGN BODY

Ian C. May • Carl G. Skinner



BASICS

DESCRIPTION

- Foreign material on or in the corneal epithelium
- Corneal epithelium disrupted:
 - Abrasion if only epithelium disrupted
 - Scar if deeper layers of cornea involved

ETIOLOGY

- Foreign material causes inflammatory reaction:
 - May develop conjunctivitis, corneal edema, iritis, necrosis
- Poorly tolerated:
 - Organic material (plant material, insect parts)
 - Inorganic material that oxidizes (iron, copper)
- Well tolerated:
 - Inert objects (paint, glass, plastic, fiberglass, nonoxidizing metals)



DIAGNOSIS

SIGNS AND SYMPTOMS

- Foreign body (FB) sensation
- Eye pain
- Conjunctiva and sclera injection
- Tearing
- Blurred or decreased vision
- Photophobia
- Visible FB or rust ring
- Iritis

History

Common complaint: Something fell, flew, or otherwise landed in my eye:

- Hot, high-speed projectiles may not produce pain initially.

Physical-Exam

- Complete eye exam:
 - Visual acuity
 - Visual fields
 - Extraocular movements

- Lids and lashes
- Pupils
- Sclera
- Conjunctiva
- Anterior chamber
- Fundi:
 - Slit-lamp
 - Fluorescein exam
 - Perform Seidel test (visualization of flow of aqueous through corneal perforation during fluorescein slit-lamp exam)
 - Intraocular pressure if no evidence of perforation

ESSENTIAL WORKUP

- Injury history to determine type of FB and likelihood of perforation
- Exclude intraocular FB:
 - Suspect intraocular FB with high-speed mechanisms, such as machine operated or hammering metal on metal, or positive Seidel test.

DIAGNOSIS TESTS & INTERPRETATION

Imaging

- Orbital CT scan or B-mode US when suspect intraocular FB
- Orbital plain radiograph to screen for intraocular metallic FB

ALERT

Avoid MRI for possible metallic FBs.

DIFFERENTIAL DIAGNOSIS

- Conjunctival FB
- Corneal abrasion
- Corneal perforation with or without intraocular FB
- Corneal ulcer
- Keratitis



TREATMENT

PRE HOSPITAL

Place a Fox shield and position the patient upright.

INITIAL STABILIZATION/THERAPY

Apply topical anesthetic to stop eye discomfort and assist in exam.

ED TREATMENT/PROCEDURES

- Deep FBs:
 - Refer those penetrating the Bowman membrane (next layer under epithelium) to an ophthalmologist, because permanent scarring may occur.
- Superficial FBs:
 - Irrigation removal technique
- Apply topical anesthetic
- Try to wash FB off cornea by directing a stream of 0.9% NS at an oblique angle to cornea:
 - 25G needle or FB spud removal technique:
 - Using slit-lamp to immobilize patient's head and allow good visualization
 - Hold needle (bevel up) with thumb and forefinger, allowing other fingers to be stabilized on the patient's cheek.
 - Lift FB off cornea, keeping needle parallel to corneal surface.
- Rust rings removal:
 - Within 3 hr, iron-containing FBs oxidize, leaving a rust stain on adjacent epithelial cells.
 - Removal recommended as rust rings delay healing and act as an irritant focus
 - Remove with needle or pothook burr either at same time as FB or delayed 24 hr
- Postremoval therapy:
 - Recheck Seidel test to exclude corneal perforation.
 - Treat resultant corneal abrasion with antibiotic drops or ointment.
 - Initiate cycloplegic agent when suspect presence of keratitis.
 - Update tetanus.
 - Initiate analgesia (nonsteroidal anti-inflammatory drug [NSAID] or acetaminophen with oxycodone).

Pediatric Considerations

May require sedation to facilitate exam and FB removal

MEDICATION

- Cycloplegics:
 - Cyclopentolate 1–2%: 1 drop TID (lasts up to 2 days)
 - Homatropine 2% or 5%: 1 drop daily (lasts up to 3 days)
- Topical antibiotics for 3 to 5 days: Often used but unproven benefit:
 - Erythromycin ointment: Thin strip q6h
 - Sulfacetamide 10%: 1 drop q6h
 - Ciprofloxacin: 1 drop q6h
 - Ofloxacin: 1 drop q6h
 - Polymyxin/trimethoprim: 1 drop q6h
- Topical NSAIDs:

- Ketorolac: 1 drop q6h
- Diclofenac: 1 drop q6h



FOLLOW-UP

DISPOSITION

Admission Criteria

Globe penetration

Discharge Criteria

All corneal FBs

Issues for Referral

- Consult ophthalmologist for:
 - Vegetative material removal owing to risk of ulceration
 - Any evidence of infection or ulceration
 - Multiple FBs
 - Incomplete FB removal
- Ophthalmology follow-up in 24 hr for:
 - Abrasion in the visual field
 - Large abrasion
 - Abrasions that continue symptomatic or worsen the next day
 - Rust ring removal

FOLLOW-UP RECOMMENDATIONS

Return or follow-up with a physician if symptoms continue or worsen in 1 or 2 days.

PEARLS AND PITFALLS

- Consider intraocular FB, especially with history of high-projectile objects or industrial tools.
- Clinical evidence does not support eye patching for pain or healing.
- After removal, most corneal FBs can be treated as an abrasion and usually do well without further treatment.
- Topical anesthetics should not be prescribed for home use.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- [Corneal Abrasion](#)
- [Red Eye](#)



CODES

ICD9

930.0 Corneal foreign body

ICD10

- [T15.00XA Foreign body in cornea, unspecified eye, initial encounter](#)
- [T15.01XA Foreign body in cornea, right eye, initial encounter](#)
- [T15.02XA Foreign body in cornea, left eye, initial encounter](#)

COUGH

Alison Sisitsky Curcio



BASICS

DESCRIPTION

- A sudden spasmodic contraction of the thoracic cavity resulting in violent release of air from the lungs and usually accompanied by a distinctive sound:
 - Deep inspiration
 - Glottis closes
 - Expiratory muscles contract
 - Intrapulmonary pressures increase
 - Glottis opens
 - Air expiration at high pressure
 - Secretion and foreign material excretion
 - Vocal cord vibration with tracheobronchial walls, lung parenchyma, and secretions
- Defense mechanism to clear the airway of foreign material and secretions:
 - Voluntary or involuntary
 - Involuntary coughing regulated by the vagal afferent nerves:
 - Voluntary coughing under cortical control allowing for inhibition or voluntary cough
 - Because of cortical control, placebos can have a profound effect on coughing.
 - Reflex involves respiratory tissue receptor activation of afferent neurons to the central cough center followed by efferent output to the respiratory muscles.
 - Mechanical receptors in larynx, trachea, and carina sense touch and displacement.
 - Chemical receptors in larynx and bronchi are sensitive to gases and fumes.
 - Activated by irritants, mucus, edema, pus, and thermal stimuli
- Complications of severe coughing:
 - Epistaxis
 - Subconjunctival hemorrhage
 - Syncope
 - Pneumothorax
 - Pneumomediastinum
 - Emesis
 - Hernia
 - Rectal prolapse
 - Incontinence

- Seizures
- Encephalitis
- Intracranial hemorrhage
- Spinal epidural hemorrhage
- Clubbing
- Pruriginous rash

ETIOLOGY

- Acute (< 3 wk):
 - Pneumonia
 - Acute bronchitis
 - Sinusitis
 - Pertussis
 - Tuberculosis
 - Upper respiratory tract infection
 - Cough variant asthma
 - COPD exacerbation
 - Bronchiectasis
 - Pulmonary embolism
 - Left ventricular failure
 - Airway obstruction (food, pills)
 - GERD
 - Allergies
 - Bronchospasm
- Subacute (3–8 wk):
 - Postinfectious cough
 - Pertussis
 - Bronchitis
 - Bacterial sinusitis
 - Asthma
 - GERD
 - Pulmonary embolism
- Chronic (> 8 wk):
 - Postnasal drip
 - Asthma
 - GERD
 - Chronic bronchitis
 - Tuberculosis
 - Bronchiectasis
 - Eosinophilic bronchitis
 - ACE inhibitor use
 - Bronchogenic carcinoma
 - Carcinomatosis

- Sarcoidosis
- Left ventricular failure
- Aspiration syndrome
- Psychogenic/habit

Pediatric Considerations

- Most frequent causes:
 - Asthma
 - Viral illness
 - Acute bronchitis
 - Pneumonia
 - Sinusitis
 - GERD
- Less common causes:
 - Tracheobronchomalacia
 - Mediastinal tumor
 - Acyanotic congenital heart disease
 - Ventricular septal defect
 - Patent ductus arteriosus
 - Pulmonary stenosis
 - Tetralogy of Fallot
 - Lodged foreign body
 - Chronic aspiration of milk
 - Environmental exposure
- Consider:
 - Neonatal history
 - Feeding history
 - Growth and developmental history
 - Allergies
 - Eczema
 - Sleep disorders
- Indications for CXR:
 - Suspicion of foreign body ingestion
 - Suspect aspiration

DIAGNOSIS

SIGNS AND SYMPTOMS

- Sputum production:
 - Frothy (pulmonary edema)
 - Mucopurulent
 - Suggestive of bacterial pneumonia or bronchitis but also seen with viral

- infections
 - Rust colored (pneumococcal pneumonia)
 - “Currant jelly” (Klebsiella pneumonia)
 - Hemoptysis
- Post-tussive syncope or emesis (suggests pertussis)
- Shortness of breath
- Chest pain
- Chills/fever
- Night sweats
- Wheezing
- GERD:
 - Heartburn
 - Dysphagia
 - Regurgitation
 - Belching
 - Early satiety
- Malignancy:
 - Weight loss
 - Poor appetite
 - Fatigue

History

- Duration of cough to classify into acute, subacute, and chronic
- Description of sputum, if present, including hemoptysis
- Post-tussive emesis or syncope and paroxysmal cough suggests pertussis.
- History of GI symptoms pointing to GERD
- Weight loss and night sweats suggestive of tuberculosis in chronic cough

Physical-Exam

- Vital signs
- Abnormal breath sounds:
 - Absence or decreased: Reduced airflow vs. overinflation
 - Rales (crackles): Popping or rattling when air opens closed alveoli:
 - Moist, dry, fine, coarse
 - Rhonchi: Snoring-like sounds when large airways are obstructed
 - Wheezes: High-pitched sounds produced by narrowed airways
 - Stridor: Upper airway obstruction
- Evidence of respiratory distress:
 - Use of accessory muscles
 - Abdominal breathing

ESSENTIAL WORKUP

- Complete medical history:

- Duration
- Associated symptoms
- Sick contacts
- Smoking exposure
- ACE inhibitor use
- HIV/immunocompromised state
- Potential exposure to tuberculosis
- EKG:
 - History of cardiac disease
 - Associated chest pain or abnormal vital signs
 - Lack of infectious symptoms

DIAGNOSIS TESTS & INTERPRETATION

Lab

Order according to presenting signs and symptoms:

- WBC count with differential
- Sputum gram stain, cultures, and sensitivities
- Acid fast bacilli (AFB) culture
- CD4 count
- Pertussis titers
- D-Dimer
- Flu swab (for high-risk patients or those to be admitted)

Imaging

- CXR:
 - For immunosuppressed patient
 - At least 1 of the following in healthy patients with acute cough and sputum production:
 - Heart rate > 100 bpm
 - Respiratory rate > 24 breaths/min
 - Oral body temperature of > 38°C
 - Chest exam findings of focal consolidation, egophony, or fremitus
 - Ill appearing
 - Change in chronic cough
 - Continued cough after discontinuation of ACE inhibitor
- CT of chest:
 - Abnormal CXR
 - Assess for pulmonary embolism

Diagnostic Procedures/Surgery

- Peak flow

- **Bronchoscopy:**
 - For unknown mass on chest radiograph
 - Hemoptysis
 - Suspected cancer

DIFFERENTIAL DIAGNOSIS

See “Etiology.”



TREATMENT

INITIAL STABILIZATION/THERAPY

Assess airway, breathing, and circulation.

ED TREATMENT/PROCEDURES

Specific treatment related to cause:

- **Respiratory infection:** Consider antibiotics, antivirals (flu), decongestants, and antitussives.
- **Asthma:** Inhaled β_2 -agonist and steroids
- **GERD:** H₂-blockers, proton pump inhibitors, and antacids
- **Suspicion of pertussis:** Macrolide and 5 days isolation
- **Exacerbation of chronic bronchitis:** Inhaled β_2 -agonist and steroids
- **Malignancy:** Supportive care

MEDICATION

- **Antibiotics:**
 - Pick appropriate coverage for suspected bacteria.
- **Antivirals:**
 - Tamiflu: 75 mg (peds: 30–75 mg PO BID × 5 days) PO daily
- **Antitussives:**
 - Codeine: 10–20 mg (peds: 1–1.5 mg/kg/d) PO q4–6h
 - Dextromethorphan: 10–20 mg (peds: 1 mg/kg/d) PO q6–8h
 - Hydrocodone: 5–10 mg (peds: 0.6 mg/kg/d q6–8h) PO q6–8h
- **Bronchodilators:**
 - Albuterol: 2.5 mg in 2.5 NS (peds: 0.1–0.15 mg/kg/dose q20min) q20min inhaled
 - Ipratropium: 0.5 mg in 3 mL NS (peds: Nebulizer 250–500 μ g/dose q6h) q3h
- **Decongestants:**
 - Chlorpheniramine: 4–12 mg (peds: 2 mg PO q4–6h) PO q4–12h
 - Phenylpropanolamine: 25–50 mg (peds: 6.25–12.5 mg PO q4h) PO q4–8h
- **Mucolytics:**
 - Guaifenesin: 200–400 mg (peds: 2–5 yr 50–100 mg PO, 6–11 yr 100–200 mg) PO q4h PRN

- Steroids:
 - Dexamethasone: 2 sprays/nostril BID
 - Methylprednisolone: 60–125 mg IV (peds: 1–2 mg/kg/dose IV/PO q6h)
 - Prednisone: 40–60 mg (peds: 1–2 mg/kg/d q12h) PO



FOLLOW-UP

DISPOSITION

Admission Criteria

- Hypoxemia or critical illness
- Suspected tuberculosis with positive chest radiograph result
- Immunocompromised with fever
- Risk of bacteremia or sepsis

Discharge Criteria

- Oxygenation at baseline for patient
- Oral medications
- Safe environment at home

Issues for Referral

Close follow-up by primary care physician for outpatient management

FOLLOW-UP RECOMMENDATIONS

- Stop smoking, avoid being around smokers or other harmful substances such as asbestos.
- Change diet:
 - Avoid coffee, tea, and soda.
 - Avoid eating for at least 4 hr prior to sleeping.
- Use pillows to keep head elevated at night.
- Seek care immediately with:
 - Chest pain
 - Coughing blood
 - Shortness of breath
 - Fainting

PEARLS AND PITFALLS

- For patients fitting the clinical profile for cough due to GERD, it is recommended that treatment be initially started in lieu of testing.
- For patients with a presumed diagnosis of acute bronchitis, routine treatment with antibiotics is not justified and should not be offered.

ADDITIONAL READING

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- Irwin RS, Baumann MH, Bolser DC, et al. Diagnosis and management of cough executive summary: ACCP evidence-based clinical practice guidelines. *Chest.* 2006;129:1S.
- Schroeder K, Fahey T. Over-the-counter medications for acute cough in children and adults in ambulatory settings. *Cochrane Database Syst Rev.* 2004;18(4):CD001831.



CODES

ICD9

- 306.1 Respiratory malfunction arising from mental factors
- 786.2 Cough
- 786.30 Hemoptysis, unspecified

ICD10

- F45.8 Other somatoform disorders
- R04.2 Hemoptysis
- R05 Cough

CROUP

Dale W. Steele

BASICS

DESCRIPTION

- Viral infection of the upper respiratory tract
- Most commonly presents in children 6 mo–3 yr:
 - Laryngotracheitis/laryngotracheobronchitis
 - Inspiratory stridor owing to extrathoracic airway obstruction
 - Expiratory wheeze suggests lower airway involvement.
 - Inflammatory edema of subglottic region
 - Narrowest part of pediatric airway
- May progress to respiratory failure

ETIOLOGY

- Parainfluenza types 1, 2, and 3
- Human coronavirus NL63
- Influenza A and B
- Adenoviruses
- Respiratory syncytial virus
- Measles
- *Mycoplasma pneumoniae*
- Herpes simplex

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Nonspecific upper respiratory prodrome with or without fever
- Duration of illness
- History of tracheal intubation
- Possibility of foreign body aspiration
- Previous episodes
- History of wheeze
- Immunization status (*Haemophilus influenzae* type b [HIB]; diphtheria, pertussis, and tetanus [DPT]), influenza

Physical-Exam

- Rarely toxic appearing

- Cyanosis (not present in majority of patients. If present, suggests severe disease)
- Prefer upright position
- Quality of cry/voice
- Drooling/trismus/limited *neck extension*
- *Mental status*
- Stridor at rest, increased work of breathing
- Hydration status
- Westley croup score (max. total points: 17):
 - Stridor (inspiratory or biphasic):
 - 0 = None
 - 1 = Audible with stethoscope at rest
 - 2 = Audible without stethoscope at rest
 - Retractions:
 - 0 = None
 - 1 = Mild
 - 2 = Moderate
 - 3 = Severe
 - Air entry:
 - 0 = Normal
 - 1 = Decreased
 - 2 = Severely decreased
 - Cyanosis:
 - 0 = None
 - 4 = With agitation
 - 5 = At rest
 - Level of consciousness:
 - 0 = Normal
 - 5 = Altered

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Continuous pulse oximetry
- Other tests are not routinely indicated.

Imaging

Anteroposterior (AP) and lateral neck radiographs:

- Steeple sign indicates narrowing of subglottic trachea.
- Imaging not routinely indicated, unless atypical presentation or clinical course
- Subject to misinterpretation and should not be used as sole means to exclude epiglottitis
- Should not delay definitive visualization and intubation in OR in child with concern for epiglottitis or bacterial tracheitis

- Monitor child during imaging, if done.

DIFFERENTIAL DIAGNOSIS

- Infection:
 - Bacterial tracheitis
 - Retropharyngeal or parapharyngeal abscess
 - Epiglottitis
 - Peritonsillar abscess
 - Diphtheria
- Foreign body (airway or esophageal)
- Angioedema
- Congenital airway anomaly:
 - Laryngomalacia, tracheomalacia, laryngeal cleft
- Acquired subglottic stenosis
- Vocal cord paralysis
- Thermal or chemical injury to upper airway
- Hemangioma
- Laryngeal papillomatosis
- Vocal cord dysfunction (VCD) (adolescents)



TREATMENT

PRE HOSPITAL

- Allow child to maintain position of comfort.
- Defer interventions that may distress child such as:
 - IV access
 - IM injections
- If severe distress:
 - Immediate nebulized epinephrine

INITIAL STABILIZATION/THERAPY

- Nebulized racemic epinephrine or l-epinephrine if distress or stridor at rest:
 - l-epinephrine containing only the active isomer; has been shown to be therapeutically equivalent to racemic epinephrine
- Oxygen (via blow-by) for suspected or documented hypoxia suggesting severe disease
- Mist therapy often used, but no evidence for efficacy
- Dexamethasone:
 - Reduces need for intubation, shortens length of stay, and reduces admissions and return visits and may have effects within 30 min
 - Effective even in mild croup (Westley croup score ≤ 2)
- If poor response to nebulized racemic epinephrine or l-epinephrine:

- Consider trial of heliox:
 - Heliox, when available, has been used to decrease the work of breathing in patients with an incomplete response to epinephrine.
- If impending or existing respiratory failure despite aforementioned therapy:
 - Tracheal intubation by most experienced person available
 - Use uncuffed endotracheal tube (ETT) 0.5--1 mm smaller than usual size.
- If epiglottitis or foreign body suspected:
 - Ideally, to OR for inhalational anesthesia, direct laryngoscopy, and intubation
 - Surgeon standing by for emergent tracheostomy

ED TREATMENT/PROCEDURES

See “Initial Stabilization.”

MEDICATION

- Racemic epinephrine 2.25%: 0.25–0.5 mL nebulized in 2.5 mL NS
- l-epinephrine 1:1,000: 5 mL (5 mg) nebulized
- Dexamethasone: Single dose of 0.6 mg/kg (max. 10 mg) PO (use crushed tablet) or IV preparation (4 mg/mL) PO with flavored syrup. Equally effective when given PO, IV, or IM. Lower doses may be effective.
- Heliox (70% helium: 30% oxygen mixture administered via face mask or tent house)
- Antibiotics: Not indicated



FOLLOW-UP

DISPOSITION

Admission Criteria

- Young infants, pre-existing upper airway obstruction
- Persistent or recurrent stridor at rest unresponsive to nebulized epinephrine, or recurring during 2–3 hr observation
- Pediatric intensive care unit:
 - Persistent severe obstruction
 - Need for frequent epinephrine treatments and/or heliox
 - Tracheal intubation with assisted ventilation

Discharge Criteria

- Normal oxygenation in room air
- No stridor at rest after brief observation
- Children initially given epinephrine who no longer have stridor at rest should be observed for a min. of 2–3 hr

- Reliable caretaker, communication, and transport

Issues for Referral

- Concern for underlying anatomic abnormality (young age, history of intubation, frequent recurrence)
- Infants <1 year with stridor unassociated with laryngotracheobronchitis may require endoscopic evaluation

FOLLOW-UP RECOMMENDATIONS

- Most children with croup do not require specific follow-up.
- Patients who have had prolonged stridor, or acute worsening of stridor should seek care with their primary care physician or return to the ED.

PEARLS AND PITFALLS

- Beware young infants with stridor
- High incidence of congenital abnormalities
- Mild and early epiglottitis or bacterial tracheitis may mimic croup

ADDITIONAL READING

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- Zoumalan R, Maddalozzo J, Holinger LD. Etiology of Stridor in infants. *Ann Otolaryng, Rhinology, Laryngology*. 2007;116(5):329–334.



See Also (Topic, Algorithm, Electronic Media Element)

Epiglottitis



CODES

ICD9

- 464.4 Croup
- 464.20 Acute laryngotracheitis without mention of obstruction

ICD10

- J04.2 Acute laryngotracheitis
- J05.0 Acute obstructive laryngitis [croup]

CUSHING SYNDROME

Rami A. Ahmed • Stephen L. Chesser

BASICS

DESCRIPTION

- Cushing disease: Pituitary adenoma producing excess adrenocorticotrophic hormone (ACTH)
- Cushing syndrome: Excessive glucocorticoid effects

RISK FACTORS

Genetics

- Multiple endocrine neoplasia type I
- Carney complex (pigmented lentigines, atrial myxoma, germ-cell tumors with Cushing disease)

ETIOLOGY

- Most commonly exogenous administration of glucocorticoids either therapeutically or surreptitiously
- Pituitary adenoma secreting ACTH
- Adrenal production of cortisol from adenoma, carcinoma, or micronodular disease
- Tumor-producing ectopic ACTH:
 - Small cell lung carcinoma:
 - Most common
 - Uterine cervical carcinoma
 - Islet cell tumor of pancreas:
 - Multiple endocrine neoplasia (MEN) I-type syndrome
 - Medullary thyroid cancer
 - Pheochromocytoma
 - Ganglioneuroma
 - Melanoma prostate carcinoma
 - Carcinoid tumor:
 - Lung
 - Pancreas
 - GI tract
 - Thymus
 - Ovary

DIAGNOSIS

SIGNS AND SYMPTOMS

ALERT

- The most important aspect of Cushing syndrome in the ED is recognizing the potential for Addisonian (adrenal) crisis during periods of stress.
- Although nonemergent, the early recognition of Cushing syndrome may prevent morbidity and mortality.

Pediatric Considerations

Suspect if increasing in obesity while failing to maintain height on the growth chart

Pregnancy Considerations

Cushing syndrome rarely complicates pregnancy, but has been associated with severe pre-eclampsia and HELLP syndrome (*hemolysis, elevated liver function, and low platelets*)

History

- Cushing disease previously diagnosed
- Prior use of corticosteroids
- Characteristic appearance should lead to questions concerning change in weight, facial appearance, hirsutism, or psychiatric symptoms

Physical-Exam

- Diagnosis suggested by:
 - _ Abnormal fat deposition with moon facies
 - _ Buffalo hump
 - _ Central obesity with thin extremities
 - _ Supraclavicular fat deposition:
 - Above findings raise suspicion in a stressed patient of potentially developing Addisonian (adrenal) crisis
- Cardiovascular:
 - _ Uncontrolled hypertension
- Neurologic:
 - _ Atherosclerotic or embolic stroke
 - _ Pseudotumor cerebri (primarily with exogenous glucocorticoid administration):
 - Check fundi
- Spinal lipomatosis with cord or nerve-root compression
- Gastroenterologic:
 - _ Peptic ulcers
 - _ GI hemorrhage
 - _ Pancreatitis (primarily with exogenous glucocorticoid administration)

- Fatty liver
- Psychiatric:
 - Toxic psychosis
 - Mood disorders (40%)
 - Depression
 - Memory impairment
 - Euphoria
- Musculoskeletal:
 - Myopathy (proximal weakness)
 - Pathologic fractures
 - Osteoporosis
 - Aseptic necrosis humeral or femoral heads (primarily with exogenous glucocorticoid administration)
- Endocrine:
 - Glucose intolerance
 - Hyperlipidemia
 - Amenorrhea, female with male pattern balding, or hirsutism
- Hematologic:
 - Increased neutrophils
 - Decreased lymphocytes and eosinophils
 - Opportunistic infections
- Ophthalmologic:
 - Cataracts (primarily with exogenous glucocorticoid administration)
 - Glaucoma (primarily with exogenous glucocorticoid administration)
- Dermatologic:
 - Purple striae > 1 cm in diameter
 - Hyperpigmentation: Especially of buccal mucosa (from excess ACTH production)
 - Facial plethora
 - Thin skin
 - Impaired wound healing
 - Ecchymoses
 - Acne
 - Hyperhidrosis

ESSENTIAL WORKUP

- Cannot confirm diagnosis in ED
- Anticipate impending Addisonian (adrenal) crisis:
 - Most frequent and common problem with Cushing syndrome is its recognition with concurrent illness to prevent acute Addisonian crisis
- Search for life-threatening conditions:
 - MI
 - Stroke

- Sepsis
- Pathologic fracture
- Uncontrolled DM
- Psychiatric emergency necessitating admission

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Electrolytes, BUN, creatinine, glucose:
 - Hypokalemia
 - 10% with metabolic alkalosis
 - Diminished glucose tolerance (75%)
 - 20% overt DM
- Urinalysis:
 - 50% have glycosuria
- CBC:
 - Increased WBCs
 - Decreased eosinophils

Imaging

- ECG for myocardial ischemia
- CXR for tumor-causing ectopic ACTH
- Plain films if suspect possible pathologic fractures:
 - Delayed bone age

Diagnostic Procedures/Surgery

Nonemergent testing:

- MRI for pituitary tumor
- CT for adrenal carcinoma, adenoma, or hyperplasia
- Dexamethasone-suppression test (follow-up study with primary physician):
 - If suspicion of endogenous Cushing syndrome exists
 - Low-dose (screening test): 1 mg at 11:00 pm with an 8 am cortisol level drawn:
 - Low specificity
- False-positive results from alcohol, estrogens, spironolactone, phenytoin, barbiturates, and rifampin:
 - High-dose dexamethasone-suppression test needed to confirm the diagnosis:
 - 2 mg QID of dexamethasone with cortisol level 6 hr later
 - Compare day 2 urine-free cortisol and 17-hydroxyketosteroids with baseline levels.

DIFFERENTIAL DIAGNOSIS

- Alcohol-induced pseudo-Cushing syndrome

- Obesity
- Psychiatric states:
 - Depression
 - Obsessive–compulsive disorder
 - Panic disorder
- Physiologic states:
 - Chronic stress
 - 3rd-trimester pregnancy
 - Chronic strenuous exercise



TREATMENT

PRE HOSPITAL

- Acute addisonian (adrenal) crisis under stress may develop with iatrogenic Cushing syndrome
- Patients may have extremely labile behavior with violent behavior
- Leading causes of death in untreated Cushing syndrome are:
 - Infection
 - Stroke
 - MI

INITIAL STABILIZATION/THERAPY

- Anticipate addisonian (adrenal) crisis.
- Initiate treatment for associated complications:
 - MI
 - Stroke
 - Psychiatric stabilization

ED TREATMENT/PROCEDURES

- IV rehydration
- Glucose-lowering agents for hyperglycemia
- Appropriate cultures and antibiotics for suspected infection
- Antihypertensive agents for uncontrolled BP
- Administer steroids (hydrocortisone) with iatrogenic Cushing if patient under stress to prevent addisonian crisis.
- Medications to lower cortisol levels (bromocriptine, ketoconazole, aminoglutethimide, metyrapone):
 - Used rarely with severe symptoms in patients awaiting surgery
 - Institute under the direction of an endocrinologist.
 - Definitive therapy:
- Iatrogenic:
 - Taper steroids as rapidly as possible

- Calcium, vitamin D, and estrogen supplementation if possible
- Pituitary Cushing:
 - Transsphenoidal surgery
 - Radiation for surgical failures and a few select patients
- Adrenal adenoma/carcinoma:
 - Adrenal resection with medical therapy for metastatic lesions not resectable
- Ectopic ACTH:
 - Tumor resection (if possible) with medical therapy for metastatic lesions not resectable

MEDICATION

First Line

ONLY if in adrenal crisis: Hydrocortisone: 100 mg (peds: 1–2 mg/kg) IV q6h

Second Line

- In consultation with an endocrinologist
- SYMPTOMATIC TREATMENT ONLY as adjunctive therapy in patients awaiting surgery or refractory to other treatment
- Steroidogenic inhibitors:
 - Ketoconazole 200 mg PO BID
 - Methyrapone 0.5–1 g/d PO in 4 div. doses
 - Aminoglutethimide 250 mg PO q6h
 - Mifepristone 300 mg PO daily
- Adrenolytics:
 - Mitotane 500 mg PO daily
- ACTH release inhibitors:
 - Cyproheptadine 4 mg PO BID
 - Bromocriptine 2.5–30 mg/d
- Other:
 - Pasireotide 0.6 mg SQ initial
 - Spironolactone for symptomatic relief of HTN or hypokalemia



FOLLOW-UP

DISPOSITION

Admission Criteria

- Complications that require admission such as:
 - MI
 - Stroke
 - Sepsis

- Pathologic fracture
- Uncontrolled DM
- Psychiatric emergency
- Impending Addisonian (adrenal) crisis

Discharge Criteria

Well-appearing, stable patient without admission criteria

Issues for Referral

- Any patient suspected of Cushing syndrome for further evaluation
- Conditions secondary to Cushing requiring treatment

FOLLOW-UP RECOMMENDATIONS

Follow-up testing to confirm diagnosis

PEARLS AND PITFALLS

- Keep a high index of suspicion in the physiologically stressed patient by history or from body habitus and for the need to prevent against Addisonian crisis
- Suspect Cushing disease when there are supraclavicular fat pads

ADDITIONAL READING

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CODES

ICD9

255.0 Cushing's syndrome

ICD10

- E24.0 Pituitary-dependent Cushing's disease
- E24.2 Drug-induced Cushing's syndrome
- E24.9 Cushing's syndrome, unspecified

CYANIDE POISONING

Paul E. Stromberg • Kirk L. Cumpston

BASICS

DESCRIPTION

- Toxicity through inhalation, or GI tract absorption
- Intracellular toxin that inhibits aerobic metabolism through interruption of oxidative phosphorylation:
 - _ Leads to decreased O₂ utilization and ATP production
- Detoxification:
 - _ Rhodanese: Hepatic mitochondrial enzyme responsible for the metabolism:
 - Combines cyanide (CN) with sulfur (rate-limiting step) covalently (irreversible) to form less toxic and water-soluble thiocyanate (T-CN)
 - Forms less toxic reversible cyanhemoglobin when combined with hemoglobin (Fe 3+)
 - Forms nontoxic cyanocobalamin (B₁₂) when combined with hydroxocobalamin (B_{12a})
 - Rate of CN removal requires adequate bioavailability of sulfur compounds (thiosulfate [TS]).

ETIOLOGY

- Fires:
 - _ Combustion by-product of natural and synthetic products
- Industry:
 - _ Metal plating, microchip manufacturing
 - _ Chemical synthesis
 - _ Plastic manufacturing
 - _ Pesticides
- Solvents:
 - _ Artificial nail remover
 - _ Metal polishes
- By-product of nitroprusside metabolism (nonenzymatic)
- By-product of *Pseudomonas aeruginosa* and pyocyanus infections
- Amygdalin (converted by intestinal flora to CN), CN-containing plants (apricot and peach pits, apple and pear seeds, and cassava)
- Jewelry making

DIAGNOSIS

SIGNS AND SYMPTOMS

- Heart and brain—most sensitive organs—1st to show manifestation of toxicity
- CNS:
 - Headache
 - Confusion
 - Syncope
 - Seizures
 - Coma
- Cardiovascular:
 - Dyspnea
 - Chest pain
 - Cardiorespiratory collapse and death
- Other:
 - Nausea/vomiting
- Oral exposure: Can be caustic, 50 mg has caused death.
- Inhalational exposure:
 - 50 ppm causes anxiety, palpitations, dyspnea, headache.
 - 100–135 ppm < 1 hr is lethal.

ESSENTIAL WORKUP

- History of exposure:
 - Smoke inhalation
 - Industrial exposure
 - Intentional suicide
 - Intentional homicide
- Clinical clues (frequently absent):
 - Peculiar odor of bitter almonds
 - Bright red (arterialization) retinal vessels
 - Abrupt onset and/or deteriorating toxic effects
 - Lactic acidosis
 - High venous O₂ saturation (secondary to blocked cellular O₂ consumption); arterialization of venous blood gases

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC
- Electrolytes, BUN, creatinine, glucose:
 - Anion gap acidosis
- Liver profile
- Creatine phosphokinase (CPK)
- Carboxyhemoglobin (CO) level
- Methemoglobin (MH) level

- CN level:
 - Send out lab that is not usually available in a clinically relevant time period.
 - Levels > 0.5–1 mg/L: Toxic
 - Levels 2.5–3 mg/L: Fatal
- Blood gas determinations:
 - Elevated mixed venous O₂: MvO₂ (normal about 35–40)
 - Elevated mixed venous O₂ saturation (co-oximeter): SmvO₂ (normal about 75%)
 - Decreased arteriovenous O₂ difference: AVO₂D (normal about 3–4.8 mL/dL)
- Elevated lactate level > 8 mmol/L:
 - An elevated lactate is a surrogate marker for the presence of CN with the appropriate history and physical exam.

Imaging

CXR

DIFFERENTIAL DIAGNOSIS

- Carbon monoxide
- Hydrogen sulfide
- Methemoglobinemia
- Sulfhemoglobinemia
- Inert gases “asphyxiants”
- Other causes of high anion gap metabolic acidosis



TREATMENT

PRE HOSPITAL

- Remove source of CN.
- Prevent others from becoming contaminated.
- Remove and bag all contaminated clothing and wash affected areas copiously with soap and water if a liquid exposure. If vapor contamination, removal of the patient from the CN environment may be all that is necessary.

INITIAL STABILIZATION/THERAPY

- ABCs:
 - Administer 100% oxygen:
 - Even in presence of normal PaO₂
 - Acts synergistically with antidotes
- Gastric decontamination for oral ingestions if within 1 hr:
 - Perform gastric lavage and administer activated charcoal (AC) if ingestion

of solid CN or CN-containing products and no contraindications.

– Do not induce emesis.

- Dermal exposure: Standard decontamination

ED TREATMENT/PROCEDURES

- Hydroxocobalamin (B_{12a}) Cyanokit®:

– Administer if manifesting significant CN toxicity with persistent high anion gap metabolic acidosis and hyperlactatemia, with any syncope, seizures dysrhythmias, and hypotension.

– Administration often instituted empirically; CN levels not immediately available

– Binds to CN:

- Forms nontoxic cyanocobalamin (B₁₂); renally excreted

– Advantages:

- No MH induction
- Does not cause hypotension
- Intracellular distribution

– Limitations:

- Cost

– Incompatible in the same IV line with:

- Diazepam
- Dobutamine
- Dopamine
- Fentanyl
- Nitroglycerin
- Pentobarbital
- Propofol
- Sodium thiosulfate
- Sodium nitrite
- Ascorbic acid
- Blood products

– Side effects of hydroxocobalamin:

- HTN
- Red skin and all secretions
- Interference of colorimetric assays of AST, ALT, total bilirubin, creatinine, Mg, iron

- CN antidote kit:

– Administer if manifesting significant CN toxicity with persistent high anion gap metabolic acidosis, hyperlactatemia with any syncope, seizures dysrhythmias, and hypotension.

– Administration often instituted empirically; CN levels not immediately available

- Contents: Amyl nitrite pearls, sodium nitrite, and sodium thiosulfate
- Nitrite action:
 - o Induce a CN-scavenging MH by oxidizing hemoglobin (Fe^{2+} to Fe^{3+}), which attracts extracellular CN away from the mitochondria-forming CN-MH, which is less toxic.
 - o Do not administer empirically or prophylactically.
- Sodium thiosulfate action:
 - o Substrate for the enzyme rhodanese
 - o Combines with CN to form a less toxic T-CN
- Hyperbaric oxygen therapy:
 - Can be used to treat CN exposures
 - Maximizes tissue oxygenation despite toxic MH level

MEDICATION

AC: 1 g/kg PO

First Line

Hydroxocobalamin (B_{12a}):

- 70 mg/kg IV, max. 5 g
- The kit contains either two 2.5 grams/bottle or one 5 gram/bottle. The starting dose is 5 grams.
- Reconstitute the powder by gently rolling the bottle after filling with 100 mL of 0.9% NS.
- Infuse each 2.5 gram bottle over 7.5 minutes, or one 5 gram bottle over 15 minutes. The 5 gram dose can be repeated.

Consider adjunctive use of sodium thiosulfate

Second Line

- CN antidote kit: Amyl nitrite, sodium nitrite, and sodium thiosulfate:
- Amyl nitrite pearls:
 - Crush 1 or 2 ampules in gauze and hold close to nose, in lip of face mask, or within Ambu bag.
 - Inhale for 30 sec–1 min until IV access obtained.
- Sodium nitrite (NaNO_2): 10 mL (300 mg) (peds: 0.15–0.33 mL/kg) IV as 3% solution over 5–20 min:
 - May repeat once at half dose within 30–60 min
 - Keep MH level < 30%.
 - Dilute; infuse slowly if hypotensive.
- Sodium thiosulfate: 50 mL: 12.5 g (peds: 0.95–1.65 mL/kg) IV over 10–15 min of 25% solution:
 - 1/2 initial dose may be given after 30–60 min.

Pregnancy Considerations

- Hydroxocobalamin is class C.
- Amyl nitrite is class X.
- Sodium nitrite is unknown.
- Sodium thiosulfate is class C.

Geriatric Considerations

- ~ 50 known or suspected CN victims aged 65 or older received hydroxocobalamin and it had similar safety and efficacy as younger patients.
- Hydroxocobalamin is renally excreted unchanged in the urine so renal impairment could prolong the elimination half-life.
- The safety and effectiveness of hydroxocobalamin is unknown in hepatic impairment.
- Sodium thiosulfate is metabolized in the liver and excreted by the kidney. Impairment in either organ may prolong elimination.
- The nitrites are short acting. Hepatic or renal impairment may prolong elimination.

Pediatric Considerations

The safety and effectiveness of hydroxocobalamin has not been established in children, but the 70 mg/kg dose has been used.

ALERT

- Sodium nitrite has weight-based dosing for children.
- Sodium nitrite dosing can be based on serum hemoglobin when the clinical scenario does **NOT** require life-saving administration of the antidote before lab testing:

Hgb Nitrite (mg/kg) Nitrite (mL/kg)

7	5.8	0.19
8	6.6	0.22
9	7.5	0.25
10	8.3	0.27
11	9.1	0.30
12	10.0	0.33
13	10.8	0.36
14	11.6	0.39



FOLLOW-UP

DISPOSITION

Admission Criteria

ICU admission of all symptomatic exposures

Discharge Criteria

- Asymptomatic patients after at least 4 hr of observation
- Survival after 4 hr of acute exposure usually associated with complete recovery

Issues for Referral

Psychiatry referral for intentional overdose and suicidal patients

PEARLS AND PITFALLS

- In a patient with hypotension, high anion gap metabolic acidosis, hyperlactatemia, seizures, syncope, altered mental status consider CN in the differential diagnosis and treat presumptively.
- Use serum lactate as a surrogate marker for CN exposure.
- Victims of smoke inhalation may have combination of:
 - _ CN toxicity
 - _ MH
 - _ CO toxicity
 - _ If the COHgb concentration is extremely elevated, considered a concomitant CN exposure as well
 - _ To avoid further reduction in oxygen transport; initially treat with hydroxocobalamin or sodium thiosulfate, without sodium nitrite to avoid methemoglobinemia.

ADDITIONAL READING

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CODES

ICD9

- 987.7 Toxic effect of hydrocyanic acid gas
- 989.0 Toxic effect of hydrocyanic acid and cyanides

ICD10

- T65.0X1A Toxic effect of cyanides, accidental (unintentional), initial encounter
- T65.0X2A Toxic effect of cyanides, intentional self-harm, initial encounter
- T65.0X4A Toxic effect of cyanides, undetermined, initial encounter

CYANOSIS

Michael S. Murphy



BASICS

DESCRIPTION

Abnormal bluish discoloration of the skin or mucous membranes

- Caused by abnormal elevations of deoxygenated hemoglobin or hemoglobin derivatives in the capillaries:
 - Deoxygenated hemoglobin > 5 g/dL
 - Methemoglobin > 1.5 g/dL
 - Sulfhemoglobin > 0.5 g/dL
- The absolute amount of deoxygenated hemoglobin is the pigment that creates the bluish tint
 - The amount of oxyhemoglobin does not affect the blood's color
 - Cyanosis is more common in patients with polycythemia and less common in patients with anemia.
- Cyanosis varies based on skin thickness or pigment
- Accumulation of deoxygenated hemoglobin may be systemic producing central cyanosis or localized producing peripheral cyanosis
 - Central cyanosis
 - Hypoxemia
 - Anatomic right to left shunts
 - Abnormal hemoglobin derivatives
 - Peripheral cyanosis
 - Tissue extracts more than normal amounts of O₂ from the blood
 - Hypoperfusion
 - Vasoconstriction to cold air or water
 - Arterial insufficiency
 - Venous insufficiency
 - Acrocyanosis: Painless, symmetrical, cyanosis in distal parts of body, the pathophysiologic cause of which is not known

ETIOLOGY

Central cyanosis

- Impaired pulmonary function:
 - Hypoventilation:
 - Pneumonia
 - Chronic obstructive pulmonary disease
 - Pulmonary edema
 - Ventilation/perfusion mismatch:

- Asthma
- Pulmonary embolus
- Diffusion problems:
 - Interstitial lung disease
 - Anatomic shunts
- Pulmonary arteriovenous fistula:
 - Hereditary hemorrhagic telangiectasia
- High-altitude related, with decreased atmospheric pressure at 16,000 ft
- Cardiac abnormalities with right to left shunt
 - Eisenmenger syndrome
 - Pulmonary hypertension
 - Longstanding intracardiac shunt (VSD, patent ductus arteriosus, ASD)
 - Reversal of flow through detected when pulmonary artery pressure exceeds threshold
- Abnormal hemoglobin
 - Low-oxygen affinity hemoglobin mutants:
 - Hb Kansas
 - Hb Beth Israel
 - Hb St. Mandé
 - Congenital methemoglobinemia:
 - Cytochrome *b5* reductase deficiency
 - Hemoglobin M disease
 - Acquired methemoglobinemia:
 - Aniline dyes
 - Chloroquine, primaquine
 - Dapsone
 - Local anesthetic agents such as lidocaine
 - High doses of methylene blue
 - Naphthalene
 - Nitrites, nitroglycerine
 - Sulfonamides
 - Fava beans
 - Sulfhemoglobin:
 - Generally benign
 - Irreversible alteration of hemoglobin
 - Caused by many medications
 - Dimethyl sulfoxide
 - Paint
 - Phenacetin
 - Phenazopyridine
 - Phenylenediamine
 - Phenylhydroxylamine
 - Sulfanilamide

- Sulfapyridine
- Sulfathiazole
- Sulfur compounds

Peripheral cyanosis

- Shock
- Exposure to cold
- Arterial insufficiency
- Venous insufficiency
- Raynaud phenomenon
- Acrocyanosis

Pediatric Considerations

- Cardiac:
 - _ Cyanotic congenital defects:
 - Tetralogy of Fallot
 - Transposition of great vessels
 - Truncus arteriosus
 - Pulmonary and tricuspid atresia
 - Ebstein anomaly
 - Pseudocoarctation
 - Patent ductus arteriosus
 - _ Total anomalous pulmonary venous return
- Pulmonary stenosis:
 - _ Any right-to-left shunting
- Respiratory:
 - _ Upper airway disorders:
 - Croup
 - Bacterial tracheitis
 - Epiglottitis
 - Retropharyngeal abscess
 - Foreign body
 - _ Lower airway disorders:
 - Asthma
 - Bronchiolitis
 - Pneumonia
 - Cystic fibrosis
 - Pulmonary edema/CHF
 - Pulmonary embolism
- Neurologic:
 - _ Breath holding



SIGNS AND SYMPTOMS

- A bluish discoloration of the skin and mucous membranes that blanches with pressure:
 - Chocolate color:
 - Methemoglobinemia
 - Slate gray color:
 - Methemoglobinemia, sulfhemoglobin
 - Reddish blue
 - Venous stasis

History

- Establish timing of onset of cyanosis
- Associated symptoms
 - Pain
 - Dyspnea
 - Fatigue
 - Headache
 - Changes in mental status
- Medication list
- Occupational exposure or use of chemicals or drugs

Physical-Exam

- General appearance and vital signs for shock and respiratory distress
- Does the discoloration blanch with pressure?
 - Distinguishes cyanosis from abnormal skin pigmentation
- Location of discoloration
 - Symmetrical involving extremities and mucus membranes
 - Central cyanosis
 - Face, neck, and upper extremities
 - Superior vena cava syndrome
 - Lower extremities with upper extremities unaffected
 - Differential cyanosis
 - Pseudocoarctation and patent ductus arteriosus
 - Inferior vena cava syndrome
 - Single extremity
 - Arterial or venous insufficiency
 - Symmetrical, painful, involving extremities
 - Raynaud phenomenon
 - Symmetrical, painless, involving extremities and face with hyperhidrosis
 - Acrocyanosis
- Clubbing
 - Chronic hypoxemia

- Pulmonary exam
- Cardiac exam
- Extremities for edema, pulses, and temperature

ESSENTIAL WORKUP

- Assess airway and ventilation as 1st priority:
 - _ Stabilize airway and provide adequate ventilation.
- Investigate hypoxemia causes:
 - _ Cardiac and respiratory most common
 - _ Consider methemoglobinemia

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Pulse oximetry:
 - _ Does not assess ventilation
 - _ Results inaccurate with:
 - Abnormal hemoglobins
 - Nail polish
 - Pigmented skin
 - Hypoperfusion
 - Use of vital dyes
- Arterial blood gas:
 - _ Oxygen tension
 - _ Measured hemoglobin saturation
 - _ Cyanosis in face of normal PO₂, think methemoglobinemia
 - _ Blood in methemoglobinemia is chocolate color.
 - _ Methemoglobin level
- Complete blood chemistry:
 - _ Check hemoglobin.
- Hyperoxia test for congenital cyanosis of newborn:
 - _ If PO₂ fails to increase to 100 mm Hg after 100% O₂, suspect congenital heart disease.

Imaging

- CXR to investigate respiratory or cardiac pathology:
 - _ Inspiratory/expiratory views if foreign body
 - _ Expiratory view if occult pneumothorax suspected
- Radiograph of neck for upper airway disorders:
 - _ Foreign body
 - _ Steeple sign (croup)
 - _ Prevertebral swelling (retropharyngeal abscess)

- Epiglottic swelling
- EKG:
 - Dysrhythmia, injury, or ischemia
- Echo:
 - Bubble study if septal defect/shunt suspected
 - Wall motion/valvular abnormalities
 - Pericardial fluid

DIFFERENTIAL DIAGNOSIS

- Abnormal skin pigmentation (fails to blanch with pressure)
 - Amiodarone
 - Minocycline
 - Chronic high-dose chlorpromazine
 - Argyria (silver deposits)
 - Arsenic
 - Alkaptonuria
 - Chrysiasis (secondary to parenteral administration of gold salts)
 - Tattoos
- Chromhidrosis
 - Rare condition characterized by the secretion of colored sweat



TREATMENT

PRE HOSPITAL

- Assess and establish patent airway.
- Correct any airway obstruction.
- Recognize an incorrectly placed airway.
- 100% O₂ using a nonrebreathing device
- Ensure adequate ventilation.
- Recognize need to establish definitive airway.
- Protect cervical spine if trauma suspected.
- IV line, monitor, pulse oximetry
- Albuterol nebulizer for bronchospasm
- Racemic epinephrine nebulizer for severe croup
- Management of pulmonary edema per protocol

INITIAL STABILIZATION/THERAPY

- Oxygen supplied through a 100% nonrebreathing device
- Immediately assess and address airway issues.

TREATMENT GENERAL MEASURES

- Recognize and manage cardiopulmonary disorders.

- Methylene blue for methemoglobinemia exceeding 30%:
 - _ Do not use if patient has G6PD deficiency.

MEDICATION

- Albuterol nebulized: 0.03 mL/kg (5 mg/mL)
- Dexamethasone: (For croup) 0.75–9 mg/d in div. doses q6–12h
- Furosemide: 0.5 mg/kg IV over 1–2 min. May double the dose after 1 hr if unsatisfactory response.
- Magnesium: 2 g IV over 10 min (40 mg/kg IV over 20 min)
- Methylene blue: 1–2 mg/kg IV of 1% solution over 5 min
- Methylprednisolone: 1–2 mg/kg IV q6h
- Morphine: 2–4 mg IV (0.05–0.1 mg/kg IV q2h PRN)
- Nitroglycerine: USE NON-PVC tubing. 5 µg/min, titrate up by 5 µg/min every 3–5 min until desired effect
- Prostaglandin E₁: 0.05–0.1 µg/kg/min IV; max. 0.4 µg/kg/min
- Racemic epinephrine nebulized: 0.25–0.75 mL of 2.25% solution diluted in 2 mL NS



FOLLOW-UP

DISPOSITION

Admission Criteria

- Most affected patients should be admitted to the hospital.
- ICU admission is required for any instability or cyanosis.

Discharge Criteria

Reversible causes of hypoxia:

- Reactive airway disease responsive to β-agonists
- Pulmonary edema in patient with known CHF but no suspicion of myocardial injury and diuresis

PEARLS AND PITFALLS

- First assume hypoxemia and immediately assess airway and breathing
- Chocolate-colored blood or unchanging oxygen saturation despite aggressive administration of oxygen: Think methemoglobinemia.

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- Stack A. Cyanosis. *Synopsis of Pediatric Emergency Medicine.* 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2002:64–67.



CODES

ICD9

- 770.83 Cyanotic attacks of newborn
- 782.5 Cyanosis

ICD10

- P28.2 Cyanotic attacks of newborn
- R23.0 Cyanosis

CYSTIC FIBROSIS

Joseph Kahn

BASICS

DESCRIPTION

- Defect of the cystic fibrosis transmembrane conductance regulator (CFTR)
- CFTR functions as an ATP-regulated chloride channel that regulates the activity of chloride and sodium channels on the cell surface:
 - Abnormal electrolyte transport in exocrine glands and secretory epithelia
 - Decreased exocrine pancreatic function with malabsorption
 - Thickened mucus, recurrent pulmonary infections, and progressive obstructive damage to the lungs
 - Recurrent sinus disease
- Occurs in 1:3,600 live births in White population, 1:29,000 in African American population; 1:6,500 in Hispanic population
- 30% of cases diagnosed by newborn screening
- 75% cases diagnosed in the 1st 2 yr of life
- ~ 30,000 children and young adults in US have CF.
- Median life expectancy in US about 40 yr.
- 40% of CF patients are older than 18 yr.
- 10 million Americans are unknown, asymptomatic carriers of the defective gene.
- 16% of lung transplants in US due to CF

RISK FACTORS

Genetics

Recessively inherited genetic disease, involving the CFTR gene on the long arm of chromosome 7:

- Different mutations; variable phenotypes.
- Classic disease: Homozygous DF508 mutation.
- Most common lethal genetic disease in US

ETIOLOGY

Common organisms in patients with pneumonia; often multiple drug resistance:

- *Staphylococcus aureus*:
 - MSSA/MRSA
- *Pseudomonas aeruginosa*:
 - Prevalence increases with age; >70% of adults are chronically infected.
- *Haemophilus influenzae*
- *Stenotrophomonas maltophilia*

- *Burkholderia cepacia*:
 - Prevalence 3%
 - Associated with rapid clinical deterioration
- *Achromobacter xylosoxidans*
- Mycobacteria (nontuberculous):
 - *Mycobacterium avium* complex, *Mycobacterium abscessus*
- *Aspergillus*

DIAGNOSIS

SIGNS AND SYMPTOMS

- General:
 - Failure to thrive
 - Recurrent respiratory tract infections
 - Anasarca in infancy
 - Salty taste of skin
- Head, ears, eyes, nose, and throat (HEENT):
 - Nasal polyps; severe headaches due to sinusitis; otitis media
- Pulmonary:
 - Persistent cough. Initially dry, then productive
 - Recurrent pneumonitis or bronchiolitis in 1st yr of life
 - Wheezing
 - Hemoptysis
 - Pneumonia
 - Chronic bronchitis
 - Bronchiectasis
 - Respiratory distress
 - Pneumothorax
 - Pneumomediastinum
 - Most common cause of CF hospitalization
- Cardiac:
 - CHF
 - Cor pulmonale; pulmonary hypertension
- GI:
 - Abdominal pain
 - Meconium ileus
 - Distal intestinal obstructive syndrome (DIOS) or “meconium ileus equivalent”
 - Gastroesophageal reflux
 - Cholelithiasis
 - Pancreatitis/pancreatic insufficiency
 - Ileocecal intussusception

- Foul smelling, fatty stools
- Jaundice/cirrhosis
- Rectal prolapse
- Hematemesis
- Small intestine bacterial overgrowth
- Extremities:
 - Bone pain
 - Edema/joint effusions
 - Decreased thickness of cortical bone
- Recurrent venous thrombosis
- Cardiorespiratory failure is most common cause of death.

ESSENTIAL WORKUP

- Sweat chloride test
- DNA analysis if sweat test equivocal
- Nasal potential difference if DNA inconclusive

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Sweat chloride test:
 - Chloride concentration > 60 mEq/L
 - With classic signs and symptoms, a positive test result confirms the diagnosis.
- Stool sample:
 - Decreased elastase, trypsin, or chymotrypsin
 - Increased fat in 72-hr fecal fat excretion
- Immunoreactive trypsin (IRT):
 - Defines increased risk and/or diagnosis
 - May be falsely positive or negative
- DNA analysis:
 - Indicated if symptoms are highly suggestive, but sweat test result is negative
 - 90% of CF chromosomes identified
 - Positive if 2 abnormal genes present
 - Genotyping cannot establish the diagnosis.
 - 1,300 CFTR mutations listed
 - Ameliorating or neutralizing 2nd mutation may be present.
- CBC:
 - Thrombocytopenia
- Serum electrolytes:
 - Hyponatremic, hypochloremic alkalosis
- Serum glucose:
 - Hyperglycemia and new-onset diabetes in adolescents and adults;

ketoacidosis is rare.

- Liver function tests and PT:
 - Obtain if hematemesis or hemoptysis or signs of liver failure
- ABG:
 - Hypoxemia. Metabolic alkalosis
- Sputum culture:
 - May have pseudomonal colonization.
- Studies indicated in high-risk patients with unclear diagnosis:
 - Nasal potential-difference measurements:
 - Complex and time-consuming study
 - Semen analysis:
 - Azoospermia

Imaging

- Chest radiograph:
 - Hyperaeration
 - Peribronchial thickening
 - Atelectasis
 - Hilar lymphadenopathy
 - Pneumothorax/pneumomediastinum
 - Bronchiectasis
 - Blebs
 - Chest CT identifies blebs/bronchiectasis
- Abdominal radiographs and/or CT:
 - Indicated if abdominal pain, vomiting, or abdominal distention
 - Distal intestinal obstruction syndrome
 - Intussusception
- Barium enema:
 - Indicated if suspicion of intussusception
- Sinus films:
 - Limited use because routine sinus films are always cloudy
 - CT scan is needed to assess sinuses for pre-operative planning.

Diagnostic Procedures/Surgery

Bronchoalveolar lavage:

- High percentage of neutrophils and absolute neutrophil count
- Unnecessary if obvious pulmonary symptoms

DIFFERENTIAL DIAGNOSIS

- Respiratory:
 - Asthma
 - Recurrent pneumonia
 - Bronchiectasis

- Pertussis
- Immunodeficiency
- Foreign body aspiration
- α_1 -Antitrypsin deficiency
- Ciliary agenesis
- GI:
 - Chronic diarrhea
 - Gastroenteritis
 - Milk allergy
- Elevated electrolyte levels in sweat:
 - Fucosidosis
 - Glycogen storage disease type I
 - Mucopolysaccharidosis
 - Hypothyroidism
 - Vasopressin-resistant diabetes insipidus
 - Adrenal insufficiency
 - Familial cholestasis
 - Familial hypoparathyroidism
 - Malnutrition
 - Ectodermal dysplasia
 - Atopic dermatitis
 - Infusion of prostaglandin E₁



TREATMENT

PRE HOSPITAL

- Transcutaneous pacing for unstable type II 2nd- or 3rd-degree block
- Atropine:
 - Avoid with type II 2nd-degree block because it may precipitate 3rd-degree block.

ED TREATMENT/PROCEDURES

- Stabilize airway, breathing, and circulation:
 - Correct fluid, respiratory, electrolyte, and glucose abnormalities.
 - Bronchodilators/steroids if wheezing.
- Pneumothorax:
 - Observe if <5–10%.
 - Thoracostomy
- Consultation with the primary CF physician or pulmonary specialist
- Right heart failure:
 - Diuretics
- Hemoptysis:

- Blood products as indicated (check INR)
- Ventilatory support
- DIOS:
 - Usually requires surgery
- Hematemesis:
 - Packed RBCs
 - Blood products for coagulation abnormalities
 - Early consultation with endoscopist
- Intussusception:
 - Correct with barium/air enema
 - May require surgery
- Rectal prolapse:
 - Manual reduction
 - Consider surgical consult
- Respiratory care:
 - Pulmonary toilet/physical therapy
 - Mucous thinning inhaled agents
- Antibiotics for pneumonia:
 - Based on culture and sensitivity
 - *S. aureus* (MSSA):
 - Cephalothin or Nafcillin
 - *S. aureus* (MRSA):
 - Vancomycin or linezolid
 - *P. aeruginosa*:
 - (Tobramycin or amikacin or colistin) + (piperacillin/tazobactam or ticarcillin/clavulanate or ceftazidime or imipenem/cilastatin or meropenem)
 - *S. aureus* (MSSA) and *P. aeruginosa*:
 - (Piperacillin/tazobactam or ticarcillin/clavulanate or cefepime or imipenem/cilastatin or meropenem) + (tobramycin or amikacin or colistin)
 - *S. aureus* (MRSA) and *P. aeruginosa*:
 - (Vancomycin or linezolid) + coverage for Pseudomonas alone
 - *B. cepacia*:
 - Trimethoprim–sulfamethoxazole and/or meropenem and/or cipro and/or minocycline and/or chloramphenicol
 - *H. influenzae*:
 - Cefotaxime or ceftriaxone
 - Sinusitis
 - Based on cultures and sensitivities

Note: Ciprofloxacin may replace the aminoglycoside if sensitive pseudomonas

- CFTR modulation: Ivacaftor
 - Repair of protein function

- Restore airway surface liquid:
 - Nebulized hypertonic saline
- Mucous alteration:
 - Dornase alpha to thin mucus in lungs
- Future directions:
 - Gene therapy: Compacted DNA
 - Anti-inflammatory: High-dose ibuprofen
 - Anti-infective agents:
 - Inhaled tobramycin, aztreonam, colistin
 - Continuous vancomycin infusion
 - Transplantation: Inhaled cyclosporine
 - Nutrition and exercise

MEDICATION

- Amikacin: 7.5–10 mg/kg IV q8h
- Cefazolin: 100 mg/kg/d IV (max.: 6 g/d)
- Cefepime: 50 mg/kg IV q8h (max.: 2 g/8hr)
- Ceftazidime: 50 mg/kg IV q8h (max.: 6 g/d)
- Colistin: 2.5–5 mg/kg/d IV, div. BID–QID
- Imipenem/cilastatin: 15–25 mg/kg IV q6h
- Meropenem: 40 mg/kg IV q8h (max.: 2 g/8hr)
- Nafcillin: 25–50 mg/kg IV q6h (max. 2–3 g q6h)
- Piperacillin/tazobactam: 350–450 mg/kg/d IV (max.: 4.5 g q6h)
- Ticarcillin/clavulanate: 300–400 mg/kg/d IV q6h
- Tobramycin: 2.5–3.3 mg/kg/dose IV q8h
- TMP–SMX: 5–10 mg/kg IV q12h (max.: 160 mg TMP q12h)
- Vancomycin: 15 mg/kg q6h (max.: 1 g q6h)
- Note: Because many patients are undernourished, pharmacokinetics of antibiotics (especially aminoglycosides, penicillins, and cephalosporins) may be altered, requiring careful monitoring.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Pulmonary exacerbation with significant deterioration from baseline, hypoxemia, resistant bacteria, failure of outpatient therapy
- Pneumothorax
- Hemoptysis
- Hematemesis
- Intussusception or unexplained abdominal pain or bowel obstruction

- Hyperglycemia

Discharge Criteria

- Close follow-up to verify the sensitivities of culture results and change therapy as needed
- Avoid hot weather.
- Oral salt supplement when profuse sweating

Issues for Referral

All patients followed by a pediatric pulmonary center. Consultation during acute exacerbations.

FOLLOW-UP RECOMMENDATIONS

- Team approach of specialists
- Breathing treatments, chest PT, exercise programs, antibiotics, replacement of pancreatic enzymes

PEARLS AND PITFALLS

- With CF patients in respiratory distress, always consider pneumothorax: Obtain CXR.
- For CF patients with abdominal pain/vomiting, always consider DIOS and intussusception

ADDITIONAL READING

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CODES

ICD9

- 277.00 Cystic fibrosis without mention of meconium ileus
- 277.02 Cystic fibrosis with pulmonary manifestations
- 277.03 Cystic fibrosis with gastrointestinal manifestations

ICD10

- E84.0 Cystic fibrosis with pulmonary manifestations

- E84.9 Cystic fibrosis, unspecified
- E84.19 Cystic fibrosis with other intestinal manifestations

DACRYOCYSTITIS AND DACRYOADENITIS

Shari Schabowski

BASICS

DESCRIPTION

- Dacryoadenitis and dacryocystitis are inflammatory conditions affecting the lacrimal system of the eye:
 - Dacryoadenitis is inflammation or infection of the lacrimal gland from which tears are secreted.
 - Dacryocystitis is an infection within the lacrimal drainage system.
- Dacryoadenitis may be a primarily inflammatory condition or an infectious process resulting from contiguous spread from a local source or systemic infection.
- Dacryocystitis is a suppurative infection involving an obstructed lacrimal duct and sac.

EPIDEMIOLOGY

Dacryoadenitis is an uncommon disorder more commonly seen on the left:

- Acquired:
 - Uncommon

Dacryocystitis is a more common disorder most often occurring in adult females > 30 yr old but may be seen in infants

Etiology—Dacryoadenitis

- Most commonly caused by systemic inflammatory conditions:
 - Autoimmune diseases
 - Sjögren syndrome
 - Sarcoidosis
 - Crohn's disease
 - Tumor
- Infectious causes may be primary or may occur secondary to contiguous spread from bacterial conjunctivitis or periorbital cellulites
- Acute, suppurative:
 - Bacteria most common cause in adults:
 - *Staphylococcus aureus*
 - *Streptococci*
 - *Chlamydia trachomatis*
 - *Neisseria gonorrhoea*
- Chronic dacryoadenitis:
 - Nasal flora > ocular flora

Pediatric Considerations

- Viruses most common cause in children:
 - Mumps
 - Measles
 - Epstein–Barr virus
 - Cytomegalovirus
 - Coxsackievirus
 - Varicella-zoster virus
- Slowly enlarging mass may be dermoid

Etiology—Dacryocystitis

- Under normal conditions, tears drain via pumping action at the lacrimal duct, moving tears to lacrimal sac and then into middle turbinate/sinuses.
- Symptoms begin when duct to lacrimal sac becomes partially or completely obstructed:
 - In acquired form, chronic inflammation related to ethmoid sinusitis is a commonly implicated cause but many nasal and systemic inflammatory conditions have been correlated with this process:
 - May also occur secondary to trauma, a dacryolith, after nasal or sinus surgery or by any local process that might obstruct flow
 - Stasis in this conduit results in overgrowth of bacteria and infection.
 - Infection may be recurrent and may become chronic:
 - Most common bacteria: Sinus > ocular flora
 - *S. aureus* is the most common organism

Complications may include formation of draining fistulae, recurrent conjunctivitis, and even abscesses or orbital cellulitis

Pediatric Considerations

- In congenital form, presentation occurs in infancy as a result of dacryocystoceles
- High morbidity and mortality associated with this form:
 - Caused by systemic spread of infectious process or bacterial overgrowth in a partially obstructed gland
- The most common organism is *Streptococcus pneumoniae*.

DIAGNOSIS

Both will present as a unilateral, red, painful eye.

SIGNS AND SYMPTOMS

Dacryoadenitis

May present as an acute or indolent swelling and erythema of upper eyelid

- Swelling and tenderness greatest in temporal aspect of upper lid under orbital rim:
 - S-shaped lid
- Mass may be palpable

- May be associated with:
 - Extensive cellulitis
 - Conjunctival injection and discharge
 - Increase or decrease in tear production
 - Ipsilateral conjunctival injection and chemosis
 - Ipsilateral preauricular adenopathy
 - Systemic toxicity may be present
- Normal visual acuity, slit-lamp, and funduscopy exams
- May cause pressure on the globe or globe displacement:
 - Visual distortion may occur.
- Chronic form: Slowly progressive, painless swelling

ALERT

Promptly determine clinical probability of spread *from N. gonorrhoea* conjunctivitis:

- Morbidity very high:
 - Visual loss likely
 - Systemic illness probable
- Treatment differs significantly from other causes.

Dacryocystitis

Presents as an acutely inflamed, circumscribed mass extending inferiorly and medially from inner canthus:

- Epiphora or excessive tearing—hallmark symptom:
 - Tear outflow is obstructed.
- Discharge from punctum:
 - Pressure on the inflamed mass may result in purulent material from the punctum.
 - This may be diagnostic.
- Cellulitis extending to lower lid may be present
- Low-grade fever may be present, but patient rarely appears toxic.

ESSENTIAL WORKUP

Complete eye exam, including visual acuity, extraocular movements, slit-lamp, and funduscopy exam:

- Flip lids
- Examine nasal passages

Pediatric Considerations

Careful inspection for evidence of extension to orbital cellulitis or meningitis is essential.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Tests of expressed material (used to help direct specific antibiotic treatment):
 - Gram stain
 - Culture and sensitivity
 - Chocolate agar plating if GC suspected
- CBC and blood cultures

Imaging

CT of orbit/sinus to evaluate deep-tissue extension or possible underlying disorder in dacryoadenitis particularly with recurrent cases or in children at risk for orbital cellulitis extending from dacryocystitis.

DIFFERENTIAL DIAGNOSIS

- Dacryoadenitis:
 - Autoimmune diseases
 - Lacrimal gland tumor
 - Hordeolum
 - Periorbital cellulitis
 - Severe blepharitis
 - Orbital cellulitis
 - Insect bite
 - Traumatic injury
 - Orbital or lacrimal gland tumor
- Dacryocystitis:
 - Insect bite
 - Traumatic injury
 - Acute ethmoid sinusitis
 - Periorbital cellulitis
 - Acute conjunctivitis



TREATMENT

ED TREATMENT/PROCEDURES

- Early diagnosis and initiation of treatment will reduce risk of extension of infection to adjacent structures and systemic infection.
- Topical antibiotics may be considered to treat or avoid conjunctivitis.

Dacryoadenitis

- Cool compresses to decrease inflammation and nonsteroidal pain medication
- Viral etiology:
 - Typically self-limited inflammation
- Bacterial etiology:

- Antibiotics
- Oral for mild infection:
 - Cephalexin
 - Amoxicillin/clavulanate
- IV for severe infection:
 - Cefazolin
 - Ticarcillin/clavulanate
- Tetanus toxoid if necessary
- Incision and drainage rarely necessary except in very severe cases:
 - Perform with consultation to facial surgery service or ophthalmology

Pediatric Considerations

- Cool compresses
- Analgesics
- If cause unclear, treat with antibiotics as with adults

Dacryocystitis

- Drainage of infected sac is essential:
 - Warm compresses and gentle massage to relieve obstruction
 - May facilitate outflow from obstructed tract with nasal introduction of vasoconstricting agent
 - Incision and drainage only in severe cases:
 - Typically done by ophthalmology
 - Avoid in ED when possible
 - May result in fistula formation
 - Duct instrumentation to facilitate drainage is not indicated in acute setting:
 - Reserve instrumentation for nonacute setting, if necessary at all
 - Manipulation while duct is inflamed may cause injury to duct and permanent obstruction from scarring and stenosis.
 - Topical ophthalmic antibiotic drops to prevent secondary conjunctivitis
- Systemic antibiotics to resolve infection and prevent spread to adjacent structures:
 - Oral for mild infection
 - Intravenous when febrile or severe infection
- Analgesics

Pediatric Considerations

- Newborns respond well to massage and topical antibiotics in ~95% of cases.
- If no resolution in 1st yr of life, may require probing of duct by ophthalmologist
- Children <4 yr old who develop dacryocystitis:
 - At increased risk for *Haemophilus influenzae* infection, if not immunized:
 - Given typical age of presentation, complete immunization is unlikely at primary presentation.

- Recommended schedule 2, 4, 6, and 12–15 mo
- *H. influenzae* type B carries high risk for bacteremia, septicemia, and meningitis.
- Treat afebrile, well-appearing children with responsible parent with oral cefaclor or amoxicillin/clavulanate.
- Administer cefuroxime IV in acutely ill patients.

MEDICATION

- Amoxicillin/clavulanate (Augmentin): 500 mg (peds: 20–40 mg of amoxicillin/kg/24h) PO q8h
- Cefaclor: 500 mg (peds: 20–40 mg/kg/24h) immediate release PO TID
- Cefazolin: 500–1,000 mg (peds: 50–100 mg/kg/24h) IV q6–8h
- Cefuroxime: 750–1,500 (peds: 50–100 mg/kg/24h) mg IV q8h
- Cephalexin: 500 mg (peds: 25–100 mg/kg/24h) PO QID
- Erythromycin ophthalmic ointment: 2 drops QID to affected eye
- Tetracaine and phenylephrine topical solution single-dose nasal spray
- Ticarcillin/clavulanate: 3.1 g (peds: 200–300 mg of ticarcillin/kg/24h) IV q4–6h
- Trimethoprim-polymyxin ointment: 2 drops QID to the affected eye



FOLLOW-UP

DISPOSITION

Admission Criteria

- Adults:
 - Febrile or toxic appearance
 - Concomitant medical problems including diabetes or immunosuppression
 - Extensive cellulitis
 - Suspicion of adjacent spread with deep tissue involvement or meningitis or *Neisseria meningitidis*
- Children:
 - Acutely ill appearance
 - Concomitant medical problems
 - Extensive cellulitis
 - High risk for *H. influenzae* (nonvaccinated)
 - If reliable follow-up within 24 hr cannot be arranged

Issues for Referral

Dacryoadenitis and dacryocystitis should be referred promptly to ophthalmology:

- Patients with dacryocystitis require further evaluation to confirm complete drainage of sac and to assess need for further intervention to avoid recurrence.
- Availability of follow-up should be confirmed and ophthalmologic consultation

should be completed prior to discharge.

PEARLS AND PITFALLS

- In cases of red eye with lid swelling, specifically examine the lacrimal structures for evidence of involvement.
- Skin incision and drainage of dacryocystitis should be avoided whenever possible to avoid fistula formation:
 - Intranasal vasoconstricting agents should be used primarily to facilitate drainage.

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- Wald ER. Periorbital and orbital infections. *Infect Dis Clin North Am*. 2007;21:393–408.



See Also (Topic, Algorithm, Electronic Media Element)

- Conjunctivitis
- Hordeolum and Chalazion
- Periorbital and Orbital Cellulitis
- Red Eye



CODES

ICD9

- 375.00 Dacryoadenitis, unspecified
- 375.30 Dacryocystitis, unspecified
- 375.32 Acute dacryocystitis

DECOMPRESSION SICKNESS

Catherine M. Visintainer • Nicole L. Lunceford • Peter J. Park

BASICS

DESCRIPTION

Multisystemic disease process resulting from escape of inert gas bubbles (nitrogen) out of solution into body fluids and tissues

ETIOLOGY

Mechanism:

- Pathophysiology:
 - Increases in ambient pressure cause increase in partial pressure of nitrogen inspired (as per Henry law, below).
 - Nitrogen accumulates in tissues in increasing concentrations the longer ambient pressures remain elevated.
 - Decompression sickness (DCS) results when ambient pressure keeping nitrogen in solution decreases too rapidly (on ascent), preventing gradual removal of excess body burden of nitrogen.
 - As the nitrogen removal gradient is overwhelmed, tissues become supersaturated and bubble formation occurs.
- Henry law:
 - Amount of gas that will dissolve in a solution at a given temperature is directly proportional to partial pressure of that gas.
 - Increases in partial pressure result in larger amount of gas dissolved in tissue.
 - Decreases in partial pressure result in gas coming out of solution.
- Bubbles are viewed as foreign material by body inciting inflammatory and coagulation responses
 - Leads to increased vascular permeability and decreased intravascular volume and hemoconcentration
- Bubble location determines clinical effects:
 - Blood flow and lymphatic obstruction leading to ischemia, infarction, or lymphedema
 - Mechanical distention of tissues leading to pain
- Risk factors for DCS:
 - Dive factors:
 - Greater depth
 - Longer bottom time
 - Multiple dives in a day
 - Rapid ascent

- Cold water
- Human factors:
 - Obesity
 - Intercurrent illness
 - Pulmonary disease
 - Dehydration
- Proper use of dive tables and computers does *not* eliminate risk for DCS.
- Pre-dive vigorous exercise may reduce risk
- 50% of patients develop symptoms in 1 hr, 90% develop symptoms within 6 hr.
- Airplane flight following diving can precipitate DCS owing to lower cabin pressure.

DIAGNOSIS

SIGNS AND SYMPTOMS

- Cutaneous:
 - Scarlatiniform, erysipeloid, or mottled rash:
 - Mottling (*Cutis marmorata*) often indicates more severe disease
 - Peau d'orange appearance owing to lymphatic obstruction
- Musculoskeletal (the bends):
 - Pain:
 - Dull, deep muscular aching
 - Often in a joint (elbow and shoulder most common)
 - Typically not exacerbated by movement or reproduced with palpation
- GI:
 - Nausea and vomiting
 - Abdominal pain
- Pulmonary (the chokes):
 - Pulmonary vasculature obstruction from bubble burden (venous gas embolism)
 - Acute respiratory distress
 - Substernal chest pain/pressure
 - Cough
 - Dyspnea
 - Hypoxia
- CNS:
 - Weakness and fatigue
 - Numbness and paresthesia
 - Agitation
 - Headache
 - Dizziness
 - Vertigo

- Convulsion
- Bladder and/or bowel incontinence
- Lethargy
- Visual disturbances
- Most commonly affects spinal cord (lower thoracic and lumbar regions)
- Inner ear (the staggers):
 - Vestibular damage
 - Dizziness, vertigo, tinnitus, nausea
 - Similar symptoms to inner ear barotrauma but with worse prognosis

History

Meticulous dive history including time at depth, ascent history, and onset of symptoms.

Physical-Exam

Thorough physical exam including a detailed neurologic exam

ESSENTIAL WORKUP

- Clinical diagnosis: Recognize risk factors and various clinical presentations.
- Careful neurologic exam to document possible waning symptoms
- Trial of pressure:
 - Rapid relief of symptoms upon recompression in a hyperbaric chamber may be the only way to diagnose DCS conclusively.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC:
 - Increased hematocrit secondary to hemoconcentration
- Electrolytes, BUN, creatinine, glucose
- Urinalysis
 - Increased specific gravity may indicate intravascular volume depletion
- ABG and pulse oximetry:
 - Monitor oxygenation

Imaging

- CXR:
 - Concomitant pulmonary barotrauma
 - Noncardiogenic pulmonary edema from DCS
- Extremity x-ray
 - Rule out trauma as cause of pain
- Head CT when altered mental status or neurologic deficit

DIFFERENTIAL DIAGNOSIS

- Musculoskeletal injury unrelated to bubble formation
- Inner or middle ear barotraumas
- Arterial gas embolism
- Cerebrovascular accident (CVA)
- Trauma



TREATMENT

PRE HOSPITAL

- Cautions:
 - _ Recognize DCS:
 - Postdive extremity pain often attributed to muscle strain
 - Serious neurologic complaints often minimized because diver does not consider DCS
 - _ If air evacuation required:
 - Limit altitude to less than 1,000 ft or use pressurized aircraft
- Controversies:
 - _ In-water recompression:
 - Return injured diver/patient to depth where symptoms are ameliorated.
 - Extremely difficult
 - Need large amount of surface support

INITIAL STABILIZATION/THERAPY

- Airway, breathing, and circulation management (ABCs)
- Provide normobaric (100%) oxygen via mask or endotracheal tube (ETT):
 - _ Increases inert gas (nitrogen) elimination from tissues, reducing gas bubble size
 - _ Increases oxygen delivery to injured tissue
- Maintain patient in supine position to prevent further cerebral involvement
- Early recompression in hyperbaric chamber

ED TREATMENT/PROCEDURES

- IV rehydration with 0.9% normal saline (NS) to maintain goal urine output of 1–2 mL/kg/h:
 - _ Diver usually dehydrated owing to diuretic effect of pressure, exercise, breathing dry compressed air, and increased vascular permeability
 - _ Increased fluid assists with gas removal and dissolution of nitrogen
- Hyperbaric oxygen recompression therapy (see Hyperbaric Oxygen Therapy):
 - _ Arrange transportation to nearest hyperbaric facility.
 - _ Prophylactic chest tube for simple pneumothorax to prevent conversion to tension pneumothorax in chamber

- Fill endotracheal and Foley catheter balloons with water or saline to avoid shrinkage/damage during recompression.
- Recompression therapy protocols found in US Navy diving manual
- Divers Alert Network (DAN):
 - Provides 24-hr emergency hotline for medical consultation on treatment of dive-related injuries and for referrals to hyperbaric chambers ([919] 684-9111)
- Analgesics and antiemetics
- Diazepam (Valium) for severe vertigo
- Adjunctive therapy with NSAIDs and/or heliox may reduce number of recompression treatments required



FOLLOW-UP

DISPOSITION

Admission Criteria

Refer all patients with suspected or diagnosed DCS for hyperbaric therapy.

Discharge Criteria

- Stable patients with mild symptoms may be discharged posthyperbaric oxygen treatment.
- Air travel may exacerbate symptoms as ambient pressure decreases.

FOLLOW-UP RECOMMENDATIONS

Hyperbaric referral

PEARLS AND PITFALLS

- Difficult to distinguish musculoskeletal DCS from musculoskeletal pain
- Significant fatigue may be the only symptom of DCS
- Even minor symptoms or resolving symptoms suspected of being DCS should be treated with hyperbaric recompression therapy
- Do not delay recompression therapy for lab testing and imaging when DCS is most likely diagnosis
- Avoid in-water recompression therapy.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Arterial Gas Embolism
- Barotrauma
- Hyperbaric Oxygen Therapy



CODES

ICD9

993.3 Caisson disease

ICD10

T70.3XXA Caisson disease [decompression sickness], initial encounter

DEEP VEIN THROMBOSIS

Sarah K. Flaherty



BASICS

DESCRIPTION

- A constant balance exists between intravascular clot formation and clot dissolution, clot forming when the former overpowers the latter.
- Clot can be superficial (to the fascia) or deep. The latter is called deep vein thrombosis (DVT).
- Pulmonary embolism (PE) and DVT are different ends of the clinical spectrum of the same disease process (venous thromboembolism, VTE).
- DVT can be upper or lower extremity, as well as distal or proximal (to the popliteal vein)
- Incidence is ~2 1st time VTE episodes per 1,000 person yr.
- Prevalence increases with advancing age
- Common in both medical and surgical hospitalized patients
- Diagnosis is more accurate using active surveillance rather than clinical suspicion.

Pediatric Considerations

DVT in children is unusual, but when cases do occur, search for an underlying reason for hypercoagulability. Also, upper-extremity DVT is associated with central IV lines in children.

ETIOLOGY

- Clot formation/dissolution is an intricately balanced system which can be influenced by many factors which must be considered
- Hypercoagulable states:
 - Cancer
 - Myeloproliferative disorders
 - Nephrotic syndrome
 - Sepsis
 - Inflammatory conditions:
 - Ulcerative colitis
 - Increased estrogen:
 - Pregnancy
 - Exogenous hormones (OCPs, HRT)
 - Antiphospholipid syndrome
 - Protein S, C, and antithrombin III deficiencies, factor V Leiden, prothrombin gene mutations, lupus, others
- Stasis:

- Prolonged bed rest
- Immobility (such as from a cast)
- Long plane, car, or train rides
- Neurologic disorders with paralysis
- CHF
- Obesity
- Vascular concerns/damage:
 - Trauma
 - Surgery
 - Anatomic anomalies (May–Thurner syndrome)
 - Central lines:
 - Especially with upper extremity DVT
- Multifactorial issues:
 - Advancing age
 - Medications (hydralazine, procainamide, phenothiazines)
 - Tobacco use
 - Prior DVT or PE
- Genetics:
 - Important with respect to some of the risk factors; ask about family history of clotting.
 - There is no consensus about which patients with VTE to test for inherited thrombophilias

Pregnancy Considerations

Pregnancy is a risk factor for DVT, especially in the 3rd trimester up to the 2nd wk postpartum.

Geriatric Considerations

Age in and of itself is a risk for DVT (and PE). As with many diseases, the presentation may be atypical in the elderly.

DIAGNOSIS

SIGNS AND SYMPTOMS

- Leg swelling:
 - > 1 cm difference is usually significant.
- Leg warmth and redness
- Leg pain and tenderness
- Palpable cord
- In superficial thrombophlebitis, a red pipe cleaner–like cord may be visible and palpable.
- Arm swelling, warmth, or tenderness:

- Upper extremity or subclavian vein involved
- Phlegmasia cerulea dolens:
 - Cold, tender, swollen, and blue leg (secondary arterial insufficiency, venous gangrene)
- In phlegmasia alba dolens:
 - Cold, tender, and white leg (secondary arterial insufficiency)

ESSENTIAL WORKUP

- Determination of a patient's clinical (pretest) risk is a key step in a workup for DVT.
- A careful history and physical exam, interpreted in the context of the risk-factor profile, is the most important driver of subsequent diagnostic evaluation as individual clinical findings are poorly predictive in isolation.
- Consider further evaluation for underlying malignancy when appropriate as VTE may be initial manifestation.

DIAGNOSIS TESTS & INTERPRETATION

Lab

D-dimer testing:

- D-dimer, a byproduct of endogenous clot formation, is becoming increasingly used in evaluation of patients for DVT and PE.
- *Only useful when the result is negative* (to exclude DVT). Positive D-dimer does not make the diagnosis; it only mandates further testing.
- Methods of measuring D-dimer levels:
 - Latex agglutination (1st-generation tests) and microlatex agglutination (2nd-generation) are generally insufficient.
 - Whole-blood latex agglutination (SimpliRED) is valuable if negative in low probability patients (using Wells criteria).
 - Enzyme-linked immunosorbent assay (ELISA) testing gives a quantitative result and has been validated in large clinical studies in ED patients; particularly when combined with assessment of pretest probability

Imaging

- Contrast venography:
 - Once the imaging test of choice; now rarely performed because it is invasive, expensive, and has complications.
 - Involves injection of contrast medium into a leg vein, which can cause thrombophlebitis in patients undergoing the procedure; as well as contrast dye reactions and possible renal damage
- Compression US:
 - Standard 1st-line diagnostic test

- Venous study. Normal veins compress; those with clots do not.
- Color Doppler can be useful for identifying the vein but does not add substantially to accuracy. Duplex scanning refers to the combination of compression B-mode US and color Doppler.
- Has a sensitivity in the high 90% range
- Should be repeated (or followed up with contrast venography) in high-risk patients with negative US.
- Other tests include radionuclide venography and impedance plethysmography; however, these are not commonly used in clinical practice

DIFFERENTIAL DIAGNOSIS

- Superficial thrombophlebitis
- Cellulitis
- Torn muscle and/or ligaments (including plantaris and gastrocnemius tears)
- Ruptured Baker cyst
- (Bilateral) edema secondary to heart, liver, or kidney disease
- Venous valvular insufficiency
- Drug-induced edema (calcium channel blockers)
- (Unilateral) edema from abdominal mass (gravid uterus or tumor) or lymphedema
- Postphlebotic syndrome (from prior thrombophlebitis)



TREATMENT

INITIAL STABILIZATION/THERAPY

In cases of phlegmasia cerulea, or alba dolens:

- IV access
- Supplemental oxygen
- Surgical or vascular consultation

ED TREATMENT/PROCEDURES

- Systemic anticoagulation:
 - In patients without contraindications as PE will occur in ~50% of untreated DVT
 - Use either unfractionated heparin or low-molecular-weight heparin (LMWH), fondaparinux or adjusted dose subcutaneous heparin
 - Carefully selected patients can be primarily treated with as outpatients
- Warfarin:
 - Started shortly after a heparin has been administered
 - Not before heparin because of the theoretic risk for inducing a transient hypercoagulable state
 - Insufficient evidence exists to safely recommend other oral anticoagulants (e.g., dabigatran, rivaroxaban, apixaban)

- Vena cava filters:
 - Two main indications:
 - Contraindications to systemic anticoagulation
 - New thromboembolic event while on adequate anticoagulation
 - Vena cava filters can be placed transcutaneously, usually by a vascular or trauma surgeon or radiologist.
 - Empiric filter placement may be useful in certain settings:
 - Ongoing risk such as cancer, polytrauma.
 - Risk for a recurrent PE could be fatal because of poor cardiopulmonary reserve or a recent PE.
 - Randomized data suggest that filter placement is no more effective than anticoagulation.
 - Filters can also be deployed in the superior vena cava in the setting of upper-extremity DVT.
- Thrombolysis:
 - Rarely indicated
 - Roughly a 3-fold increase in bleeding complications
 - Catheter-administered lytic therapy is used more commonly in upper-extremity DVT
- Thrombectomy (surgical or percutaneous):
 - Occasionally recommended for patients with extensive disease
 - Consult a vascular surgeon
- Septic thrombophlebitis:
 - Surgical excision of the vein or IV antibiotics

MEDICATION

- Maintain treatment with IV or SQ therapy until INR has been therapeutic for 2 consecutive days.
- Warfarin: 5 mg/d starting dose with a prothrombin time being checked on the 3rd day.
- Heparin (unfractionated): 80 U/kg bolus followed by an 18 U/kg/h drip, with the activated partial thromboplastin time (aPTT) titrated 1.5–2.5 times normal.
- LWMH (enoxaparin): 1 mg/kg SC BID for outpatients (alternative: 1.5 mg/kg SCQD).
- Tinzaparin: 175 U/kg/d SC.
- Dosing regimens are based on total body weight; however, in obese patients alternative dosing should be considered.
- Treatment usually maintained for at least 3 mo, total length is individualized.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients with DVT unable to receive LMWH as an outpatient or poor follow-up
- Patients with concomitant PE or other serious diseases (i.e., renal failure)
- Patients thought to be at especially high bleeding risk
- Patients with phlegmasia

Discharge Criteria

- Outpatient treatment with an LMWH:
 - No serious concomitant disease that requires hospitalization.
 - Patient has means of communication and transportation to return to the hospital if needed, as well as appropriate follow-up.
 - Patient (or family member) is willing and able to inject the medication.
 - aPTT does not need to be checked.
 - Heparin-induced thrombocytopenia is less common with the LMWH but still occurs.
- Patients with superficial or distal thrombophlebitis can be discharged with close follow-up.

Issues for Referral

- Consult vascular surgery if there is any question about arterial insufficiency.
- Consider need for inferior vena cava filter in patients who have contraindications to full anticoagulation or form new clots on adequate anticoagulation.

ALERT

When the clinical suspicion is high but the US is negative, remember to advise the patient to follow-up with his or her primary care physician, and to have a follow-up US in ~1 wk.

FOLLOW-UP RECOMMENDATIONS

Outpatient treatment with an LMWH:

- Patient needs hematocrit, platelet count, and INR checked in 2–3 days.
- INR needs to be checked at about day 3.

PEARLS AND PITFALLS

- Do not use a negative Homans sign to exclude the diagnosis of DVT.
- Use some measure (whether clinical gestalt or a formal scoring system such as the Wells score) to determine pretest probability for DVT.
- In high pretest probability patients, do not rely on D-dimer testing; instead, perform venous imaging, generally compression US.
- In medium-risk patients with a negative D-dimer or negative US, arrange or recommend a repeat study in 1 wk.

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- Lawall H, Hoffmanns W, Hoffmanns P, et al. Prevalence of deep vein thrombosis (DVT) in non-surgical patients at hospital admission. *Thromb Haemost.* 2007;98(4):765–770.
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CODES

ICD9

- 453.40 Acute venous embolism and thrombosis of unspecified deep vessels of lower extremity
- 453.41 Acute venous embolism and thrombosis of deep vessels of proximal lower extremity
- 453.82 Acute venous embolism and thrombosis of deep veins of upper extremity

ICD10

- I82.409 Acute embolism and thrombosis of unspecified deep veins of unspecified lower extremity
- I82.609 Acute embolism and thrombosis unsp vn unsp upper extremity
- I82.4Y9 Acute embolism and thrombosis unsp deep vn unsp prox low extrem

DEFIBRILLATORS, IMPLANTABLE

Robert D. Sidman • Lawrence S. Rosenthal



BASICS

DESCRIPTION

- An implantable cardiac device (ICD) is a small battery-powered electrical impulse generator implanted SC in patients at risk of cardiac arrest from cardiac arrhythmias.
- Lead(s) are positioned via venous return to heart and are endocardial (RA and RV) or epicardial (LV via coronary sinus).
- The device is able to detect and convert ventricular and atrial arrhythmias to sinus rhythm with electric shocks delivered between the ICD can and coil(s) in the RV (single coil) and the SVC/RA juncture (dual coil).
- Similar method of implantation as a pacemaker
- Newly released devices (S-ICD) no longer have endocardial leads reducing the risk of blood infection.
- 450,000 individuals experience sudden cardiac death yearly in US:
 - >100,000 devices implanted in US each year
 - ICDs have been shown to reduce mortality more effectively than antiarrhythmic drug therapy in patients with left ventricular dysfunction:
 - Absolute risk reduction of mortality of 7% in the 1st 2 yr
 - Benefit over antiarrhythmic drug therapy is limited to patients with ejection fractions of <35%
 - Effective in reducing mortality in hypertrophic cardiomyopathy
 - Both ischemic and nonischemic dilated cardiomyopathy patients show survival benefit with ICD
- Immediate postimplant complications:
 - Pneumothorax
 - Vascular perforation
 - Acute lead dislodgement
- Appropriate shocks:
 - 5% a year for primary prevention
 - 20% a year for secondary prevention
- Electrical storm:
 - ≥ 2 appropriate shocks delivered within a 24-hr period
- Inappropriate shocks:
 - 10–20% of ICD recipients
 - Oversensing
 - Inappropriate classification of rapid supraventricular tachycardia
- Device infection:

- 1–12% of patients
- Acute 1–30 days—think staph
- Subacute > 30 days—think *Staphylococcus epidermidis* or gram negatives
- 31–66% mortality if the device is left in place
- Infection may involve the skin, the generator, the defibrillation pocket, or the leads.
- Coagulase-negative staphylococci (42%)
- Methicillin-sensitive staphylococci (25%)
- MRSA (4%)
- Gram-negative bacilli (9%)
- Pocket hematoma do not aspirate
- Vascular occlusion

ETIOLOGY

- Electrical storm: (≥ 2 appropriate shocks delivered within a 24-hr period)
 - Unknown
 - Decompensated heart failure
 - Acute ischemia
 - Metabolic disturbances
 - Drug proarrhythmia
 - Thyrotoxicosis
 - Fever with dilated cardiomyopathy
 - Genetic channelopathies, Brugada syndrome, Long QT, catecholaminergic polymorphic VT, arrhythmogenic RV cardiomyopathy
 - Postcardiac surgery
 - ICD induced from left ventricular or T-wave pacing
- Inappropriate shocks:
 - Oversensing:
 - QRS, T-wave, P-wave, myopotential, electromagnetic interference (EMI)
 - Frequent nonsustained ventricular dysrhythmias
 - Lead fracture
 - Loose setscrew
 - Chatter between leads
 - Header (device circuitry) problem
 - Inappropriate classification of rapid supraventricular tachycardia:
 - Atrial fibrillation
 - Sinus tachycardia
 - Atrial flutter
 - Other supraventricular tachycardias (SVT)
- Device/site-related:
 - Wound infection:
 - *Staphylococcus aureus* (most aggressive and seen early)

- *S. epidermidis* (more indolent and later)
- *Escherichia coli*, *Pseudomonas* species, and *Streptococcal* species (less common)
- Pocket hematomas
- Vascular (venous thrombosis/embolism secondary to impedance of venous flow as a result of the ICD lead[s])

DIAGNOSIS

SIGNS AND SYMPTOMS

- Felt bad before shock and good after: Likely appropriate therapy
- Felt good before and after shock: Likely inappropriate therapy
- Felt bad before and after shock: Consider ongoing arrhythmia or ischemia
- Appropriate shocks:
 - Syncope or near syncope
 - Lightheadedness or dizziness
 - Shortness of breath
 - Palpitations (non-SVT)
 - Chest discomfort or pain
 - Diaphoresis
- Inappropriate shocks:
 - Palpitations (SVT)
 - No symptoms (Lead-related fractures, inappropriate sensing)
- Device infection:
 - Fever
 - Chills
 - Malaise
 - Anorexia
 - Nausea
 - Diaphoresis
 - Hypotension
 - Heart murmur
 - Wound infection:
 - Pain
 - Erythema
 - Purulent drainage
 - Warmth
 - Fluctuance
 - Skin erosion
- Hematoma at the insertion site (pocket hematoma):
 - Pain (mild)
 - Swelling

- Vascular (thromboembolic phenomena):
 - Unilateral swelling in upper extremity
 - Superficial varicosities

History

- Therapy-related:
 - Recent angina, heart failure
- Device-related:
 - Recent implant (<14 days)
 - Skin trauma to wound
 - Lead-related:
 - Repetitive arm motions
 - “Twiddler’s syndrome” (inadvertent manipulation of the device)
 - Vascular:
 - Recent implant
 - Multiple leads

Physical-Exam

- Vital signs
- Evidence of heart failure/acute coronary syndrome:
 - Displaced point of maximal impulse
 - Left ventricular heave
 - Presence of an S3 or S4
 - Presence of basilar rales
 - Dullness to percussion
 - Determination of jugular venous pressure
 - Hepatojugular reflex
 - Peripheral edema
- Device/site-related:
 - Exam of wound/pocket:
 - Demarcation of pocket (erythema)
 - Purulent drainage
 - Exam of affected upper extremity

ESSENTIAL WORKUP

- Following ICD therapy:
 - ICD interrogation will determine whether therapy was appropriate and can determine lead fracture if present.
 - EKG (transient ST-segment changes and elevations of the cardiac enzymes may be seen after shock delivery and do not necessarily indicate myocardial damage)
 - CXR may diagnose lead fracture.
- Device/site-related:

- Signs and symptoms of local vs. systemic infection
- Upper-extremity swelling suggests venous thrombosis.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Therapy-related:
 - 12-lead EKG
 - Cardiac enzymes
- Device-related:
 - CBC with differential
 - Blood cultures
 - Do not aspirate pocket

Imaging

- PA and lateral chest radiograph:
 - Lead fractures
 - Lead dislodgement
- Vascular US of upper extremity
- MRI absolutely contraindicated:
 - Magnetic field may damage ICDs and cause heating at lead tip.

Diagnostic Procedures/Surgery

- Therapy-related:
 - Device interrogation by electrophysiologist/cardiologist
 - Application of magnet inhibits tachy therapies (does not affect brady support pacing).
- Device/site-related (pocket hematoma/infection):
 - Referral to surgeon/electrophysiologist
 - Electrocautery should generally be avoided in patients with ICDs unless device is deactivated with programming or with magnet application.
- External defibrillation is safe, but avoids shocking directly over ICD (see below).

DIFFERENTIAL DIAGNOSIS

- Appropriate therapies:
 - Single shock following an episode of VT or VF with restoration of normal rhythm
- Inappropriate therapies:
 - Usually due to SVT (afib), lead fracture, or EMI
- Phantom shocks:
 - Patient awakened from sleep by a perceived shock(s)



TREATMENT

PRE HOSPITAL

Following an ICD electrical discharge:

- IV access
- Continuous EKG monitoring
- Advanced cardiac life support (ACLS) protocols

INITIAL STABILIZATION/THERAPY

- ACLS protocols
- Magnet application inhibits ICD therapies.
- Device-related:
 - _ Pain management
 - _ Elevation of affected extremity (upper-extremity thrombosis)

ED TREATMENT/PROCEDURES

- Patients with devices should receive treatment according to standard ACLS protocols.
- Electrical storm may require IV antiarrhythmic agents such as amiodarone.
- Inappropriate therapies:
 - _ Treatment of supraventricular dysrhythmia to prevent ICD shocks with β -blockers or calcium channel blockers
- Lead-related problems may require further surgical intervention or device reprogramming; magnet application will inhibit tachy therapies.
- Device infections:
 - _ Broad-spectrum antibiotics
 - _ Obtain blood cultures 1st

MEDICATION

- Amiodarone 150 or 300 mg IVP followed by an infusion 1 mg/kg/h for 6 hr, then reduce to 0.5 mg/kg/h. Can rebofus (150 mg) as often as required
- Metoprolol: 5 mg IV as needed to control heart rate
- Diltiazem: 5–20 mg IV, then a maintenance drip to control heart rate
- Cefazolin: 1 g IV q8h
- Vancomycin: 1 g IV q12h
- Cephalexin: 500 mg PO QID
- Warfarin for documented venous occlusion, INR 2–3 for 3 mo



FOLLOW-UP

DISPOSITION

Admission Criteria

- Therapy-related:
 - Ongoing/suspected cardiac ischemia or heart failure
 - Multiple ICD shocks and initiation of antiarrhythmic agents for VF/VT or other SVT
 - Treat underlying process and consult with electrophysiologist to determine if immediate interrogation is warranted.
- Device/site-related:
 - Skin erosion
 - Wound dehiscence
 - Systemic infection/endocarditis
 - Need for lead revision
 - Expanding pocket hematoma
 - Upper-extremity thrombosis

Discharge Criteria

- Therapy-related:
 - If patient is hemodynamically stable without evidence of active ischemia or heart failure, interrogation usually not required:
 - Single-shock, appropriate therapy
 - Consult with electrophysiologist and arrange appropriate follow-up.
 - Device reprogrammed to avoid inappropriate therapy
- Device/site-related:
 - Localized infection
 - No signs of skin erosion
 - Pocket not expanding:
 - Prophylactic antibiotics are not indicated for pocket hematomas.
 - Wound stable

FOLLOW-UP RECOMMENDATIONS

- Therapy-related:
 - Cardiologist or electrophysiologist
- Device-related:
 - Surgeon or cardiologist/electrophysiologist

PEARLS AND PITFALLS

- Aspiration of device pocket is not recommended.
- Care should be taken not to deliver external shocks directly over the device, as it may shunt energy away from the heart.

ADDITIONAL READING

- Epstein AE, DiMarco JP, Ellenbogen KA, et al. 2012 ACCF/AHA/HRS focused update incorporated into the ACCF/AHA/HRS 2008 guidelines for device-based therapy of cardiac rhythm abnormalities: A report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines and the Heart Rhythm Society. *J Am Coll Cardiol*. 2013;61(3):e6–e75.
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CODES

ICD9

- V45.02 Automatic implantable cardiac defibrillator in situ
- V53.32 Fitting and adjustment of automatic implantable cardiac defibrillator
- 996.04 Mechanical complication of automatic implantable cardiac defibrillator

ICD10

- T82.518A Breakdown (mechanical) of other cardiac and vascular devices and implants, initial encounter
- Z45.02 Encounter for adjustment and management of automatic implantable cardiac defibrillator
- Z95.810 Presence of automatic (implantable) cardiac defibrillator

DELIRIUM

Lori A. Stolz • Arthur B. Sanders

BASICS

DESCRIPTION

- Delirium is a clinical syndrome characterized by acute changes in awareness, cognition, and perception with a waxing and waning course.
- Delirium is a syndrome secondary to an underlying medical condition.
- Pathophysiology unknown:
 - Diffuse cerebral dysfunction
 - Derangements of cerebral acetylcholine
 - CNS dopamine, γ -aminobutyric acid, and serotonin may be involved.
- Frequently missed by emergency medicine physicians due to atypical chief complaints.
- Associated with increased mortality for inpatients and increased length of stay.

ETIOLOGY

- Neurologic:
 - Meningitis or encephalitis
 - Seizure
 - Wernicke encephalopathy
 - Hypoxia and hypoperfusion of the brain
 - Intracranial bleed or mass
- Pulmonary:
 - Pneumonia
 - Other pulmonary etiology of hypoxia
- Cardiovascular:
 - Hypertensive crisis
 - Acute coronary syndromes
 - Arrhythmia
- GI:
 - Hepatic encephalopathy
 - Dehydration
- Renal:
 - UTI
 - Acute renal failure
- Endocrine:
 - Hypoglycemia
 - Hyperglycemia
 - Hypothyroid

- Rheumatologic:
 - Collagen vascular disorder
- Toxicologic:
 - Environmental toxins
 - Medications
 - Withdrawal from barbiturates or alcohol
- Other:
 - Electrolyte abnormalities
 - Vitamin deficiencies
 - Hypothermia
 - Hyperthermia
 - Trauma

Geriatric Considerations

- Common presentation in older ED patients
- Up to 10% of older ED patients may have delirium.
- Many patients will present with subtle symptoms and vague chief complaints:
 - Fall, dizzy, or not feeling well
- Waxing and waning symptoms
- Cause may be life-threatening condition.

DIAGNOSIS

SIGNS AND SYMPTOMS

- Disturbed consciousness:
 - Hyperalert:
 - Combative
 - Agitation
 - Hypoactive:
 - Lethargic
 - Stupor
 - Coma
 - Can have mixed hyperalert and hypoactive state with rapid oscillations
- Cognitive changes:
 - Disorientation
 - Impaired memory
 - Disorganized thinking and speech
 - Misperceptions, illusions, delusions, and hallucinations
- Reduced awareness of environment
- Inattention:
 - Difficulties in focusing, shifting, and maintaining attention
 - Restlessness

- Distractibility
- Lability

History

- History from caregivers is essential.
- Time course:
 - Hours to days
 - Fluctuating course
- Medications:
 - Prescribed, over-the-counter and illicit drugs
 - Dosing
 - Recently added medications
 - Recently discontinued medications
- Associated signs, symptoms, pre-existing conditions that would indicate underlying etiology

Physical-Exam

- Vital signs
- Complete neurologic exam:
 - Careful attention to changes in mental status
 - Orientation
 - Focal deficits
 - Hallucinations
- Psychiatric exam
- Cardiovascular, pulmonary, GI systems.
- Use physical exam to determine possible underlying medical illness and to focus further workup, especially sources of infection and sepsis.
- Several screening tools are available to evaluate for delirium:
 - Confusion assessment method consists of 4 key features:
 - 1: Acute onset or fluctuating course
 - 2: Inattention
 - 3: Disorganized thinking
 - 4: Altered level of consciousness
 - Diagnosis is made when features 1 and 2 are present with either 3 or 4
 - Mini-mental state exam:
 - Can be administered serially and will fluctuate; formal cognitive assessment may be difficult to accomplish due to patient cooperation.

ESSENTIAL WORKUP

- Awareness of delirium as syndrome is key.
- Workup should be broad to determine underlying organic disease.
- Ancillary studies as determined by history, physical, and initial workup

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Initial testing:
 - Electrolytes, calcium
 - Renal function
 - Hepatic function
 - Glucose
 - CBC
 - Urinalysis with culture and sensitivity
 - Toxicology screens
- Further studies based on signs and symptoms:
 - Arterial blood gas
 - Thyroid-stimulating hormone
 - Cardiac enzymes

Imaging

- ECG
- Head CT scan
- CXR
- Other imaging based on history, physical exam, and possible etiologies

Diagnostic Procedures/Surgery

- As indicated by potential underlying cause
- Lumbar puncture if indicated
- EEG if indicated by potential seizure activity

DIFFERENTIAL DIAGNOSIS

- Other disease processes that should be distinguished from delirium include:
 - Psychiatric illness:
 - Symptoms do not have fluctuating course that is typical of delirium.
 - Usually there are no changes in level of consciousness.
 - Delirium is classically associated with visual hallucinations and psychiatric illness with auditory hallucinations.
 - Dementia:
 - Delirium has rapid onset, while dementia has a slowly progressive, insidious course without fluctuation of symptoms.
 - Dementia is not associated with acute changes in consciousness.
- Once identified as delirium, the differential for the underlying cause is quite extensive.



TREATMENT

PRE HOSPITAL

- IV access:
 - Pulse oximetry to monitor respiratory status:
 - Glucose measurement
 - ECG monitoring
- Naloxone if associated respiratory insufficiency
- Monitor patient:
 - Advanced life support (ALS) transport with all medications
- Look for signs of an underlying cause:
 - Medications
 - Medical alert bracelets
- Document basic neurologic exam:
 - Glasgow coma scale score
 - Pupils
 - Extremity movements

ED TREATMENT/PROCEDURES

- When delirium is identified, seek the underlying cause intensely.
- Treatment should be targeted at underlying medical condition.
- IV line access
- Oxygen if indicated by hypoxia
- Cardiac, pulse oximetry, and BP monitoring
- Thiamine should be administered to alcoholic and malnourished patients.
- In patients who are significantly agitated, chemical treatment of agitation may help facilitate ED workup.

MEDICATION

- Treatment of delirium should be aimed at underlying condition.
- Benzodiazepines should be 1st line for patients with alcohol or benzodiazepine withdrawal.
- Benzodiazepines should be avoided in patients with all other causes of delirium, if possible.

First Line

- Assess the patient for prolonged QT syndrome before administering antipsychotic agents. Haloperidol: 5–10 mg IV or IM:
 - Lower doses (0.5–2 mg) are appropriate for elderly patients.
- Recent studies show that atypical antipsychotics may be equally effective to typical antipsychotics.
- Thiamine: 100 mg IV, IM, or PO

Second Line

- Alprazolam: 0.25–0.5 mg PO

- Lorazepam: 0.5–2 mg IV, IM, or PO



FOLLOW-UP

DISPOSITION

Admission Criteria

- When cause is unclear, admit.
- If delirium has not resolved, admit.

Discharge Criteria

Patient could be discharged if:

- Treatable cause is found and treated
- Mental status clears while in the ED
- Reliable caregivers are available
- Follow-up is ensured

FOLLOW-UP RECOMMENDATIONS

- Follow-up depends on underlying condition.
- When delirium has resolved within ED stay, close follow-up with primary care provider, preferably in <2 days.
- Patients and caregivers should be counseled carefully regarding return precautions:
 - Any recurrence of delirium should prompt a return to the ED.
 - Delirium can be a life-threatening condition.

PEARLS AND PITFALLS

- Identify underlying cause
- Delirium is often missed by emergency physicians and maintaining an awareness of delirium as a syndrome is critical.

ADDITIONAL READING

- Han JH, Zimmerman EE, Cutler N, et al. Delirium in older emergency department patients: Recognition, risk factors, and psychomotor subtypes. *Acad Emerg Med.* 2009;16:193–200.
- Inouye SK. Delirium in older persons. *N Engl J Med.* 2006;354:1157–1165.
- Lonergan E, Luxenberg J, Areosa Sastre A. Benzodiazepines for delirium. *Cochrane Database Syst Rev.* 2009;(4):CD006379.



CODES

ICD9

- 291.0 Alcohol withdrawal delirium
- 293.0 Delirium due to conditions classified elsewhere
- 780.09 Other alteration of consciousness

ICD10

- F05 Delirium due to known physiological condition
- F10.231 Alcohol dependence with withdrawal delirium
- R41.0 Disorientation, unspecified

DELIVERY, UNCOMPLICATED

Jonathan B. Walker • James S. Walker

BASICS

ETIOLOGY

- Delivery in ED is rare:
 - Incidence of ED deliveries in US is not known.
 - Health care systems in which patients have little prenatal care tend to have greater incidence of ED deliveries.
- ED deliveries usually occur in 1 of the following 3 scenarios:
 - Multiparous patient with history of prior rapid labor
 - Nulliparous patient who does not recognize symptoms of labor
 - Patients with lack of prenatal care, lack of transportation, or premature labor

DIAGNOSIS

SIGNS AND SYMPTOMS

- True labor presents as uterine contractions occurring at least every 5 min and lasting 30–60 sec.
- Significant vaginal bleeding with labor demands immediate assessment for placenta previa or abruption.

History

- Last menstrual period and estimated gestational age (EGA)
- Recent infections
- Pregnancy history, complications
- Prior C-section
- Prenatal care
- Abdominal/pelvic cramping
- Ruptured membranes (amniotic sac)
- May report incontinence
- Urge to push or have a bowel movement
- Bloody show—loss of mucous plug

Physical-Exam

- Signs of imminent delivery:
 - Fully effaced and dilated cervix (~ 10 cm in term infant)
 - Palpable fetal parts
 - Bulging of perineum

- Widening of vulvovaginal area
- Try to determine fetal position and presenting part by palpation of the uterus

ESSENTIAL WORKUP

- *Sterile* bimanual pelvic exam is the most useful tool to assess presence of labor and possibility of imminent delivery:
 - Assess dilation, station, and effacement
 - No pushing until full dilation
 - Bimanual exam should *not* be done with vaginal bleeding until ultrasound (US) can rule out placenta previa.
- Fetal heart tones (FHTs) should be obtained by Doppler

DIAGNOSIS TESTS & INTERPRETATION

Lab

- If patient is in active labor, CBC, blood typing, and Rh screen should be sent:
 - Kleihauer-Betke testing should be ordered after delivery if Rh-negative mother gives birth to Rh-positive child
 - Rh immunoglobulin can be administered to mother within 72 hr of delivery
- Urinalysis if there is concern about urinary tract infection or preeclampsia

Imaging

- Imaging studies are not needed for uncomplicated vaginal deliveries
- 3rd-trimester vaginal bleeding should have emergent US to evaluate for placental abruption or placenta previa
- If time permits, US can help locate the position and anatomy of the placenta

DIFFERENTIAL DIAGNOSIS

- Braxton Hicks contractions:
 - Irregular uterine contractions that do not result in cervical dilation or effacement
- Muscular low back pain
- Round uterine ligament pain
- Other causes of abdominal pain, such as torsion of the ovary, appendicitis, nephrolithiasis



TREATMENT

PRE HOSPITAL

- Place patients in left lateral recumbent position
- Emergency medical services (EMS) personnel should be adequately trained and have proper equipment available for delivery

- EMS transportation of high-risk obstetric patients *before* delivery:
 - Lower neonatal morbidity and mortality
 - Faster and less expensive when compared with transportation of neonate *after* delivery
- Use of air transport for obstetric patients has been shown to be safe and effective:
 - Altitude during flight can result in hypoxia for fetus; pregnant patients should be placed on supplemental oxygen

INITIAL STABILIZATION/THERAPY

- Immediate sterile pelvic exam to assess for cervical dilation, effacement, station, or presenting parts (if no vaginal bleeding)
- Patients in active labor should be transferred to labor and delivery immediately unless delivery is imminent
- If patient is completely dilated and fetal parts are on perineal verge, prepare for ED delivery

ED TREATMENT/PROCEDURES

- Obstetrician should be notified that delivery will be occurring in ED
- Pediatrician or neonatologist and NICU should be notified
- Prepare for neonatal resuscitation
- Place patient in supine position or Sims position
- Begin IV saline or D5NS and supplemental oxygen, and place patient in lithotomy position
- Assemble obstetric (OB) pack:
 - Bulb syringe
 - 2 sterile Kelly clamps
 - Sterile Mayo scissors
 - Umbilical clamp
- Neonatal resuscitative equipment should also be available
- If time permits, sterilize vaginal area with povidone-iodine (Betadine)
- Uncomplicated vaginal delivery should occur as follows:
 - As crowning occurs, deliver head in controlled fashion, guiding it through introitus with each contraction.
 - Routine episiotomy is not necessary; however, if perineum is tearing, perform midline episiotomy by placing 2 fingers behind perineum and make straight incision toward (but not including) rectum with sterile Mayo scissors.
 - After fetal head is delivered, quickly suction nasopharynx, then feel around neck for nuchal cord:
 - If present, manually reduce over head
 - If nuchal cord is too tight, double clamp, cut cord, and deliver infant immediately
 - Apply gentle downward pressure on fetal head with uterine contractions:

- Deliver anterior shoulder
- Posterior shoulder and remainder of infant will rapidly deliver
- After delivery, infant should be held at level of uterus and oropharynx suctioned again
- Double clamp cord with sterile Kelly clamps and cut between them
- Infant should be stimulated, warmed, and dried:
 - If cyanosis is present, infant should be given oxygen and resuscitated
 - Follow neonatal resuscitation protocols if necessary
- Place umbilical clamp
- Placenta will spontaneously deliver in 20–30 min:
 - Observe mother closely for postpartum hemorrhage
- Uterine massage can aid in separation of placenta from uterus and limit uterine atony:
 - Avoid placing traction on umbilical cord because this can lead to inversion of uterus or rupture cord
- If patient has severe bleeding and placenta is not passing spontaneously, patient should be taken immediately to operating room
- After delivery of placenta, it should be examined for any irregular or torn areas suggestive of retained placental products
- In uncomplicated delivery, use of drugs is not necessary:
 - Massage of uterus is all that is needed to facilitate cessation of bleeding after placenta has been delivered
- Postpartum uterine bleeding is common:
 - Uterus, vagina, and perineum should be inspected for laceration
 - If no laceration is found, assume uterine atony
 - If uterus does not contract in response to uterine massage, administer oxytocin IV
 - Continued massage of uterus may be helpful if bleeding still persists; then give methylergonovine maleate (Methergine) IM
 - If bleeding is not responding to these measures, then carboprost tromethamine (Hemabate) can be administered IM

MEDICATION

- Carboprost tromethamine (Hemabate): 0.25 mg IM q15–60min (up to 2 doses)
- Methylergonovine maleate (Methergine): 0.2 mg IM
- Oxytocin: 20–40 U IV in 1 L of normal saline infused at 250–500 mL/h IV



FOLLOW-UP

DISPOSITION

Admission Criteria

- All women with uncomplicated deliveries and no significant postpartum bleeding should be admitted to labor and delivery or postpartum unit for care and monitoring
- Obtain pediatric or neonatal consultation and admit to neonatal ICU:
 - All infants with respiratory distress
 - Gestational age < 36 wk
 - Weight < 5 lb
 - Low Apgar scores
- Term infants with none of above complications may be admitted to the nursery or with mother to combined maternal–fetal unit
- If transferring the mother and infant after delivery, consider using 2 ambulances

Discharge Criteria

- After adequate recovery from delivery, patient can be taken labor and delivery or postpartum unit
- Patient should not be discharged home from ED

PEARLS AND PITFALLS

- Be ready for complications such as cord prolapse, shoulder dystocia, breech delivery
- Be prepared to treat 2 patients after delivery—mother and infant

ADDITIONAL READING

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- Marx JA, Hockberger RS, Walls RM, et al. *Rosen's Emergency Medicine: Concepts and Practice.* 7th ed. St. Louis, MO: Mosby; 2009.
- Mirza FG, Gaddipati S. Obstetric emergencies. *Semin Perinatol.* 2009;33:97–103.
- Roberts JR, Hedges JR, Chanmugan AS, et al., eds. *Clinical Procedures in Emergency Medicine.* 4th ed. Philadelphia, PA: Saunders; 2004.



CODES

ICD9

- 650 Normal delivery
- 661.30 Precipitate labor, unspecified as to episode of care or not applicable
- V23.7 Supervision of high-risk pregnancy with insufficient prenatal care

ICD10

- O09.30 Suprvsn of preg w insufficient antenat care, unsp trimester
- O62.3 Precipitate labor

- O80 Encounter for full-term uncomplicated delivery

DEMENTIA

Gabriel Wardi



BASICS

DESCRIPTION

- Progressive deterioration in cognition, behavior, or both without impaired consciousness that is severe enough to interfere with activities of daily living due to alteration in cortical brain function. A chronic and progressive form of organic brain syndrome.
- Over 50 different causes, but > 60% caused by Alzheimer disease
 - Involves increased neurofibrillary tangles and elevated beta amyloid plaques
- Prevalence 1% at age 60 yr to 30–50% by age 85 yr
- Characterized by gradual decline in cognitive functioning:
 - Generally evolves over period of years
 - Course is highly variable, months to years in duration
 - Rapid decline indicative of other causes, or rare rapid onset causes of dementia (prion diseases, progressive supranuclear palsy)
- Variable hereditary
 - Increased risk of Alzheimer disease in 1st-degree relatives of patients with Alzheimer
 - Apolipoprotein ε4 is the only well-established mutation with late-onset Alzheimer

ETIOLOGY

- Primary dementia:
 - Cortical (Alzheimer disease, frontotemporal dementia)
 - Subcortical (Huntington disease, Parkinson disease, progressive supranuclear palsy)
- Secondary dementia:
 - Cerebrovascular disease (multi-infarct dementia)
 - Toxic, metabolic, nutritional derangements
 - Prion disorders (Creutzfeldt-Jakob or bovine spongiform encephalopathy and variants)
 - Infectious agents (HIV, syphilis, encephalitis)
 - Vasculitis (systemic lupus erythematosus, thrombotic thrombocytopenic purpura)
 - Traumatic (chronic subdural hematomas, pugilistic dementia)
 - Structural (normal pressure hydrocephalus, brain masses)
 - Binswanger disease

- Reversible (~15%) causes include normal pressure hydrocephalus, medications, intracranial masses, and alcohol abuse syndromes
- Pseudodementia:
 - Depression in elderly can present with dementia-like symptoms
 - Common in mildly demented patients, look for pin-point event with short duration of symptoms
 - Generally with history of psychiatric conditions, emphasis on failures and disabilities

DIAGNOSIS

SIGNS AND SYMPTOMS

- Insidious onset, with initial complaints of anxiety, depression, frustration, increased forgetfulness
- Generally preceded by “mild cognitive impairment,” an intermediate state of cognitive function between normal aging and those meeting criteria for dementia
- Can be grouped into 3 categories:
 - Early: Difficulty concentrating, memory deficits, difficulty with complex tasks, social withdrawal
 - Moderate: Major memory difficulties, need assistance with activities of daily living
 - Severe: Minimal ability to speak or communicate, difficulty eating, loss of psychomotor skills
- Diagnostic criteria (from American Psychiatric Association):
 - Development of multiple cognitive deficits manifested by both:
 - Memory impairment
 - One (or more) of the following cognitive disturbances: Aphasia, apraxia, agnosia, disturbance in executive functioning
 - Cognitive deficits that cause significant impairment in social or occupational functioning and are a decline from prior levels of functioning
 - Deficits do not occur during course of delirium

History

- Must include input from family and friends
- Complete list of medications
- Comorbid diseases
- Prior history of similar behavior
- Onset and progression
- Consider use of Montreal Cognitive Assessment, Short Test of Mental Status (alternative to mini-mental status exam)

Physical-Exam

Full and complete physical exam:

- Head-to-toe evaluation, all organ systems
- Meticulous neurologic exam:
 - _ Mental status evaluation
 - _ Cranial nerves
 - _ Reflexes
 - _ Motor, sensory, cerebellar, gait

ESSENTIAL WORKUP

- Must eliminate acute reversible or exacerbating factors
- Extent of workup is related to history and course of illness:
 - _ Extensive evaluation for new diagnosis
 - _ Directed evaluation for sudden change of dementia
 - _ Limited evaluation for stable disease previously assessed
- Must be able to identify signs and symptoms of the reversible causes of dementia

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Extent of evaluation dependent on patient condition and suspected cause
- New diagnosis or sudden deterioration:
 - _ CBC
 - _ ESR/CRP
 - _ CMP
 - _ Ammonia
 - _ Urinalysis
 - _ Toxicology screen
 - _ Thyroid-stimulating hormone
 - _ Vitamin B₁₂ level
 - _ Syphilis serology (RPR)
 - _ HIV
 - _ Blood cultures if fever present
 - _ Urine cultures if fever present
 - _ Antinuclear antibody if SLE suspected
- Established diagnosis with stable disease: No tests may be required.

Imaging

- New diagnosis or sudden deterioration in established dementia:
 - _ CXR if infection considered
 - _ Head CT, without and with contrast
 - _ EEG if suspicion of seizure disorder
 - _ Brain MRI/MRA in selected cases

- More advanced imaging (PET, etc.) should be reserved for use by specialists
- Established diagnosis with stable disease: Studies may not be required.

Diagnostic Procedures/Surgery

- Lumbar puncture and CSF analysis, syphilis serology
- EEG if seizure suspected

DIFFERENTIAL DIAGNOSIS

- Toxic, metabolic, nutritional abnormalities:
 - Narcotics, sedatives, hypnotics
 - Alcohol
 - Heavy metals
 - Dehydration
 - Electrolyte abnormalities
- Pseudodementia
- Delirium (high suspicion for UTI and pneumonia in febrile patients)
- Senescent aging



TREATMENT

PRE HOSPITAL

- Obtain history from friends, family
- Provide for patient and staff safety
- Manage agitation
- Attentiveness to comorbid conditions
- Treat acute toxic and metabolic disorders:
 - Hypoglycemia
 - Hypothermia
 - Hyperthermia

INITIAL STABILIZATION/THERAPY

- Ensure adequate airway
- Administer O₂ if hypoxic
- Ensure normal vital signs
- Establish IV access if required
- In agitated patients, provide for patient and staff safety

ED TREATMENT/PROCEDURES

- Must determine if patient presents with acute change in mental status
- Consider full differential diagnosis—evaluate and treat appropriately:
 - Treat hypoglycemia with PO or IV dextrose.
 - Treat narcotic overdose or excess with naloxone.

- Rewarm if hypothermic.
- Antipyretic for hyperthermia
- IV fluids for dehydration
- Correct electrolyte abnormalities
- Administer antibiotics for infection:
 - UTI and pneumonia most common occult infections; look for wounds and decubitus ulcers
- Treat seizures:
 - Lorazepam, other agents as needed
 - Long-term management in conjunction with neurology
- Sedation for agitation:
 - Start with low doses and increase as necessary to achieve clinical result.
 - Neuroleptics: Haloperidol, risperidone, ziprasidone
 - Benzodiazepines: Lorazepam, midazolam
- Soft restraints if chemical sedation ineffective
- Attempt to limit number of medications:
 - Reduced likelihood of toxicity
 - Reduced likelihood of drug–drug interaction
 - If agitation not an issue, eliminate all sedative-hypnotics
- Treat depression

MEDICATION

- Alzheimer’s agents: Always start at lowest dose:
 - Donepezil: 5–10 mg PO at bedtime
 - Rivastigmine: 1.5–6 mg PO BID
 - Galantamine: 4–12 mg PO BID
 - Above 3 anticholinergics without clear superior agent, watch for side effects including nausea, vomiting, diarrhea
 - Consider memantine (NMDA receptor antagonist) in those with poor response to anticholinergics: 5 mg PO QID–10 mg PO BID
 - Effects generally modest, best started, and changed by primary provider
 - Numerous trials showing inconsistent or negative benefit of anti-inflammatory agents, estrogens, and statins for Alzheimer
- Antidepressants: Start with lowest dose:
 - Oversedation a problem
 - May worsen dementia
 - Useful in patients who cannot sleep
- Sedative agents: Always start with lowest dose
 - Droperidol: 0.625–2.5 mg IV—advantage, rapid onset; disadvantage, risk for QT prolongation
- Haloperidol: 0.5–2 mg PO BID; start with lowest dose 0.5–2.5 mg IM or IV if rapid onset required
- Lorazepam: 0.5–1 mg IV, 0.5–2 mg PO

- Midazolam: 0.5–2 mg IV slow push
- Naloxone: 0.4–2 mg IVP
- Risperidone: 0.5–2 mg PO BID; start with lowest dose
- Ziprasidone: 20–80 mg PO BID, 10–20 mg IM q4h; start with lowest dose



FOLLOW-UP

DISPOSITION

Admission Criteria

- Unstable vital signs
- Significant comorbid condition requiring parenteral medications:
 - Pneumonia
 - UTI
 - Fluid and electrolyte disorder
- Uncertain diagnosis requiring evaluation and management that is not suitable for outpatients
- Inadequate home support coupled with inability to arrange suitable placement from ED

Discharge Criteria

- Stable vital signs
- No significant unstable comorbid conditions
- Secure diagnosis or elimination of life-threatening organic disease
- Adequate home support, watch for caregiver burnout
- Reliable access to follow-up care

Issues for Referral

- Patients may need assistance with transportation, finances, etc.
- Patients with other comorbidities need referral to appropriate specialists.

FOLLOW-UP RECOMMENDATIONS

- Primary care
- Geriatrician
- Psychiatrist
- Neurologist

PEARLS AND PITFALLS

- Primary dementia is characterized by slow, steady progression:
 - Course is generally 5–10 yr from diagnosis to death.
- Can fluctuate as consequence of intervening illness and comorbid conditions

- Cholinesterase medications can improve functional status in patients with Alzheimer disease.
- Careful attention to medications, secondary illnesses, and prompt intervention for infections can improve quality of life and longevity.
- Death is generally consequence of infection, cardiovascular disease, or injury.

ADDITIONAL READING

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- Petersen RC. Mild cognitive impairment. *N Engl J Med*. 2011;364:2227–2234.
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See Also (Topic, Algorithm, Electronic Media Element)

- Altered Mental Status
- Delirium



CODES

ICD9

- 294.10 Dementia in conditions classified elsewhere without behavioral disturbance
- 294.20 Dementia, unspecified, without behavioral disturbance
- 331.0 Alzheimer’s disease

ICD10

- F02.80 Dementia in other diseases classified elsewhere without behavioral disturbance
- F03.90 Unspecified dementia without behavioral disturbance
- G30.9 Alzheimer’s disease, unspecified

DENGUE FEVER

Jessica Freedman



BASICS

DESCRIPTION

- Dengue fever occurs secondary to dengue viral infection.
- Most prevalent mosquito-borne viral infection.
- Poorly understood immunopathologic response causes dengue hemorrhagic fever (DHF) and dengue shock syndrome (DSS).
- DHF and DSS usually occur in patients with previous exposure to dengue virus.
- Hemorrhagic manifestations occur after defervescence of fever.
- Vascular permeability increases.
- Plasma extravasates into extravascular space, including pleural and abdominal cavities.
- Bleeding tendency
- Shock may ensue.
- Disseminated intravascular coagulation (DIC) may develop.
- Dengue fever, DHF, and DSS are all self-limited.
- World Health Organization—required criteria for the diagnosis of DHF:
 - Fever
 - Bleeding evidenced by one of the following: Positive tourniquet test, petechiae, ecchymosis, purpura, GI tract bleeding, injection site bleeding
 - Increased vascular permeability and plasma leakage as evidenced by an elevated hematocrit ($> 20\%$), decreased hematocrit $> 20\%$ after volume replacement or pleural effusions, ascites or hypoproteinemia
 - Thrombocytopenia ($< 100,000/\text{mm}^3$)
- World Health Organization—required criteria for diagnosis of DSS:
 - All 4 criteria of DHF +
 - Rapid and weak pulse
 - Narrow pulse pressure or hypotension for age
 - Cold, clammy skin
 - Restlessness

ETIOLOGY

- Occurs in tropical and subtropical regions: Asia, Africa, Central and South America, and the Caribbean
- Caused by dengue virus serotypes 1–4
- Transmitted by mosquitoes: *Aedes aegypti* and *Aedes albopictus*
- Incubation period of 3–14 days
- There is only transient and poor cross protection among the 4 serotypes

DIAGNOSIS

SIGNS AND SYMPTOMS

- Fever:
 - Abrupt in onset rising to 39°C or higher
 - 2–7 days duration
 - Biphasic (“saddleback”) curve, returning to almost normal after 2–7 days
 - Associated with frontal or retro-orbital headache
- Rash:
 - Generalized maculopapular rash occurs with onset of fever in 50% of patients.
 - After 3–4 days, rash becomes diffusely erythematous.
 - Faded areas appear.
 - Areas of desquamation may appear.
 - After defervescence of fever, scattered petechiae may develop over trunk, extensor surfaces of limbs, and axillae.
 - Palms and soles spared
- Musculoskeletal:
 - Arthralgias and myalgias after onset of fever
 - Severe lumbar back pain
- GI:
 - Anorexia
 - Nausea and vomiting
 - Abdominal pain (sometimes severe)
 - Altered taste
 - Hepatomegaly/ascites
 - GI bleeding
- Miscellaneous:
 - Epistaxis
 - Gingival bleeding
 - Hemoptysis
 - Hypotension
 - Narrowed pulse pressure (< 20 mm Hg)
 - Retro-orbital pain

ESSENTIAL WORKUP

- Primarily a clinical diagnosis
- Suspect in endemic areas
- Suspect in patients with history of travel

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC:
 - Thrombocytopenia
 - Elevated hematocrit
- Electrolytes, BUN, creatinine:
 - Elevated BUN
 - Hyponatremia
- Liver function tests:
 - Elevated aspartate transaminase (AST; or serum glutamic-oxaloacetic transaminase [SGOT])
- Coagulation profiles:
 - Prolonged INR, prothrombin time (PT), and partial thromboplastin time (PTT)
 - Low fibrinogen:
 - D-dimer
 - Virus isolation or detection of dengue virus–specific antibodies (available in only a few labs) through hemagglutination inhibition (HI) assay

Imaging

CXR:

- Pleural effusions

Diagnostic Procedures/Surgery

Tourniquet test:

- Inflate BP cuff to median BP in patient's extremity.
- Test is positive when 3 or more petechiae appear per square centimeter.

DIFFERENTIAL DIAGNOSIS

- Viral illness, nonspecific
- Influenza
- Rubella
- Measles
- Malaria
- Rocky Mountain spotted fever
- Typhoid
- Kawasaki disease
- Scarlet fever
- Erythema infectiosum
- Mononucleosis
- Roseola infantum
- Secondary syphilis
- Enterovirus
- West Nile virus

- HIV
- Leptospirosis
- Chikungunya fever
- Toxic shock syndrome
- Hepatitis
- Appendicitis
- Meningitis



TREATMENT

INITIAL STABILIZATION/THERAPY

- IV access
- IV crystalloids for hypotension
- O₂ and monitor for unstable patients

ED TREATMENT/PROCEDURES

- Treatment is supportive.
- IV fluids
- Acetaminophen (Tylenol) for fever
- Analgesics for pain
- Platelet transfusion for severe thrombocytopenia
- DIC therapy, if necessary

Pediatric Considerations

- Neonatal dengue can occur by vertical transmission if mother infected 0–8 days before delivery:
 - Infants may develop DHF or DSS because of passive maternal immunity.
- DHF and DSS most common in children 7–12 yr of age



FOLLOW-UP

DISPOSITION

Admission Criteria

- ICU admission for the following:
 - Hypotension
 - DIC
 - Thrombocytopenia
 - Hemoconcentration
- Regular admission for the following:
 - 15 yr of age or younger

- All patients with previous dengue exposure
- Any patient where close follow-up is not available

Discharge Criteria

- Close follow-up guaranteed
- Tolerating PO
- Pain controlled

PEARLS AND PITFALLS

- Consider dengue in patients presenting with fever and rash who recently traveled to endemic regions.
- Chikungunya fever is an emerging infectious disease also seen in travelers and must be considered in the differential:
 - Found in Asia and Africa

ADDITIONAL READING

- Halstead SB: Dengue. *Lancet*. 2007;370:1644–1652.
- Istúriz RE, Gubler DJ, Brea del Castillo J. Dengue and dengue hemorrhagic fever in Latin America and the Caribbean. *Infect Dis Clin North Am*. 2000;14(1):121–140.
- Pincus LB, Grossman ME, Fox LP. The exanthem of dengue fever: Clinical features of two US tourists traveling abroad. *J Am Acad Dermatol*. 2008;58(2):308–316.
- Simmons CP, Farrar JJ, Nguyen vV, et al. Dengue. *N Engl J Med*. 2012;366:1423–1432.
- Wilder-Smith A, Schwartz E: Dengue in travelers. *N Engl J Med*. 2005;353:924–932.



CODES

ICD9

061 Dengue

ICD10

A90 Dengue fever [classical dengue]

DENTAL TRAUMA

Brian N. Corwell

BASICS

DESCRIPTION

- Primary teeth:
 - Eruption begins between 6–10 mo of age and concludes by 30 mo
 - Eruption is bilaterally symmetric
 - 20 total teeth
- Permanent teeth:
 - Begin to erupt at age 6
 - 32 total (4 central and 4 lateral incisors, 4 canines, 8 premolars, 12 molars)
 - Number from 1–32 starting with upper right 3rd molar (1) to upper left 3rd molar (16) and lower left 3rd molar (17) to lower right 3rd molar (32)
 - Better and often easier to describe the involved tooth anatomically
- Most commonly injured teeth:
 - Maxillary central incisors, maxillary lateral incisors, and the mandibular incisors
- Tooth fractures:
 - Fractures of the crown are classified as uncomplicated (involve only the enamel or both the enamel and dentin) or complicated (involves the neurovascular pulp)
 - Fractures can be classified using the *Ellis classification system*
 - Class I fracture (uncomplicated fracture):
 - Involves only the superficial enamel
 - Fracture line appears chalky white
 - Painless to temperature, air, percussion
 - Class II fracture (uncomplicated fracture):
 - Involves enamel and dentin
 - Fracture line will appear ivory or pale yellow compared to whiter enamel
 - May be sensitive to heat, cold, or air
 - Not tender
 - Class III fracture (complicated fracture):
 - True dental emergency
 - Involves enamel, dentin, and pulp
 - Pulp has pinkish, red, fleshy hue
 - Frank bleeding or a pink blush after wiping tooth surface indicates pulp violation
 - May be exquisitely painful or desensitized (with associated

neurovascular disruption)

- Luxation injuries
 - Involve the supporting structures
 - Includes the periodontal ligament (PDL) and alveolar bone
- Several types of injuries exist:
 - Concussed teeth:
 - Tooth neither loose nor displaced
 - Sensitivity with chewing or percussion
 - Subluxed teeth:
 - Tooth is loose but not displaced
 - Bleeding from gingival sulcus
 - Sensitivity with chewing or percussion
 - PDL is damaged
 - Intrusion:
 - Tooth is driven into socket
 - Alveolar socket fractured
 - PDL compressed
 - Avulsed tooth:
 - Total displacement from alveolar ridge
 - PDL severed
 - Extrusion:
 - Partial central dislocation from socket
 - PDL damaged
 - Lateral luxation:
 - Nonaxial displacement of the tooth
 - PDL damaged
 - Associated with alveolar socket fracture
- Alveolar bone fractures:
 - Fractures of tooth-bearing portions of mandible or maxilla
 - Bite malocclusion, painful bite, tooth mobility en bloc
 - Diagnosed clinically or radiographically

ETIOLOGY

- Nearly 50% of children sustain a dental injury
- Age periods of greatest predilection:
 - Toddlers (falls and child abuse)
 - School-aged children and preteens (falls, bicycle, and playground accidents)
 - Adolescents (athletics, altercations, MVCs)
 - Mouth guard use greatly reduces sport-associated dental injury
- Assault, domestic violence, or multiple trauma
- Motor vehicle, motorcycle, bicycle accidents
- Child abuse
 - Frequently associated with orofacial injury

- Laryngoscopy
- Certain predisposing anatomic factors increase risk:
 - Anterior overbite >4 mm increases risk for traumatic injury 2–3 times
 - Short or incompetent upper lip, mouth breathing, physical disabilities, use of fixed orthodontic appliances

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Tooth mobility, avulsion or laxity
- Bite malocclusion or trismus
- Exacerbating factors (may indicate pulp exposure or PDL damage):
 - Chewing or drinking
 - Extremes of temperature
 - Pain on palpation
- Mechanism:
 - Sufficient mechanism necessitates complete evaluation for multiple trauma and associated local injuries (e.g., jaw fracture)
- Exact time of injury:
 - May affect treatment and prognosis

Physical-Exam

- Examine all teeth for trauma or fracture
- Examine fractured teeth for pulp exposure:
 - Dry the tooth with gauze; observe for frank bleeding or pink blush
- Inspect each tooth surface and percuss for mobility, sensitivity, or fracture
- Assess for malocclusion and midface stability
- Account for all missing teeth
 - Tooth fragments and prostheses may have been swallowed, aspirated, embedded into adjacent soft tissue or impacted into alveolus
- Inspect oral cavity carefully:
 - Adjacent soft tissue or bone injuries
 - Suspect a mandible fracture in those unable to open mouth >5 cm or with a positive tongue blade bite test
 - Associated injuries:
 - Salivary glands, ducts, blood vessels
 - Mental and infraorbital nerves

ESSENTIAL WORKUP

- Thorough physical exam

- Imaging as necessary
- Stabilization and proper referral

DIAGNOSIS TESTS & INTERPRETATION

Imaging

- Plain dental radiograph:
 - Complicated fractures
- Panorax indications:
 - Foreign bodies
 - Displacement of teeth
- CT indications:
 - Trauma with malocclusion or trismus
 - Suspected alveolar or mandibular fracture
- CXR:
 - Indicated for missing teeth or fragments
 - Teeth visualized below the diaphragm do not require removal
- Bronchoscopy:
 - Indicated removal of aspirated tooth

DIFFERENTIAL DIAGNOSIS

Rule out other significant concurrent facial or systemic injuries.



TREATMENT

PRE HOSPITAL

- Avulsed teeth:
 - Only replace avulsed secondary teeth
 - Rinse tooth with cold running water
 - Immediate attempt to reimplant permanent tooth into socket by 1st capable person:
 - *Time is tooth:* Each minute tooth is out of socket reduces tooth viability by 1%
 - Best chance of success if reimplant done within 5–15 min
 - Poor tooth viability if avulsed for >1 hr
 - If unsuccessful, place tooth in a transport solution (from most to least desirable):
 - Hanks balanced salt solution (HBSS)
 - Balanced pH culture media available commercially in the Save-A-Tooth kit
 - Effective hours after avulsion
 - Cold milk:

- Best alternative storage medium
- Place tooth in a container of milk that is then packed in ice (prevents dilution)
- _ Saliva:
 - Store in a container of child's saliva
- _ Never use tap water or dry transport

INITIAL STABILIZATION/THERAPY

- Ensure patent airway
- Have patient bite on gauze to control bleeding
- Account for all teeth and tooth fragments
- Reimplant avulsed tooth immediately

ED TREATMENT/PROCEDURES

- General considerations:
 - _ Splint before attempting laceration repair
 - _ Occlusion is always the best guide to proper tooth position
 - _ Tetanus prophylaxis:
 - Consider as a nontetanus-prone wound
 - Indicated for dirty wounds, deep lacerations, avulsed teeth, intrusion injuries, bone fracture
 - _ Antibiotic indications:
 - Open dental alveolar fractures
 - Treatment of secondary infection
 - Persons at risk for subacute bacterial endocarditis
 - Not indicated for infection prophylaxis
 - _ Dental fracture management:
 - Determined by patient age and extent of associated trauma
- Ellis class I:
 - _ No emergency treatment indicated
 - _ File/smooth sharp edges with an emery board:
 - Prevents further injury to soft tissue
 - _ Dental referral for elective cosmetic repair
- Ellis class II:
 - _ Treatment goal is to prevent bacterial pulp contamination through exposed dentin
 - _ Cover exposed surface with calcium hydroxide paste or similar barrier agent
 - Dry tooth surface prior to application
 - Use cyanoacrylate tissue adhesive if no such agent exists
 - _ Next, cover and wrap tooth with dental foil
 - _ Liquid diet until follow-up
 - _ Pain control
 - _ Dental referral within 48 hr

- Ellis class III:
 - Immediate referral to dentist or endodontist
 - If dentist/oral surgeon is not available:
 - Cover exposed surface and wrap with dental foil as with class II injuries
 - For brisk bleeding, have patient bite into gauze soaked with topical anesthetic and epinephrine or inject solution into pulp
 - Pain control
- Concussed tooth:
 - No splinting required
 - Soft diet
 - Follow-up with dentist as needed
- Subluxed tooth:
 - Splinting only required for excess laxity
 - Soft diet for 1 wk
 - Follow-up with dentist
- Extrusion:
 - Reposition with digital pressure
 - Splinting for 2 wk
 - Soft diet for 1 wk
 - Follow-up with dentist
- Lateral luxation:
 - Repositioning may be forceful/traumatic
 - May need to disengage from bony lock
 - May require local anesthetic
 - Use 2-finger technique:
 - 1st finger guides the apex down and back while 2nd finger repositions crown
 - Soft diet for 2 wk
 - Splinting usually required for up to 4 wk
 - Follow-up with dentist
- Intrusion:
 - Do not manipulate
 - Pain control
 - Dental follow-up within 24 hr
- Partial tooth avulsion:
 - May require local anesthetic
 - Carefully reduce to normal position
 - Consider manual removal of extremely loose teeth in neurologically impaired patients to prevent aspiration
- Avulsed tooth:
 - Never replace avulsed primary teeth
 - Handle the tooth only by the crown

- Avoid touching the root
- Remove debris by gentle rinsing in saline or tap water
- Do not wipe, scrub, curette, or attempt to disinfect tooth
- Administer local anesthesia if needed
- Gently irrigate or suction clots
 - Use care not to damage socket walls
- Manually reimplant tooth with firm but gentle pressure
 - Tooth should “click” into place
- Once tooth inserted, have patient bite gently onto folded gauze pad to help maneuver into proper position
- Splinting may be required
 - Apply to anterior or both anterior and posterior surfaces of the avulsed tooth/gingiva and adjacent 2 teeth
- Attempt reimplant regardless of time avulsed
- Liquid diet until follow-up
- Definitive stabilization by a dentist
- If tooth reimplanted pre-hospital:
 - Assure correct position and alignment
- Alveolar bone fracture:
 - Oral surgery/dental consultation for reduction and fixation (arch bar)
 - Pain control
 - Prophylactic antibiotics
 - Liquid diet, avoid straws

MEDICATION

- Acetaminophen with codeine: 30–60 mg/dose 1–2 tabs PO q4–6h PRN (peds: Codeine: 0.5–1 mg/kg/dose [max. 30–60 mg] PO q4–6h)
- Acetaminophen with oxycodone: 1–2 tabs PO q4–6h PRN (peds: Oxycodone: 0.05–0.15 mg/kg/dose [max. 5 mg/dose] PO q4–6h)
- Penicillin V: 250–500 mg PO q6h (peds: 25–50 mg/kg/24h [max. 3 g] PO q6h)
- Clindamycin (use if penicillin allergic): 150–300 mg PO q6h (peds: 10–25 mg/kg/24h PO q6h)
- Tetanus prophylaxis: 0.5 mL IM

ALERT

The dose of acetaminophen and all acetaminophen products should not exceed 4 g/24h



FOLLOW-UP

DISPOSITION

Admission Criteria

- Admission for other associated injuries
- Suspected child or elder abuse and those with no available safe environment

Discharge Criteria

All hemodynamically stable patients with dental injury without associated traumatic injury

Issues for Referral

- Ellis III injuries: Immediate dental referral
- Loose, displaced, or missing teeth
- Document recommendations and arrangements for dental follow-up care

FOLLOW-UP RECOMMENDATIONS

All patients with avulsions and Ellis II and III injuries should see dentist within 24 hr

PEARLS AND PITFALLS

- Avulsed teeth should never be transported in a dry medium or in tap water
- Occlusion is the best guide to proper tooth position after reimplantation
- Warn patients with dental trauma of risks of tooth resorption, color change, potential tooth loss, and/or need for future root canal

ADDITIONAL READING

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- Wolfson AB, Hendey GW, Ling LJ, et al., eds. *Harwood Nuss' Clinical Practice of Emergency Medicine.* 5th ed. Philadelphia, PA: Lippincott; 2010.



See Also (Topic, Algorithm, Electronic Media Element)

Tooth Pain



CODES

ICD9

- 525.8 Other specified disorders of the teeth and supporting structures
- 525.11 Loss of teeth due to trauma
- 873.63 Open wound of tooth (broken) (fractured) (due to trauma), without mention of complication

ICD10

- K03.81 Cracked tooth
- S02.5XXA Fracture of tooth (traumatic), init for clos fx
- S03.2XXA Dislocation of tooth, initial encounter

DEPRESSION

Jonathan Florman



BASICS

DESCRIPTION

Major depression:

- Depressed mood and associated signs and symptoms lasting more than 2 wk
- Significant morbidity and mortality, including risk of suicide
- Often coexists with other medical illness

ETIOLOGY

- Biologic illness associated with derangements in several neurotransmitter systems including serotonin, norepinephrine, and dopamine
- Contributing factors:
 - _ Genetic predisposition
 - _ Medical illness
 - _ Medication effects
 - _ Psychosocial stress: Depression may follow adverse life event, trauma, loss of important relationship, or life role
- Higher prevalence in women. (Women make more suicide attempts; men are more likely to complete suicide successfully)



DIAGNOSIS

SIGNS AND SYMPTOMS

- 5 or more symptoms for at least 2 wk. (One of the symptoms must be depressed mood or loss of interest or pleasure):
 - _ Depressed mood
 - _ Diminished interest or pleasure
 - _ Change in appetite, weight loss/gain
 - _ Sleep disturbance
 - _ Fatigue or loss of energy
 - _ Diminished concentration
 - _ Feeling of worthlessness or guilt
 - _ Recurrent thoughts of death or suicide
 - _ Psychomotor agitation or retardation
- Subtypes: Psychotic features, melancholic, catatonic, atypical, postpartum, seasonal
- May be anxious/agitated or withdrawn
- Associated somatic complaints:

- Weakness, malaise
- Weight loss
- Headache, back pain

History

- Time course, acuity, stressors
- Review depressive symptoms (see above)
- Past medical history
- Past psychiatric history
- Medications (prescribed and over-the-counter)
- Substance use
- Family history
- Social and occupational history; losses, transitions, trauma, and other major life events
- Safety assessment:
 - Suicide risk
 - Risk of violence to others
 - Assess ability to care for self, nutrition
- Collateral from family or outpatient providers
- Cultural and language differences may complicate evaluation; use interpreter when appropriate

Physical-Exam

- Vital signs
- Neurologic exam:
 - Motor exam: Station, gait, strength, tone, abnormal movements
 - Cognitive exam: Orientation, attention, memory, language, executive function
- Mental status exam: Affect and mood, thought form and content

Pediatric Considerations

- Depression may be difficult to diagnose in children and adolescents. Indicators of major depression in children may include:
 - Changes in school, home, and social functioning
 - Changes in sleep
 - Social withdrawal
 - Somatic complaints
- Consult with a child psychiatrist

ALERT

Rule-out bipolar disorder: May require different treatment (mood stabilizers, antipsychotics), also antidepressants may precipitate mania in bipolar patients

ESSENTIAL WORKUP

- Identify signs and symptoms of major depression (see “Signs and Symptoms”)
- Use history and physical exam to guide further workup
- Rule-out associated or coexisting psychiatric and medical conditions, substance use
- Safety assessment

DIAGNOSIS TESTS & INTERPRETATION

Lab

- 1st line:
 - CBC; chemistries including electrolytes, BUN/creatinine, glucose, calcium, liver function tests
 - Urinalysis
 - Serum and urine toxicology screen
 - Thyroid function tests
 - B₁₂ and folate
- 2nd line, guided by history and initial findings:
 - HIV testing
 - RPR
 - ESR/CRP/ANA

Imaging

- Brain imaging: Recommended for atypical presentation or if focal neurologic findings
- MRI brain preferred over CT for detecting tumors, cerebrovascular accident, white matter changes

DIFFERENTIAL DIAGNOSIS

- Psychiatric illnesses:
 - Dysthymic disorder
 - Adjustment disorder
 - Bipolar disorder
 - Anxiety disorders, including acute stress reactions, PTSD
 - Schizophrenia, schizoaffective disorder
 - Personality disorder
 - Eating disorder
 - Substance-induced mood disorder
- Medical conditions that may cause or mimic depression:
 - Drug induced:
 - Antihypertensives
 - Oral contraceptives
 - Steroids

- Sedative-hypnotics
- Opioids
- Psychostimulants (in withdrawal phase)
- β -Blockers
- Metoclopramide
- Endocrine disorders:
 - Hypothyroidism
 - Adrenal insufficiency
 - Diabetes mellitus
 - Postpartum, perimenopausal, and premenstrual syndromes
- Tumors:
 - Pancreatic
 - Lung
 - Brain
- Neurologic disorders:
 - Dementia (early phase or frontal type)
 - Epilepsy
 - Parkinson disease
 - Multiple sclerosis
 - Huntington disease
 - Stroke
 - Head trauma; subdural hematoma
 - Normal pressure hydrocephalus
- Infections:
 - Hepatitis
 - HIV
 - Mononucleosis
- Nutritional disorders:
 - Folate deficiency
 - Pellagra
 - Vitamin B₁₂ deficiency
- Electrolyte disturbances
- End-stage renal, hepatic, pulmonary, and cardiovascular diseases
- Obstructive sleep apnea
- Chronic pain syndromes



TREATMENT

PRE HOSPITAL

- Ensure safety of patient and providers
- Understand local laws for involuntary commitment to hospital

INITIAL STABILIZATION/THERAPY

- Safety: Assess risk of suicide, violence
- General medical evaluation
- Management:
 - 1-to-1 observation and suicide precautions when appropriate
 - Work up potential medical causes

ED TREATMENT/PROCEDURES

- Psychological management:
 - Listen empathically to understand context and relevant stressors
 - Reassurance and education (e.g., depression is a treatable condition)
- Initiate medications:
 - Antidepressant medication may be initiated for some patients with clear diagnosis and established follow-up
 - Usually takes weeks for antidepressant medications to resolve major depression
 - Low-dose benzodiazepines or neuroleptics may be used for associated agitation, insomnia, or psychosis
- Choice of drug determined by:
 - Indications, efficacy
 - Side-effect profile and risks
 - Convenience, cost, availability
- Selective serotonin reuptake inhibitors (SSRIs: fluoxetine, paroxetine, sertraline, citalopram, escitalopram):
 - Well tolerated
 - Side effects may include:
 - Mild nausea
 - Headache
 - Anxiety, restlessness, insomnia
 - Somnolence
 - Sexual dysfunction
 - Weight gain
 - Minimal overdose risk
- Serotonin norepinephrine reuptake inhibitors (SNRIs: venlafaxine, duloxetine):
 - Well tolerated
 - May be helpful for some pain syndromes
 - Side effects similar to SSRIs
- Dopamine norepinephrine reuptake inhibitor (bupropion):
 - Agitation, insomnia
 - Tremor
 - Decreased seizure threshold
 - Well-tolerated; no sexual side effects
- Norepinephrine serotonin modulator (mirtazapine):

- Weight gain
- Sedation
- Orthostasis
- Constipation
- Tricyclic antidepressants (amitriptyline, imipramine, nortriptyline, clomipramine):
 - Anticholinergic effects
 - Weight gain
 - Postural hypotension
 - Sedation
 - Decreased seizure threshold
 - Cardiac risk; overdose can be fatal
 - Nortriptyline is best tolerated
- Monoamine oxidase inhibitors (phenelzine, tranylcypromine, selegiline transdermal):
 - Dietary and other medication restrictions to avoid hypertensive crisis
 - Dangerous in overdose

MEDICATION

Medication dosage ranges are for adults.

Dose may be titrated over weeks as indicated.

- Amitriptyline: Initial 25–50 mg/d PO
- Bupropion: 75–400 mg/d PO
- Citalopram: 20–40 mg/d PO
- Desvenlafaxine: 50 mg/d PO
- Duloxetine: 30–120 mg/d PO
- Escitalopram: 10–20 mg/d PO
- Fluoxetine: 20–60 mg/d PO
- Imipramine: Initial 25–50 mg/d PO
- Mirtazapine: 15–45 mg/d PO
- Nortriptyline: Initial 25 mg/d PO
- Paroxetine: 20–40 mg/d PO
- Phenelzine: 15–90 mg/d PO
- Sertraline: 50–200 mg/d PO
- Tranylcypromine: 10–60 mg/d PO
- Venlafaxine: 75–300 mg/d PO

First Line

SSRIs, SNRIs, bupropion, mirtazapine

Second Line

- Tricyclics and monoamine oxidase inhibitors
- Use with caution in geriatric or medically ill
- Consider ECT for severe or treatment-resistant depression, psychotic depression, or

Geriatric Considerations

- Older patients may require lower dose; pay careful attention to potential drug interactions
- Caution with orthostatic hypotension and cholinergic blockade

Pediatric Considerations

FDA “Black box” warning: Antidepressants may increase risk of suicidal thinking and behavior in some children, adolescents, or young adults with depression

Pregnancy Considerations

In pregnant or breast-feeding women pay special attention to risks and benefits of medication treatments—consider consultation with a specialist in perinatal psychiatry



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patient is suicidal or at high risk for suicide. See “Suicide, Risk Evaluation”
- Minimal or unreliable social supports
- Symptoms so severe that continual observation or nursing supportive care is required
- Psychotic features
- *Civil commitment* for psychiatric hospitalization is necessary if the patient is refusing treatment and is at risk to harm self or others

Discharge Criteria

- Low suicide risk
- Adequate social support
- Close follow-up available

Issues for Referral

- Outpatient mental health appointments and/or partial (day) hospital for patients not admitted
- Insurance carrier may determine inpatient disposition and options for other levels of care
- Case management or social services in ED may be helpful for disposition issues
- Communicate and coordinate care with other providers including primary care

FOLLOW-UP RECOMMENDATIONS

Follow-up depends on severity of illness and risk:

- If not admitted, patients with significant symptoms should follow up in 1–2 wk
- When medication is initiated, patient should be seen in follow-up in 1–2 wk
- More stable patients or those with minor symptoms may be seen with less urgency

PEARLS AND PITFALLS

- Patients with depression experience significant morbidity and may present a risk of self-harm
- Consider other conditions that mimic depression; also coexisting psychiatric and medical conditions, substance use
- Know hospitalization and involuntary commitment criteria in your area

ADDITIONAL READING

- American Psychiatric Association. Practice guideline for the treatment of patients with major depressive disorder, third edition. *Am J Psychiatry*. 2010;167(suppl 10):1–152.
- Belmaker RH, Agam G. Major depressive disorder. *N Engl J Med*. 2008;358:55–68.
- Cassem NH. Mood disordered patients. In: Stern TA, Fricchione GL, Cassem NH, eds. *MGH Handbook of General Hospital Psychiatry*. 6th ed. St. Louis, MO: Mosby; 2010.
- Stewart DE. Clinical practice. Depression during pregnancy. *N Engl J Med*. 2011;365:1605–1611.



See Also (Topic, Algorithm, Electronic Media Element)

- Bipolar Disorder
- Psychosis, Medical vs. Psychiatric
- Psychiatric Commitment
- Suicide, Risk Evaluation



CODES

ICD9

- 296.20 Major depressive affective disorder, single episode, unspecified
- 296.24 Major depressive affective disorder, single episode, severe, specified as with psychotic behavior
- 296.30 Major depressive disorder, recurrent episode, unspecified degree

ICD10

- F32.3 Major depressv disord, single epsd, severe w psych features
- F32.9 Major depressive disorder, single episode, unspecified

- F33.9 Major depressive disorder, recurrent, unspecified

DERMATOMYOSITIS/POLYMYOSITIS

Sean-Xavier Neath



BASICS

DESCRIPTION

- Dermatomyositis (DM) and polymyositis (PM) are systemic inflammatory myopathies, which represent the largest group of acquired and potentially treatable causes of skeletal muscle weakness
- Patients experience a marked progression of muscle weakness over weeks to months
- Can lead to respiratory insufficiency from respiratory muscle weakness
- Aspiration pneumonia can occur owing to a weak cough mechanism, pharyngeal muscle dysfunction, and esophageal dysmotility
- Cardiac manifestations include myocarditis, conduction defects, cardiomyopathy, and congestive heart failure (CHF)
- Arthralgias of the hands, wrists, knees, and shoulders
- Ocular muscles are not involved but facial muscle weakness may be seen in advanced cases

ETIOLOGY

- The exact cause is unknown, although autoimmune mechanisms are thought to be largely responsible
- Incidence ~ 1:100,000 with a female preponderance
- Association with HLA-B8 and HLA-DR3
- There may be an association between PM and certain viral, bacterial, and parasitic infections
- DM/PM occurs with collagen vascular disease about 20% of the time
- In DM, humoral immune mechanisms are implicated, resulting in a microangiopathy and muscle ischemia
- In PM, a mechanism of T-cell-mediated cytotoxicity is posited. CD8 T cells, along with macrophages surround and destroy healthy, non-necrotic muscle fibers that aberrantly express class I major histocompatibility complex (MHC) molecules
- Deposition of complement is the earliest and most specific lesion, followed by inflammation, ischemia, microinfarcts, necrosis, and destruction of the muscle fibers

Pediatric Considerations

- Although DM is seen in both children and adults, PM is rare in children
- Similar to adult DM, juvenile DM (JDM) primarily affects the skin and skeletal muscles

- Juvenile form may include vasculitis, ectopic calcifications (calcinosis cutis), and lipodystrophy
- The juvenile form may be associated with coxsackievirus

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- PM is distinguished from DM by the absence of rash
- Patients with PM present with muscle pain and proximal muscle weakness
- DM presents with skin rash, muscle pain, and weakness
- Constitutional symptoms include weight loss, fever, anorexia, morning stiffness, myalgias, and arthralgias
- Patients often note fatigue doing customary tasks:
 - Brushing hair, climbing stairs, reaching above the head, rising from a chair
 - May also complain of dysphagia, dyspnea, and cough
- Progressive weakness of the proximal limb and girdle muscles is seen early; distal muscle weakness can occur late in the disease

Physical-Exam

- General:
 - Fatigue
 - Fever
 - Weight loss
- Dysphagia
- Progressive muscle weakness:
 - Involves proximal muscles primarily
 - Symmetrical
- Skin findings of DM:
 - Skin rash occurs with or precedes muscle weakness
 - Heliotrope rash (lilac discoloration) on the upper eyelids associated with edema
 - Gottron sign: Violaceous or erythematous papules over the extensor surfaces of the joints, particularly knuckles, knees, and elbows
 - Shawl sign: A V-shaped erythematous rash occurring on the back and shoulders
 - Periungual telangiectasias: Nail-bed capillary changes that include thickened irregular and distorted cuticles
 - “Machinist hands”: Darkened horizontal lines across the lateral and palmar aspects of the fingers

ESSENTIAL WORKUP

- Assess airway and breathing for any signs of aspiration or compromise
- Assess for any signs of cardiac involvement and complications

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Serum muscle enzymes:
 - Creatine phosphokinase (CPK) is elevated, other muscle enzymes such as aldolase, can also be elevated
- Diagnostic criteria established in 1975 by Bohan and Peter:
 - Symmetric proximal muscle weakness with dysphagia and respiratory muscle weakness
 - Elevation of serum muscle enzymes
 - Electromyographic features of myopathy
 - Muscle biopsy showing features of inflammatory myopathy
 - Confidence limits for diagnosis (typical rash must be seen for diagnosis of DM):
 - Definite diagnosis: 3 or 4 criteria
 - Probable diagnosis: 2 criteria
 - Possible diagnosis: 1 criterion
- Newer diagnostic criteria using autoantibody profiles (Anti-Jo-1, Anti-SRP, Anti-Mi-2) or immunohistologic characterization may prove to be more specific for diagnosis of specific disease subgroups

Imaging

- Chest radiograph may show interstitial lung disease, evidence of aspiration pneumonia, CHF, or cardiomyopathy
- EMG studies show myopathic potentials that may support the diagnosis but are not specific for DM/PM
- Increasing role for MRI in determining regions of inflammation best suited for biopsy

Diagnostic Procedures/Surgery

- Muscle biopsy is the definitive test:
 - In PM, inflammatory infiltrates are often endomysial, although they may be perivascular
 - In DM, inflammatory infiltrates are mostly perivascular and include a high percentage of B cells
- Renal biopsies of patients may show focal proliferative glomerulonephritis
- Pulmonary function tests are useful in following the progression of interstitial lung disease

DIFFERENTIAL DIAGNOSIS

- Collagen vascular diseases
- Muscular dystrophies
- Spinal muscular atrophy
- Myasthenia gravis
- Amyotrophic lateral sclerosis
- Poliomyelitis
- Guillain–Barré syndrome
- Hypothyroidism
- Hyperthyroidism
- Cushing syndrome
- Drug-induced:
 - Colchicine
 - Zidovudine (AZT)
 - Penicillamine
 - Ipecac
 - Ethanol
 - Chloroquine
 - Corticosteroids
- Infection:
 - Toxoplasmosis
 - Trichinosis
 - Coxsackievirus
 - HIV, influenza
 - Epstein–Barr virus
- Electrolyte disturbances:
 - Hypokalemia
 - Hypercalcemia
 - Hypomagnesemia
- Vasculitis
- Paraneoplastic neuromyopathy
- Hypereosinophilic myalgia syndrome



TREATMENT

PRE HOSPITAL

- Assess ABCs
- Transport with elevation of head of bed

INITIAL STABILIZATION/THERAPY

- Intubation and mechanical ventilation as required
- Nasogastric (NG) suction to prevent aspiration

- Pneumothorax has been described as a rare occurrence in childhood DM

ED TREATMENT/PROCEDURES

- Elevate head of the bed to prevent aspiration
- Begin high-dose corticosteroids to suppress inflammation and improve muscle weakness
- Avoid triamcinolone and dexamethasone because they may cause a drug-associated myopathy
- Efficacy of prednisone determined by objective increase in muscle strength, not change in CK levels
- Some clinicians start glucocorticoid sparing immunosuppressive medications at onset, others reserve these agents for failure to respond to corticosteroids
- Azathioprine and methotrexate are used with limitations based on side-effect profiles
- Cyclosporine and monoclonal antibody therapies have been used but with limited success
- Do not base treatment decisions solely upon CPK level

MEDICATION

First Line

- Prednisone: 60 mg/d PO (peds: 1–2 mg/kg/d PO) (in severe illness consider methylprednisolone pulse 1,000 mg/d for 3 days):
 - Length of treatment and taper individualized to clinical response and normalization of CK

Second Line

- Methotrexate: 15–25 mg PO per week (peds: 15 mg/m²/wk PO not > 25 mg)
- Azathioprine: Start at 50 mg/d then in 2 wk, increase by 50 mg until a dose of 1.5 mg/kg/d.
 - After 3 mo, may increase dose to 2.5 mg/kg/d if tolerated
- Intravenous immunoglobulin (IVIG), plasmapheresis, and cyclosporine are also used by some rheumatologists



FOLLOW-UP

DISPOSITION

Admission Criteria

- Respiratory insufficiency
- Aspiration pneumonia
- Profound muscle weakness

- Weakened cough mechanisms
- Pharyngeal dysfunction
- CHF

Discharge Criteria

- Well-appearing patients with no respiratory dysfunction and no risk for aspiration
- Patients who can take oral corticosteroids and immunosuppressive agents as outpatients

Issues for Referral

Consultation with a rheumatologist should be made when the diagnosis is suspected for assistance with definitive diagnosis and further treatment.

FOLLOW-UP RECOMMENDATIONS

- Compared to the general population, the incidence of malignant conditions appears to be increased in patients with DM (but not in those with PM)
- A complete annual physical exam with pelvic, breast, and rectal exams; urinalysis; CBC; blood chemistry tests; and a chest film are often recommended for cancer surveillance in patients with a history of DM

PEARLS AND PITFALLS

- The diagnosis of an inflammatory myopathy is largely clinical supported by selected lab testing and muscle biopsy
- Most patients improve with therapy, and many make a full functional recovery, which is often sustained with maintenance therapy
- Up to 30% may be left with some residual muscle weakness
- It is important to keep in mind that relapses may occur at any time despite successful response to therapy

ADDITIONAL READING

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- Casciola-Rosen L, Mammen AL. Myositis autoantibodies. *Curr Opin Rheumatol*. 2012;24:602–608.
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- Longo DL, Kasper DL, Jameson JL, et al. Polymyositis, dermatomyositis, and inclusion body myositis: Introduction. In *Harrison's Principles of Internal Medicine*. 18th ed. New York, NY: McGraw-Hill; 2012:2103–2103.

- Wedderburn LR, Rider LG. Juvenile dermatomyositis: New developments in pathogenesis, assessment and treatment. *Best Pract Res Clin Rheumatol*. 2009;23:665–678.



See Also (Topic, Algorithm, Electronic Media Element)

- Hypokalemia
- Hypothyroidism
- Myasthenia Gravis
- Systemic Lupus Erythematosus



CODES

ICD9

- 710.3 Dermatomyositis
- 710.4 Polymyositis

ICD10

- M33.20 Polymyositis, organ involvement unspecified
- M33.90 Dermatopolymyositis, unspecified, organ involvement unspecified
- M33.92 Dermatopolymyositis, unspecified with myopathy

DIABETES INSIPIDUS

Melissa H. White • Carolyn Maher Overman



BASICS

DESCRIPTION

- Disorder in which large volumes of dilute urine are excreted (polyuria) as an inappropriate response to arginine vasopressin (AVP)
- Polyuria defined as >3 L in 24 hr
- Often characterized by excessive fluid intake (polydipsia)
- 2 types:
 - Central diabetes insipidus (DI, CDI; failure or deficiency of AVP release):
 - 4 types:
 - No AVP to release (loss or malfunction of posterior pituitary neurons)
 - Defective osmoreceptors—release AVP only in response to severe dehydration
 - Elevated threshold for AVP release
 - Subnormal amount of AVP released
 - Familial cases have been reported (autosomal dominant).
 - Nephrogenic DI (lack of renal response to AVP):
 - Differentiate from primary polydipsia.
 - Some cases are X-linked recessive in males.

ETIOLOGY

- Central DI:
 - Any condition that disrupts the osmoreceptor-hypothalamus-hypophyseal axis:
 - Highest incident in ages 10–20 yr
 - Trauma (skull fractures, hemorrhage)
 - Pituitary or hypothalamic surgery
 - CNS neoplasm: DI can be considered a tumor marker:
 - Pituitary adenomas
 - Craniopharyngiomas
 - Germinomas
 - Pinealomas
 - Meningiomas
 - Metastatic tumors:
 - Leukemia/lymphoma
 - Granulomatous:
 - Histiocytosis

- Sarcoidosis
 - Congenital CNS defects
 - CNS infections (e.g., meningitis, encephalitis)
 - Pregnancy (Sheehan syndrome)
 - Idiopathic (autoantibodies, occult tumor)
 - Wolfram syndrome (DI, DM, optic atrophy, deafness)
 - Ethanol
- Nephrogenic DI:
 - Any condition that disrupts the kidney:
 - Congenital renal disorders
 - Obstructive uropathy
 - Renal dysplasia
 - Polycystic kidney disease
 - Systemic disease with renal involvement
 - Sickle cell disease
 - Sarcoidosis
 - Amyloidosis
 - Drugs:
 - Amphotericin
 - Phenytoin
 - Lithium (most common and persists past discontinuation of drug)
 - Aminoglycosides
 - Methoxyflurane
 - Demeclocycline
 - Electrolyte disorders:
 - Hypercalcemia
 - Hypokalemia

Pregnancy Considerations

- Transient in the 2nd trimester:
 - Unclear etiology, but there is an increase of circulating vasopressinase.
 - Leads to a decrease in AVP and transient DI
 - Watch patient closely during anesthesia and periods of water restriction.
 - Typically clears after 2–6 wk after delivery
 - Desmopressin (DDAVP) resists this vasopressinase.
- Sheehan syndrome may cause DI.



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Polyuria (up to 16–24 L/d of urine):
 - Note the voiding frequency.
- Polydipsia (often craves cold fluids):
 - Note the amount of PO fluid intake per day.
- Drug ingestion
- Signs and symptoms of hypothalamic tumors:
 - Headache
 - Visual disturbances
 - Growth disturbances
 - Obesity
 - Hyperpyrexia
 - Sleep disturbances
 - Sexual precocity
 - Emotional disturbances

Physical-Exam

- Dehydration
- Cachexia
- Head trauma
- Visual field defects
- Seizures

Pediatric Considerations

- Polyuria and polydipsia may not be recognized by caregivers until symptoms of dehydration develop.
- In neonates:
 - Often present at birth
 - If unrecognized, dehydration and hypernatremia may cause permanent CNS damage.
- In infants:
 - Irritability
 - Poor feeding/weight loss
 - Constipation
 - Growth failure
 - Intermittent high fever
 - Abnormal behavior (hyperactivity, restlessness, excessive crying)
- In children:
 - Enuresis
 - Difficulty with toilet training

ESSENTIAL WORKUP

- Clinical diagnosis in the ED:

- Elevated serum sodium concentration
- Copious amounts of dilute urine
- History:
 - Usually an increased amount of PO fluid intake per day
 - Voiding frequency
 - Medication use history
- Physical exam
- Labs below

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Urinalysis:
 - Specific gravity will be low.
- Serum and urine osmolality:
 - High serum osmolality
 - Low urine osmolality
- Electrolytes, BUN, creatinine, and glucose:
 - Hyponatremia
 - Hypercalcemia
 - Hypokalemia
- CBC:
 - Anemia may be a sign of a neoplasm.
- Serum and urine AVP tests are expensive and unnecessary in the ED.

Imaging

- As needed to evaluate for trauma or search for neoplasm
- CXR
- CT of brain
- MRI of pituitary axis is usually outpatient.

Diagnostic Procedures/Surgery

Water deprivation test (dehydration test):

- Unnecessary in the emergency setting
- Can be dangerous in cases of hypotension or small children
- Performed as a confirmatory test for those receiving treatment
- Measures urine and plasma osmolality after fluid restriction
 - Urine osmo < 300 is significant for DI
 - Desmopressin is administered
 - Central DI—urine osmo increased by $> 50\%$
 - Nephrogenic DI—urine osmo increased by $< 50\%$
 - Further testing is needed if urine osmo 300–800

- Primary polydipsia if urine osmo > 800

DIFFERENTIAL DIAGNOSIS

- Primary water deficit:
 - Inadequate access to free water
 - Increased insensible water loss (e.g., premature infants)
 - Inadequate breast-feeding
- Primary sodium excess:
 - Excessive sodium bicarbonate during resuscitation
 - Hypernatremic enemas
 - Ingestion of seawater
 - Hypertonic saline administration
 - Accidental substitution of salt (sodium chloride) for glucose in infant formulas
 - Intentional salt poisoning
 - High breast milk sodium
- Primary polydipsia (psychogenic polydipsia):
 - Solute-induced polyuria
 - Diuretic use
 - Resolving acute renal failure
 - Osmotic diuresis
 - Uncontrolled *DM*



TREATMENT

PRE HOSPITAL

- ABCs
- Immobilize if trauma is suspected.
- Serum blood glucose
- IV access and fluids if signs of dehydration exist
- Control seizures according to medical direction guidelines.

INITIAL STABILIZATION/THERAPY

- Manage ABCs.
- Manage traumatic injuries accordingly.
- High index of suspicion for head trauma

ED TREATMENT/PROCEDURES

- Correction of hypotension:
 - Use of 0.9% NaCl is indicated for shock.
 - Intravascular losses represent only about 1/12 of total water losses.
- Central DI (vasopressin deficient):
 - AVP (aqueous vasopressin):

- Half-life is too short.
- May induce coronary vasospasm
- Used only for dehydration test
- Lysine vasopressin (lypressin):
 - Can be given intranasally
 - Frequent instillation needed
- Desmopressin:
 - Drug of choice to control symptoms
 - Administer intranasally, SC, IV, or PO in 2 divided doses as necessary to control polyuria or polydipsia.
 - Caution in postoperative patients as cerebral edema may develop
- Chlorpropamide (Diabinese):
 - Enhances effect of vasopressin at renal tubule
 - May stimulate AVP release
 - Useful only in partial CDI
 - Clofibrate stimulates the release of endogenous vasopressin.
- Nephrogenic DI:
 - Diuretics:
 - Induce natriuresis
 - Thiazides 1st line
 - Amiloride often used in combination with thiazides
 - Dietary sodium restriction
 - Restrict solutes and avoid excessive drinking to prevent water intoxication.
 - Avoid alcohol (especially beer) intake.
 - Check daily weights.
 - NSAIDs (indomethacin)
- Parenteral correction of initial water deficit in cases where PO is not an option:
 - Usually only in symptomatic hypernatremic cases
 - For fluid replacement, refer to “Hypernatremia.”

MEDICATION

- Aqueous AVP: 5–10 U SC in the unconscious patient from head trauma or postoperative
- Amiloride: 2.5–10 mg PO BID
- Chlorpropamide (Diabinese): 200–500 mg PO daily
- Clofibrate (Atromid-S): 500 mg PO q6h
- Desmopressin: 10–20 µg/d intranasally; 1–3 µg/d SC or IV; 0.1–1.2 µg/d PO
- Hydrochlorothiazide (HCTZ): 50 mg PO daily (peds: 2–4 mg/kg QD–BID)
- Lypressin nasal spray: 1–2 nasal spray TID–QID as needed



FOLLOW-UP

DISPOSITION

Admission Criteria

- AMS
- Seizure
- Severe dehydration
- Electrolyte abnormalities
- Associated trauma
- Patients requiring DDAVP testing or a trial of water restriction

Discharge Criteria

- Known diagnosis of DI
- Stable electrolytes
- Adequately hydrated

FOLLOW-UP RECOMMENDATIONS

Referral to specialist depends on underlying etiology of DI.

PEARLS AND PITFALLS

- Check urine osmolality and consider DI in polyuria.
- Central DI will typically respond to desmopressin.
- Nephrogenic DI will not respond to ADH:
 - Treat the underlying electrolyte abnormality, discontinue concerning drugs, and consult nephrology for further management.

ADDITIONAL READING

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- Makaryus AN, McFarlane SI. Diabetes insipidus: Diagnosis and treatment of a complex disease. *Cleve Clin J Med*. 2006;73:65–71.



See Also (Topic, Algorithm, Electronic Media Element)

- Head Trauma

- Hyponatremia



CODES

ICD9

- 253.5 Diabetes insipidus
- 588.1 Nephrogenic diabetes insipidus

ICD10

- E23.2 Diabetes insipidus
- N25.1 Nephrogenic diabetes insipidus

DIABETES MELLITUS, JUVENILE

Madeline M. Joseph



BASICS

DESCRIPTION

- Decrease in effective circulating insulin
- Increase in counter regulatory hormones including glucagon, catecholamines, cortisol, and growth hormone
- Hyperglycemia owing to:
 - Decreased peripheral glucose utilization
 - Increased hepatic gluconeogenesis
- Hyperosmolality and osmotic diuresis due to hyperglycemia
- Ketoacidosis produced by increased lipolysis, with ketone body (β -hydroxybutyrate, acetoacetate) production, causes ketonemia and metabolic acidosis, which is augmented with lactic acidosis from poor tissue perfusion
- Potassium deficit:
 - Intracellular shifts into extracellular space owing to hydrogen ion exchange
 - Loss from osmotic diuresis

ETIOLOGY

Mechanism:

- Immune-mediated pancreatic islet β -cell destruction
- The overall incidence has been increased worldwide by 2–5% over the past 20 yr.
- Precipitating events leading to diabetic ketoacidosis (DKA):
 - Infection, often minor acute illness such as virus, group A streptococcal pharyngitis, or UTI
 - Stress
 - Endocrine: Pregnancy, puberty, hyperthyroidism
 - Psychiatric disorders, including eating disorders
 - Medication noncompliance, inappropriate interruption of insulin pump therapy, or treatment error
- Risk factors for cerebral edema:
 - Attenuated rise in measured serum sodium during DKA therapy (unrelated to the volume or sodium content of IV fluid or rate of change in serum glucose)
 - Bicarbonate treatment for acidosis correction
 - Hypocapnia
 - Increased serum urea nitrogen
 - No association with degree of hyperglycemia
 - Demographic factors that have been associated with an increased risk of

cerebral edema include younger age, longer duration of symptoms, and new onset diabetes mellitus. These factors are also associated with increased risk of severe DKA.



DIAGNOSIS

SIGNS AND SYMPTOMS

- Polydipsia
- Polyuria (may have good urine output despite dehydration)
- Nocturia
- Polyphagia
- Malaise, weight loss
- DKA:
 - Initial presentation in 20–40% of patients
 - Often associated with tachypnea (Kussmaul respiration), tachycardia, orthostatic BP changes
 - Nausea
 - Vomiting
 - Abdominal pain, often resolving with reduction in ketosis/acidosis
 - Hyperpnea
 - Fruity breath secondary to ketones
- Rapid onset of DKA can occur within 7–8 hr with the use of insulin pump therapy if there is an infusion set or insulin delivery malfunction. This is due secondary to lack of long acting insulin to provide a safety net (more commonly seen in female > 10 yr of age).
- Findings with more advanced disease
 - Dehydration, drowsiness, altered mental status, and ultimately, late stage coma and shock
- Cerebral edema:
 - The incidence ranges from 0.87–1.1%.
 - Cerebral edema accounts for 57–87% of all DKA deaths.
 - It typically occurs 4–12 hr after treatment is initiated, but can be presenting (subclinical) before treatment has started.
 - Headache
 - Change in neurologic status, such as drowsiness, irritability, or specific neurologic deficit, such as pupillary responses or cranial nerve palsies
 - Inappropriate slowing in pulse rate
 - Increase in BP
- Hyperglycemic hyperosmolar nonketotic coma:
 - Glucose level of 800–1,200 mg/dL
 - Rare in children; more common in adults

ESSENTIAL WORKUP

For DKA:

- The International Society for Pediatric and Adolescent Diabetes (ISPAD) defines DKA as blood bicarbonate level <15 mmol/L or venous pH <7.3 and hyperglycemia (>200 mg/dL) with related ketonemia or ketonuria.
- DKA classification:
 - Mild DKA: Venous pH <7.2 – 7.3 or $\text{HCO}_3^- <10$ – 15 mmol/L
 - Moderate DKA: Venous pH <7.1 – 7.2 or $\text{HCO}_3^- <5$ – 10 mmol/L
 - Severe DKA: Venous pH <7.1 or $\text{HCO}_3^- <5$ mmol/L
- Hourly vital signs and neurologic checks
- Frequent blood chemistries
- ECG monitoring (in severe DKA) to assess T-waves for evidence of hyperkalemia or hypokalemia
- Accurate fluid input and output. Consider urinary catheterization in patients with impaired level of consciousness.

DIAGNOSIS TESTS & INTERPRETATION

Lab

For DKA:

- Glucose, serum: Hyperglycemia
- Urinalysis:
 - Glycosuria
 - Ketonuria
 - Exclude UTI
- Blood chemistries every 2–4 hr until acidosis has resolved (more frequent as clinically indicated in the more severe cases)
- Electrolytes and venous pH
- Anion gap metabolic acidosis:
 - Potassium—high or normal (artifactual owing to extracellular shift)
 - Serum potassium rises 0.5 mEq/L for each 0.1 decrease in pH
 - Sodium—low or normal (may be artifactual owing to hyperglycemia)
 - Corrected Na (mEq/L) = [measured serum Na (mEq/L) + plasma glucose (mg/dL) – 100] \times 0.016
 - Bicarbonate—low
 - Calculation: $\text{Na} - (\text{Cl} + \text{HCO}_3^-)$
- Serum ketones—elevated. β -hydroxybutyrate (BHOB) is a quantitative test that is available to replace the classic nitroprusside test for serum ketones
- Serum osmolality
- CBC:
 - WBC often elevated owing to stress or infection
- Calcium
- Phosphate

- Cultures as indicated: Group A streptococcal pharyngeal swab, urine, etc.
- Pregnancy test if indicated
- ECG if potassium markedly abnormal

Imaging

- CXR if any suggestion of pneumonia
- Head CT if there are concerns about cerebral edema

DIFFERENTIAL DIAGNOSIS

- Infection (may precipitate):
 - UTI
 - Gastroenteritis
 - Appendicitis
 - Sepsis
- Ingestion (salicylates, alcohols, glycols)
- Diabetes insipidus



TREATMENT

PRE HOSPITAL

For DKA:

- ABCs
- Airway protection
- Establish IV access and initiate fluid therapy.

INITIAL STABILIZATION/THERAPY

For DKA:

- Oxygen
- Cardiac monitor
- IV access and volume resuscitation

ED TREATMENT/PROCEDURES

- For DKA:
 - Fluid replacement:
 - Assume fluid deficit of 10% of body weight.
 - Initial volume expansion with 10–20 mL/kg of 0.9% NaCl or lactated Ringer; may repeat to achieve hemodynamic stability
 - Correct 50% of fluid deficit over 1st 8 hr, remainder over 24–48 hr.
 - Do not give $> 3 \text{ L/m}^2$ over 1st 24 hr.
 - Begin IV insulin infusion after ketoacidosis confirmed:
 - Initial rate of continuous infusion (regular insulin) 0.1 U/kg/h IV
 - Adjust rate to drop serum glucose 50–100 mg/dL/h.

- Add dextrose to infusion fluid when serum glucose < 300 mg/dL.
 - Change to SC insulin when no longer significantly acidotic and able to eat.
- Some clinicians prefer IM route, commonly initially using regular insulin at a dose of 0.1–0.2 U/kg/h.
- Replace potassium and phosphate losses:
 - Verify adequate urine output.
 - Add to fluids as K-acetate (or KCl if acetate not available) and K_3PO_4 in equal amounts.
 - Large doses of K^+ may be necessary; guide therapy by frequent monitoring of K^+ .
- Monitor serum sodium:
 - Risk for cerebral edema if Na^+ fails to rise as glucose falls
- Bicarbonate therapy:
 - Not recommended in most cases since generally it does not alter outcome and it increases risk for cerebral edema with its use
 - Use it with caution in patients with severe acidosis (pH < 6.9) in whom peripheral vasodilation and decreased cardiac contractility may further impair tissue perfusion and in potentially life-threatening hyperkalemia.
- Cerebral edema:
 - Treat cerebral edema as soon as the condition is suspected due to its high mortality and morbidity rates: 21–25% and 10–26%, respectively.
 - Decrease fluid administration rate.
 - Mannitol (0.25–1 g/kg over 20 min): No large studies to date demonstrate definitive beneficial or detrimental effects. Consider its use in patients with signs of cerebral edema before impeding respiratory failure. Dose can be repeated in 2 hr if there is no initial response.
 - Endotracheal intubation and ventilation: Avoid aggressive hyperventilation since it has been associated with poor outcome in DKA-related cerebral edema (similar to that found in head trauma).

MEDICATION

- Insulin drip: Start regular insulin 0.1 U/kg/h IV (some clinicians prefer the IM dosing and route).
- Mannitol: 0.25–1 g/kg IV



FOLLOW-UP

DISPOSITION

Admission Criteria

For DKA:

- ICU:
 - Altered mental status
 - Shock or cardiac dysrhythmia
 - Initial glucose > 700 mg/dL
 - Initial pH < 7
 - Risk factors for cerebral edema (age < 5 yr, prolonged symptoms, high BUN)
- Inpatient unit:
 - Stable new-onset diabetic patients requiring intensive education
 - Patients with ketoacidosis not meeting requirements for ICU care
 - Compliance concerns or other social issues

Discharge Criteria

- Known diabetic patients who respond well to therapy with normalization of glucose, pH, and ketosis
- Tolerating oral fluids
- Reliable parents
- Reliable follow-up within 24 hr including appropriate education

Issues for Referral

- Critically ill
- Persistent abnormal mental status
- Poorly controlled diabetes

FOLLOW-UP RECOMMENDATIONS

- Close follow-up with the primary care physician is important even after the resolution of DKA to ensure appropriate management of the patient's diabetes to prevent further occurrence of DKA.
- Many children with diabetes are followed at comprehensive diabetes centers in collaboration with primary care physician.

PEARLS AND PITFALLS

- Mortality from DKA is predominately related to the occurrence of cerebral edema. Therefore, early and appropriate treatment is of most importance in managing children with DKA.
- In children, avoid using an insulin bolus since it increase the risk of cerebral edema. Recently, some data suggest that starting insulin drip at 0.05 U/kg/h may reduce the risk for rapid fluid shifts and theoretically for cerebral edema.

ADDITIONAL READING

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CODES

ICD9

- 250.01 type I diabetes mellitus [insulin dependent type] [IDDM] [juvenile type], not stated as uncontrolled, without mention of complication
- 250.03 Diabetes mellitus without mention of complication, type I [juvenile type], uncontrolled
- 250.11 type I diabetes mellitus [insulin dependent type] [IDDM] [juvenile type], not stated as uncontrolled, with ketoacidosis

ICD10

- E10.9 Type 1 diabetes mellitus without complications
- E10.10 Type 1 diabetes mellitus with ketoacidosis without coma

- E10.65 Type 1 diabetes mellitus with hyperglycemia

DIABETIC KETOACIDOSIS

Joseph M. Weber

BASICS

DESCRIPTION

Insulin deficiency and excess of counterregulatory hormones (catecholamines, glucagon, growth hormone, and cortisol) resulting in:

- Dehydration (osmotic, hyperglycemic, diuresis, and decreased oral intake)
- Acidosis (anion gap metabolic acidosis)
- Ketone formation (unrestrained lipolysis and ketogenesis)
- Hyperglycemia (unrestrained glycogenolysis and gluconeogenesis)
- Electrolyte disturbances (hypokalemia, hypo/hyponatremia, hypophosphatemia)

ETIOLOGY

- Medication noncompliance (> 50%)
- New-onset diabetes (type I or II)
- Underlying medical illness (increased counterregulatory hormones and insulin resistance):
 - Infectious process
 - MI
 - GI bleed
 - CNS event
- Pregnancy (relative insulin deficiency and counterregulatory hormone excess)
- Medications (protease inhibitors and atypical antipsychotics: Olanzapine, clozapine)
- Alcohol abuse

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Medication noncompliance
- Polyuria, polydipsia
- Weakness
- Abdominal pain, nausea, vomiting
- Altered mental status
- Chest pain
- Febrile illness

Physical-Exam

- Tachycardia
- Hypotension (dehydration, sepsis)
- Tachypnea (hyperpnea)
- Kussmaul respirations
- Hyperthermia/hypothermia (coexisting infection)
- Dehydration:
 - Poor skin turgor
 - Dry mucous membranes
- Odor of ketones on breath
- Diffuse abdominal tenderness

ESSENTIAL WORKUP

- Diagnostic criteria:
 - pH < 7.3 with ketonemia
 - Bicarbonate < 15 mEq/L
 - Glucose > 250 mg/dL
- Bedside glucose measurement
- Venous blood gas
- Urine dip for ketones
- Serum electrolytes, glucose, BUN/creatinine
- Search for precipitating cause

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Serum glucose measurement:
 - Confirm bedside test.
- Electrolyte measurement:
 - Increased anion gap metabolic acidosis: $[\text{Na} - (\text{Cl} + \text{HCO}_3)] > 12$
 - Sodium:
 - Pseudohyponatremia (from hyperglycemia) correction factor; add 1.6 mEq/L to the measured sodium for every 100 mg/dL of blood glucose > 100 mg/dL.
 - Potassium:
 - Initial serum level may be normal to high owing to extracellular shift as compensation for acidosis.
 - Total body deficit usually 3–5 mEq/kg
 - As acidosis improves, for every 0.1 increase in the pH, serum potassium decreases 0.5 mEq/L.
 - Can drop precipitously with insulin and fluids
 - Bicarbonate:
 - Usually < 15 mEq/L

- May be higher owing to coexisting volume contraction alkalosis
- BUN/creatinine:
 - Usually shows prerenal azotemia owing to dehydration
- Serum ketones:
 - Must be present to make diagnosis of DKA.
 - β -Hydroxybutyrate is the predominant ketoacid, but acetoacetate and acetone are also present:
 - β -Hydroxybutyrate is not measured by most hospital serum and urine ketone tests (nitroprusside reaction measures only acetoacetate and acetone), thus there is a theoretical risk of missing the presence of ketones using these tests.
 - Urine ketone dip test (UKDT) is 97% sensitive for presence of serum ketones and a negative UKDT has a negative predictive value of 100% in ruling out the presence of DKA.
 - Point-of-care capillary testing for β -hydroxybutyrate is 98% sensitive for serum ketones:
 - May be used with capillary glucose testing in triage to detect DKA early in the ED course.
- Urinalysis:
 - Ketonuria, glucosuria
 - Pregnancy (UhCG)
- Venous blood gas:
 - Essential to assess patient's pH
 - pH correlates well with arterial pH
 - Avoids need for repeated arterial sticks
 - ABG should be performed if oxygenation/ventilation needs assessment.
- Serum osmolarity:
 - May be measured in the lab and calculated
 - Calculated: $2(\text{Na}) + \text{glucose}/18 + \text{BUN}/2.8$ (normal 285–300 mOsm/L)
 - Significant hyperosmolarity > 320
- CBC:
 - Leukocytosis may be present without infection.
 - If left shift in differential, suspect infection.
- Other lab tests:
 - Amylase: Elevation is nonspecific in DKA
 - Lipase: Elevation specific for pancreatitis
 - Calcium, Mg, Phosphate: All usually decreased as is K^+

Imaging

- CT head to rule out other causes of altered mental status.
- CXR if pneumonia suspected as precipitant or hypoxia present
- EKG to rule out ischemia as a precipitant and look for signs of hyper/hypo K^+

DIFFERENTIAL DIAGNOSIS

- Other causes of anion gap acidosis
- Use ACAT MUD PILES mnemonic:
 - _ Alcoholic ketoacidosis
 - _ Carbon monoxide/cyanide
 - _ Aspirin
 - _ Toluene
 - _ Methanol
 - _ Uremia
 - _ Diabetic ketoacidosis
 - _ Paraldehyde
 - _ Iron/isoniazid
 - _ Lactic acidosis
 - _ Ethylene glycol
 - _ Starvation/sepsis
- Hyperglycemic hyperosmolar nonketotic syndrome



TREATMENT

PRE HOSPITAL

- Fluid bolus often initiated in field
- Quantify amount given by paramedics to guide further ED fluids.

INITIAL STABILIZATION/THERAPY

- ABCs for patients with altered mental status
- Coma cocktail for AMS: Naloxone, thiamine, blood sugar
- 0.9% NS bolus for hypotension/tachycardia

ED TREATMENT/PROCEDURES

- Cardiac monitor and pulse oximetry for patients with abnormal vitals
- Fluids:
 - _ Average adult water deficit is 100 mL/kg (5–10 L).
 - _ Initial 1–2 L bolus of 0.9% NS to restore intravascular volume over 1st hr.
 - _ If corrected serum sodium is low, continue with 0.9% NS, giving 1–2 more liters over the next 2–4 hr.
 - _ If corrected serum sodium is normal or elevated, use 0.45% NS giving 1–2 more liters over next 2–4 hr.
 - _ Be careful to avoid fluid overload in patients with cardiac disease.
 - _ Avoid precipitous falls in serum sodium/osmolality, as this may contribute to cerebral edema.
 - _ Total fluid replacement should take 24–36 hr.
- Insulin:

- Reverses ketogenic state and down-regulates counterregulatory hormones
- Administered as continuous IV infusion of regular insulin at 0.1 U/kg/h:
 - Adjust infusion in response to changes in glucose and anion gap
- Continue until pH >7.3 and resolution of anion gap
- Serum glucose will fall sooner than resolution of acidosis and should be kept >250 mg/dL with glucose-containing fluids such as D₅ 45% NS.
- Potassium:
 - Administration is essential.
 - Total body deficit of 3–5 mEq/kg
 - Will drop precipitously with administration of fluid and insulin
 - Administer KCl, 10 mEq/h IV once renal function is established and K⁺ is known to be <5.5 mEq/L.
 - May need to give up to 20–40 mEq/h IV in cases where initial K⁺ is <3.5 mEq/L
 - In hypokalemic patients, insulin therapy should be delayed until K⁺ is >3.5 mEq/L.
 - Should measure q1–2h during 1st 4–6 hr of therapy
- Bicarbonate:
 - No studies have shown clinical benefit in DKA, and its routine use is not advocated.
 - Complications include hypokalemia, alkalosis, cerebral acidosis, and edema.
 - Some advocate its use for pH <6.9 with cardiac instability.
- Phosphate:
 - Not routinely replaced during initial ED therapy
 - May supplement if <1 mg/dL and symptomatic muscle weakness.
 - Administer as potassium phosphate.
- Magnesium:
 - May supplement if <1.2 mg/dL
 - Administer 2 g MgSO₄ IV over 1 hr.
- Identify and treat precipitating cause.

Pediatric Considerations

- Fluids:
 - Average fluid deficit is 100 mL/kg.
 - Initial 10–20 mL/kg bolus of 0.9% NS to restore intravascular volume
 - May repeat once in severely dehydrated children
 - Should not exceed 40–50 mL/kg of fluid in 1st 4 hr of therapy
 - Replace remainder of deficit at 1.5–2 times maintenance over 24–36 hr.
 - Overzealous fluid administration is thought to contribute to cerebral edema.
- Cerebral edema:
 - Occurs in 1–2% of children with DKA

- Causes 31% of deaths associated with DKA
- Exact causes unclear
- Suspect with coma, fluctuating mental status, bradycardia, HTN, severe headache, decreased urine output, or quickly falling corrected Na^+ or osmolality to below normal levels
- Mannitol: 0.25–1 g/kg IV over 30 min should be given immediately and can be repeated hourly.
- Fluid rate should be decreased and other supportive measures instituted.

MEDICATION

- D_{50} : 1 amp (25 g) of 50% dextrose IVP (peds: 2–4 mL/kg D_{25})
- Insulin (100 U regular insulin in 100 mL NS) run at 0.1 U/kg/h
- MgSO_4 : 2 g of 20% solution



FOLLOW-UP

DISPOSITION

Admission Criteria

- ICU admission for pH < 7, altered mental status, serious comorbid illness, and extremes of age (< 2 yr or > 60 yr)
- Monitored unit for moderate DKA (pH 7.01–7.24) with CHF or cardiac history
- General floor (nurses skilled with insulin infusions) for moderate DKA without comorbidities
- Observation unit (< 23 hr admission) for mild DKA (pH 7.25–7.30) without precipitating illness

Discharge Criteria

- Resolution of anion gap acidosis
- Tolerating PO fluids
- No evidence of precipitating event
- Clear instructions on home insulin regimen
- Close primary care follow-up arranged

PEARLS AND PITFALLS

- Decreasing or discontinuing insulin drip when glucose normalizes is a pitfall. Insulin should only be stopped when pH improves and anion gap normalizes.
- Failure to replete potassium is a pitfall.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Hyperosmolar Syndrome



CODES

ICD9

- 250.10 type II diabetes mellitus [non-insulin dependent type] [NIDDM type] [adult-onset type] or unspecified type, not stated as uncontrolled, with ketoacidosis
- 250.11 type I diabetes mellitus [insulin dependent type] [IDDM] [juvenile type], not stated as uncontrolled, with ketoacidosis
- 250.12 Diabetes with ketoacidosis, type II or unspecified type, uncontrolled

ICD10

- E10.10 Type 1 diabetes mellitus with ketoacidosis without coma
- E10.11 Type 1 diabetes mellitus with ketoacidosis with coma
- E13.10 Oth diabetes mellitus with ketoacidosis without coma

DIALYSIS COMPLICATIONS

Christopher B. Colwell

BASICS

DESCRIPTION

Dialysis complications may be:

- Vascular access related (infection, bleeding)
- Nonvascular access related (hypotension, hyperkalemia)
- Peritoneal (abdominal pain, infection)

ETIOLOGY

- Vascular access related:
 - _ Infections:
 - Infections (largely access related or peritonitis) are a major cause of death in dialysis patients.
 - Often caused by *Staphylococcus aureus*
 - Can present with signs of localized infection or systemic sepsis
 - Can also present with minimal findings
 - _ Thrombosis or stenosis:
 - Often presents with loss of bruit or thrill over access site
 - Must be addressed quickly (within 24 hr) to avoid loss of access site
 - _ Bleeding:
 - Can be life-threatening
 - Aneurysm
- Nonvascular access related:
 - _ Hypotension:
 - Most common complication of hemodialysis
 - After dialysis: Often owing to acute decrease in circulating blood volume
 - During dialysis: Hypovolemia (more commonly) or onset of cardiac tamponade owing to compensated effusion suddenly becoming symptomatic after correction of volume overload
 - MI, sepsis, dysrhythmias, hypoxia
 - Hemorrhage secondary to anticoagulation, platelet dysfunction of renal failure
 - _ Shortness of breath:
 - Volume overload
 - Development of dyspnea *during* dialysis owing to tamponade, pericardial effusion, hemorrhage, anaphylaxis, pulmonary embolism, air embolism

- Chest pain:
 - Ischemic:
 - Dialysis patients are often at high risk for having atherosclerotic disease
 - Dialysis is an acute physiologic stressor with transient hypotension and hypoxemia that increases myocardial oxygen demand.
 - Pleuritic:
 - Pericarditis, pulmonary embolism
- Neurologic dysfunction: Disequilibrium syndrome:
 - Rapid decrease in serum osmolality during dialysis leaves brain in comparatively hyperosmolar state.
- Peritoneal:
 - Peritonitis:
 - Owing to contamination of peritoneal dialysate or tubing during exchange
 - *S. aureus* or *Staphylococcus epidermidis* (70%)
 - Perforated viscus with abdominal pain that can be severe, fever, brown or fecal material in effluent, or localized tenderness
 - Fibrinous blockage of catheter resulting from infection or inflammation



DIAGNOSIS

SIGNS AND SYMPTOMS

- Vascular access related:
 - Bleeding from puncture sites
 - Loss of bruit in graft
 - Local infection, cellulitis, fever
 - Decreased sensation and strength distal to access
 - New or increasing size mass adjacent to access site
- Nonvascular access related:
 - Hypotension before, during, or after procedure
 - Palpitations
 - Syncope
 - Chest pain:
 - Ischemic
 - Pleuritic
 - Hemorrhage:
 - GI
 - Pleural
 - Retroperitoneal
 - Shortness of breath:

- Neurologic symptoms (disequilibrium syndrome):
 - Headache
 - Malaise
 - Seizures
 - Coma
- Peritoneal:
 - Abdominal pain
 - Cloudy dialysis effluent
 - Nausea and vomiting
 - Exudates or inflammation at insertion site of Tenckhoff catheter

ESSENTIAL WORKUP

- Careful physical exam:
 - Complete set of vital signs including auscultated BP, pulse, respiratory rate, accurate temperature, and pulse oximetry
 - Careful physical exam for occult infectious sources (odontogenic, perirectal abscess)
 - Auscultation of lungs for evidence of infection (rhonchi) or volume overload (rales)
 - Search for other evidence of volume overload (edema)
 - Careful cardiac exam including listening for murmurs or rubs
- EKG: Look for signs of electrolyte balance or conduction disturbances.
- Infection:
 - Blood and wound cultures
 - Cell count, Gram stain, culture of peritoneal fluid
- Bleeding:
 - CBC to evaluate anemia and platelet count
 - Coagulation studies
- Chest pain or shortness of breath:
 - Chest radiograph
 - ABG
 - EKG, cardiac enzymes (if appropriate, based on history)
- Neurologic dysfunction: CT of brain for intracranial hemorrhage

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Glucose, electrolytes, BUN, and creatinine
- CBC

Imaging

- ECG for suspected:
 - Hyperkalemia

- Pericarditis
- Effusion
- Tamponade
- US of access for possible clotted graft or fistula
 - ECHO to assess for pericardial effusion/tamponade
- Peritoneal cathetergram for blockages
- CT scan for pulmonary embolism:
 - Dialysis patients are at risk for both bleeding and clotting problems.
 - Problematic in renal insufficiency owing to contrast dye load:
 - Can be done in renal failure, but contrast is then a fluid bolus and may need to be dialyzed off
 - Communicate contrast load to renal team, as dialysis may need to occur for longer-than-normal duration.

DIFFERENTIAL DIAGNOSIS

- Hypotension:
 - Sepsis
 - Cardiogenic shock, acute MI, tamponade, primary dysrhythmias
 - Electrolyte abnormalities leading to dysrhythmias (hyperkalemia and hypokalemia)
 - Embolism: Air or pulmonary
 - Hypovolemia
 - Vascular instability: Autonomic neuropathy, drug related, dialysate related
- Neurologic complications:
 - Cerebrovascular accident
 - Disequilibrium syndrome
 - Hyperglycemia or hypoglycemia
 - Hyponatremia or hypernatremia
 - Hypoxemia
 - Intracranial bleed
 - Meningitis or abscess
 - Uremia
- Peritoneal complications:
 - Peritonitis
 - Hernia incarceration
 - Perforated viscus
 - Acute abdominal process: Appendicitis, cholecystitis



TREATMENT

PRE HOSPITAL

ALERT

- Do not perform IV access and BP measurement in extremity with functioning AV graft or fistula.
- Run IV fluids slowly and keep to min., if possible.
- Administer furosemide in pulmonary edema (anuric patients: Use high doses ≤ 200 mg).

INITIAL STABILIZATION/THERAPY

- Check airway, breathing, and circulation.
- Vascular access related:
 - Bleeding:
 - Firm pressure to site(s)
 - Do not totally occlude access; may cause clotting.
 - Will likely need pressure applied for at least 5–10 min to stop even minor bleeding
 - Document presence or absence of thrill after pressure was applied.
 - Apply Gelfoam.
- Nonvascular access related:
 - Hypotension:
 - Search for underlying cause.
 - Vasopressors, fluids
 - Shortness of breath:
 - Preload and afterload reduction with nitrites and ACE inhibitors.
 - Attempt diuresis if fluid overload is suspected cause.
 - Arrange for dialysis.
 - Hyperkalemia:
 - Administer IV calcium, bicarbonate, insulin, and glucose when appropriate (see “Hyperkalemia”).
 - Monitor cardiac rhythm.
 - Administer ion-exchange resin (Kayexalate).
 - Arrange for dialysis.
 - Neurologic complications:
 - Administer naloxone, thiamine, dextrose (or Accu-Chek) for altered mental status.
 - Control seizures with benzodiazepines.

ED TREATMENT/PROCEDURES

- Vascular access related:
 - Infection:
 - Initiate antistaphylococcal IV antibiotics.
 - Clotted access:
 - Analgesia
 - Warm compresses

- Vascular surgery consult
- Hemorrhage:
 - Control bleeding.
 - Correct coagulopathies.
 - Administer IV fluids and blood products.
- Nonvascular access related:
 - Electrolyte imbalances:
 - Treat hypercalcemia or hypermagnesemia with saline infusion if tolerated (dilution).
 - Diuresis with furosemide after preload and afterload reduction (nitroglycerin, enalapril)
 - Arrange for dialysis.
 - Volume overload:
 - Attempt diuresis with nitrites and furosemide.
 - Arrange for dialysis.
 - Pericardial effusion or tamponade:
 - Emergent pericardiocentesis may be necessary in unstable patient.
 - Arrange for dialysis.
 - Acute MI:
 - Thrombolytics or angioplasty if patient is appropriate candidate
 - Nitrates to decrease myocardial workload
 - Disequilibrium syndrome:
 - Rule out other causes of altered mental status.
 - Generally resolves over time
- Peritoneal:
 - Peritonitis: IV or intraperitoneal antibiotics
 - Culture catheter or tunnel infection, visible exudates:
 - Oral antibiotics (antistaphylococcal)
 - If recurrent or tunnel, may need to be unroofed
 - Meticulous site care
 - Perforated viscous:
 - IV antibiotics
 - Surgical consultation

MEDICATION

- Calcium gluconate: 1 g slowly IV (cardioprotective in hyperkalemia with widened QRS complex)
- Cefazolin: 1 g IV or IM followed by 250 mg/2 L bag for 10 days (peritonitis)
- Captopril: 25 mg sublingually
- Dextrose D₅₀W: 1 amp: 50 mL or 25 g (peds: dextrose D₂₅W: 2–4 mL/kg)IV
- Dopamine: 2–20 µg/kg/min IV
- Enalapril: 1.25 mg IV
- Furosemide: 20–100 mg IV (may require doses of ≥ 30 mg to effect diuresis in

chronic renal failure)

- Insulin: 5–10 U regular insulin IV (with D₅₀ for hyperkalemia)
- Naloxone (Narcan): 2 mg (peds: 0.1 mg/kg) IV or IM initial dose
- Nitroglycerin: 0.4 mg sublingually; 5–20 µg/min IV
- Sodium bicarbonate: 1 mEq/kg up to 50–100 mEq IV PRN
- Sodium polystyrene sulfonate (Kayexalate): 1 g/kg up to 15–60 g PO or 30–50 g retention enema q6h PRN (for hyperkalemia)
- Thiamine (vitamin B₁): 100 mg (peds: 50 mg) IV or IM
- Tobramycin: 1.7 mg/kg IV or IM followed by 10 mg/2 L bag for 10 days (peritonitis)
- Vancomycin: 1 g IV or IM followed by 50 mg/2 L bag for 10 days (peritonitis)



FOLLOW-UP

DISPOSITION

Admission Criteria

- ICU admission:
 - Severe hyperkalemia
 - Pulmonary edema
 - Volume overload
 - Persistent hypotension
 - Uncontrolled seizures
 - Acute MI
 - Cardiovascular accident
 - Pericarditis
 - Sepsis
 - Peritonitis with toxic or systemic symptoms
- Regular admission:
 - Fever
 - Vomiting
 - Peritonitis without toxic or systemic symptoms
 - Non–life-threatening electrolyte disturbances
 - Inability to provide self-care for continuous ambulatory peritoneal dialysis with antibiotics

Discharge Criteria

- Mild infections of access site
- Same-day surgery for some thrombectomy procedures
- Hemostasis at puncture sites

FOLLOW-UP RECOMMENDATIONS

Most patients on dialysis are followed closely by their nephrologists.

PEARLS AND PITFALLS

- Consider cardiac tamponade in dialysis patients, even when they don't exhibit classic symptoms.
- Always consider hyperkalemia in dialysis patients.
- Infections can have very subtle presentations in dialysis patients and are a common cause of morbidity and mortality
- Early vascular surgery consultation is important for patients with clotted or ruptured access sites

ADDITIONAL READING

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- Zink JN, Netzley R, Erzurum V, et al. Complications of endovascular grafts in the treatment of pseudoaneurysms and stenoses in arteriovenous access. *J Vasc Surg.* 2013;57:144–148.
- Padberg FT Jr, Calligaro KD, Sidawy AN. Complications of arteriovenous hemodialysis access: Recognition and management. *J Vasc Surg.* 2008;48:55S–80S.



See Also (Topic, Algorithm, Electronic Media Element)

- Renal Failure
- Hyperkalemia



CODES

ICD9

- 996.1 Mechanical complication of other vascular device, implant, and graft
- 996.62 Infection and inflammatory reaction due to other vascular device, implant, and graft
- 999.9 Other and unspecified complications of medical care, not elsewhere classified

ICD10

- T80.29XA Infct fol oth infusion, transfuse and therapeutc inject, init
- T80.90XA Unsp comp following infusion and therapeutic injection, init
- T82.9XXA Unspecified complication of cardiac and vascular prosthetic device, implant and graft, initial encounter

DIAPER RASH

Francesco Mannelli



BASICS

DESCRIPTION

- Very common dermatologic disorder of infancy
- Most common in 1st month of life and again at 12–24 mo
- Incidence in adult incontinent patients is reported from 5.7% to more than 42% and appears to be strongly associated with age
- Primary irritant/contact dermatitis:
 - Outer skin layers are broken down, leading to inflammation, impairment of normal skin microflora, and loss of protective barrier function.
 - Increased skin moisture encourages growth of microorganisms on the surface of the skin.
 - Secondary fungal or bacterial infection can cause more severe forms of diaper dermatitis.
- Also known as irritant diaper dermatitis

ETIOLOGY

- Irritants:
 - Moisture:
 - Prolonged overhydration owing to infrequent diaper changes, poorly absorbing diapers or cloth diapers, urinary or faecal incontinence in adults
 - Friction:
 - Diaper rubbing on skin or loose-fitting diaper
 - Chemicals:
 - Prolonged exposure to stool enzymes and urine
 - Scents or moisturizers in wipes or soap
 - Diaper material or adhesive used to hold diaper in place
- Infection:
 - *Candida albicans*:
 - Isolated in up to 80% of infants
 - Overgrowth common after systemic antibiotic use
 - Bacterial
 - Often complication of other causes of dermatitis:
 - *Staphylococcus aureus*, *Streptococcus*, *Escherichia coli* are common; *Peptostreptococcus* and *Bacteroides* may also be encountered.
- Seborrheic diaper dermatitis
- Atopic diaper dermatitis (contact dermatitis)

- Risk factors:
 - Oral thrush
 - Number of previous episodes of diaper rash
 - Duration of use of diapers
 - Diarrhea



DIAGNOSIS

Diagnosis often empiric based on appearance of rash

SIGNS AND SYMPTOMS

History

Child may cry with diaper changes or wiping diaper area or may be irritable.

Physical-Exam

- Irritant:
 - Beefy-red confluent patches with distinct borders at diaper edges, typically sparing skin folds
- Infectious:
 - *Candida*—demarcated erythematous rash with satellite pustules or papules, typically involves skin folds
 - Bacterial—superficial erosions with yellow crust and occasionally bullae
- Seborrheic diaper dermatitis:
 - Lesions with erythematous base and greasy yellow or gray scale
 - Infant will likely have similar lesions on other body surfaces, especially scalp.
- Atopic diaper dermatitis:
 - Similar appearance to irritant dermatitis, but lesions also on other body surfaces such as the face.
- Variations include:
 - Jacquet form—erosive variant with ulcers or erosions with elevated margins usually seen with persistent diarrhea or adult urinary incontinence.
 - Psoriasiform—erythema, silvery surface scales and spared skin folds; also likely to have similar lesions on other body surfaces.
 - Granuloma gluteale infantum—violaceous papules and nodules on the buttocks and in the groin with a self-limited course, resolving in weeks or months, often with residual scarring.

ESSENTIAL WORKUP

- Inquire about diaper-changing habits and urinary and fecal habits.
- Examine other body areas to identify associated rashes.
- Consider child abuse or neglect:

- Child's overall hygiene
- Burns or other trauma

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Lab evaluation usually not necessary for management of diaper dermatitis.
- Bacterial cultures usually not indicated except in complicated cases.
- Skin surface scrapings with KOH prep and/or culture may help distinguish between *Candida* and atypical seborrheic dermatitis:
 - Look for budding yeast and/or pseudohyphae.

DIFFERENTIAL DIAGNOSIS

- Child abuse or neglect
- Infection:
 - Impetigo
 - Scabies
 - Herpes simplex
 - Varicella
 - Congenital syphilis
- Psoriasis
- Atopic dermatitis
- Seborrheic dermatitis
- Papular urticaria
- Bullous pemphigoid
- Epidermolysis bullosa
- Acrodermatitis enteropathica
- Acrodermatitis enteropathica–like eruption
- Langerhans cell histiocytosis



TREATMENT

ED TREATMENT/PROCEDURES

The management of diaper dermatitis should include reducing moisture in the diaper area, minimizing contact with urine and feces and eradicating infectious microorganism

- Environmental adjustments:
 - Education of parents and caregivers is essential:
 - Cleanse skin frequently using cotton balls and water.
 - Wet wipes and talcum powders are not recommended.
 - Frequent diaper changes, up to q1h for neonates and q3–4h for infants and adults.
 - Gentle rinsing of affected area with warm water or saline.

- Avoid harsh soaps or alcohol wipes.
- Leave area uncovered as much as possible; allow time to air dry.
- Highly absorbant diapers have less incidence of diaper rash than cloth diapers.
- Cloth diapers are not recommended for patients with irritant diaper dermatitis.
- New diapers that are “breathable” or contain top sheet of zinc oxide/petroleum and stearyl alcohol lining have been shown to decrease incidence.
- Barrier creams:
 - Many preparations available containing zinc oxide, petroleum, lanolin.
 - Should be applied after each diaper change and continued after rash resolves to minimize recurrence
 - A substantial negative relationship exists between barrier cream use and number of previous episodes of diaper dermatitis.
 - If *Candidal* infection present, apply over antifungal medication.
- Corticosteroids:
 - For moderate to severe cases not responding to other therapy
 - Should not be stronger than 1% hydrocortisone: Anything stronger can cause serious side effects.
 - Discontinue after 3–5 days.
- Antifungals:
 - Nystatin cream, powder, or ointment:
 - Expect improvement in 1–2 days.
 - Ointment best tolerated on macerated skin.
 - Clotrimazole applied topically after diaper change.
 - Miconazole applied topically after diaper change.
 - Lotion is preferred in intertriginous areas.
 - Cream should be applied sparingly to avoid maceration effects.
 - Ciclopirox applied topically after diaper change.
 - Generally continue 1–2 days after clearing
 - Antifungal agent also found to have some antibacterial activity and anti-inflammatory properties.
 - Consider oral agent if concurrent cutaneous or oral candidiasis is present or in recalcitrant case because stool may be colonized with *C. albicans*.
- Antibacterials:
 - Typically concurrent with other therapies if suspicion of bacterial infection
 - Mupirocin (Bactroban) applied after diaper changes
 - Systemic antibiotics rarely needed

MEDICATION

- Ciclopirox 0.77% cream, gel, or suspension: Applied topically BID after diaper change

- Clotrimazole 1% cream: Applied topically BID after diaper change
- Hydrocortisone 0.5–1% topical cream: Applied BID
- Miconazole topical 2% cream: Applied BID after diaper change
- Miconazole nitrate 0.25% ointment: Apply after diaper change and bathing
- Mupirocin 2% ointment or cream (Bactroban): Applied topically 3–5 times daily after diaper changes (for infants > 3 mo of age)
- Nystatin 100,000 U/g cream, powder, or ointment: Apply BID after diaper change



FOLLOW-UP

DISPOSITION

Admission Criteria

- Evidence of child abuse or neglect
- Evidence of sepsis

ADDITIONAL READING

- Adalat S, Wall D, Goodyear H. Diaper dermatitis-frequency and contributory factors in hospital attending children. *Pediatr Dermatol*. 2007;24(5):483–488.
- Adam R. Skin care of the diaper area. *Pediatr Dermatol*. 2008;25(4):427–433.
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- Ravanfar P, Wallace JS, Pace NC. Diaper dermatitis: A review and update. *Curr Opin Pediatr*. 2012;24(4):472–479.
- Van L, Harting M, Rosen T. Jacquet erosive diaper dermatitis: A complication of adult urinary incontinence. *Cutis*. 2008;82(1):72–74.



CODES

ICD9

691.0 Diaper or napkin rash

ICD10

L22 Diaper dermatitis

DIAPHRAGMATIC TRAUMA

Jennifer Cullen



BASICS

DESCRIPTION

- Penetrating injury:
 - Violation of the diaphragm by penetrating object (most commonly stab and gunshot wounds)
 - May involve any portion of diaphragm
 - Smaller defect compared with blunt injuries (more likely to be missed)
- Blunt injury:
 - Increased intra-abdominal or intrathoracic pressure is transmitted to diaphragm, causing rupture.
 - Usually due to motor vehicle crashes
 - Injuries are more commonly left-sided:
 - Left hemidiaphragm has posterolateral embryologic point of weakness.
 - Right hemidiaphragm is protected by liver.
 - Injuries are larger than with penetrating injury (frequently between 5 and 15 cm in length).
- Diaphragmatic defects do not heal spontaneously because of pleuroperitoneal pressure gradient:
 - May exceed 100 cm H₂O during maximal respiratory effort
 - Promotes herniation of abdominal contents through rent in diaphragm and into chest

EPIDEMIOLOGY

Incidence

Uncommon; <1% of all traumatic injuries

ETIOLOGY

- Lateral torso impact is 3 times more likely to result in ipsilateral diaphragmatic rupture than frontal impact.
- Suspect diaphragmatic injury:
 - Penetrating trauma to thoracoabdominal area
 - Injuries that cross plane of the diaphragm



DIAGNOSIS

ALERT

In acute phase, there may be no abdominal visceral herniation:

- This injury may even be missed on initial laparotomy or laparoscopy.

SIGNS AND SYMPTOMS

- Vary depending on whether phase is acute, latent, or obstructive:
 - _ Acute:
 - Tachypnea
 - Hypotension
 - Absent or diminished breath sounds
 - Abdominal distention
 - Bowel sounds in chest
 - _ Latent:
 - Abdominal discomfort from intermittent herniation of abdominal contents into thorax
 - Abdominal pain that is worse postprandially
 - Exacerbated by lying supine
 - Pain radiating to left shoulder
 - Nausea, vomiting, or belching
 - _ Obstructive:
 - Severe abdominal pain
 - Obstipation
 - Nausea, vomiting
 - Abdominal distention
- Strangulated abdominal organs may perforate and spill abdominal contents into chest
- Respiratory compromise, sepsis, and death
- Obstructive injuries may present in delayed fashion

ESSENTIAL WORKUP

CXR may reveal herniated loops of bowel or other abdominal viscera in thorax:

- Pathognomonic finding is presence of nasogastric tube above diaphragm.
- Findings are often nonspecific:
 - _ Elevated hemidiaphragm
 - _ Irregular diaphragmatic contour
 - _ Mediastinal shift away from affected side
 - _ Unilateral pleural thickening or pleural effusion
 - _ Areas of atelectasis or consolidation at bases
 - _ Small hemothorax or pneumothorax
- 50% of initial CXRs may be normal.
- Diagnosis may be difficult in latent phase because of intermittent nature of herniation.
- Contrast studies of GI tract may be helpful.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- If diagnostic peritoneal lavage (DPL) is performed:
 - Red blood cell count of 1,000 RBC/mm³ is considered positive for diaphragmatic injury after penetrating trauma.
 - May provide false-negative result in up to 40% of patients with isolated diaphragmatic injury
- No lab studies confirm or rule out presence of diaphragmatic injury.

Imaging

- CXR is diagnostic in 90% of cases in which herniation is present, but sensitivity is limited in absence of acute hernia.
- GI contrast studies are the most useful in diagnosing chronic herniation of abdominal contents through diaphragm.
- US may be used, particularly on right side with accompanying hepatic herniation.
- Conventional CT is rarely diagnostic and has poor sensitivity.
- New helical and multidetector CT (MDCT) modalities have much more success in diagnosing subtle diaphragmatic injuries.
- MRI is useful in its ability to visualize the diaphragm as a discrete structure, but is not practical in acute settings.

Diagnostic Procedures/Surgery

- Diagnostic pneumoperitoneography:
 - Air is injected through DPL catheter.
 - Pneumothorax on subsequent CXR is diagnostic of diaphragmatic injury.
 - Poorly tolerated by unstable patients and may require chest tube placement.
- Thoracoscopic and laparoscopic exploration may be indicated
 - Especially when suspicion is high despite negative imaging results
 - Facilitates minimally invasive repair

DIFFERENTIAL DIAGNOSIS

- Atelectasis
- Hemothorax
- Pneumothorax
- Pulmonary contusion
- Gastric dilation, intra-abdominal fluid
- Traumatic pneumatocele
- Subdiaphragmatic abscess
- Intrathoracic cyst
- Empyema
- Congenital eventration of the diaphragm



TREATMENT

ALERT

- Herniation of abdominal contents into chest wall may mimic hemothorax or tension pneumothorax
- Bowel sounds in chest may help distinguish
- Be suspicious of diaphragmatic injury with lateral compression of chest:
 - _ Be cautious in placement of needle or tube thoracostomies.
- Fecal thorax has been reported with bowel rupture.

INITIAL STABILIZATION/THERAPY

- Follow advanced trauma life support (ATLS) protocols.
- If respiratory distress is present, immediate placement of a nasogastric tube may decompress herniated abdominal contents.

ED TREATMENT/PROCEDURES

- Palpate within the chest cavity for visceral organs before inserting a chest tube.
- Patients with visceral perforations are septic and need aggressive resuscitation and antibiotic therapy.
- Empiric broad-spectrum antibiotics are indicated in the case of perforated viscera.
- Early surgical intervention is paramount.
- Minimally invasive repair may be possible in selected circumstances

MEDICATION

- Gram-negative aerobes:
 - _ Gentamicin: Adults/peds: 2–5 mg/kg IV initial dose
- Gram-negative anaerobes:
 - _ Clindamycin: 900 mg (peds: 20–40 mg/kg/24h) IV q8h
 - _ Metronidazole: 1 g (peds: 15 mg/kg) IV load, then 500 mg (peds: 7.5 mg/kg) IV q6h
- Both aerobic and anaerobic:
 - _ Ampicillin/sulbactam: 1.5–3 g (peds: 100–400 mg/kg/24h) IV q6h
 - _ Cefotetan: 2 g (peds: 40–80 mg/kg/24h) IV q12h
 - _ Cefoxitin: 2 g (peds: 80–160 mg/kg/24h) IV q12h
 - _ Ticarcillin/clavulanate: 3.1 g (peds: 50 mg/kg/dose) IV q6h



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients with suspicion for diaphragmatic injury must be admitted to trauma

surgery.

- Patients should be admitted to the monitored or ICU setting.

Discharge Criteria

Patients with diaphragmatic injury or any significant suspicion for it must not be discharged from ED.

FOLLOW-UP RECOMMENDATIONS

Patients with diaphragmatic injuries s/p repair must be followed by trauma surgeon to monitor for recurrence.

Pediatric Considerations

- Pediatric anatomic differences predispose to diaphragmatic injury via less severe mechanisms:
 - Thinner abdominal wall
 - More horizontal orientation of diaphragm
 - Greater cartilaginous rib component
- Incidence of right- and left-sided injury is equal.
- More likely to be isolated injury

PEARLS AND PITFALLS

- Overall mortality is 18–40% depending on mechanism.
- Highly associated with concomitant severe injuries to spleen and liver, hemothorax, pneumothorax, and pelvic fractures.
- Must have high suspicion for diaphragmatic injury with left-sided upper abdominal and lower thoracic penetrating trauma.
- Delayed diagnosis is associated with increased risk for herniation and strangulation of abdominal organs.
- Always obtain chest imaging.

ADDITIONAL READING

- Al-Salem AH. Traumatic diaphragmatic hernia in children. *Pediatr Surg Int*. 2012;28:687–691.
- Blaivas M, Brannam L, Hawkins M, et al. Bedside emergency ultrasonographic diagnosis of diaphragmatic rupture in blunt abdominal trauma. *Am J Emerg Med*. 2004;22(7):601–604.
- Desir A, Ghaye B. CT of blunt diaphragmatic rupture. *Radiographics*. 2012;32:477–498.
- Hanna WC, Ferri LE. Acute traumatic diaphragmatic injury. *Thorac Surg Clin*. 2009;19:485–489.
- Lewis JD, Starnes SL, Pandalai PK, et al. Traumatic diaphragmatic injury:



CODES

ICD9

- 862.0 Injury to diaphragm, without mention of open wound into cavity
- 862.1 Injury to diaphragm, with open wound into cavity

ICD10

- S27.802A Contusion of diaphragm, initial encounter
- S27.803A Laceration of diaphragm, initial encounter
- S27.809A Unspecified injury of diaphragm, initial encounter

DIARRHEA, ADULT

Isam F. Nasr

BASICS

DESCRIPTION

Bowel movements characterized as frequent ($> 3/\text{day}$), loose, and watery owing to an infectious or toxin exposure

ETIOLOGY

- Viruses:
 - 50–70% of all cases
- Invasive bacteria:
 - *Campylobacter*:
 - Contaminated food or water, wilderness water, birds, and animals
 - Most common bacterial diarrhea
 - Gross or occult blood is found in 60–90%.
 - *Salmonella*:
 - Contaminated water, eggs, poultry, or dairy products
 - Typhoid fever (*Salmonella typhi*) characterized by unremitting fever, abdominal pain, rose spots, splenomegaly, and bradycardia
 - *Shigella*:
 - Fecal or oral route
 - *Vibrio parahaemolyticus*:
 - Raw and undercooked seafood
 - *Yersinia*:
 - Contaminated food (pork), water, and milk
 - May present as mesenteric adenitis or mimic appendicitis
- Bacterial toxin:
 - *Escherichia coli*:
 - Major cause of traveler's diarrhea
 - Ingestion of food or water contaminated by feces
 - *Staphylococcus aureus*:
 - Most common toxin-related disease
 - Symptoms 1–6 hr after ingesting food
 - *Bacillus cereus*:
 - Classic source—fried rice left on steam tables
 - Symptoms within 1–36 hr
 - *Clostridium difficile*:
 - Antibiotic-associated enteritis linked to pseudomembranous colitis
 - Incubation period within 10 days of exposure or initiation of

antibiotics

- *Aeromonas hydrophila*:
 - Aquatic sources primarily
 - Affects children < 3 yr of age
 - Fecal leukocytes absent
- *Cholera*:
 - Caused by enterotoxin produced by *Vibrio cholerae*
 - Profuse watery stools with mucus (classic appearance of rice-water stools)
- Protozoa:
 - *Giardia lamblia*:
 - Most common cause of parasite gastroenteritis in North America
 - High-risk groups: Travelers, children in day care centers, institutionalized people, homosexual men, and campers who drink untreated mountain water
 - *Cryptosporidium parvum*:
 - Commonly carried in patients with AIDS
 - *Entamoeba histolytica* (entamebiasis):
 - 5–10% extraintestinal manifestations (hepatic amebic abscess)

Pediatric Considerations

- Most are viral in origin and self-limited.
- Rotavirus accounts for 50%.
- *Shigella*: Infections associated with seizures
- Focus evaluation on state of hydration.



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Loose, watery bowel movements
- Bloody stools with mucus
- Abdominal pain and cramps, tenesmus, flatulence
- Fever, headache, myalgias
- Nausea, vomiting
- Dehydration, lethargy, and stupor

Physical-Exam

- Dry mucous membranes
- Abdominal tenderness
- Perianal inflammation, fissure, fistula

ESSENTIAL WORKUP

- Digital rectal exam to determine presence of gross or occult blood
- Fecal leukocyte determination:
 - Present with invasive bacteria
 - Absent in protozoal infections, viral, toxin-induced food poisoning

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC—indications:
 - Significant blood loss
 - Systemic toxicity
- Electrolytes, glucose, BUN, creatinine—indications:
 - Lethargy, significant dehydration, toxicity, or altered mental status
 - Diuretic use, persistent diarrhea, chronic liver, or renal disease
- Stool culture—indications:
 - Presence of fecal leukocytes
 - Historical markers: Immunocompromised, travel, homosexual
 - Public health: Food handler, day care or health care worker, institutionalized
- Blood cultures—indications:
 - Suspected bacteremia or systemic infections
 - Ill patients requiring admission
 - Immunocompromised
 - Elderly patients and infants

Imaging

Abdominal radiographs:

- No value unless obstruction or toxic megacolon suspected

DIFFERENTIAL DIAGNOSIS

- Ulcerative colitis
- Crohn's disease
- Mesenteric ischemia
- Diverticulitis, anal fissures, hemorrhoids
- Irritable bowel syndrome
- Milk and food allergies
- Malrotation with midgut volvulus
- Meckel diverticulum
- Intussusception
- Appendicitis
- Drugs and toxins:
 - Mannitol

- Sorbitol
- Phenolphthalein
- Magnesium-containing antacids
- Quinidine
- Colchicine
- Mushrooms
- Mercury poisoning



TREATMENT

PRE HOSPITAL

- Difficult IV access with severe dehydration
- Avoid exposure to contaminated clothes or body substances.

INITIAL STABILIZATION/THERAPY

- ABCs
- IV fluid with 0.9% normal saline (NS) resuscitation for severely dehydrated

ED TREATMENT/PROCEDURES

- Oral fluids for mild dehydration (Gatorade/Pedialyte)
- IV fluids for:
 - Hypotension, nausea and vomiting, obtundation, metabolic acidosis, significant hypernatremia or hyponatremia
 - 0.9% NS bolus: 500 mL–1 L (peds: 20 mL/kg) for resuscitation, then 0.9% NS or D₅W 0.45% NS (peds: D₅W 0.25% NS) to maintain adequate urine output
- Bismuth subsalicylate (Pepto-Bismol):
 - Antisecretory agent
 - Effective clinical relief without adverse effects
- Kaolin-pectin (Kaopectate):
 - Reduces fluidity of stools
 - Does not influence course of disease
- Antimotility drugs: Diphenoxylate (Lomotil), loperamide (Imodium), paregoric, codeine:
 - Appropriate in noninfectious diarrhea
 - Initial use of sparse amounts to control symptoms in infectious diarrhea
 - Avoid prolonged use in infectious diarrhea—may increase duration of fever, diarrhea, and bacteremia and may precipitate toxic megacolon
- Antibiotics for infectious pathogens:
 - *Campylobacter*: Quinolone or erythromycin
 - *Salmonella*: Quinolone or trimethoprim–sulfamethoxazole (TMP-SMX)
 - *Typhoid fever*: Ceftriaxone

- *Shigella*: Quinolone, TMP-SMX, or ampicillin
- *V. parahaemolyticus*: Tetracycline or doxycycline
- *C. difficile*: Metronidazole or vancomycin
- *E. coli*: Quinolone or TMP-SMX
- *G. lamblia*: Metronidazole or quinacrine
- *E. histolytica* (entamebiasis): Iodoquinol or metronidazole

MEDICATION

- Ampicillin: 500 mg (peds: 20 mg/kg/24h) PO or IV q6h
- TMP-SMX (Bactrim DS): 1 tab (peds: 8–10 mg TMP/40–50 mg SMX/kg/24h) PO or 4–5 mg/kg TMP IV BID
- Ceftriaxone: 1 g (peds: 50–75 mg/kg/12h) IM or IV q12h.
- Ciprofloxacin (quinolone): 500 mg PO or 400 mg IV q12h (> 18 yr)
- Doxycycline: 100 mg PO or 100 mg IV q12h
- Erythromycin: 500 mg (peds: 40–50 mg/kg/24h) PO QID
- Iodoquinol: 650 mg (peds: 30–40 mg/kg/24h not to exceed 2 g daily) PO TID
- Metronidazole: 250 mg (peds: 35 mg/kg/24h) PO TID (> 8 yr)
- Quinacrine: 100 mg (peds: 6 mg/kg/24h) PO TID
- Tetracycline: 500 mg PO or IV q6h
- Vancomycin: 125–500 mg (peds: 40 mg/kg/24h) PO q6h



FOLLOW-UP

DISPOSITION

Admission Criteria

- Hypotension, unresponsive to IV fluids
- Significant bleeding
- Signs of sepsis or toxicity
- Intractable vomiting or abdominal pain
- Severe electrolyte imbalance or metabolic acidosis
- Altered mental status
- Children with > 10–15% dehydration

Discharge Criteria

- Mild cases requiring oral hydration
- Dehydration responsive to IV fluids

Issues for Referral

Cases of prolonged diarrhea may be referred to a gastroenterologist for further workup.

FOLLOW-UP RECOMMENDATIONS

Since diarrhea is self-limiting, follow-up is optional.

PEARLS AND PITFALLS

- Avoid prolonged use of antimotility drugs in infectious diarrhea.
- TMP-SMX (Bactrim DS), ciprofloxacin, doxycycline, and tetracycline are contraindicated in pregnancy. Metronidazole may be used in the 3rd trimester.
- Health care providers and food handlers with documented infectious diarrhea may need clearance to return to work from their local health department.
- Infectious diarrhea with *C. difficile* is on the rise, especially in nursing home patients.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Gastroenteritis



CODES

ICD9

- 008.5 Bacterial enteritis, unspecified
- 008.8 Intestinal infection due to other organism, not elsewhere classified
- 787.91 Diarrhea

ICD10

- _ A04.9 Bacterial intestinal infection, unspecified
- _ A08.4 Viral intestinal infection, unspecified
- _ R19.7 Diarrhea, unspecified

DIARRHEA, PEDIATRIC

Rajender Gattu • Richard Lichenstein



BASICS

DESCRIPTION

- One of the most common pediatric complaints; 2nd only to respiratory infections in overall disease frequency for ED visits
- Leading cause of illness and death in children worldwide
- Acute infectious enteritis (AIE):
 - Vomiting and diarrhea
 - Children <5 yr in US typically have 2 episodes annually.
 - Responsible for ~10% of all pediatric ED visits and hospital admissions
- Acute change in the “normal” bowel pattern that leads to increased number or volume of stools and lasts <7 days; World Health Organization (WHO) defines case as 3 or more loose or watery stools per day.
 - Chronic if the diarrhea persists for >2 wk

ETIOLOGY

- Acute enteritis:
 - Infectious:
 - Viruses: 70–80% of cases:
 - Rotavirus most common
 - Enteric adenovirus
 - Norovirus (foodborne outbreaks)
 - Bacteria: 10–20%:
 - *Escherichia coli*, *Yersinia*, *Clostridium difficile*
 - *Salmonella*, *Shigella*, *Campylobacter*
 - *Vibrio*
 - *Aeromonas*
 - Parasites 5%:
 - Cryptosporidiosis (waterborne)
 - *Giardia lamblia*
 - Noninfectious:
 - Postinfectious
 - Food allergies and intolerance:
 - Cow’s milk protein
 - Soy protein
 - Methyl xanthines
 - Lactose intolerance
 - Chemotherapy/radiation induced

- Drug induced:
 - Antibiotics, laxatives, antacids
- Ingestion of heavy metals—copper, zinc
- Ingestion of plants—hyacinth, daffodils, amanita species
- Vitamin deficiency: Niacin, folate
- Vitamin toxicity: Vitamin C
- Associated with other infections
 - Otitis media, UTI, pneumonia, meningitis, appendicitis.
- Chronic diarrhea:
 - Dietary factors: Excessive consumption of sorbitol or fructose from fruit juices
 - Enteric infections in immunocompromised
 - Malnutrition
 - Endocrine: Thyrotoxicosis, pheochromocytoma
 - Inflammatory bowel diseases: Crohn's disease, ulcerative colitis
 - Malabsorption syndromes (cystic fibrosis, celiac disease)
 - Irritable bowel syndrome

DIAGNOSIS

SIGNS AND SYMPTOMS

- Frequent, loose stools
- Signs of dehydration:
 - Watery
 - Bloody
 - Mucoïd
 - Sometimes abdominal pain, fever, anorexia
 - Tenesmus
- Signs of dehydration reflect degree of loss of total body water and vary with the degree of dehydration: Mild < 5%, moderate 5–10%, severe > 15%
- Severe dehydration:
 - Mental status change: Often depressed with significant dehydration associated with impaired muscle tone
 - Mucous membrane: Dry
 - Skin turgor: Decreased
 - Anterior fontanel: Depressed
 - Blood pressure: Decreased
 - Pulse: Tachycardia
 - Capillary refill: Prolonged (> 2 sec)
 - Urine output: Decreased
 - Eyes: Sunken and absent tears
 - Thirst

History

- Onset and duration
- Mental status and muscle tone
- Fever and associated symptoms (e.g., abdominal pain, emesis)
- Stool frequency and character with blood and mucus
- Urine output
- Feeding
- Recent antibiotics
- Recent travel
- Possible ingestions
- Immunodeficiency
- Underlying intestinal anomalies (e.g., Hirschsprung disease)

Physical-Exam

- Abnormal capillary refill > 2 sec
- Absent tears
- Dry mucus membranes
- 3 best exam signs for determining dehydration in children are an abnormal respiratory pattern, abnormal skin turgor, and prolonged capillary refill time:
 - Clinical dehydration scales based on a combination of physical exam findings are better predictors than individual signs.

ESSENTIAL WORKUP

Majority of children with acute diarrhea do not require any lab tests. Consider workup if:

- Temperature > 103°F
- Systemic illness
- Bloody diarrhea
- Prolonged course > 2 wk
- Tenesmus
- Dehydration greater than mild, usually requiring parenteral therapy
- Diarrhea with blood or mucus suggests an enteroinvasive inflammatory or cytotoxin-mediated process (*Salmonella*, invasive *E. coli*).

DIAGNOSIS TESTS & NTERPRETATION

Lab

- CBC with differential, blood culture, urine culture, and UA—if any signs of systemic infection
- Basic metabolic panel including electrolytes, BUN, creatinine, bicarbonate, for any child treated with IV hydration for severe dehydration or with those patients with abnormal physical signs:
 - Recent evidence suggests that serum bicarbonate is particularly helpful in

- detecting moderate dehydration.
- Stool pH <5.5 or positive stool-reducing substances are positive in lactose intolerance.
- Stool occult blood
- Stool microscopy:
 - >5 fecal leucocytes per high-power field are suggestive of invasive bacterial infection:
 - *Shigella*
 - *Salmonella*
 - *Campylobacter*
 - *Yersinia*
 - Invasive *E. coli*
- Stool culture:
 - Unnecessary in most cases unless there is a high likelihood of identifying bacterial pathogens (positive guaiac and/or fecal leucocytes) for which the clinical course and period of contagion may be altered by antibiotic therapy
- Consider urine culture in febrile children ≤12 mo.

Imaging

Imaging is usually not indicated. Abdominal x-ray or ultrasound may be useful if the clinical suspicion is high for other diagnoses such as intussusception, ileus, appendicitis.

Diagnostic Procedures/Surgery

Usually not indicated unless high clinical suspicion for other diagnoses based on history and physical exam

DIFFERENTIAL DIAGNOSIS

- Postinfectious:
 - Follows acute or bacterial or viral gastroenteritis; often associated with malabsorption, especially lactose
- *C. difficile* following use of antibiotics.
- Milk allergy
- Malrotation with midgut volvulus
- Inflammatory bowel disease
- Intussusception
- Malabsorption syndromes
- Extra intestinal infections
- Medications altering intestinal flora such as antibiotics (e.g., amoxicillin—clavulanate)



TREATMENT

INITIAL STABILIZATION/THERAPY

- For severely dehydrated children in shock or near shock, IV or intraosseous access with 20 mL/kg 0.9% NS and 1 g/kg dextrose if hypoglycemic
- Alternatively, fluids can be subcutaneously administered using recombinant hyaluronidase human injection using strict protocols
- Pulse oximetry
- Endotracheal intubation may be required for children in shock.

ED TREATMENT/PROCEDURES

- For mild to moderate dehydration, correct dehydration using oral rehydration therapy (ORT), 50 mL/kg and 100 mL/kg, respectively, over a 4-hr period:
 - Replace ongoing losses with 10 mL/kg of ORT for each stool.
 - Ideal ORT solution has a low osmolarity (210–250), glucose of about 2 g/dL, and sodium content of 50–60 mmol/L.
- For moderate to severe dehydration, correct dehydration using parenteral fluids combining maintenance and deficit requirements.
- If diarrhea is not associated with dehydration, use 10 mL/kg of ORT for each stool alone.
- Antibiotics only for defined acute enteritis: Routine use is not recommended; use only in either severe or invasive disease or patients who are immunocompromised or who have significant underlying GI conditions
 - Erythromycin for *Campylobacter jejuni*
 - TMP-SMX for:
 - *Salmonella*—complicated (infant <6 mo old, disseminated, bacteremia, immunocompromised host, enteric fever)
 - *Shigella*
 - *Yersinia*
 - *E. coli*—enteroinvasive
 - Metronidazole or vancomycin for:
 - *C. difficile* (severe and/or prolonged enteritis)
 - Neomycin for *E. coli*—enteroadherent
 - Furazolidone or metronidazole for *G. lamblia*
- Antidiarrheal agents *not* recommended
- Probiotics: *Lactobacillus GG*
 - Probiotics degrade and modify dietary antigens and balance the anti-inflammatory response to cytokines. They reduce the duration of diarrhea
- Post-ED diet:
 - While rehydrating, feed children with diarrhea age-appropriate diets.
 - Well-tolerated foods:
 - Rich in complex carbohydrates (rice, potatoes, bread)
 - Lean meats
 - Yogurt
 - Fruits

- Vegetables
- Full-strength milk and formula unless there is a strong suspicion of lactose intolerance
- Avoid fatty foods and foods high in simple sugars.

MEDICATION

- Ampicillin: 50–200 mg/kg/24h IV/PO q6h
- Erythromycin: 40 mg/kg/24h PO q6h; 10–20 mg/kg/24h IV q6h
- Metronidazole: 30 mg/kg/24h PO divided QID × 7 d
- Neomycin: 50–100 mg/kg/24h PO q6–8h
- TMP-SMX: 8–10 mg/kg/24h as TMP PO divided BID
- Vancomycin: 40–50 mg/kg/24h PO q6h
- Loperamide (not for use in children <6 yr old or in those with heme-positive stools): Age 6–8 yr, 2 mg PO div. BID; age 8–12 yr, 2 mg PO div. TID
- Cefixime: 8 mg/kg/d PO per day for 7–10 days
- Ceftriaxone: 50 mg/kg/d IV/IM for 7–10 days
- *Lactobacillus GG* and *Saccharomyces boulardii*: 5 billion doses/d
- Zinc: 10–20 mg/d for 10–14 days (children <5 yr)

First Line

- TMP-SMX for *Salmonella* and *Shigella* sp.
- Doxycycline for *Vibrio cholerae*
- Metronidazole for *C. difficile*

Second Line

- Ceftriaxone and Cefotaxime for *Salmonella* and *Shigella* sp.
- Erythromycin for *V. cholerae*.
- Vancomycin for resistant *C. difficile*



FOLLOW-UP

DISPOSITION

Admission Criteria

- Surgical abdomen
- Inability to tolerate oral fluids
- 10% dehydration or greater
- Suspected complicated *Salmonella* enteritis
- Toxic-appearing child

Discharge Criteria

- Improvement in the patient's condition

- Caregivers of child can follow through with appropriate ORT and diet.
- Caregivers able to identify signs and symptoms of dehydration

Issues for Referral

- Immunocompromised host
- Conditions associated with complications such as seizures
- Underlying bowel disorders

FOLLOW-UP RECOMMENDATIONS

Follow-up care depends on the length and severity of diarrhea, age of the child, and caregiver's ability to comply with instructions:

- Uncomplicated diarrhea does not typically need follow-up.
- Neonates require strict follow-up care in a few days.

PEARLS AND PITFALLS

- History and PE assists in differentiating uncomplicated diarrhea from other, often more serious conditions in children.
- Vast majority of children with acute diarrhea do not need extensive lab tests, which are unlikely to affect the management.
- Treatment with antidiarrheals and antibiotics has very limited role in childhood diarrhea.
- Diagnoses like appendicitis, intussusception, UTI, and sepsis may need to be considered.

ADDITIONAL READING

- Canavan A, Arant BS Jr. Diagnosis and management of dehydration in children. *Am Fam Physician*. 2009;80(7):692–696.
- Levy JA, Bachur RG, Monuteaux MC, et al. Intravenous dextrose for children with gastroenteritis and dehydration: A double-blind randomized controlled trial. *Ann Emerg Med*. 2013;61:281–288.
- Spandorfer PR, Alessandrini EA, Joffe MD, et al. Oral versus intravenous rehydration of moderately dehydrated children: A randomized, controlled trial. *Pediatrics*. 2005;115:295–301.
- Steiner MJ, DeWalt DA, Byerley JS. Is this child dehydrated? *JAMA*. 2004;291(22):2746–2754.



See Also (Topic, Algorithm, Electronic Media Element)

Vomiting, Pediatric



CODES

ICD9

- 008.8 Intestinal infection due to other organism, not elsewhere classified
- 008.61 Enteritis due to rotavirus
- 787.91 Diarrhea

ICD10

- A08.0 Rotaviral enteritis
- A08.4 Viral intestinal infection, unspecified
- R19.7 Diarrhea, unspecified

DIGOXIN, POISONING

Michelle M. Troendle • Kirk L. Cumpston



BASICS

DESCRIPTION

- Acute digitalis effects (elevated levels in children and intentional overdose):
 - Inhibits sodium-potassium ATPase pump in cell membranes
 - Allows more calcium ions to enter cell and cardiac cells to contract more strongly
 - Increases K^+ extracellularly
 - Increases vagal tone
 - Slows atrioventricular (AV) node conduction (vagotonic)
 - Increases automaticity and conduction system refractory period
 - Bradydysrhythmias
- Chronic digitalis effects (therapeutic to toxic levels in elderly patients):
 - Inhibits sodium-potassium ATPase pump in cell membranes
 - Increases intracellular calcium
 - Increases vagal tone
 - Bradydysrhythmias
 - Increases automaticity
 - Usually hypokalemic secondary to diuretic use
 - Tachydysrhythmias

ETIOLOGY

- Digoxin/digitoxin pharmaceuticals
- Plants and animals containing cardiac glycosides:
 - Foxglove
 - Oleander (white and yellow)
 - Lily of the valley
 - Dogbane
 - Red squill
 - Cane toad, Colorado River toad



DIAGNOSIS

SIGNS AND SYMPTOMS

- Toxicity onset: 2 hr after PO ingestion and 15 min following IV
- Toxicity:
 - Occurs with normal digoxin levels (chronic)
 - May be absent with elevated digoxin levels (acute)

- Cardiovascular:
 - Dysrhythmias:
 - Paroxysmal atrial tachycardia (PAT) with AV block
 - Bidirectional ventricular tachycardia (VT) is pathognomonic
 - Premature ventricular contractions (PVCs) most common
 - Nonparoxysmal accelerated junctional tachycardia
 - VT
 - Ventricular fibrillation
 - Atrial fibrillation/flutter
 - Bigeminy
 - Bradycardia
 - Nonparoxysmal atrial tachycardia
 - AV blocks
 - Sinus arrhythmia
 - Premature atrial contraction
 - CHF exacerbation
 - Hypotension
 - Shock
 - Cardiovascular collapse
 - Syncope
- CNS:
 - Mental status changes:
 - Agitation
 - Lethargy
 - Psychosis
- Visual perception:
 - Blurred
 - Scotoma
 - Green to yellow halo
 - Photophobia
 - Color perception changes
- GI:
 - Anorexia, nausea, vomiting, abdominal pain

History

- Accidental adult or pediatric overdose of a known amount
- Intentional acute overdose in a patient not taking digoxin chronically
- Intentional acute or chronic overdose in a patient taking digoxin chronically
- Unintentional chronic ingestion of digoxin in which renal clearance decreases or the dose chronically increases
- Unintentional toxicity from recent antibiotic use (esp. macrolides) that alter GI flora, primarily by decreasing *Eubacterium lentum*, increasing absorption

Physical-Exam

- Altered mental status
- Bradycardia
- Tachycardia
- Irregular rhythm
- Hypotension

ESSENTIAL WORKUP

- ECG:
 - For dysrhythmia
- Digoxin level:
 - Normal range: 0.5–2 ng/mL
 - Distribution after oral intake not complete until 6 hr; therefore, >6-hr level is most accurate steady state concentration.
 - False elevations possible with spironolactone use, pregnancy, hyperbilirubinemia, chronic renal failure, liver failure, CHF
 - May be falsely elevated after digoxin-specific Fab fragments given

DIAGNOSIS TESTS & NTERPRETATION

Lab

- Electrolytes, BUN, creatinine, glucose:
 - Hypokalemia contributes to digitalis toxicity.
 - Hyperkalemia seen in acute toxicity and correlates with acute digitalis mortality better than digoxin serum levels.
 - Follow K⁺ serially
- Calcium, magnesium

ALERT

Serum digoxin concentration (SDC) should not be obtained after digoxin-specific antibody Fab fragments have been administered because it will be inaccurate.

DIFFERENTIAL DIAGNOSIS

- Overdoses:
 - Calcium channel blockers
 - β -Blockers
 - Quinidine, procainamide
 - Clonidine
 - Organophosphates
 - Antidysrhythmics
 - Other antihypertensives
- Primary cardiac dysrhythmias
- Acute gastroenteritis



TREATMENT

PRE HOSPITAL

- Establish IV access
- Continuous cardiac monitoring
- Apply pads for potential cardioversion

ALERT

- If cardioversion is necessary for tachydysrhythmias, use low levels (50 J)
- May precipitate refractory tachydysrhythmias

INITIAL STABILIZATION/THERAPY

ABCs:

- IV, oxygen, monitor:
 - IV fluid bolus if hypovolemic
- Administer naloxone, thiamine, dextrose for altered mental status.

ED TREATMENT/PROCEDURES

- Cardiac arrest resuscitation:
 - Defibrillate for ventricular fibrillation, pulseless VT.
 - Standard advanced cardiac life support (ACLS) protocol
 - Administer digoxin-specific antibody Fab fragments (Digibind), up to 5–20 vials IV push (IVP).
 - MgSO₄, 2 g IVP
 - Continue resuscitation for 30 min after digoxin-specific antibody Fab fragments.
- General measures:
 - Activated charcoal if acute ingestion
 - Replenish magnesium.
 - Treat hyperkalemia with insulin, dextrose, bicarbonate, sodium polystyrene sulfonate.
 - Calcium can probably be used to treat hyperkalemia, but because other safer alternatives exist it is not recommended, unless life-saving membrane stabilization is needed secondary to hyperkalemia, in the unstable patient.
 - If the patient has hyperkalemia from digoxin toxicity treatment with digoxin-specific Fab fragments are indicated 1st in the hemodynamically stable patient.

DYSRHYTHMIA MANAGEMENT

- 1st choice: Digoxin-specific antibody Fab fragments (Digibind, DigiFab)
 - Indications:
 - SDC ≥ 15 ng/mL at any time or ≥ 10 ng/mL at steady state (6 hr)

- Ingestion of >10 mg in adults or 0.2 mg/kg or 4 mg in children
- Hyperkalemia >5–5.5 mEq/L
- Hemodynamically unstable or life-threatening dysrhythmias
- VT, ventricular fibrillation
- Atrial tachycardia
- Variable AV block
- Bradycardia with no response to atropine
- Hypotension
- Onset: 20–30 min
- Digoxin levels may increase, decrease, or stay in therapeutic range after therapy owing to Fab digoxin complexes and redistribution.
- Renal clearance of drug–antibody complexes:
 - Too large to be removed by dialysis
- 2nd dose if rebound toxicity
- Complications:
 - Exacerbation of CHF
 - Hypokalemia
 - Atrial fibrillation with rapid ventricular response
- If digoxin-specific antibody Fab fragments not immediately available initiate the following:
 - Lidocaine:
 - For ventricular dysrhythmias without AV block
 - Not harmful but not very effective
 - For bradydysrhythmias:
 - Atropine
 - Pacing for symptomatic bradydysrhythmia
 - MgSO₄ for ventricular dysrhythmias with torsades de pointes
 - Quinidine, procainamide contraindicated
- Cardioversion is last resort for severe, life-threatening tachydysrhythmia:
 - Start at low energy 10–50 J, then increase to high levels if ineffective.
 - Safe if digoxin level <2 ng/mL

MEDICATION

- Activated charcoal slurry:
 - 1 g/kg if within 1 hr
- Digoxin-specific antibody Fab fragments:
 - 40-mg vial neutralizes 0.5 mg of digoxin.
 - If amount ingested known:
 - Number of vials needed to treat equals [amount ingested (mg)/0.5 (mg/vial)]
 - If steady serum level known:
 - Number of vials needed equals [SDC (ng/mL) × weight (kg)]/100
 - If neither amount ingested nor serum level known:

- Acute toxicity: 5–10 vials adults or children
- Chronic toxicity: 1--2 vials in adults or children
- Bolus digoxin-specific antibody Fab fragments for cardiac arrest
- Additional doses as needed
- Standard treatment for hyperkalemia and bradycardia (calcium only if necessary)

Geriatric Considerations

- Dosage is based on weight and serum concentration. There is no change in the setting of renal or hepatic dysfunction.
- Recrudescence of toxicity has been reported in patients with concomitant renal failure. Redosing of digoxin-specific antibody Fab fragments should be used again when indicated.

Pediatric Considerations

- Weight-based dosing for children is the same as it is for adults.
- On some occasions, accidental dose ingested by a child is known and the number of vials is indicated by the amount of digoxin bound by each vial. See dosing.

Pregnancy Considerations

Digoxin-specific Fab fragments are pregnancy class C.



FOLLOW-UP

DISPOSITION

Admission Criteria

- ICU:
 - Unstable cardiovascular status in acute or chronic toxicity
- Telemetry:
 - Asymptomatic or mildly symptomatic dysrhythmia
 - High risk for developing toxicity

Discharge Criteria

Acute/chronic ingestion:

- Digoxin level < 2 ng/mL
- Asymptomatic for 6 hr and no ECG abnormalities

FOLLOW-UP RECOMMENDATIONS

Psychiatric referral for stable patients who are suicidal

PEARLS AND PITFALLS

When it is known that the patient is on digoxin and presents with cardiovascular instability, and/or hyperkalemia, treatment should begin with the antidote: Digoxin-specific Fab fragments.

ADDITIONAL READING

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- Levine M, Nikkanen H, Pallin DJ. The effects of intravenous calcium in patients with digoxin toxicity. *J Emerg Med.* 2011;40(1):41–46.



CODES

ICD9

972.1 Poisoning by cardiotonic glycosides and drugs of similar action

ICD10

- T46.0X1A Poisoning by cardi-stim glycos/drug similar act, acc, init
- T46.0X4A Poisoning by cardi-stim glycos/drug similar act, undet, init

DIPLOPIA

Jonathan A. Edlow



BASICS

DESCRIPTION

- Double vision
 - Simultaneous perception of 2 images
 - Can be oriented horizontally, vertically, or diagonally from one another.
- Diplopia is usually due to abnormal movement of the extraocular muscles (EOMs), which are innervated by 3 cranial nerves (CNs):
 - CN 3 – superior, inferior, and medial rectus and inferior oblique muscles
 - CN 4 – superior oblique muscle
 - CN 6 – lateral rectus muscle
- Brainstem lesions can damage CN nuclei or their connections (medial longitudinal fasciculus, MLF), causing an internuclear ophthalmoplegia (INO)
- CN dysfunction
 - Compression as they traverse the subarachnoid space and venous sinuses
 - Inflammation
 - Elevation (or reduction) of CSF pressure can cause CN 6 palsy
- Disease affecting the orbits and the bony skull can cause restriction of motion of one or both eyes or EOMs

ETIOLOGY

- Traumatic diplopia
 - Orbital fracture
 - Contusions
 - Hematoma
 - Rarely brainstem contusion or hematoma
- Monocular diplopia
 - Nearly always due to an intrinsic eye problem
 - Corneal surface keratoconus
 - Subluxation of the lens
 - Structural defect within the eye
 - Functional disorders such as conversion disorder, factitious disorder, or somatization.
- Nontraumatic binocular diplopia
 - Brain and brainstem dysfunction
 - Stroke
 - Multiple sclerosis
 - Cerebral cortical problems (e.g., migraine) are rare

- CN dysfunction
 - Aneurysm of posterior communicating artery (CN 3 palsy)
 - Chronic lymphocytic meningitis (multiple CN deficits)
 - Pseudotumor cerebri (CN 6 palsy)
 - Low pressure (spontaneous intracranial hypotension) (CN 6 palsy)
- Bony skull and orbits:
 - Tumor
 - Thyroid disease
 - Inflammation (Tolosa-Hunt)
- Neuromuscular junction (NMJ) of EOMs:
 - Myasthenia gravis (MG)

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Determine if the diplopia is following head injury, if it is constant or intermittent and its duration.
- Determine if diplopia is monocular or binocular.
- Ask about HA or other neurologic or visual symptoms.
- Are the 2 images aligned horizontally, vertically, or diagonally?

Physical-Exam

- Differentiate monocular from binocular:
 - If the diplopia goes away with covering either eye, it is binocular
 - If it persists in one eye, it is monocular (problem in the eye with diplopia)
- Differentiate traumatic and nontraumatic diplopia
- Monocular diplopia
 - Careful ocular exam
 - Visual acuity
- Binocular diplopia:
 - Examine the eye completely (is there ptosis, anisocoria, limitation of EOMs, proptosis, exophthalmos?). Is VA normal?
 - CN 3 – pupil involving – diagonal diplopia with ptosis and dilated pupil
 - CN 3 – pupil sparing – diagonal diplopia with normal lids and pupils
 - CN 4 – vertical or diagonal diplopia, least common of all
 - CN 6 – horizontal diplopia; images separate more as gaze goes out laterally on affected side.
 - Do a complete neurologic exam
- Traumatic diplopia:
 - Facial anesthesia

- Anisocoria
- Proptosis
- Decreased visual acuity

ALERT

- Patients may appear well with benign exams; one must look carefully for neurologic or ophthalmologic findings.
- Do a systematic physical exam to try to localize the site of the lesion
- If CN 3 palsy, differentiate a pupil-sparing (usually microvascular infarct) from a pupil-involving palsy (usually aneurysmal). This is often due to an expanding but unruptured aneurysm.
- If facial numbness (CN 5) with diplopia, consider cavernous sinus or superior orbital fissure syndromes
- If decreased vision, suspect orbital or superior orbital fissure syndrome

ESSENTIAL WORKUP

Accurate history and physical exam are the cornerstones of diagnosis. Establishing a history of trauma is key. In spontaneous cases, the physical exam should be directed at establishing:

- Is the diplopia isolated or not?
- If isolated, which CN is involved?
- If other neurologic deficits exist, try to localize the site of the lesion.
- Some form of cerebral angiography (CTA, MRA, DSA) if there is a CN 3 palsy and dilated pupil.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Lab testing in the ED is not generally useful:
 - Occasionally, thyroid function tests are helpful. Other serologic tests (e.g., for myasthenia) are not necessary in the ED).

Diagnostic Procedures/Surgery

- If MG is possible, consider an edrophonium (Tensilon) test or an ice test.
- Lumbar puncture – if considering SAH, chronic lymphocytic meningitis, high or low CSF pressure.
- Brain and cerebrovascular imaging – if considering mass lesion, aneurysm, stroke, MS. Specific tests depend on the specific differential diagnosis.

DIFFERENTIAL DIAGNOSIS

- Post-traumatic diplopia:
 - Orbital fracture/hematoma with direct damage to CNs or hematoma limiting EOM excursions

- Rarely, brainstem contusion affecting CN nuclei or MLF
- Monocular diplopia:
 - Nearly any ocular problem (corneal, lens, iris, retinal problems, and refractive error)
 - Rarely, bilateral monocular diplopia due to cortical dysfunction
- Binocular diplopia:
 - Brain and brainstem
 - Stroke or MS involving the brainstem, often with a nuclear CN palsy or an INO. This pattern is rarely seen after head injury and contusion
 - Wernicke encephalopathy
 - Rare: “Cortical” diplopia from migraine or irritative lesion and botulism
 - CN lesions
 - CN palsy due to stretch, contusion, ischemia, CSF inflammation, or abnormal pressure
 - CN 3 – KEY to differentiate pupil involving (must r/o aneurysm) vs. pupil sparing (microvascular infarct from diabetes/hypertension).
 - CN 6 palsy is a nonlocalizing finding.
 - When multiple CNs are involved, think about MG, lymphocytic meningitis, cavernous sinus pathology.
 - When medial rectus is involved (decreased adduction) but there is no clear CN 3 palsy, consider INO
- Skull and orbits:
 - Infiltrative disorders of orbit (thyroid, tumor, abscess, and Tolosa-Hunt)
- NMJ:
 - MG and botulism



TREATMENT

INITIAL STABILIZATION/THERAPY

The vast majority of patients with diplopia do not require stabilization. Initial steps are entirely based on the etiology in an individual patient.

ED TREATMENT/PROCEDURES

- Lumbar puncture:
 - In cases of possible lymphocytic meningitis, pseudotumor cerebri, spontaneous intracranial hypotension, do LP. ALWAYS measure the opening pressure.
- Edrophonium test:
 - In cases of possible MG, consider performing an edrophonium test (see reference Scherer et al. for technique).
- Eye patch:

- Consider eye patch in discharged patients for symptom control

Pediatric Considerations

Same differential diagnosis.

Pregnancy Considerations

- Pregnant women with hyperemesis gravidarum can get Wernicke encephalopathy or orbital hemorrhage, both of which can present with diplopia.
- Postpartum women can develop diplopia due to cavernous sinus thrombosis, postdural puncture headache, or orbital hemorrhage.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Admission is predicated upon the cause.
- Many patients are admitted to facilitate a rapid workup for serious causes (including advanced brain and vascular imaging and specialty consultation).

Discharge Criteria

Most patients with monocular diplopia or with traumatic diplopia whose cause is *clearly established* and does not require urgent surgery can be safely discharged.

FOLLOW-UP RECOMMENDATIONS

All patients who are discharged with diplopia will require some form of follow-up, usually with either a neurologist or ophthalmologist.

PEARLS AND PITFALLS

- Diplopia can present as “blurred vision” if the 2 images are not far off from one another.
- Never assume diplopia is an isolated cranial neuropathy without doing a very careful neurologic exam.
- Check pupils to avoid missing a cerebral aneurysm. Aneurysmal CN 3 palsies are often due to *unruptured* aneurysm (CT and LP normal).
- MG can present with intermittent diplopia. The pupils are *always* normal.
- Test facial sensation; hypoesthesia and diplopia localize the lesion to the cavernous sinus or superior orbital fissure.
- Test vision; decreased VA and diplopia are often an orbital or superior orbital fissure lesion (may be surgical emergency!)

ADDITIONAL READING

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- Pelak VS. Evaluation of diplopia. *Hospital Practice.* 2004;3:16–25.
- Scherer K, Bedlack RS, Simel DL. Does this patient have myasthenia gravis? *JAMA.* 2005;293:1906–1914.
- Tilikete C. When is diplopia strongly suggestive of a vascular event? *Exp Rev Ophthalmol.* 2009;4(4):357–361.



See Also (Topic, Algorithm, Electronic Media Element)

Potential – MS, MG, stroke, aneurysm, SAH, lymphocytic meningitis, Wernicke encephalopathy



CODES

ICD9

- 368.2 Diplopia
- 368.15 Other visual distortions and entoptic phenomena

ICD10

H53.2 Diplopia

DISSEMINATED INTRAVASCULAR COAGULATION

Steven H. Bowman • Ernesto J. Romo

BASICS

DESCRIPTION

- Normal coagulation: Series of local reactions among blood vessels, platelets, and clotting factors
- Disseminated intravascular coagulation (DIC) is systemic activation of coagulation and fibrinolysis by some other primary disease process.
- Coagulation system activation results in systemic circulation of thrombin and plasmin.
- Role of thrombin in DIC:
 - Tissue factor/factor VIII(a) activate the extrinsic pathway, leads to thrombin formation.
 - Thrombin circulates and converts fibrinogen to fibrin monomer.
 - Fibrin monomer polymerizes into fibrin (clot) in the circulation.
 - Clots cause microvascular and macrovascular thrombosis with resultant peripheral ischemia and end organ damage.
 - Platelets become trapped in clot with resultant thrombocytopenia.
- Role of plasmin in DIC:
 - Plasmin circulates systemically converting fibrinogen into fibrin degradation products (FDPs).
 - FDPs combine with fibrin monomers.
 - FDP-monomer complexes interfere with normal polymerization and impair hemostasis.
 - FDPs also interfere with platelet function.
- Role of impaired anticoagulation in DIC.
 - Failure of physiologic anticoagulation is necessary for DIC to occur.
 - Antithrombin III, protein C system, and tissue factor pathway inhibitor all impaired.
- Acute DIC—uncompensated form:
 - Clotting factors used more rapidly than body can replace them
 - *Hemorrhage* predominant clinical feature, which overshadows ongoing thrombosis
- Chronic DIC—compensated form:
 - Body able to keep up with pace of clotting factor consumption
 - *Thrombosis* predominant clinical feature

ETIOLOGY

- Precipitated by many disease states

- Complications of pregnancy:
 - Retained fetus
 - Amniotic fluid embolism
 - Placental abruption
 - Abortion
 - Eclampsia
 - HELLP syndrome
- Sepsis:
 - Gram negative (endotoxin-mediated meningococemia)
 - Gram positive (mucopolysaccharide-mediated)
 - Other microorganisms (e.g., viruses, parasites)
- Trauma:
 - Crush injury
 - Severe burns
 - Severe head injury
 - Fat embolism
- Malignancy:
 - Solid tumor or metastatic disease
 - Hematologic malignancy (e.g., leukemia)
- Intravascular hemolysis:
 - Transfusion reactions
 - Massive transfusion
- Organ destruction:
 - Severe pancreatitis
 - Severe hepatic failure
- Vascular abnormalities:
 - Kasabach–Merritt syndrome
 - Large vascular aneurysm
- Thrombocytopenia:
 - Thrombotic thrombocytopenic purpura
 - Idiopathic thrombocytopenic purpura
- Miscellaneous:
 - Snake bites
 - Recreational drugs



DIAGNOSIS

SIGNS AND SYMPTOMS

- Excessive bleeding:
 - Petechiae
 - Purpura
 - Hemorrhagic bullae

- Wound bleeding
- Bleeding from venipuncture/arterial lines
- Epistaxis
- Hemoptysis
- GI bleeding
- Excessive thrombosis:
 - Large vessels
 - Microvascular thrombosis and end organ dysfunction
 - Cardiac, pulmonary, renal, hepatic, CNS
 - Thrombophlebitis
 - Pulmonary embolus
 - Nonbacterial thrombotic endocarditis
 - Gangrene
 - Ischemic infarcts of kidney, liver, CNS, bowel
- Acute DIC:
 - Hemorrhagic complications predominate.
- Chronic DICL:
 - Thrombotic complications predominate.

History

- Previous history of bleeding disorder
- Pregnancy/last menstrual period
- History of malignancy or immunocompromised

Physical-Exam

- Neurologic:
 - Altered MS, confusion, lethargy
- Cardiovascular:
 - Hypotension, tachycardia
- Respiratory:
 - Tachypnea, rhonchi, rales
- GI:
 - Upper or lower GI bleeding, abdominal distension
- GU:
 - Oliguria, hematuria
- Skin:
 - Petechiae, purpura, jaundice, necrosis

ESSENTIAL WORKUP

- Depends on precipitating illness
- Diagnosis generally not made in ED

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Platelet count:
 - Important to note rapid decrease
 - $< 100,000/\text{mm}^3$
 - May be normal in chronic DIC
- Prothrombin time (PT)/partial thromboplastin time (PTT):
 - Increased
 - May be normal in chronic DIC
- Fibrinogen:
 - Decreased
 - $< 150 \text{ mg/dL}$ in 70%
 - Low sensitivity, as levels can remain normal
 - May be normal in chronic DIC
- FDPs:
 - Increased
 - $> 40 \mu\text{g/mL}$
- D-dimer increased
- CBC/peripheral smear:
 - Red cell fragments
 - Low platelets
 - Peripheral smear confirms disease in chronic DIC
- Electrolytes, BUN, creatinine, glucose:
 - Elevated BUN, creatinine owing to renal insufficiency
- ABGs:
 - Oxygen, acid–base status
- ISTH scoring system
 - Underlying disorder associated with DIC
 - no = 0, yes = 2
 - Platelet count
 - $> 100 = 0$, $< 100 = 1$, $< 50 = 2$
 - Fibrin markers (D-dimer, FDP)
 - Normal = 0, moderate increase = 1, strong increase = 2
 - Prolonged PT
 - $< 3 = 0$, > 3 but $< 6 = 1$, $> 6 = 2$
 - Fibrinogen
 - $1 \text{ g/L} = 0$, $< 1 \text{ g/L} = 1$
 - Score > 5 overt DIC, associated with increased mortality.

Imaging

- CXR for suspected pneumonia
- Head CT for altered mental status
- OB US in pregnant patients

DIFFERENTIAL DIAGNOSIS

- Inherited coagulation disorders:
 - Factor deficiencies
- Other acquired coagulation disorders:
 - Anticoagulant therapy
 - Drugs
 - Hepatic disease
 - Vitamin K deficiency
 - Massive blood loss
- Platelet dysfunction:
 - TTP/HUS
 - HIT
 - ITP
- Platelet dysfunction:
 - TTP/HUS
 - HI



TREATMENT

INITIAL STABILIZATION/THERAPY

- Airway management and resuscitation measures:
 - Control bleeding
 - Establish IV access
 - Restore and maintain circulating blood volume.
- Initiate therapy of precipitating disease:
 - Antibiotics in sepsis
 - Evacuate uterus of retained products of conception
 - Chemotherapy in malignancy
 - Débridement of devitalized tissue in trauma

ED TREATMENT/PROCEDURES

- Therapy of DIC is controversial and should be individualized based on:
 - Age
 - Hemodynamic status
 - Severity of hemorrhage
 - Severity of thrombosis
- Involve admitting service before initiating specific DIC therapy.
- Replace depleted blood components:
 - Fresh frozen plasma (FFP):
 - For prolonged PT
 - Provides clotting factors and volume replacement
 - Dose: 2 U or 10–15 mL/kg

- Platelets:
 - If platelet count < 20,000 or platelet count < 50,000 with ongoing bleeding
 - Dose: 1 U/10 kg body weight
- Cryoprecipitate:
 - Higher fibrinogen content than whole plasma
 - For severe hypofibrinogenemia (< 50 mg/dL) or for active bleeding with fibrinogen < 100 g/dL
 - Dose: 8 U
- Recombinant factor VIIa
 - Successful use reported, benefit and safety unknown.
- Washed packed cells
- Albumin
- Nonclotting volume expanders
- Inhibit intravascular clotting with heparin:
 - Use is controversial.
 - Consider when thrombosis predominates.
 - May be effective in mild to moderate DIC
 - Efficacy undetermined in severe DIC. Possible indications:
 - Purpura fulminans (gangrene of digits, extremities)
 - Acute promyelocytic leukemia
 - Dead fetus syndrome—several weeks after intrauterine fetal death
 - Thromboembolic complications of large vessels
 - Before surgery with metastatic carcinoma
 - Administer activated protein C (controversial):
 - No mortality benefit.
 - Antithrombin
 - No mortality benefit found in patients also receiving heparin.
 - Lack of evidence to support use at this time.
- Inhibit fibrinolysis:
 - Block secondary compensatory fibrinolysis that accompanies DIC
 - Use complicated by severe thrombosis
 - Use only when DIC accompanied by primary fibrinolysis:
 - Promyelocytic leukemia
 - Giant hemangioma
 - Heat stroke
 - Amniotic fluid embolism
 - Metastatic carcinoma of prostate
 - Initiate in extreme cases only:
 - Profuse bleeding not responding to replacement therapy
 - Excessive fibrinolysis present (rapid whole blood lysis/short euglobulin lysis time)
 - E-aminocaproic acid (EACA)

- Tranexamic acid

MEDICATION

Specific DIC treatment is usually not initiated in the ED. Underlying precipitating diseases should be treated initially:

- Heparin:
 - Low-dose regimen: 5–10 U/kg/h IV for causes where thrombosis predominates.



FOLLOW-UP

DISPOSITION

Admission Criteria

Severe precipitating illness in combination with DIC requires ICU admission.

Discharge Criteria

None

FOLLOW-UP RECOMMENDATIONS

Follow-up involves following platelets and coagulation factors.

PEARLS AND PITFALLS

- Suspect DIC as a complicating factor in severe, life-threatening illness.
- Establish early clinical suspicion since the sequelae of DIC can be devastating.
- Remember to consider treating the underlying cause of DIC when the thromboembolic and bleeding complications of the process seem to be dominating the clinical picture.

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See Also (Topic, Algorithm, Electronic Media Element)

- Sepsis
- Idiopathic Thrombocytopenic Purpura
- Thrombotic Thrombocytopenic Purpura



CODES

ICD9

286.6 Defibrination syndrome

ICD10

D65 Disseminated intravascular coagulation

DISULFIRAM REACTION

Timothy J. Meehan • Sean M. Bryant



BASICS

DESCRIPTION

- Inhibits various enzymes and its active metabolites exert additional effects.
- Disulfiram–ethanol reaction:
 - Usually occurs 8–12 hr after taking the drug; should not be observed > 24 hr after dosing
 - Competitively and irreversibly inactivates aldehyde dehydrogenase
 - Ethanol metabolism is blocked, resulting in accumulation of acetaldehyde
 - Acetaldehyde produces release of histamine resulting in vasodilation and hypotension
 - Severe reactions may occur in drinkers with ethanol levels of 50–100 mg/dL
 - Severity and duration of reaction is proportional to amount of ethanol ingested
- Disulfiram blocks dopamine β -hydroxylase and limits synthesis of norepinephrine from dopamine:
 - Relative excess of dopamine may contribute to altered behavior
 - Relative depletion of norepinephrine may contribute to hypotension
- Disulfiram metabolite (carbon disulfide) interacts with pyridoxal 5-phosphate:
 - Diminishes concentration of pyridoxine available for formation of γ -aminobutyric acid (GABA) in CNS
 - Potentially lowers seizure threshold
 - Carbon disulfide is also cardiotoxic, hepatotoxic, and inhibits cytochrome P-450 (CYP2E1)
- Disulfiram metabolites may chelate important metals (copper, zinc, iron) essential in various enzyme systems
- Disulfiram metabolites may cause peripheral neuropathies that are dose and duration dependent

ETIOLOGY

- Disulfiram is used as a deterrent in the treatment of chronic ethanol abuse
- Many users of the medication wear a medical alert bracelet
- Other agents producing disulfiram-like reactions:
 - Antibiotics:
 - Metronidazole
 - Cephalosporins (with nMTT side chain)
 - Cefoperazone, Cefotetan, Cefmetazole
 - Nitrofurantoin

- Oral hypoglycemics:
 - o Sulfonylureas
- Industrial agents:
 - o Carbon disulfide
 - o Hydrogen sulfide
- Mushrooms:
 - o *Coprinus atramentarius*
 - o *Clitocybe clavipes*

DIAGNOSIS

SIGNS AND SYMPTOMS

- Disulfiram–ethanol reaction:
 - Hypotension, tachycardia, tachypnea
 - Flushing of face, neck, torso
 - Pruritus, diaphoresis, sensation of warmth
 - Nausea, vomiting, abdominal pain, diarrhea
 - Headache, ataxia, confusion, anxiety, dizziness
 - Dyspnea, pulmonary edema, chest pain, dysrhythmias, myocardial infarction
- Disulfiram overdose:
 - Symptoms rare with < 3 g ingested
 - 10–30 g may be lethal
 - May mimic shock and/or sepsis
 - Tachycardia, hypotension, tachypnea
 - Abdominal pain, diarrhea, garlic, or rotten-egg breath
 - Agitation, irritability, ataxia
 - Dysarthria, hallucinations
 - Lethargy, coma, seizures, flaccidity
 - Parkinsonism

History

Ingestion of disulfiram or agents listed above may provide essential clues to diagnosis

Physical-Exam

- Vital signs:
 - Hypotensive, tachycardic, tachypneic
- Cardiovascular:
 - Tachycardia, arrhythmias
- Pulmonary:
 - Pulmonary edema, dyspnea
- Abdominal:

- Diffuse abdominal pain, nausea, vomiting
- Skin:
 - Flushed, diaphoretic
- Neurologic:
 - Dysphoria, confusion, signs of cerebellar dysfunction, seizures

ESSENTIAL WORKUP

Suspect disulfiram–ethanol reaction with the following:

- Typical signs and symptoms are present
- Treatment for chronic ethanol abuse in conjunction with recent ethanol ingestion, or exposure to ethanol-containing foods or medications, including mouthwash

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Ethanol level
- Electrolytes, BUN, creatinine, and glucose
- Liver function tests if hepatitis is suspected
- Creatine phosphokinase (CPK) if considering rhabdomyolysis in light of seizures or agitation
- Urinalysis (myoglobin)
- Serum levels of offending agent are NOT clinically useful

Imaging

- ECG to assess cardiac ischemia or dysrhythmia
- CT scan or MRI:
 - Indicated with altered mental status/seizure
 - Basal ganglia ischemia and infarction have been reported
- EEG:
 - Diffuse slowing without focal abnormalities has been seen in cases of acute toxicity with coma

DIFFERENTIAL DIAGNOSIS

- Sepsis
- Meningitis, encephalitis
- Cardiogenic shock secondary to acute coronary syndrome
- Anaphylactoid/anaphylactic reaction
- Gastroenteritis/pancreatitis with dehydration
- Ethanol withdrawal

Pediatric Considerations

- Acute poisonings yield mainly severe CNS toxicity
- Ataxia, weakness, lethargy, seizures

- Reye syndrome-like encephalopathy in severe cases
- Adult symptoms may also be present



TREATMENT

PRE HOSPITAL

- ABCs, IV access
- Begin resuscitation with IVF if no signs or symptoms of pulmonary edema
- Rapid glucose determination (Accu-Chek)

INITIAL STABILIZATION/THERAPY

- ABCs:
 - Airway protection if necessary
 - Supplemental oxygen
 - Mechanical ventilation as needed
 - Resuscitation with 0.9% NS IV for hypotension
- Pressor support with norepinephrine for refractory hypotension

ED TREATMENT/PROCEDURES

- Management is primarily supportive with aggressive, appropriate care:
 - No specific antidote available
- GI decontamination:
 - Activated charcoal in cases of disulfiram overdose:
 - Caution if mental status depression
 - Caution if vomiting (potential for aspiration)
 - Do not intubate solely to give activated charcoal
 - Gastric lavage is unnecessary
 - Whole-bowel irrigation is not indicated
- Alleviation of flushing:
 - Antihistamines (H₁ and H₂ antagonists)
 - Prostaglandin inhibitors (indomethacin, ketorolac)
- Antiemetics for intractable vomiting (ondansetron, metoclopramide)
- Seizures:
 - Benzodiazepines (diazepam, lorazepam)
 - Pyridoxine (vitamin B₆)
 - 4-methylpyrazole:
 - Inhibits ethanol metabolism at alcohol dehydrogenase enzyme
 - Not indicated for routine disulfiram–ethanol reactions or mild disulfiram overdose
 - May improve the hemodynamic profile in moderate to severe overdoses
- Hemodialysis:

- Consider after massive ingestion of disulfiram and ethanol with refractory hypotension
- No studies documenting beneficial effect

MEDICATION

- Diazepam: 5–10 mg (peds: 0.2–0.5 mg/kg) IV
- Diphenhydramine: 25–50 mg (peds: 1–2 mg/kg) IV
- Indomethacin: 50 mg PO (peds: 0.6 mg/kg PO for age > 14 yr)
- Lorazepam: 2–6 mg (peds: 0.03–0.05 mg/kg) IV
- Metoclopramide: 10 mg (peds: 1–2 mg/kg) IV
- Norepinephrine: 4 mL in 1,000 mL of D₅W, infused at 0.1–0.2 µg/kg/min
- Ondansetron: 4 mg (peds: 0.1 mg/kg for > 2 yr old) IV
- Pyridoxine: 1 g (peds: 500 mg) IV, repeat PRN



FOLLOW-UP

DISPOSITION

Admission Criteria

- ICU admission for mechanical ventilation, coma, refractory hypotension requiring pressors, cardiac ischemia, refractory seizures, and severe agitation
- Persistent vomiting, abdominal pain, or flushing
- Elderly patients or those who have pre-existing cardiac disease

Discharge Criteria

- Mild reactions that resolve with supportive care after observation period of 8–12 hr:
 - Symptoms may recur on rechallenge with ethanol up to 7–10 days after last dose of disulfiram or agents that cause disulfiram-like reactions
 - Abstain from ethanol use until at least 2 wk after last dose of such agents
- Appropriate follow-up needed to assess development of hepatic or neurologic sequelae

FOLLOW-UP RECOMMENDATIONS

- Psychiatry follow-up for intentional overdose with disulfiram
- Detox follow-up for patients with disulfiram–ethanol reactions

PEARLS AND PITFALLS

- Educate patients who are prescribed medications with potential for disulfiram-like reactions to avoid ALL alcohol
 - Includes: Mouthwash, alcohol-based hand gels, alcohol-based aftershave,

some cough syrups, and elixir-based liquid medications

- Recommend abstinence for 3 days longer than the course of treatment to ensure low likelihood of reaction

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See Also (Topic, Algorithm, Electronic Media Element)

Alcohol Poisoning



CODES

ICD9

977.3 Poisoning by alcohol deterrents

ICD10

- T50.6X1A Poisoning by antidotes and chelating agents, acc, init
- T50.6X4A Poisoning by antidotes and chelating agents, undet, init
- T50.6X5A Adverse effect of antidotes and chelating agents, initial encounter

DIVERTICULITIS

Ronald E. Kim

BASICS

DESCRIPTION

- Micro- or macroscopic perforation of diverticulum
 - Uncomplicated (75%) vs. complicated
- Incidence increasing
 - Obesity is a risk factor

ETIOLOGY

- Fecal material in diverticulum hardens, forming fecalith, increasing intraluminal pressure
- Erosion of diverticular wall leads to inflammation
- Focal necrosis leads to perforation
- Microperforation: Uncomplicated diverticulitis:
 - Colonic wall thickening
 - Inflammatory changes (fat stranding on CT)
- Macroperforation: Complicated diverticulitis:
 - Abscess
 - Bowel obstruction
 - Fistulas after recurrent attacks
 - Colovesical fistula (most common) presents with dysuria, frequency, urgency, pneumaturia, and fecaluria.
 - Peritonitis

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Symptoms typically develop over days
 - Almost 50% have had prior episodes of pain
- Left lower quadrant pain in 70% of cases in Western countries
 - Initially vague, then localizes
 - RLQ in 75% of Asian patients
- Nausea/vomiting, constipation, diarrhea, urinary symptoms (in decreasing order)

Physical-Exam

- +/- low-grade fever

- Tenderness at left lower quadrant with occasional (20%) mass palpated (phlegmon):
 - *Phlegmon*—inflamed bowel loops or abscess
- Abdominal distension
- Bowel sounds variable
- Rectal tenderness with heme-positive stool:
 - Massive gross rectal bleeding (rare)
- Peritoneal signs if:
 - Perforation has occurred
- Unremarkable exam if:
 - Elderly
 - Immunocompromised
 - Taking corticosteroids

ESSENTIAL WORKUP

- CBC
- UA
- Blood cultures and lactate
 - If showing signs of sepsis
- CT of abdomen/pelvis
 - Preferred diagnostic modality
 - Ability to diagnose nondiverticular causes of abdominal pain
 - Accuracy enhanced with use of IV and PO/PR contrast
 - Gastrografin PO/PR (per rectum) contrast may be used; avoid barium, especially when perforation is suspected
- Plain radiographs: Chest/abdomen

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC
 - Leukocytosis common, but absence does not exclude diagnosis
- UA
 - Sterile pyuria is possible
 - Colonic flora (bacteria) suggests colovesical fistula

Imaging

- Abdominal (supine and upright) and chest radiographs
 - Perforation indicated by free air
 - Obstruction indicated by air–fluid levels
- CT
 - Diagnostic criteria include:
 - Wall thickening >5 mm

- Inflammation of pericolic fat
- Pericolic abscess
- Nondiagnostic criteria include:
 - Stricture
 - Diverticula
 - Fistula
- CT-guided percutaneous needle aspiration of localized abscesses avoids further surgery.
- Endoscopy
 - Not necessary to diagnose acute illness
 - Rigid sigmoidoscopy aids in diagnosing nondiverticular causes of abdominal pain (spasm, stricture, edema, pus, or peridiverticular erythema).
- US
 - For diagnosing colonic wall thickening, inflammation, mass, abscess, or fistula
 - Greatly operator dependent
 - Not reliable in presence of intestinal gas
- Barium enema
 - Indicated after resolution of acute illness to rule out fistula or other colonic pathology (e.g., carcinoma)

DIFFERENTIAL DIAGNOSIS

- Colon carcinoma with perforation
- Ischemic colitis
- Bacterial colitis
- Appendicitis
 - Left-sided pain if peritonitis from ruptured appendix
 - Right-sided diverticular pain with cecal diverticulum (rare) or redundant sigmoid colon
- Inflammatory bowel disease
- Irritable bowel syndrome
- Ruptured or torsed ovarian cyst
- Pancreatic disease
- Pelvic inflammatory disease
- Peptic ulcer disease
- Renal colic



TREATMENT

PRE HOSPITAL

IV fluids

INITIAL STABILIZATION/THERAPY

- Fluid resuscitation with 0.9% normal saline
- Bowel rest
 - NPO or clear liquid diet
 - Nasogastric tube (NG) tube if persistent vomiting or bowel obstruction suspected

ED TREATMENT/PROCEDURES

- Uncomplicated diverticulitis
 - Most respond to medical therapy, but 30% may require surgery
- Complicated diverticulitis
 - Most require percutaneous drainage or surgery
- Analgesia
 - Anticholinergics (dicyclomine):
 - Reduces colonic spasm
 - Does not mask underlying pathology
 - Opiates for more aggressive pain management (theoretically increase intraluminal pressure, leading to perforation)
 - Do not use if hemodynamically unstable
- Antibiotics to cover gram-negative aerobic and anaerobic bacteria:
 - Mild, uncomplicated cases (peridiverticulitis) for outpatient management:
 - Ciprofloxacin or levaquin + metronidazole or clindamycin
 - Trimethoprim/sulfamethoxazole (TMP/SMX) DS + metronidazole
 - Amoxicillin/clavulanate
 - Duration of therapy is 10–14 days
 - Moderate uncomplicated and mild complicated cases for inpatient management:
 - Ceftriaxone or other 3rd-generation cephalosporin + metronidazole or clindamycin
 - Ampicillin/sulbactam
 - Piperacillin/tazobactam
 - Ticarcillin/clavulanate
 - Ciprofloxacin or levaquin + metronidazole or clindamycin
 - Aztreonam
 - Complicated cases (with peritonitis from perforation), consider:
 - Imipenem/cilastatin
 - Meropenem
 - Aztreonam + metronidazole or clindamycin
 - Gentamicin + metronidazole or clindamycin ± ampicillin
 - Trovafloxacin (alternative)
- Surgery:
 - Emergent surgery:
 - Indicated for generalized peritonitis from perforation

- 2-stage procedure with resection of diseased segment of colon and proximal colostomy followed later with reanastomosis
- Elective surgery:
 - Indicated for multiple recurrent attacks (>2) without generalized peritonitis (controversial); fistula formation; intractable pain; unresolved obstruction; failure of medical therapy; single serious attack in patient <50 yr of age (controversial)
 - 1-stage procedure following resolution of inflammation from medical therapy
 - Nonoperative management may be considered for complicated diverticulitis.
- Peridiverticular abscess drainage:
 - Indicated if well circumscribed and easily accessible
 - Accomplished by CT- or ultrasound-guided percutaneous needle aspiration
- Outpatient therapy:
 - Clear liquids with follow-up in 2–3 days
 - When acute condition has resolved:
 - High-fiber, low-fat diet to decrease recurrence of attacks

MEDICATION

- Amoxicillin/clavulanate: 500/125 mg PO TID or 875/125 mg PO BID
- Ampicillin: 2 g IV q6h
- Ampicillin/sulbactam: 3 g IV q6h
- Cefotetan: 2 g IV q12h
- Cefoxitin: 2 g IV q8h
- Ciprofloxacin: 400 mg IV q12h or 500 mg PO BID
- Dicyclomine: 20 mg PO QID (up to 40 mg PO QID) or 20 mg IM q6h (*not* for IV use)
- Gentamicin: Multiple daily dose (MDD) regimen, 2 mg/kg load, then 1.7 mg/kg IV q8h, or once-daily dose (OD) regimen, 5–7 mg/kg IV q24h (assuming normal renal function)
- Imipenem/cilastatin: 500 mg IV q6h
- Meropenem: 1 g IV q8h
- Metronidazole: 1 g (15 mg/kg) IV load then 500 mg IV q8h or 500 mg PO q8h
- Piperacillin/tazobactam: 3.375 g IV q6h or 4.5 g IV q8h
- Ticarcillin/clavulanate: 3.1 g IV q6h
- Trimethoprim/sulfamethoxazole DS: 1 tablet PO BID
- Trovafloxacin: 300 mg IV for 1st dose, then 200 mg IV/PO daily

First Line

- Uncomplicated diverticulitis (outpatient), 10–14 days
 - Amoxicillin–clavulanate 875/125 mg PO BID

- Trimethoprim/sulfamethoxazole DS 1 tablet PO BID AND metronidazole 500 mg PO q6h
- Ciprofloxacin 500 mg PO BID AND metronidazole 500 mg PO q8h
- For patients intolerant of metronidazole, consider clindamycin
- Complicated diverticulitis
 - Ticarcillin/clavulanate: 3.1 g IV q6h or
 - Ampicillin/sulbactam: 3 g IV q6h or
 - Ceftriaxone 1 g IV q24h AND metronidazole 500 mg IV q8h
 - Levofloxacin 500 mg or 750 mg IV q24h (or ciprofloxacin 400 mg IV q12h) AND metronidazole 1 g IV q12h
 - Imipenem 500 mg IV q6h or meropenem 1 g IV q8h



FOLLOW-UP

DISPOSITION

Admission Criteria

- Intractable pain and/or vomiting
- High fever
- Peritonitis
- Failure to respond to outpatient management
- Severe disease on CT scan
- Significant leukocytosis
- Immunocompromised or steroid-dependent patients
- Recurrent episodes
- Comorbidities: Renal insufficiency, liver dysfunction, COPD, diabetes with end-organ damage
- Extremes of age
- Uncertainty of diagnosis

Discharge Criteria

- Mild cases (low-grade fever, mild discomfort) of known diverticular disease
- Minimal comorbidities
- Tolerating PO

Issues for Referral

Massive diverticular bleeding requiring GI or surgical consultation

FOLLOW-UP RECOMMENDATIONS

- Clear liquids
- Clinical improvement should be seen in 3 days, after which diet can be advanced
- Advise patients to call for increasing pain, fever, or inability to tolerate PO

- Colonoscopy (or contrast enema x-ray with flexible sigmoidoscopy) should be obtained after resolution of initial episode
- Patients do NOT need to avoid seeds and nuts

PEARLS AND PITFALLS

- CT scanning differentiates diverticulitis as complicated or uncomplicated:
 - Surgery reserved for complicated cases, but nonoperative management becoming more prevalent
- Most cases of uncomplicated diverticulitis rarely progress to complicated disease
 - Multiple attacks do not seem to lead to increased complications.
- Diverticulitis does not seem to be a progressively worsening process
 - Acute episodes can present at any stage.
- Severe disease on initial CT scan
 - Increased risk of failure of medical therapy
 - High risk of secondary complications

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See Also (Topic, Algorithm, Electronic Media Element)

Diverticulosis



CODES

ICD9

- 562.11 Diverticulitis of colon (without mention of hemorrhage)
- 562.13 Diverticulitis of colon with hemorrhage

ICD10

- K57.20 Diverticulitis of large intestine with perforation and abscess without bleeding
- K57.32 Diverticulitis of large intestine without perforation or abscess without bleeding
- K57.92 Diverticulitis of intestine, part unspecified, without perforation or abscess without bleeding

DIVERTICULOSIS

Ronald E. Kim

BASICS

DESCRIPTION

- Single (diverticulum) or multiple (diverticula) colonic wall outpouchings from colonic muscle dysfunction, usually acquired
- Sequence:
 - Insufficient amounts of dietary fiber cause diminished stool bulk
 - Increased colonic contractions to propel stool through colon cause increase in intraluminal pressure
 - Increased pressure forces mucosa and submucosa to herniate through muscularis propria at its weakest point, where vasa recta penetrate

ETIOLOGY

- Occurs anywhere in GI tract, although generally a colonic disease:
 - Left sided 95% (Western countries)
 - Right sided 70% (Asian countries)
 - Sigmoid colon most common site
- Pseudodiverticula:
 - Outpouchings of mucosa and submucosa only
 - Most common form of colonic diverticula
 - True congenital diverticula (uncommon) contain all bowel wall layers.
- Common in Western society, owing to refined diet and low intake of fiber
- Prevalence is age-dependent
 - 30% by 50 yr old, 65% by 85 yr old
- Complications
 - 70% are asymptomatic
 - 15–25% develop diverticulitis
 - 5–15% develop bleeding; obesity is a risk factor
 - Bleeding stops spontaneously in 75% of cases
 - Inflammation (diverticulitis)
 - Massive arterial bleeding usually from right colon:
 - Fecalith (dry, hard stool) erodes through arterial branch.
 - Perforation
 - Abscess
 - Obstruction

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Chronic or intermittent abdominal pain
 - _ Often precipitated by eating
 - _ Sometimes relieved by flatulence or bowel movement
- Change in bowel pattern
 - _ Constipation or diarrhea
- Dyspepsia
- Painless hematochezia; 75% self-limiting
 - _ Left colon origin: Bright red
 - _ Right colon origin: Dark or maroon colored, mixed with stool
- Diverticulitis and diverticular bleeding are separate entities and rarely coexist.

Physical-Exam

- Afebrile
- Abdomen typically benign, but presentation variable
 - _ Tenderness in left lower quadrant
 - _ Firm sigmoid colon in left lower quadrant
- Rectal exam variable
 - _ Heme-negative stool
 - _ Blood if diverticular bleed

ESSENTIAL WORKUP

Thorough history and physical exam essential to avoid excessive workup

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Asymptomatic diverticulosis
 - _ Requires no workup
- Recurrent uncomplicated painful disease
 - _ Requires no workup
- New onset uncomplicated painful disease
 - _ Requires workup to rule out carcinoma (if weight loss, anorexia, heme-positive stool)
 - _ CBC for leukocytosis or anemia
 - _ Urinalysis to exclude hematuria or pyuria
- Hemorrhagic diverticulosis
 - _ CBC
 - _ Electrolytes, BUN, creatinine, glucose, calcium
 - _ Type and cross for 4 units of packed RBCs
 - _ PT, PTT, INR

- ECG

Imaging

- Uncomplicated painful diverticulosis—outpatient options
 - Flexible sigmoidoscopy, then barium enema
 - Sigmoidoscopy: Rule out carcinoma (before barium studies for optimal visualization)
 - Barium enema: Search for classic diverticula and exclude carcinoma or polyps
 - Colonoscopy
- Hemorrhagic diverticulosis
 - Anoscopy
 - If mild bleeding, to rule out hemorrhoids
 - Massive bleeding from hemorrhoids is rare
 - Proctosigmoidoscopy
 - If no blood in stool above rectum, assume rectal bleed
 - Colonoscopy
 - Cannot perform if bleeding excessive (difficult to visualize pathology)
 - Allows for therapeutic intervention
 - Usually done prior to radionuclide imaging or angiography
 - Radionuclide imaging
 - Safe, no bowel prep needed
 - Poor localization of bleeding site
 - Ideal for detecting intermittent bleeding, owing to long half-life of radioisotope (24–36 hr)
 - No potential for therapeutic intervention, but helpful prior to angiography
 - Angiography
 - Helpful if bleeding site cannot be identified by colonoscopy; must be actively bleeding at least 0.5 mL/min
 - Localizes site of bleeding (more exact after radionuclide scanning)
 - Allows for therapeutic intervention
 - Risk of intestinal infarction
 - Barium enema
 - Rarely indicated, but most sensitive for diagnosis
 - Identifies diverticula but not bleeding (can hinder visualization of other imaging techniques)

DIFFERENTIAL DIAGNOSIS

- Painful diverticulosis
 - Irritable bowel syndrome (almost identical clinical presentation)
 - Diverticulitis
 - Colon carcinoma

- Crohn's disease
- Urologic (renal colic)
- Gynecologic (ruptured or torsed ovarian cyst)
- Hemorrhagic diverticulosis
 - Hemorrhoids
 - Anal fissure
 - Proctitis
 - Colitis
 - Carcinoma
 - Polyps
 - Ischemic enteritis
 - Angiodysplasia
 - Amyloidosis
 - Vascular-enteric fistula
 - Upper GI source



TREATMENT

PRE HOSPITAL

- Avoid opiates in abdominal pain when underlying cause is uncertain.
- Establish 2 large-bore IV lines
- For significant bleeding or hypotension:
 - 1–2 L (20 mL/kg) bolus 0.9% NS intravenously
 - Trendelenburg position

INITIAL STABILIZATION/THERAPY

- Hemorrhagic diverticulosis (massive):
 - Airway control (100% O₂ or intubate if unresponsive)
 - Intravenous access with at least 1 large-bore catheter or 2 if unstable
 - 0.9% NS bolus 1–2 L (20 mL/kg) for hypotension
 - Central catheter placement if unstable following initial fluid resuscitation for more efficient delivery of fluids and monitoring of central venous pressure
 - Consider nasogastric tube to rule out upper GI bleed
 - Bladder catheter to monitor urine output
 - Transfuse O-negative RBCs immediately if arrest is impending
 - Consult surgeon for persistent bleeding, impending hemorrhagic shock (most diverticular bleeding stops spontaneously)

ED TREATMENT/PROCEDURES

- Uncomplicated symptomatic diverticulosis
 - High-fiber diet and/or hydrophilic bulk laxative (i.e., psyllium)

- Warm compresses to abdomen
- Reassurance
- Avoid cathartic laxatives
- No evidence to support use of antispasmodic (dicyclomine)
- Hemorrhagic diverticulosis (massive):
 - Initial stabilization (see above)
 - Colonoscopy is diagnostic and potentially therapeutic
 - Radionuclide scan; sensitive and noninvasive, but requires active bleeding
 - Selective angiography with injection of vasopressin to control bleeding
 - Embolization, interventional radiology; consider before surgery
 - Surgical intervention for segmental colectomy

MEDICATION

- Dicyclomine: 20 mg PO/IM QID (not for IV use)
- Propantheline: 15 mg PO 30 min ac/qhs



FOLLOW-UP

DISPOSITION

Admission Criteria

- ICU if unstable with massive hemorrhagic diverticulosis
- Mild or intermittent hemorrhagic diverticulosis that is otherwise stable so as to determine site of bleeding and evaluate need for definitive treatment

Discharge Criteria

- Uncomplicated, symptomatic diverticulosis
- Stable with trace heme-positive stool, negative gastric aspirate, no anemia, and no other complaints

Issues for Referral

GI follow-up for colonoscopy

FOLLOW-UP RECOMMENDATIONS

- Colonoscopy within 48 hr of initial presentation for stable patients
- Discontinue aspirin and NSAIDs
- Increase intake of dietary fiber
- No evidence for avoidance of nuts, corn, popcorn

PEARLS AND PITFALLS

- 15% with hematochezia have an upper GI source

- Most cases (75–95%) resolve spontaneously or with conservative management
- Massive blood loss seen in 9–19% of patients, especially those with comorbid diseases or advanced age
- Colonoscopy is the initial diagnostic procedure of choice in stable patients

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See Also (Topic, Algorithm, Electronic Media Element)

- Diverticulitis
- GI Bleeding



CODES

ICD9

- 562.10 Diverticulosis of colon (without mention of hemorrhage)
- 562.12 Diverticulosis of colon with hemorrhage
- 751.5 Other anomalies of intestine

ICD10

- K57.30 Dvrtclos of lg int w/o perforation or abscess w/o bleeding
- K57.31 Dvrtclos of lg int w/o perforation or abscess w bleeding
- Q43.8 Other specified congenital malformations of intestine

DIZZINESS

Michael Bouton • Jonathan A. Edlow

BASICS

DESCRIPTION

- Patients' descriptions of symptom quality (vertigo, lightheadedness, disequilibrium, or "other") are frequently misleading and should not be the basis of clinical decision making.
- An approach based on associated symptoms, timing and triggers of the dizziness followed by a targeted physical exam looking for telltale signs is less prone to subjective errors of language and possibly more likely to yield a specific diagnosis.
- There are 4 "timing and triggers" categories:
 - Acute vestibular syndrome (AVS)
 - Abrupt onset of persistent dizziness
 - Episodic vestibular syndrome (EVS)
 - Spontaneous episodes of dizziness lasting many minutes to hours
 - Positional vestibular syndrome (PVS)
 - Very brief episodes (usually lasting 20–50 sec) that are triggered by head or body position movement
 - Chronic vestibular syndrome (CVS)
 - Gradual onset of dizziness lasting weeks to months or longer

ETIOLOGY

- General medical (49%):
 - Arrhythmia
 - Hypoglycemia and other toxic metabolic causes
 - Hypovolemia of any cause
 - Sepsis and infections
 - Low cardiac output states of any cause
- Otologic/vestibular (33%):
 - Benign paroxysmal positional vertigo (BPPV)
 - Labyrinthitis and vestibular neuritis
- Neurologic (11%):
 - Stroke and transient ischemic attack (TIA)
 - Vestibular migraine
- Psychiatric (7%):
 - Anxiety and depression

DIAGNOSIS

SIGNS AND SYMPTOMS

History

Define the timing and triggers category and determine if the ROS suggests a particular serious diagnosis:

- Is the dizziness abrupt or gradual in onset?
- Is the dizziness intermittent or persistent?
- If intermittent, how long do episodes last?
- If intermittent, are the episodes triggered by head or body position movement?
- Are there any hearing or neurologic symptoms?
- Has the patient had recent head injury or started any new medications?
- Does the ROS suggest an acute medical issue; not an encyclopedic list, but examples include:
 - Headache – stroke, dissection, or tumor
 - Ear pain – mastoiditis, otitis media
 - Hearing changes – Ménière disease or labyrinthitis
 - Neck pain – vertebral dissection
 - Fever – systemic infection
 - Dyspnea – pulmonary embolism, pneumonia, or anemia
 - Chest pain – ACS or pulmonary embolism
 - Fluid losses – orthostatic hypotension, hypovolemia
 - Pregnancy – ectopic pregnancy, pre-eclampsia

ALERT

Exacerbation of dizziness with head motion occurs with both central and peripheral causes. However, new dizziness with head motion in a patient who is entirely asymptomatic at rest suggests a peripheral cause.

Physical-Exam

- Vital signs
- Stand patient to test for clinical signs of orthostatic hypotension
- Otoscopic evaluation
- Cardiac exam – is there a murmur or S3?
- Neurologic exam
 - CN II-XII. In particular, is there nystagmus, and if so, what type (see below)?
 - Observe gait
 - Cerebellar exam (finger to nose/heel to shin)
 - Dix-Hallpike maneuver only for intermittent symptoms
- HINTS exam (only for patients with AVS)
 - This is a 3-part more detailed oculomotor exam (head impulse test, nystagmus testing, and test for skew deviation)
 - For acute (< 48 hr) of symptoms this exam has been shown to be more

sensitive than MRI. If exam is concerning obtain MRI or neurology consultation.

- Head impulse testing (vestibulo-ocular reflex)
 - Patient fixes gaze on examiner's nose
 - Move patient's heads rapidly about 20° in the horizontal plane
- If reflex is intact their eyes will stay fixed on your nose (vestibulo-ocular reflex is intact) and a central cause such as cerebellar stroke may be at play. If there is a corrective saccade (eye moves with head and then snaps back toward your nose), this suggests a peripheral cause (vestibular neuritis or labyrinthitis)
- Nystagmus
 - Have patient track your finger to all visual fields.
 - Does the direction of horizontal nystagmus change with change in direction of gaze? (i.e., when patient looks left, is fast component beating to left; when patient looks right, is fast component toward the right)?
 - Direction-changing, vertical or torsional nystagmus (in a patient with the AVS) strongly suggests a central cause.
 - Direction-fixed nystagmus (always in same direction independent of direction of gaze) suggests peripheral cause.
- Tests of skew
 - Alternating cover test
 - Have the patient look at your nose and cover one of their eyes with your hand
 - Rapidly uncover the 1st eye; cover the other one and observe if there is a rapid vertical eye movement (the amplitude can be quite small).
 - Continue to alternately cover and uncover each eye (focusing on 1 eye) in rapid succession.
 - A rapid vertical corrective saccade (up or down) strongly suggests a central process.

ALERT

Each of the components of HINTS individually is not sufficiently sensitive to rule out a central cause. If any one of them is worrisome, assume stroke. Remember that it is the negative head impulse test (no corrective saccade) that is worrisome in patients with AVS.

ESSENTIAL WORKUP

The only mandatory workup is history and physical exam. Using these, one can often make a specific diagnosis.

- Triage: Identify abnormal vital signs, changes in mentation or gross focal deficits in primary survey
 - Focused history to elicit other complaints such as chest pain, headache, and change in hearing that will guide evaluation

- **Timing:** Distinguish between intermittent and chronic symptoms considering relevant conditions for each
- **Triggers:** For intermittent symptoms consider the immediate context of episodes
- **Telltale signs:** HINTS exam for acute dizziness

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Serum glucose
- Hematocrit, if suspected anemia/blood loss
- Electrolytes and renal function
 - VBG if considering CO poisoning or CO₂ narcosis
- UA to evaluate for infection
- Toxicologic screen, if suspected exposure

Imaging

- CT head if acute bleed suspected
 - CT only ~ 40% sensitive for ischemic posterior circulation stroke
- MRI if no other etiology found and HINTS exam concerning in a patient with the AVS

Diagnostic Procedures/Surgery

- Dix-Hallpike maneuver, head thrust maneuver, and test for skew deviation.
- EKG to detect arrhythmia, MI
- Lumbar puncture in setting of unexplained infectious signs or headache

DIFFERENTIAL DIAGNOSIS

Each of the timing and triggers categories has its own differential diagnosis. Here are the common and the dangerous causes:

- **AVS – acute vestibular syndrome**
 - Benign
 - Viral labyrinthitis (hearing involved)
 - Vestibular neuritis (hearing not involved)
 - Dangerous
 - Stroke, particularly brainstem or cerebellar
 - Occasionally low cardiac output state (e.g., PE, ACS)
- **EVS – episodic (spontaneous) vestibular syndrome**
 - Benign
 - Vestibular migraine
 - Dangerous
 - TIA
 - Rarely, brief low cardiac output state (e.g., arrhythmia, PE that

breaks up and migrates)

- **PVS – positional (triggered) vestibular syndrome**
 - Benign
 - BPPV
 - Orthostatic hypotension (if benign cause)
 - Dangerous
 - Orthostatic hypotension (if serious cause)
 - Rarely, CPPV (central paroxysmal positional vertigo) caused by a posterior fossa mass
- **CVS – chronic vestibular syndrome**
 - Benign
 - Psychiatric causes (anxiety and depression)
 - Benign medication side effects
 - Dangerous
 - Rarely a posterior fossa mass



TREATMENT

INITIAL STABILIZATION/THERAPY

- Abnormal vital signs clinically managed
- Stabilization should be determined by more specific classification of dizziness based on the history, physical exam, and ancillary studies.

ED TREATMENT/PROCEDURES

Symptomatic control until diagnosis established

If BPPV suspected perform Epley maneuver

MEDICATION

- Ondansetron: 4 mg IV or ODT
- Diazepam: 2.5–5 mg IV or 2–10 mg PO
- Diphenhydramine: 25–50 mg IV, IM, or PO
- Meclizine: 25 mg PO (no more than 2–3 days)
- Promethazine: 12.5 mg IV q6h or 25–50 mg PO, IM, or PR q6h

Note: These medications are for symptom relief; response has no etiologic implications.



FOLLOW-UP

DISPOSITION

Admission Criteria

Admission or discharge of patients with dizziness should be based on the underlying etiology or associated symptoms.

Discharge Criteria

- Admission or discharge of patients with dizziness should be based on the underlying and the patient's ability to function safely at home.
- If patient has isolated complaint of dizziness with normal neurologic and oculomotor testing as described above, consider discharge with follow-up instructions

Issues for Referral

Refer for completion of workup as an outpatient to a primary care physician, ENT, or neurologist depending upon likely cause.

FOLLOW-UP RECOMMENDATIONS

- The patient should be instructed:
 - Not to drive or operate machinery if he is feeling dizzy
 - To get up slowly after sitting or lying down
- Patient should return to the ED or see his doctor right away if:
 - Symptoms of neurologic problem (worsening headache, confusion, memory loss, new motor or sensory loss)
 - Symptoms of an infection (stiff neck, fevers, or chills)
 - Symptoms of acute cardiovascular or pulmonary problem (new acute abdominal chest or back pain, new dyspnea, or hemoptysis)
 - Symptoms of fluid losses (intractable emesis or stools, GI or vaginal bleeding)

PEARLS AND PITFALLS

- Use the “timing and triggers” technique to diagnose dizzy patients.
- Advanced age and traditional stroke risk factors increase the likelihood of acute stroke as cause of dizziness.
- Noncontrast CT is NOT sensitive for acute cerebellar stroke.
- Patients with cerebellar stroke can present with isolated dizziness.
- It is a “negative” head impulse test (lack of corrective saccade) that is worrisome; a “positive” test suggests a peripheral vestibular etiology.
- The treatment for BPPV is an Epley maneuver, NOT meclizine.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Vertigo



CODES

ICD9

- 386.11 Benign paroxysmal positional vertigo
- 386.30 Labyrinthitis, unspecified
- 780.4 Dizziness and giddiness

ICD10

- H81.10 Benign paroxysmal vertigo, unspecified ear
- H83.09 Labyrinthitis, unspecified ear
- R42 Dizziness and giddiness

DOMESTIC VIOLENCE

Laura G. Burke

BASICS

DESCRIPTION

- Intimate partner violence (IPV) is the physical, sexual, or psychological abuse by a current or former partner.
- Occurs in adult and adolescent intimate relationships across the socioeconomic spectrum.

ETIOLOGY

- Most victims are women injured by male perpetrators
- Men and individuals in same-sex relationships may also be victims.
- Risk factors for IPV include female sex, young age (20–24) and being separated from partner/spouse.

DIAGNOSIS

Asking specifically about IPV increases likelihood of identifying victims.

SIGNS AND SYMPTOMS

- Traumatic injuries:
 - Wide variety of presentations
 - Unwitnessed head, neck, facial injuries are common
 - Forearm bruises or fractures suggesting a defensive posture
 - Injuries in various stages of healing
- Psychiatric:
 - Chronic pain syndromes
 - Depression
 - Somatization
 - Anxiety
 - Suicidality
 - Substance abuse

ALERT

Clinical clues:

- Discrepancy between history and physical findings
- Partner refusing to allow patient to be alone with provider
- Delay in seeking care
- Any injury during pregnancy
- Interaction between patient and partner that suggests interpersonal problems

- Multiple symptoms without obvious physical findings

History

- Screening questions for IPV may be useful in identifying victims of domestic violence.
- Controversial as to whether available evidence demonstrates that screening improves health outcomes.
- IPV screening is required by the Joint Commission and supported by some professional medical organizations.
- Screening should be direct, nonjudgmental, supportive, and private.
- There is some evidence for effectiveness of computer-based screening of ED patients for IPV.
- Consider IPV in patients with substance abuse/intoxication as they may be at greater risk and less likely to be identified.

Physical-Exam

- Careful exam for traumatic injuries
- Mental status exam

ESSENTIAL WORKUP

- After identification of IPV, a directed workup for traumatic injuries and acute medical or behavioral health illnesses is warranted.
- Assess patient's risk for future injury/victimization

DIFFERENTIAL DIAGNOSIS

Important to maintain high index of suspicion for IPV in patients with traumatic injuries, behavioral health problems, and medical complaints (e.g., GU, GYN, multiple somatic complaints)



TREATMENT

PRE HOSPITAL

- Customary trauma evaluation and treatment
- An accurate description of events by EMS should be incorporated into the medical record.

INITIAL STABILIZATION/THERAPY

- Provide timely and appropriate medical attention.
- Provide appropriate emotional support throughout workup and treatment

ED TREATMENT/PROCEDURES

- Interview the patient in a private, secure location without any family members present

- Use a medical interpreter (not family members) to conduct in an interview when there is a language barrier.
- Provide complete, careful documentation including use of the patient's exact words, as they are admissible in court.
- Carefully document extent and location of injuries. Diagrams or photographs are particularly useful.
- If stable for discharge, assess situation for lethality
 - Risk factors include violence that is increasing in frequency and severity, threats of homicide or suicide by the partner, or the availability of a lethal weapon.
 - Work with the patient to develop optimal discharge plan that is consistent with his/her wishes.
- Arrange referrals and follow-up:
 - Outpatient victim services
 - Emergency shelter information
 - Hotlines
 - Restraining order information
 - Legal services
- Mandatory reporting requirements vary among states:
 - Reporting requirements for IPV vary by state.
 - Mandatory reporting may place the victim in more danger and create ethical dilemmas for the physician when the victim does not want the case reported to police or social service agencies.
 - Inform victims of any requirement to report to authorities and possible outcomes of reporting.

MEDICATION

- Acetaminophen: 650–975 mg PO
- Morphine sulfate: 0.1 mg/kg/dose IV or IM



FOLLOW-UP

DISPOSITION

Admission Criteria

- Use appropriate admission guidelines depending on degree of trauma sustained.
- A patient who is medically stable for discharge but whose safety is at imminent risk may require hospitalization until a safe discharge plan is developed.

Discharge Criteria

A victim whose safety is ensured and whose injuries can be managed as an outpatient may be discharged.

Issues for Referral

Availability of advocacy services varies considerably.

FOLLOW-UP RECOMMENDATIONS

Provide information regarding outpatient services and emergency shelter information.

PEARLS AND PITFALLS

- Failure to consider IPV in the differential diagnosis of the patient's chief complaint.
- Failure to provide thorough, objective documentation of the details of the assault and physical exam findings.
- Failure to adequately assess patient's safety upon discharge and provide appropriate referrals
- Mandatory reporting laws remain controversial and may cause unintended consequences for the patient.

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See Also (Topic, Algorithm, Electronic Media Element)

- Elder Abuse
- Trauma



CODES

ICD9

- 995.81 Adult physical abuse
- 995.82 Adult emotional/psychological abuse

- 995.83 Adult sexual abuse

ICD10

- T74.11XA Adult physical abuse, confirmed, initial encounter
- T74.21XA Adult sexual abuse, confirmed, initial encounter
- T74.31XA Adult psychological abuse, confirmed, initial encounter

DROWNING

Colleen N. Hickey



BASICS

DESCRIPTION

- Definitions:
 - Drowning: “A process resulting in primary respiratory impairment from submersion or immersion in a liquid medium”
 - Fatal drowning: Death at any time as a result of drowning
 - Nonfatal drowning: If the victim is rescued at any time and the process of drowning is interrupted
 - Water rescue: Any submersion or immersion incident without evidence of respiratory impairment
- Scenario of drowning:
 - Now thought all drowning victims aspirate some amount of liquid
 - Previously classified as “wet” and “dry” drowning:
 - “Wet” drowning (90%): Aspiration of small amount of liquid into the lungs
 - “Dry” drowning (10%): Laryngospasm secondary to the presence of liquid in the oropharynx or larynx
 - End result: Hypoxia
 - No significant difference between freshwater and saltwater submersion
- Pathophysiology:
 - Aspiration:
 - Small volume of water
 - Decreased lung compliance causing ventilation/perfusion mismatch and intrapulmonary shunting
 - No significant electrolyte changes
 - Grossly contaminated water: Risk for pulmonary infection
 - Hypoxemia:
 - Metabolic lactic acidosis
 - Multisystem organ dysfunction
 - Noncardiogenic pulmonary edema
 - Myocardial dysfunction (arrhythmias)
 - Coagulation abnormalities (disseminated IV coagulation)
 - Renal failure (usually acute tubular necrosis)
 - Cerebral hypoxia: Cerebral edema, increased intracranial pressure

Pediatric Considerations

- Hypothermia:

- More common in young children
- Larger body surface-to-mass ratio
- Decreases the metabolic rate
- Survival with full recovery is possible (neuroprotective)
- Diving reflex:
 - Young children are more susceptible
 - Triggered by submersion of face in cold water
 - Bradycardia ensues: Redistribution of blood flow to the heart and brain
 - Delays onset of hypoxia-related damage

ALERT

Risk factors:

- Lack of proper supervision
- Alcohol or other drug abuse
- Limited swimming ability or exhaustion
- Trauma
- Seizure disorder
- Risky behavior
- Pre-existing medical problem
- Attempted suicide
- Poor education



DIAGNOSIS

SIGNS AND SYMPTOMS

- Cardiopulmonary arrest
- Cyanosis
- Dyspnea
- Copious pulmonary secretions
- Loss of consciousness
- Cerebral edema/injury
- Evidence of trauma:
 - Intracranial hemorrhage
 - Cervical spine injury rare (< 0.5%)
- Hypothermia

ESSENTIAL WORKUP

- Information from witnesses or emergency medical services personnel at the scene
- Early airway management and CPR
- Rectal temperature for hypothermia

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Arterial blood gas (pH)
- CBC
- Electrolytes, BUN, creatinine, glucose:
 - Usually normal (>85%)
 - Hyponatremia or hyponatremia
- Alcohol and toxicology screen

Imaging

- CXR:
 - Diffuse or focal infiltrates, acute respiratory distress syndrome
 - May be normal initially
- ECG:
 - Long QT interval
 - Sinus bradycardia
 - Sinus tachycardia
 - Atrial fibrillation
- CT scan:
 - Brain: Abnormality at any time during hospitalization is associated with poor neurologic outcome
 - Cervical spine: Traumatic injury

DIFFERENTIAL DIAGNOSIS

- Consider reason for submersion:
 - Dysrhythmia (long QT syndrome, familial polymorphic ventricular tachycardia)
 - Myocardial infarction
 - Seizure
 - Syncope
 - Trauma
 - Suicide attempt

Pediatric Considerations

Consider child abuse/neglect:

- Especially infants in bathtub near drowning



TREATMENT

PRE HOSPITAL

- Attention to ABCs:
 - Avoid further aspiration
 - Secure airway—intubate

- Early CPR
- Cervical spine precautions if injury suspected or concerning mechanism
- Early rewarming attempts
- 90% survival with appropriate intervention
- All drowning victims need ED evaluation
- Abdominal thrust to remove water *not* recommended:
 - Useful only if foreign body in airway
 - Increases risk for aspiration
 - Delays effective CPR

INITIAL STABILIZATION/THERAPY

- ABCs
- Core temperature:
 - Initiate rewarming (see “Hypothermia”)
 - Remove wet clothing

ED TREATMENT/PROCEDURES

- Correct hypoxemia:
 - Titrate to oxygen saturation
 - Intubate and provide mechanical ventilation with positive end-expiratory pressure
- Evaluate and treat traumatic injuries
- Correct acidosis
- Cardiopulmonary arrest:
 - Initiate advanced cardiac life support measures
 - Continue rewarming efforts:
 - Passive: Blankets, insulators
 - Active external: Warm blankets, radiant heat, warm baths
 - Active internal: Pleural or peritoneal lavage, cardiopulmonary bypass
 - Continue resuscitation until core temperature $> 32^{\circ}\text{C}$ or until spontaneous pulse and respirations return
- No value to steroids
- Poor prognostic signs:
 - Prolonged submersion time
 - Severe acidosis ($\text{pH} \leq 7.0$)
 - Need for CPR
 - Low oxygen saturation
 - Low Glasgow Coma Score (GCS)

MEDICATION

- Epinephrine: 1 mg (peds: 0.01 mg/kg) IV
- Vasopressin: 40 U (peds: 0.04 U/kg) IV
- Lidocaine: 1 mg/kg IV

- Sodium bicarbonate: 1 mEq/kg IV

Pediatric Considerations

- Hypothermia may be protective:
 - Aggressive rewarming
 - Aggressive resuscitation
- Evaluate for abuse/neglect
- Family history: Sudden death, similar episode:
 - Long QT syndrome
 - Familial polymorphic ventricular tachycardia
- Prevention is key to treatment:
 - Supervision around water
 - Empty pails and buckets

ALERT

Controversial: Therapeutic hypothermia

- Widely accepted in adult population after cardiac arrest with return of spontaneous circulation, still controversial in pediatrics
- Optimize neurologic outcome
- Suppress reperfusion injury



FOLLOW-UP

DISPOSITION

Admission Criteria

- Delayed symptomatology occurs:
 - Pulmonary edema (up to 12 hr later)
 - Neurologic abnormalities
- ICU:
 - Patients who required CPR or artificial ventilation
 - Abnormal chest radiograph
 - Arterial blood gas abnormalities
 - GCS < 13
- Admit observation status:
 - All symptomatic patients
 - Submersion for > 1 min
 - History of cyanosis or apnea
 - Patients who required brief assisted ventilation

Discharge Criteria

- Questionable history of submersion:

- Observe in ED for 8 hr:
 - No respiratory distress
 - No neurologic impairment
- Discharge to reliable home
- Home-going instructions:
 - Return for shortness of breath or mental status changes

FOLLOW-UP RECOMMENDATIONS

Close primary care follow-up for all patients discharged from ED

PEARLS AND PITFALLS

- All patients with drowning incident require at least 8 hr of observation
- Indicators of poor prognosis:
 - Acidemia (pH <7.1 on presentation)
 - Age <3 yr
 - Submersion >10 min
 - Time to basic life support care >10 min
 - GCS ≤ 5
 - Long transportation time to ED
 - Persistent apnea or need for cardiopulmonary resuscitation in the ED
 - Water temperature >10°C

ADDITIONAL READING

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- Szpilman D, Bierens JJ, Handley AJ, et al. Drowning. *N Engl J Med.* 2012;366(22):2102–2110.
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See Also (Topic, Algorithm, Electronic Media Element)

Hypothermia



CODES

ICD9

- 507.0 Pneumonitis due to inhalation of food or vomitus
- 991.6 Hypothermia
- 994.1 Drowning and nonfatal submersion

ICD10

- J69.0 Pneumonitis due to inhalation of food and vomit
- T68.XXXA Hypothermia, initial encounter
- T75.1XXA Unsp effects of drowning and nonfatal submersion, init

DUODENAL TRAUMA

Christanne H. Coffey



BASICS

DESCRIPTION

- Characteristics of duodenum:
 - 12 in long
 - C-shaped
 - From pylorus to ligament of Treitz
 - Divided into 4 sections:
 - Last 3 sections retroperitoneal along with distal portion of 1st section
 - Lies mostly over 1st 3 lumbar vertebrae
 - 2nd section is most commonly injured
- Types of injury:
 - Duodenal wall hematoma
 - Wall perforation
 - Hemorrhage, including retroperitoneal
 - Crush
- Incidence of duodenal injury is 3–5% of all traumatic abdominal injuries
- Penetrating trauma accounts for ~75% of duodenal injuries:
 - Mortality ranges from 13–28%
 - Associated with exsanguination
- Blunt duodenal trauma has a higher mortality due to greater force of injury and often delayed diagnosis due to retroperitoneal location:
 - If injury is diagnosed in < 24 hr, mortality rate is about 11%
 - If > 24 hr, mortality rate approaches 40%
 - Late mortality usually from sepsis

Pediatric Considerations

- Majority secondary to recreational injuries (e.g., bicycle handlebar injuries)
- Intramural duodenal hematomas may occur in nonaccidental trauma:
 - If suspected, prompt referral to appropriate child protective agency is required
- In children, hematoma is most commonly seen in 1st portion of duodenum

Pregnancy Considerations

- Retroperitoneal hemorrhage more common due to increased pelvic and abdominal vascularity
- Large uterus serves as protection from bowel injury.
- Peritoneal irritation is blunted in the pregnant patient; therefore, greater index of

suspicion

ETIOLOGY

- Blunt trauma:
 - Shear strain: Abrupt acceleration/deceleration at point of attachment (most common retroperitoneal injury with rapid deceleration)
 - Tensile strain: Direct compression or stretching of tissue
- Penetrating trauma:
 - Most common cause of injury
 - Creates cavitations, can lead to infection



DIAGNOSIS

SIGNS AND SYMPTOMS

- Complaints may be minimal with vague abdominal, flank, and back pain
- High GI obstruction may be seen with duodenal hematomas

History

Penetrating or blunt abdominal trauma

Physical-Exam

- Retroperitoneal: Often subtle, RUQ pain, nausea, vomiting, tachycardia, fever
- Intraperitoneal: Peritonitis

ESSENTIAL WORKUP

- Basic labs including amylase
- Acute abdominal series or CT
- Diagnostic peritoneal lavage (DPL) or ex lap if unstable, high suspicion

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Lab tests are of little value
- 50% of patients with duodenal injuries have elevated serum amylase
- An increasing leukocytosis may suggest undiagnosed duodenal injury

Imaging

- Focused assessment with sonography in trauma (FAST)
 - Validated for hemoperitoneum
 - Not reliable for duodenal injury
 - 1/3 retroperitoneal injuries with normal FAST
- Upright chest and abdominal radiographs:

- Intraperitoneal air
- Retroperitoneal air
- Air in biliary tree
- Scoliosis to the right
- Loss of psoas shadow
- Air around right kidney
- Injecting air into nasogastric tube may demonstrate retroperitoneal air more clearly
- Intramural hematomas without leakage may have coiled-spring appearance
- CT with oral and IV contrast:
 - Best imaging diagnostic test that shows small amounts of retroperitoneal gas and extravasated contrast material
 - Duodenal wall thickening, periduodenal fluid, “sentinel clot” adjacent to injury
 - Sausage-shaped mass in duodenal wall strongly suggests hematoma

Diagnostic Procedures/Surgery

- Ex lap is the ultimate diagnostic test when high suspicion remains, even after other diagnostic tests are negative
- DPL:
 - Often positive for blood, bile, or bowel content
 - Negative lavage does not exclude injury (65% false-negative rate)

DIFFERENTIAL DIAGNOSIS

- Injury to hollow organs (stomach, small and large intestines)
- Liver and biliary tree injuries
- Vascular injuries (aortic and mesenteric arteries as well as venous injuries)
- Postoperative complications from prior duodenal surgery or injury repair, such as infection and suture line dehiscence



TREATMENT

PRE HOSPITAL

- Follow trauma protocols
- Important to have pre-hospital personnel provide clear description of mechanism of injury and to transport to appropriate facility

INITIAL STABILIZATION/THERAPY

- Airway management, resuscitation as needed
- Aggressive fluid therapy with warmed normal saline or lactated Ringer solution if patient hypotensive; transfuse as indicated
- Central line may be needed for unstable patients

- Nasogastric decompression
- Early trauma surgical consultation

ED TREATMENT/PROCEDURES

- Tetanus and antibiotic prophylaxis for penetrating wounds
- Definitive treatment involves laparotomy with exploration of duodenum for injuries
- Low-grade (I or II) blunt duodenal injuries usually managed nonoperatively – 10% fail
- Broad-spectrum antibiotics to prevent sepsis in patients with perforation

MEDICATION

- Cefoxitin: 2 g (peds: 40 mg/kg) IV q6h or
- Levofloxacin 750 mg or Ciprofloxacin 400 mg q24h + Metronidazole 500 mg IV q8h



FOLLOW-UP

DISPOSITION

Admission Criteria

- All patients with duodenal injuries need admission to trauma surgical service
- Minor duodenal hematomas that do not require immediate surgery may require nasogastric decompression for obstruction (up to 7 days) and observation for possible expansion or rupture of the hematoma

Discharge Criteria

- No patient with identified traumatic duodenal injury should be discharged from the ED
- Complications: Intra-abdominal abscess, duodenal fistula, pancreatic fistula, sepsis

Issues for Referral

- Duodenal organ injury scale (DIS) by American Association for the Surgery of Trauma:

Grade Duodenal Injury Description

- | | |
|----|---|
| I | Hematoma: Single portion
Laceration: Partial thickness, no perforation |
| II | Hematoma: > 1 portion
Laceration: Disrupts < 50% circumference, spares duct
Lacerations only: |

III --Disrupts 50—75% circumference D2

--Disrupts 50—100% circumference

D1, D3, D4

Lacerations only:

IV --Disrupts >75% circumference D2

--Involves ampulla or CBD

V Laceration: Massive disruption duodenopancreatic complex Vascular-devascularization

- Majority injuries Grade II or Grade III
- 80% primary repairs

FOLLOW-UP RECOMMENDATIONS

- All patients with diagnosed duodenal injury should be admitted
- If diagnostic studies are negative, recommend follow-up with PMD within 24–48 hr
- Diet: Clear liquids, advance as tolerated

PEARLS AND PITFALLS

- Significant morbidity and mortality with delayed or missed diagnosis
- Physical exam can be misleading due to retroperitoneal location
- If continued high suspicion despite negative diagnostic tests, get surgical consult

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Abdominal Trauma, Blunt
- Abdominal Trauma, Imaging
- Abdominal Trauma, Penetrating



CODES

ICD9

- 863.21 Injury to duodenum, without open wound into cavity
- 863.31 Injury to duodenum, with open wound into cavity

ICD10

- S36.400A Unspecified injury of duodenum, initial encounter
- S36.420A Contusion of duodenum, initial encounter
- S36.430A Laceration of duodenum, initial encounter

DYSFUNCTIONAL UTERINE BLEEDING

Andrew J. French

BASICS

DESCRIPTION

- Abnormal uterine bleeding is an alteration in pattern or volume of normal menses
 - Typical blood loss during a normal menstrual cycle is 30–80 mL
 - Normal interval between menses 28 (+/– 7) days
- 2 classifications
 - **Dysfunctional uterine bleeding (DUB)**
 - Hormonally related
 - Anovulatory and ovulatory categories
 - Not due to organic or iatrogenic causes
 - Diagnosis of exclusion
 - **Organic uterine bleeding**
 - Bleeding related to systemic illness or disease of the reproductive tract

ETIOLOGY

- Anovulatory (most common):
 - Unopposed estrogen stimulation of proliferative endometrium
 - Alteration of neuroendocrine function due to:
 - Polycystic ovarian syndrome (PCOS)
 - Very low calorie diets, rapid weight change, intense exercise, anorexia
 - Psychological stress
 - Obesity
 - Drugs
 - Hypothyroidism
 - Primary hypothalamic dysfunction
- Ovulatory:
 - Inadequate uterine PGF₂ α
 - Increased uterine contractility
 - Excessive uterine prostacycline
 - Diminishes platelet function and increases uterine vasodilation

Pediatric Considerations

Anovulatory bleeding common in adolescence owing to immaturity of the hypothalamic–pituitary–ovarian axis

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Abnormal uterine bleeding in the absence of systemic or structural disease
- Most common in perimenarcheal, perimenopausal women
- Typically painless
- Anovulatory presentations:
 - Metrorrhagia:
 - Irregular bleeding between periods
 - Menorrhagia:
 - Regular periods with excess flow (> 80 mL) or >7 days of bleeding
 - Oligomenorrhea:
 - Periods with intermenstrual cycles > 35 days
 - Menometrorrhagia:
 - Excessive bleeding with and between menses

Physical-Exam

- Acne, hirsutism, obesity suggest PCOS
- Mild to moderate bleeding on pelvic exam
- Pallor, tachycardia, hypotension, orthostasis in severe cases
- Evaluate for trauma, foreign bodies

ALERT

It is rare for women to be hemodynamically unstable simply from DUB; if such instability is present, concern is for ectopic pregnancy or other cause for hemorrhage.

ESSENTIAL WORKUP

Pregnancy test

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Pregnancy test, CBC, PT/PTT
- May send iron studies, TSH, LH, FSH, prolactin level, cervical cultures for routine follow-up by primary medical doctor (PMD)/gynecology

Imaging

Pelvic ultrasound may show uterine, tubal, or ovarian abnormality; may be needed to rule out other organic or iatrogenic causes on differential diagnoses.

Diagnostic Procedures/Surgery

- Dilation and curettage (D&C) may be required for heavy bleeding unresponsive to other interventions

- Refer for endometrial biopsy if > 35 yr of age

DIFFERENTIAL DIAGNOSIS

Organic/Iatrogenic

- Pregnancy complications:
 - Threatened, incomplete, or spontaneous abortion
 - Ectopic pregnancy
 - Molar pregnancy
- Infectious:
 - Vaginitis
 - Cervicitis
 - Pelvic inflammatory disease (PID)
- Coagulopathies:
 - von Willebrand disease
 - Idiopathic thrombocytopenic purpura
 - Platelet defects
 - Thalassemia
- Medications:
 - Warfarin
 - Aspirin
 - Oral contraceptives
 - Tricyclic antidepressants
 - Major tranquilizers
- Systemic illness:
 - Adrenal, hepatic, renal or thyroid dysfunction, diabetes mellitus, other endocrinopathies
- Anatomic lesions:
 - Fibroids
 - Endometriosis
 - Polyps
 - Endometrial hyperplasia
 - Neoplasm
- Intrauterine devices
- Trauma

Hormone related

See anovulatory and ovulatory etiologies



TREATMENT

PRE HOSPITAL

IV crystalloid boluses as needed for hypotension, tachycardia secondary to heavy bleeding

INITIAL STABILIZATION/THERAPY

ABCs:

- Packed RBCs for significant bleeding unresponsive to crystalloids

ED TREATMENT/PROCEDURES

- Observation usually adequate if bleeding mild
- IV crystalloid, packed RBCs for continued bleeding, or hemodynamic instability
- Gynecology consultation if bleeding is severe and unresponsive to crystalloids, medications:
 - D&C may be necessary for hemodynamic instability
 - Endometrial ablation or hysterectomy for continued heavy bleeding unresponsive to other measures

MEDICATION

- Conjugated estrogen (Premarin) for heavy bleeding, hemodynamic instability:
 - 2.5 mg PO q6h
 - 25 mg IV, repeat in 3 hr if needed
- Ibuprofen 400–800 mg PO q8h (reduces prostaglandin synthesis)
- IV dosing has not been shown to be superior to oral route:
 - Medroxyprogesterone acetate 5–10 mg/d PO is added when bleeding subsides
- Oral contraceptive pills:
 - Ethinyl estradiol 35 µg and norethindrone 1 mg PO QID for 1 wk
- Antifibrinolytic agents:
 - Tranexamic acid: 1,300 mg PO TID × 5 days
 - May be used in conjunction with OCPs
 - Use limited by GI effects and allergy
- Medications may be deferred in mild cases with referral to gynecology
- Transdermal or long-acting estrogens are other options



FOLLOW-UP

DISPOSITION

Admission Criteria

- Significant blood loss
- Continued bleeding
- Hemodynamic instability requiring aggressive resuscitation and/or operative intervention

Discharge Criteria

Most patients can be discharged with gynecology referral once bleeding is controlled and patient is hemodynamically stable.

Issues for Referral

Endometrial biopsy if > 35 yr old:

- Follow-up with either gynecologist or primary care physician is necessary for patients with DUB
- Must evaluate for ongoing blood loss or potential malignancy as cause

PEARLS AND PITFALLS

- DUB is a diagnosis of exclusion
- Only 2% of endometrial carcinoma occur before age 40 yr
- If hemodynamic instability, unlikely diagnosis of DUB

ADDITIONAL READING

- Casablanca Y. Management of dysfunctional uterine bleeding. *Obstet Gynecol Clin North Am.* 2008;35:219–234.
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- Pitkin J. Dysfunctional uterine bleeding. *BMJ.* 2007;334:1110–1111.



See Also (Topic, Algorithm, Electronic Media Element)

- Amenorrhea
- Vaginal Bleeding



CODES

ICD9

- 626.2 Excessive or frequent menstruation
- 626.6 Metrorrhagia
- 626.8 Other disorders of menstruation and other abnormal bleeding from female genital tract

ICD10

- N92.0 Excessive and frequent menstruation with regular cycle
- N92.1 Excessive and frequent menstruation with irregular cycle
- N93.8 Other specified abnormal uterine and vaginal bleeding

DYSPHAGIA

Laura G. Burke

BASICS

DESCRIPTION

- Difficulty swallowing
- Can be neuromuscular or mechanical

ETIOLOGY

- Oropharyngeal (transfer) dysphagia:
 - Difficulty transferring from the mouth to the proximal esophagus (difficulty initiating a swallow)
 - Easier to swallow solids vs. liquids
 - Immediate, within seconds of swallowing
 - Associated with nasal or oral regurgitation, coughing, or choking
 - Usually a neuromuscular disorder resulting in bulbar muscle weakness or impaired coordination
- Esophageal (transport) dysphagia:
 - Failure of normal transit through the esophagus
 - Retrosternal sticking sensation seconds after swallowing
 - Nocturnal regurgitation/aspiration
 - Drooling or regurgitation of undigested food and liquid (characteristic of esophageal obstruction)
 - Motility disorder vs. mechanical obstruction
- Functional dysphagia:
 - Diagnosis of exclusion
 - Full workup without evidence of mechanical or neuromuscular pathology
 - Symptoms > 12 wk
- Odynophagia:
 - Pain with swallowing
 - Separate, but often related, entity
- Pain pattern:
 - Overall poor ability to localize pain with dysphagia, although oropharyngeal source is better
 - Somatic nerve fibers in the upper esophagus; better pain localization
 - Visceral pain from the lower esophagus is poorly localized and may be difficult to distinguish from that of acute coronary syndrome.

Pediatric Considerations

- Pediatric dysphagia:

- Common causes in infants/newborns include prematurity, congenital malformations, neuromuscular disease, infection (e.g., candidiasis), inflammation
- Always consider foreign body aspiration in a child presenting with dysphagia
- Other common causes in children include caustic ingestions, infections, and neurologic disorders including sequelae from head injury
- Acquired tracheoesophageal fistula in children may result from ingestions (disk battery, caustic ingestions) or prior surgery
- Other life-threatening causes of dysphagia include epiglottitis, retropharyngeal abscess, CNS infection, botulism, esophageal perforation, diphtheria

DIAGNOSIS

SIGNS AND SYMPTOMS

- Difficulty initiating swallowing
- Sensation of food stuck after swallowing
- Cough/choke after eating
- Impairment of gag reflex and ability to clear bolus
- Voice change/dysphonia
- Drooling
- Dysarthria
- Chest pain

History

- Is there difficulty swallowing solids, liquids, or both?
 - Solids and liquids suggest a neuromuscular disorder.
 - Solids only or progression from solids to liquids suggests a mechanical abnormality.
- How long after swallowing do symptoms occur?
 - Immediate onset of symptoms suggests oropharyngeal cause
 - Delay (seconds after swallowing) suggests esophageal cause
- Are symptoms intermittent or progressive?
 - Intermittent symptoms suggest rings or webs.
 - Progressive symptoms suggest peptic or malignant strictures.
 - Motility disorders can be intermittent or progressive.
- How long have the symptoms been present?
 - Acute onset is more concerning for acutely life-threatening etiology
 - Food impaction is the most common cause of acute-onset dysphagia
 - Malignancy may also progressive relatively quickly
- Are there other associated symptoms?

- e.g., nasal regurgitation, choking, heartburn, weight loss

Physical-Exam

- Often unremarkable
- Oropharyngeal inspection
- Pulmonary and cardiac auscultation
- Neurologic exam with emphasis on cranial nerves (esp. V, VII, IX, X, XII)

ESSENTIAL WORKUP

- Adequate airway evaluation
- Thorough neurologic exam

DIAGNOSIS TESTS & INTERPRETATION

EKG:

- Consider cardiac etiology for chest discomfort

Lab

No specific studies are indicated.

Imaging

- CXR:
 - Achalasia food dilating the esophagus may be seen as widened mediastinum, air–fluid level in posterior mediastinum
 - Aspiration pneumonitis
 - Extrinsic compressing mass
- Soft tissue lateral neck radiograph
- Modified barium swallow (with solid bolus) or videofluoroscopy:
 - Defines esophageal anatomy
 - Assesses function
 - Do not perform if endoscopy anticipated
- CT/MRI of the head:
 - Indicated for new-onset neuromuscular dysphagia

Diagnostic Procedures/Surgery

- Often performed in the outpatient setting
- Upper endoscopy:
 - Indicated to relieve obstruction and inspect the esophageal anatomy
 - Biopsy possible if indicated
- Esophageal manometry
- Fiberoptic nasopharyngeal laryngoscopy

DIFFERENTIAL DIAGNOSIS

- Oropharyngeal:

- Infectious:
 - Botulism
 - CNS infections
 - Mucositis
 - Lyme disease
- Mechanical:
 - Congenital
 - Malignancy
 - Pharyngeal pouch
- Medications:
 - Antibiotics (especially doxycycline)
 - Aspirin and NSAIDs
 - Bisphosphonates
 - Ferrous sulfate
 - Potassium chloride
 - Quinidine
- Neuromuscular:
 - Amyotrophic lateral sclerosis
 - Cerebrovascular accident
 - Guillain–Barré syndrome
 - Cranial nerve palsy
 - Huntington chorea
 - Multiple sclerosis
 - Myasthenia gravis
 - Parkinson disease
 - Traumatic brain injury
- Psychological/behavioral
- Esophageal:
 - Mechanical:
 - Diverticula
 - Esophageal webs
 - Foreign body
 - Neoplasm
 - Peptic esophageal stricture
 - Postsurgical (laryngeal, spinal)
 - Radiation injury
 - Schatzki ring
 - Motor:
 - Achalasia
 - Chagas
 - Cushing syndrome
 - Diffuse esophageal spasm
 - Hyperthyroidism/hypothyroidism

- Nutcracker esophagus
- Scleroderma
- Vitamin B₁₂ deficiency
- Inflammatory:
 - Eosinophilic esophagitis
 - Pill esophagitis
- Extrinsic:
 - Cardiovascular abnormalities (vascular rings, thoracic aneurysm, left atrial enlargement, aberrant subclavian artery)
 - Cervical osteophytes
 - Mediastinal mass



TREATMENT

PRE HOSPITAL

- Vigilant airway attention
- Position of comfort with suction available

INITIAL STABILIZATION/THERAPY

- Vigilant airway attention
- Position of comfort with suction available
- NPO
- 0.9% NS 500 mL (peds: 20 mL/kg) IV fluid bolus for significant dehydration
- Evaluate for life-threatening causes of dysphagia including
 - Retropharyngeal hematoma/abscess
 - Epiglottitis
 - Foreign body
 - Upper airway obstruction
 - Cardiovascular causes (thoracic aortic aneurysm)

ED TREATMENT/PROCEDURES

- Nitroglycerin for esophageal spasm
- Glucagon for impacted foreign body
- Treat complications:
 - Airway obstruction
 - Aspiration, pneumonia, lung abscess
 - Dehydration, malnutrition
- Endoscopy
- Dietary modifications:
 - Thickened liquids for neuromuscular disorder
 - Thin liquids for mechanical disorders

MEDICATION

First Line

- Glucagon for food impaction: 1 mg IV followed by 2nd dose of 1 mg after 5 min if there is no improvement in symptoms (0.02–0.03 mg/kg in children, not to exceed 0.5 mg):
 - Success rates vary from 12–50%, which may not be better than spontaneous passage.

Second Line

Calcium channel blockers and nitrates may be used in motility disorders (e.g., achalasia and nutcracker esophagus)



FOLLOW-UP

DISPOSITION

Admission Criteria

- Esophageal obstruction persists despite treatment
- Compromised fluid or nutrition status
- Inability to protect airway
- Unable to tolerate own secretions

Discharge Criteria

- Well-hydrated patient
- Urgent neurology, otolaryngology, or gastroenterology referral arranged for further evaluation and treatment

Issues for Referral

Next day follow-up with PCP or ENT/GI

FOLLOW-UP RECOMMENDATIONS

- Clear liquid diet prior to ENT follow-up
- Return if SOB, chest pain, or unable to tolerate own secretions.

PEARLS AND PITFALLS

- Consider foreign-body aspiration in children presenting with dysphagia.
- Dysphagia is a common presentation in stroke.
- Consider in patients with recurrent pneumonia.
- Assess for life-threatening causes of dysphagia before deferring definitive diagnosis to outpatient setting.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Stroke



CODES

ICD9

- 787.20 Dysphagia, unspecified
- 787.22 Dysphagia, oropharyngeal phase
- 787.24 Dysphagia, pharyngoesophageal phase

ICD10

- R13.10 Dysphagia, unspecified
- R13.12 Dysphagia, oropharyngeal phase
- R13.14 Dysphagia, pharyngoesophageal phase

DYSPNEA

Matthew M. Hall

BASICS

DESCRIPTION

Inability to breathe comfortably

- Describes a symptom of many possible underlying diseases
- Is different from signs of increased work of breathing
- Usually an unconscious activity, dyspnea is the subjective sensation of breathing, from mild discomfort to feelings of suffocation.
- Dyspnea comes from the Greek word for “hard breathing.”
- Often described as “shortness of breath”
- Common presenting complaint seen in 3.5% of ED visits
- Caused by difficulties in maintaining homeostasis with respect to gas exchange and acid–base status
- Dyspnea usually reflects an impairment in ventilation, perfusion, metabolic function, or CNS drive.
- Mechanisms that control breathing:
 - Control centers:
 - Brainstem and cerebral cortex affect both automatic and voluntary control of breathing.
 - Chemo, stretch, and irritant sensors:
 - CO₂ receptors located centrally and PO₂ receptors located peripherally.
 - Mechanoreceptors lie in respiratory muscles and respond to stretch.
 - Intrapulmonary mechanoreceptors respond to chemical irritation, engorgement, and stretch.
 - Effectors of respiratory center output are in the respiratory muscles and respond to central stimulation to move air in and out of the thoracic cavity.
 - Motor–sensory control of the diaphragm and muscles of respiration are controlled by C3–C8 nerves and T1–T12 nerves.
- Derangements of any of these neurosensory pathways produces dyspnea:
 - Many etiologies for the sensation of dyspnea are due to the complex nature of mechanisms that control breathing.

ETIOLOGY

- Upper airway:
 - Epiglottitis
 - Laryngeal obstruction
 - Tracheitis or tracheobronchitis

- Angioedema
- Pulmonary:
 - Airway mass
 - Asthma
 - Bronchitis
 - Chest wall trauma
 - CHF
 - Drug-induced conditions (e.g., crack lung, aspirin overdose)
 - Effusion
 - Emphysema
 - Lung cancer
 - Metastatic disease
 - Pneumonia
 - Pneumothorax
 - Pulmonary embolism
 - Pulmonary HTN
 - Restrictive lung disease
- Cardiovascular:
 - Arrhythmia
 - Coronary artery disease
 - Intracardiac shunt
 - Left ventricular failure
 - Myxoma
 - Pericardial disease
 - Valvular disease
- Neuromuscular:
 - CNS disorders
 - Myopathy and neuropathy
 - Phrenic nerve and diaphragmatic disorders
 - Spinal cord disorders
 - Systemic neuromuscular disorders
- Metabolic acidosis:
 - Sepsis
 - DKA
 - AKA
 - Renal failure
 - Profound thiamine deficiency
- Toxic:
 - Methemoglobinemia
 - Salicylate poisoning
 - Cellular asphyxiants:
 - Carbon monoxide
 - Cyanide

- Hydrogen sulfide
- Sodium azide
- Toxic alcohols
- Abdominal compression:
 - Ascites
 - Pregnancy
 - Massive obesity
- Psychogenic:
 - Hyperventilation
 - Anxiety
- Other:
 - Altitude
 - Anaphylaxis
 - Anemia

Geriatric Considerations

- Most common diagnoses in elderly patients presenting to the ED with dyspnea:
 - Decompensated heart failure
 - Pneumonia
 - COPD
 - Pulmonary embolism
 - Asthma

Pediatric Considerations

- Common conditions in differential diagnosis for age <2 yr:
 - Asthma
 - Croup
 - Congenital anomalies of the airway
 - Congenital heart disease
 - Foreign-body aspiration
 - Nasopharyngeal obstruction
 - Shock

DIAGNOSIS

SIGNS AND SYMPTOMS

- Difficult, labored, or uncomfortable breathing
- Upper airway:
 - Stridor
 - Upper-airway obstruction
- Pulmonary:
 - Tachypnea

- Accessory muscle use
- Wheezing
- Rales
- Asymmetric breath sounds
- Poor air movement
- Prolonged expiratory phase
- Cardiovascular:
 - S3 gallop
 - Murmur
 - Jugular venous distention
- CNS:
 - Altered levels of consciousness
- General:
 - Diaphoretic/cool vs. hot/dry skin
 - Pallor
 - Upright patient position
 - Clubbing
 - Cyanosis
 - Edema
 - Ketotic breath odor

History

- Previous history of dyspnea
- Time course, abruptness of onset, triggers, and severity
- History of stridor or wheezing
- Exercise (activity) tolerance
- Medications and recent compliance
- Exposure to allergens
- Past medical history
- Associated symptoms:
 - Chest pain
 - Fever
 - Cough
 - Hemoptysis

Physical-Exam

- Signs of acute distress:
 - Altered mental status
 - Cyanosis
 - Respiratory rate
 - Retractions suggest severe disease
- Listen for abnormal lung sounds:

- Stridor
- Rales
- Wheezing
- Decreased breath sounds

ESSENTIAL WORKUP

- Pulse oximetry:
 - May be falsely elevated due to increased ventilation or carbon monoxide
- End tidal CO₂:
 - Quickly gives hint of PaCO₂
 - Waveform can give clue to etiology
- CXR:
 - For diagnosis of pulmonary conditions
 - Assess heart size and evidence of CHF
- ABG:
 - Oxygenation
 - Calculate arterial–alveolar gradient:
 - A–a (at sea level) = $150 - (PO_2 - PCO_2)/0.8$, normal 5–20
 - Assess degree of acidosis

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC:
 - Evaluation for anemia
 - Neutrophil count helpful in evaluation of infectious processes
- Electrolyte, BUN, creatinine, glucose:
 - Consider when specific metabolic derangements are suspected
 - B-type natriuretic peptide may be elevated in CHF
- Toxicology screen
- Methemoglobin/carboxyhemoglobin level
- Thyroid function tests
- D-dimer (ELISA):
 - Useful for excluding pulmonary embolus if normal

Imaging

- Chest x-ray for infiltrate, effusion, pneumothorax, or vascular consolidation
- Ventilation–perfusion scan or CT pulmonary angiogram for suspected pulmonary embolism
- Soft tissue neck radiograph or fiberoptic visualization for suspected upper airway obstruction

Diagnostic Procedures/Surgery

- EKG for suspected myocardial ischemia, CHF, suspected pericardial effusion/tamponade
- Peak expiratory flow/spirometry to assess for reactive-airway disease
- Tensilon test for suspected myasthenia gravis

DIFFERENTIAL DIAGNOSIS

- Anticholinergic or adrenergic toxidrome
- Thyroid storm
- Munchausen syndrome



TREATMENT

PRE HOSPITAL

- Place all patients on supplemental oxygen, pulse oximetry, end tidal CO₂, and cardiac monitor.
- Initiate therapy for suspected cause of dyspnea when indicated:
 - Asthma
 - COPD
 - CHF
- Utilize advanced airways in the face of impending respiratory failure.

INITIAL STABILIZATION/THERAPY

- ABCs
- Immediate intubation for impending respiratory arrest:
 - Altered mental status
 - Unstable vital signs
- BiPAP in alert patients:
 - Contraindications:
 - Cardiac instability
 - Suspicion of upper airway obstruction
 - Inability to protect airway
 - Upper GI bleeding
 - Status epilepticus

ED TREATMENT/PROCEDURES

- Based on underlying etiology
 - Antibiotics and fluid for pneumonia
 - CPAP and diuretics for CHF
 - Bronchodilators and steroids for asthma
 - Aspirin, heparin, and lyrics/cath lab for MI
 - Other treatments as necessary for other etiologies

- Palliative care with opiates is indicated for the relief of dyspnea in terminally ill patients.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Assisted ventilation
- Hypoxia
- A-a gradient > 40
- Medical condition requiring hospital therapy

Discharge Criteria

- Adequate oxygenation
- Stable medical illness that can be managed as outpatient
- Adequate ambulatory pulse ox

Issues for Referral

Based on suspected underlying etiology

FOLLOW-UP RECOMMENDATIONS

- Patients should be told not to smoke while short of breath and to try to quit to help with some of the causes, as well as to prevent others from getting worse.
- The patient should return for any of the following problems:
 - No improvement or worsening in 24 hr
 - New chest pain, pressure, squeezing, or tightness
 - Shaking chills, or a fever > 102°F
 - New or worsening cough or wheezing
 - Abdominal (belly) pain, vomiting, severe headache
 - Dizziness, confusion, or change in behavior
 - Any serious change in symptoms, or any new symptoms that are of concern

PEARLS AND PITFALLS

- Altered mental status is an indication for immediate airway management in a patient with severe dyspnea.
- Dyspnea can and should be quantified.
- Dyspnea and tachypnea may occur without a respiratory etiology because of metabolic derangement or a catastrophic CNS event.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Respiratory Distress



CODES

ICD9

- 786.05 Shortness of breath
- 786.07 Wheezing
- 786.09 Other respiratory abnormalities

ICD10

- R06.00 Dyspnea, unspecified
- R06.02 Shortness of breath
- R06.2 Wheezing

DYSTONIC REACTION

Kenneth Jackimczyk

BASICS

DESCRIPTION

- Normal pattern of CNS neurotransmission maintained by balance between dopaminergic and cholinergic receptors:
 - Certain drugs antagonize dopamine receptors in the basal ganglia resulting in an imbalance of dopaminergic and cholinergic stimulation
 - This imbalance leads to acute involuntary muscle spasms of the face or neck (the trunk, pelvis, or extremities can also be affected)
- Although the spasms are uncomfortable and frightening, they are not life threatening except in very rare cases when laryngeal muscles are involved
- Usually occurs within hours of ingestion:
 - Almost always within 1st wk after exposure to offending drug
- Risk factors:
 - Children and young adults are at higher risk
 - Rarely occurs in patients over 45 yr of age
 - Males more often affected
 - Prior episodes of dystonia significantly increase risk
 - Recent cocaine use increases risk

ETIOLOGY

- Usually occurs after patient has taken antipsychotic, antiemetic, or antidepressant drug
- Incidence of dystonic reactions varies widely (2–25%) depending on the potency of the agent
- Higher with more potent drugs (haloperidol, fluphenazine)
- Lower with less potent drugs (chlorpromazine, thioridazine)
- Lowest with atypical antipsychotics (quetiapine, olanzapine, risperidone)
- Antiemetic agents:
 - Metoclopramide (Reglan)
 - Prochlorperazine (Compazine)
 - Promethazine (Phenergan)
 - Droperidol (Inapsine)
- Other agents:
 - Cyclic antidepressants
 - H₂ blockers
 - Some antimalarial agents
 - Antihistamines

- Some anticonvulsants
- Doxepin
- Lithium
- Phencyclidine

Pediatric Considerations

Children are particularly vulnerable to dystonic reactions when dehydrated or febrile

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Ingestion of neuroleptic, antiemetic, or other drug within week of symptom onset
 - May occur in patients on neuroleptic agents who increase their dose of neuroleptics or reduce medications (anticholinergic agents) used to treat extrapyramidal symptoms
- Difficulty with vocalization
- Completely alert and able to answer questions, although facial muscle involvement may make speech difficult.
- Involuntary muscle contractions or spasms usually involving the face or neck (see “Physical Exam”):
 - Muscles of the trunk, pelvis, or extremities can also be involved

Physical-Exam

- Characteristic involuntary muscle spasms occur
- Oculogyric crisis
 - Involves eye and periorbital muscles
 - Evolves into painful upward or lateral deviation of the eyes
- Blepharospasm
 - Involuntary eyelid closure
- Buccolingual crisis
 - Involves facial muscles and the tongue
 - May have difficulty speaking
 - Facial grimacing
 - Trismus
 - Tongue protrusion
 - Dysphagia
- Spasmodic torticollis
 - Twisting of the neck
- Torticopelvic crisis
 - Abdominal wall muscle spasm

- Opisthotonos
 - Involves muscles of trunk and back
 - Twisting and arching of spine
- Laryngeal dystonia
 - Very rare but potentially life threatening
 - May develop airway obstruction due to laryngospasm
 - Presents as dysphonia or stridor

ESSENTIAL WORKUP

- Clinical diagnosis is based on characteristic signs and symptoms with history of possible drug exposure
- Diagnosis is confirmed by response to treatment
 - Lack of response to treatment should lead one to consider alternative diagnosis

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Lab testing not routinely indicated
- If no response to treatment, hypocalcemia should be considered and calcium level obtained

Imaging

No imaging studies needed

DIFFERENTIAL DIAGNOSIS

- Tardive dyskinesia:
 - Complication of chronic antipsychotic therapy
 - Usually choreiform movements
 - Does not rapidly improve with administration of anticholinergic drug
- Akathisia:
 - Involuntary motor restlessness
 - May appear agitated
- Seizure:
 - History of prior seizures
 - Not responsive to verbal stimuli
 - Tonic-clonic-type motor movements rather than spasm
- Hysteria or pseudoseizure:
 - History of precipitating emotional event
 - Tonic-clonic motor activity rather than sustained spasm
- Tetanus
- Strychnine poisoning
- Chronic dystonias:

- Cerebral palsy, familial choreas
- Usually history of dystonia is associated with chronic neurologic process
- Scorpion envenomation:
 - Oculogyric crisis and opisthotonos are common manifestations of scorpion envenomation
 - Patient lacks history of drug exposure.
- Meningitis and encephalitis may present with atypical seizures that mimic dystonic reaction
- Mandible dislocation
- Hypocalcemia



TREATMENT

PRE HOSPITAL

- Rarely life threatening
- Direct attention toward spasm of larynx and tongue to be sure dystonic reaction is not causing respiratory compromise
- Ask family and friends about ingestions of antipsychotic medications, antiemetics, and recreational drugs
- Transport pill bottles

INITIAL STABILIZATION/THERAPY

Stabilize airway to prevent spasm of larynx or tongue from causing respiratory compromise.

ED TREATMENT/PROCEDURES

- Administer diphenhydramine (Benadryl) or benztropine mesylate (Cogentin):
 - Rapid resolution of muscular spasm by restoring cholinergic–dopaminergic balance in CNS
 - IV administration is preferred route of treatment
 - Onset of relief in 2–5 min
 - Complete resolution of symptoms in 30 min
 - IM administration is alternate route of treatment
 - Begins to work in 15–30 min
 - Continue oral administration for 3 days to prevent redevelopment of symptoms
- Diazepam (Valium):
 - Administer in cases of dystonia unresponsive to adequate doses of anticholinergic medications
 - Failure to respond to standard treatment should lead physician to consider other diagnoses

MEDICATION

- Benztropine mesylate (Cogentin): 1–2 mg either IV (over 2 min) or IM followed by 1–2 mg PO BID for 3 days:
 - Not to be used in children < 3 yr old
 - For children > 3 yr old: 0.02 mg/kg IV (over 2 min) or IM followed by 0.02 mg/kg PO BID for 3 days
- Diphenhydramine (Benadryl): 1–2 mg/kg up to 100 mg either IV (over 2 min) or IM followed by 25–50 mg (peds: 1–2 mg/kg) PO q6–8h for 3 days, *or*
- Diazepam: 5–10 mg IV followed by 5 mg PO q4–6h as necessary for 3 days

First Line

Diphenhydramine (Benadryl)

Second Line

Benzotropine mesylate (Cogentin):

- Not to be used in children < 3 yr old
- Diazepam



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients are not admitted unless symptoms do not resolve with treatment, there are concerns about maintaining the airway, or the diagnosis is not certain
- If the dystonic reaction causes laryngospasm patient should be observed for 12–24 hr after symptoms resolve

Discharge Criteria

- Discharge after resolution of symptoms
- The offending agent should be discontinued
- Patient should not drive or perform tasks that require full alertness while taking sedating medications

FOLLOW-UP RECOMMENDATIONS

Patients should follow-up with the prescribing physician of the causative agent

PEARLS AND PITFALLS

- The diagnosis of acute dystonia is made based on the history of ingestion coupled with complete resolution of the symptoms after appropriate treatment
- 1st line of therapy is diphenhydramine
- Failure to respond should lead you to consider other diagnoses

ADDITIONAL READING

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- Sachdev PS. Neuroleptic-induced movement disorders: An overview. *Psychiatr Clin North Am*. 2005;28:255–274.
- Vena J, Dufel S, Paige T. Acute olanzapine-induced akathisia and dystonia in a patient discontinued from fluoxetine. *J Emerg Med*. 2006;30:311–317.
- Wolfson AB, Hendey GW, Ling LJ, et al., eds. *Harwood Nuss’ Clinical Practice of Emergency Medicine*. 5th ed. Philadelphia, PA: Lippincott; 2010.



CODES

ICD9

333.72 Acute dystonia due to drugs

ICD10

- G24.01 Drug induced subacute dyskinesia
- G24.02 Drug induced acute dystonia
- G24.09 Other drug induced dystonia

EATING DISORDER

Rohn S. Friedman



BASICS

DESCRIPTION

Anorexia Nervosa (AN)

- Restriction of intake, leading to markedly low body weight for age, height, and/or developmental trajectory
- Intense fear of gaining weight or becoming fat, or behavior that prevents weight gain
- Severe body image disturbance, undue influence of body weight and shape on self-evaluation, or denial of seriousness of low body weight
- Lifetime prevalence: 0.5% of females in US
- Typical age of onset for AN is bimodal at 13–14 yr and 17–18 yr

Bulimia Nervosa (BN)

- Recurrent episodes of binge eating characterized by:
 - Eating an unusually large amount of food in a discrete period of time
 - A sense of loss of control over eating during the episode
- Recurrent inappropriate compensatory behaviors used to prevent weight gain:
 - Self-induced vomiting
 - Misuse of laxatives or enemas
 - Diuretics
 - Diet pills
 - Fasting
 - Excessive exercise
- Bingeing and compensation occur on average at least once a week for 3 mo
- Self-evaluation that is excessively influenced by weight or body shape
- Lifetime prevalence: 2% of females in US
- Commonly onset in late adolescence or early adulthood.

Binge Eating Disorder (BED)

- Recurrent episodes of binge eating characterized by:
 - Eating a larger than usual amount of food in a discrete period of time
 - A sense of loss of control over eating during the episode
- Binge eating episodes associated with 3 or more of the following:
 - Eating much more rapidly than normal
 - Eating until feeling uncomfortably full
 - Eating large amounts of food when not feeling physically hungry

- Eating alone because of embarrassment about how much one is eating
- Feeling disgusted with oneself, depressed, or very guilty after overeating
- Marked distress over binge eating
- Occurs on average at least once a week for 3 mo
- No compensatory behavior
- Lifetime prevalence: 3.5% of females and 2% males in US
- Onset in late adolescence or early adulthood.

ETIOLOGY

- Twin studies have supported a strong genetic component.
- Cultural emphasis on thinness as a valued attribute has been implicated
- Temperament or personality attributes of perfectionism, anxiety, and behavioral rigidity have been described
- Family conflict or stress is a frequent element
- Neurochemical (serotonin) and neuroendocrinologic (leptin, HPA axis) abnormalities have been reported
- Dieting is a frequent immediate precipitant



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Rapid or sustained weight loss
- Typical detailed day's eating pattern shows restricting and/or bingeing behavior
- Purging (vomiting, laxatives, diuretics, enemas)
- Excessive exercise
- Dizziness, syncope
- Bloating (gastroparesis), constipation, abdominal pain
- Fatigue, lethargy
- Palpitations
- Cold intolerance
- Amenorrhea, loss of libido
- Family history of eating disorders and obesity
- Comorbid psychiatric disorder (e.g., mood disorder, substance abuse, personality disorder)

Physical-Exam

- Weight < 85% IBW or BMI < 17.5 for AN
- Hypothermia
- Hypotension, orthostasis
- Bradycardia, arrhythmia

- Skin: Dry skin, lanugo (soft downy body hair on chest and arms), carotenoderma
- Breast atrophy
- Parotid swelling, submandibular swelling
- Abnormal dentition
- Abrasions of dorsum of hand
- Skin breakdown, poor wound healing
- Peripheral edema
- Muscle weakness

ESSENTIAL WORKUP

- History
- Physical exam
- Lab testing
- Nutritional assessment
- Psychiatric interview:
 - Concurrent psychiatric illness
 - Suicide risk assessment
 - Explore psychosocial context
- Family evaluation when patient lives with his or her family

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC (anemia, leukopenia, thrombocytopenia)
- Electrolytes, BUN, creatinine, glucose (hyponatremia, hypokalemia, hypoglycemia, dehydration, metabolic alkalosis)
- Calcium, magnesium, phosphorus, albumin (hypocalcemia, hypomagnesemia, hypophosphatemia, hypoalbuminemia)
- LFTs (hepatic dysfunction)
- UA including specific gravity
- Toxic screen
- β -hCG
- Amylase (salivary hyperamylasemia if vomiting, pancreatitis)
- Lipase (more accurate than amylase in predicting pancreatitis)
- Consider checking thyroid-stimulating hormone.

Imaging

Specific tests may be useful in making differential diagnoses, e.g., MRI (rule out brain tumor), abdominal CT (rule out obstruction)

Diagnostic Procedures/Surgery

- ECG (QTc prolongation, arrhythmia)
- Consider cardiac echo if substantial weight loss (cardiomyopathy from AN or

ippecac)

- Bone mineral density (osteoporosis)

DIFFERENTIAL DIAGNOSIS

- Medical conditions:
 - GI disease (e.g., Crohn's Disease, IBD, celiac disease)
 - Endocrine disorder (e.g., DM, thyroid disorder, adrenal insufficiency)
 - Cancer
- Psychiatric conditions
 - Borderline personality disorder
 - Mood disorders
 - Obsessive-compulsive disorder
 - Substance abuse



TREATMENT

INITIAL STABILIZATION/THERAPY

- ABCs
- Careful fluid resuscitation for dehydration to avoid precipitating peripheral or pulmonary edema
- Replete phosphate and thiamine since both may drop with refeeding
- Correct hypokalemia, hypomagnesemia, hypocalcemia
- Correct hypoglycemia
- Warming blankets for severe hypothermia

ED TREATMENT/PROCEDURES

- Initial workup
- Medical stabilization
- Psychiatric consultation (including assessment of suicide risk and psychiatric comorbidities)

MEDICATION

First Line

- No medication has been demonstrated to be of benefit for AN per se
 - Small trials have suggested possible benefit from atypical antipsychotics, particularly olanzapine 2.5–10 mg PO QD
 - It may be helpful to treat psychiatric comorbidities
- Only fluoxetine 20–60 mg PO QD has FDA indication for the treatment of BN, though other SSRIs are frequently used. There is also evidence for tricyclic antidepressants as well as topiramate
- There is evidence for imipramine, sertraline, citalopram/escitalopram, and



FOLLOW-UP

DISPOSITION

Admission Criteria

- Medical risk:
 - Extremely low weight ($< 75\%$ IBW)
 - Rapid weight loss
 - Serum electrolyte imbalance (K < 3 , glucose < 60)
 - Bradycardia < 40
 - BP $< 90/60$
 - Orthostasis (> 20 bpm or > 20 mm Hg/10 mm Hg)
 - Hypothermia $< 97^{\circ}\text{F}$
 - Arrhythmia or heart failure
 - Hepatic or renal dysfunction
- Psychiatric risk:
 - Severe depression, psychosis, or other comorbid psychiatric diagnosis
 - Suicidality
 - Lack of motivation or cooperation with treatment
 - Failure of outpatient treatment
 - Severe impairment in functioning
 - Toxic family environment

Discharge Criteria

- Medically and psychologically safe enough to be managed on an outpatient basis
- Multimodal, multidisciplinary team in place to manage medical, nutritional, and psychological issues

Issues for Referral

- Outpatient treatment requires a team approach composed of a:
 - Psychiatrist and/or psychologist
 - Nutritionist, preferably one who specializes in eating disorders
 - Pediatrician or internist
 - Family therapist
 - Group therapist
 - Dentist
- Prognosis:
 - AN and BN:
 - 20% chronic course
 - 30% improve

- 50% recover
- Mortality rate 5.6% per decade for AN
- Outcomes improved with early diagnosis and treatment

FOLLOW-UP RECOMMENDATIONS

- For outpatient treatment the team must establish modest goals and clear parameters, including expected weight gain for anorexic patients and compliance with follow-up appointments.
- Internist/pediatrician: Monitor vital signs, weight, BMI, electrolytes, and ECG.
- Nutritionist: Monitor diet, calorie intake, and exercise.
- Psychotherapy:
 - Cognitive behavioral therapy and interpersonal psychotherapy are the most effective forms of psychotherapy for BN.
 - Cognitive behavioral therapy, family therapy, and psychodynamic therapies are all useful for AN.
 - Family-based treatment is the preferred therapy for teenagers with AN, and it is promising for teenagers with BN as well.
- Pharmacotherapy:
 - Only indicated within the context of psychotherapy, especially with comorbid psychopathology.
 - No accepted pharmacologic treatment of AN.
 - Case studies suggest that 2nd-generation antipsychotics may be helpful in AN.
 - There is no clear evidence for specific treatment of osteoporosis in AN apart from weight restoration and nutritional calcium supplementation.
 - Antidepressant medications are shown to significantly reduce bingeing and purging behaviors:
 - Fluoxetine is the best studied

PEARLS AND PITFALLS

- Eating disorders are associated with high medical risk and risk of suicide; prioritize safety assessment
- Rapid restoration of nutrition, volume resuscitation, and/or failure to replete vitamins and electrolytes can result in potentially fatal refeeding syndrome
- Avoid trying to “out-obsess” the obsessional patient
- Coordinate care with PCP and other members of a multidisciplinary team

ADDITIONAL READING

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disorders. *World J Biol Psychiatry*. 2011;12:400–443.

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CODES

ICD9

- 307.1 Anorexia nervosa
- 307.50 Eating disorder, unspecified
- 307.51 Bulimia nervosa

ICD10

- F50.00 Anorexia nervosa, unspecified
- F50.2 Bulimia nervosa
- F50.9 Eating disorder, unspecified

ECTOPIC PREGNANCY

Ivette Motola • Aviva Jacoby Zigman

BASICS

DESCRIPTION

- Implantation of fertilized ovum outside of uterus:
 - Most commonly fallopian tube (93–97%)
- Abdominal and peritoneal implantations:
 - Associated with higher morbidity
 - Difficulty in diagnosis
 - Tendency to bleed
- Occurs in 2–2.6% of pregnancies
- Accounts for 6% of all maternal deaths (leading cause of 1st-trimester pregnancy-related death)
- 60% of women with ectopic pregnancy are subsequently able to have a normal pregnancy

ETIOLOGY

- Risk factors include:
 - Woman > 35 yr old
 - African American
 - Previous fallopian tube damage from infections, such as pelvic inflammatory disease (PID)
 - Previous tubal surgery (i.e., tubal ligation)
 - Previous ectopic pregnancy
 - Intrauterine device (IUD) use:
 - 25–50% of pregnancies with IUD are ectopic
 - Diethylstilbestrol (DES) exposure
 - In vitro fertilizations
 - Being a current smoker
- More than half of women with ectopic pregnancies have no risk factors

DIAGNOSIS

SIGNS AND SYMPTOMS

Classic triad of amenorrhea, vaginal bleeding, and abdominal pain are present in only 15% of women with ectopic pregnancies:

- Amenorrhea (75–95%)
- Abdominal pain (80–100%):
 - Frequently unilateral

- Abnormal vaginal bleeding (50–80%)
- Symptoms of pregnancy (10–25%)
- Orthostatic hypotension, dizziness, and syncope (5–35%)
- Abdominal tenderness (55–95%)
- Adnexal tenderness (75–90%)
- Adnexal mass (35–50%)
- Cervical motion tenderness (43%)

History

- Last menstrual period (LMP):
 - Majority of ectopics present 5–8 wk after LMP.
- Gestation and parity history
- Vaginal bleeding
- Location, nature, and severity of pain
- History of pelvic surgery, prior ectopic, IUD
- History of fertility treatments

Physical-Exam

- Evaluate for signs of peritoneal irritation
- Pelvic exam:
 - Note uterine size
 - Adnexal size, mass
 - Adnexal tenderness
 - Presence of tissue in vaginal vault
 - Cervical motion tenderness
 - Cervical OS open or closed

ESSENTIAL WORKUP

- Pregnancy testing:
 - Women of potential childbearing age with vaginal bleeding or abdominal pain *must* have urine or serum pregnancy test
 - Include testing of patients with history of recent elective or spontaneous abortion, tubal ligations, or IUD use
 - Quantitative β -human chorionic gonadotropin (β -hCG) in patients with positive qualitative test
- Vital signs unstable:
 - 2 large-bore IVs
 - Type and cross-match, hemoglobin (Hg)/hematocrit (Hct)
 - Bedside ultrasound (US), if immediately available, simultaneous with resuscitation (transvaginal preferred)
 - Consult obstetrics/gynecology (OB/GYN) and prepare for immediate surgical intervention
- Vital signs stable:

- Rapid Hg/Hct determination
- Type and Rh
- US (transvaginal preferred)

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Urine pregnancy tests can detect β -hCG levels of 25–50 mIU/L
- Serum can detect β -hCG levels of 25 mIU/L
- Quantitative serum β -hCG; for diagnosis and follow-up:
 - Doubles every 2 days in normal early pregnancy (early pregnancy < 10,000 β -hCG mIU/L, 8 days–7 wk)
 - β -hCG increases less in ectopic pregnancy
 - Correlation with vaginal US increases predictive value

Imaging

- Ultrasonographic evidence of IUP makes ectopic pregnancy less likely:
 - Heterotopic pregnancies are possible
- Positive IUP is indicated by double-ringed gestational sac, yolk sac, or fetal pole, and heartbeat seen in uterus
- Transvaginal US; visualization of gestational sac at 5 wk, cardiac activity at 6.5 wk
- Transabdominal US; visualization of gestational sac at 5–6 wk, cardiac activity at 8 wk
- Complex adnexal mass and fluid in cul-de-sac seen in 22% of ectopics and has 94% positive predictive value when present
- Positive pregnancy test with no confirmed IUP and fluid in pelvis; high risk for bleeding ectopic pregnancy

Diagnostic Procedures/Surgery

- US in conjunction with quantitative β -hCG
- Patients with β -hCG levels > 6,500 mIU/L and no intrauterine gestational sac seen on US have 100% chance of having ectopic pregnancy
- Patients with β -hCG levels > 6,500 mIU/L with intrauterine gestational sacs present have 94% chance of having normal pregnancy
- Patients with β -hCG < 2,000 mIU/L are too early to have gestational sac seen by abdominal US and thus cannot be ruled out for ectopic pregnancy
- Patients with β -hCG > 2,000 and < 6,500 mIU/L should have IUP visualized on transvaginal US; suspect ectopic pregnancy if IUP is absent
 - Discriminatory hCG value for transvaginal US is between 1,500 and 3,000 mIU/mL
- Culdocentesis to evaluate for intraperitoneal blood if US is unavailable

DIFFERENTIAL DIAGNOSIS

- Positive pregnancy test with vaginal bleeding:
 - Spontaneous abortion
 - Cervicitis
 - Trauma
- Positive pregnancy test with no evidence of IUP:
 - Completed spontaneous abortion
 - Early threatened abortion
- Positive pregnancy test with evidence of IUP, abdominal pain, or adnexal tenderness:
 - Septic abortion
 - Threatened abortion
 - Ruptured corpus luteal or ovarian cyst
 - Ovarian torsion
 - UTI
 - Nephrolithiasis
 - Gastroenteritis
 - Appendicitis
 - Heterotopic pregnancy (IUP + ectopic)
 - PID



TREATMENT

PRE HOSPITAL

Cautions: Female patients of childbearing age presenting in shock may have unrecognized ruptured ectopic pregnancy

INITIAL STABILIZATION/THERAPY

- Vital signs unstable:
 - Airway management, resuscitate as needed
 - Fluid therapy with 2 large-bore IVs, oxygen, and monitor
 - Type specific, or O-negative blood if hypotensive after initial fluid bolus
 - Consult gynecology and transport to OR immediately for surgery
- Vital signs stable:
 - Evidence of ectopic pregnancy on US:
 - Obstetric–gynecologic evaluation for surgery vs. outpatient methotrexate treatment
 - For patients in whom future fertility is desired, methotrexate is the best option; otherwise surgery is the definitive treatment
 - No evidence of ectopic pregnancy (pregnancy of unknown location [PUL]: Early IUP vs. early ectopic):
 - Desired pregnancy: Serial β -hCG every 48 hr in stable, reliable

- patients and in conjunction with obstetrician
- Undesired pregnancy: Dilation and curettage to evacuate uterus and confirm presence of products of conception

ED TREATMENT/PROCEDURES

Methotrexate: Initiated only in conjunction with obstetric consultant and close follow-up:

- Reliable patients with unruptured ectopic pregnancies < 3.5 cm
- β -hCG levels < 6,000–15,000
- Contraindications:
 - Breast-feeding
 - Immunodeficiency
 - Pre-existing blood dyscrasia
 - Clinically significant anemia
 - Known sensitivity to methotrexate
 - Active pulmonary disease
 - Peptic ulcer disease
 - Hepatic dysfunction
 - Renal dysfunction
 - Alcoholism
 - Alcoholic liver disease
 - Ectopic mass > 3.5 cm (relative contraindication)
 - Embryonic cardiac motion (relative contraindication)
- Most common dosing, single dose (50 mg/m²); serial β -hCG on days 2, 4, and 7
 - If < 25% decline in β -hCG from day of 1st injection, 2nd dose is given
- Multidose treatment is associated with less treatment failure
- Common side effects:
 - Worsening abdominal pain
 - Nausea, vomiting, and diarrhea
- Worsening abdominal pain usually occurs 3–7 days after methotrexate initiation.
 - These are usually tubal miscarriages
 - Follow-up USs are essential to rule out ectopic rupture
- Most common complication, tubal rupture in 4%
- Factors associated with methotrexate treatment failure:
 - Initial hCG > 5,000 mIU (5,000–9,999 mIU/mL—13% failure rate, > 15,000 mIU/mL failure rate as high as 32%)
 - Moderate to large free peritoneal fluid on US
 - Presence of fetal cardiac activity
 - Pretreatment increase in serum hCG level of more than 50% over a 48 hr period

MEDICATION

- Methotrexate: 50 mg/m² IM/IV × 1
- RhoGAM in Rh-negative women: 50 µg IM in women ≤12 wk pregnant; 300 µg IM in women >12 wk pregnant



FOLLOW-UP

DISPOSITION

Admission Criteria

- Any patient with confirmed ectopic pregnancy who is hemodynamically unstable
- Unreliable patients with increased risk factors, no available US, β-hCG >6,500 with no evidence of IUP should be admitted for observation and serial β-hCG tests

Discharge Criteria

- Decision for outpatient management should be made in conjunction with OB/GYN
- Hemodynamically stable and reliable patients with workup that cannot rule out ectopic pregnancy:
 - Strict follow-up for serial β-hCG tests every 2 days
 - Patients should be recorded in logbook with phone numbers to ensure follow-up
- *Ectopic precautions:* Patients should return to emergency room immediately for:
 - Increasing abdominal pain
 - Vaginal bleeding
 - Syncope or dizziness
 - Patients should not be left alone until diagnosis of ectopic pregnancy can be safely ruled out
 - Family and friends should also be instructed on warning signs and symptoms of ruptured/bleeding ectopic pregnancies

Issues for Referral

Phone consultation (at a minimum) with OB/GYN is essential when discharging a possible ectopic pregnancy

FOLLOW-UP RECOMMENDATIONS

All patients with positive pregnancy tests and unconfirmed IUP must be followed by an OB/GYN

PEARLS AND PITFALLS

- Always obtain a pregnancy test on women of childbearing age
- Obtain serum hCG and transvaginal ultrasonography in all women with positive pregnancy test presenting with abdominal pain or vaginal bleeding

- Recognize the possibility of heterotopic pregnancies, especially in women undergoing fertility treatment
- Secure close follow-up for any patient being evaluated and discharged for ectopic pregnancy

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Pregnancy, Uncomplicated
- Vaginal Bleeding



CODES

ICD9

- 633.00 Abdominal pregnancy without intrauterine pregnancy
- 633.10 Tubal pregnancy without intrauterine pregnancy
- 633.90 Unspecified ectopic pregnancy without intrauterine pregnancy

ICD10

- O00.0 Abdominal pregnancy
- O00.1 Tubal pregnancy
- O00.9 Ectopic pregnancy, unspecified

ECZEMA/ATOPIC DERMATITIS

James A. Nelson

BASICS

DESCRIPTION

- Atopic dermatitis is the most common cause of eczema and the terms are often used synonymously.
 - Associated with allergic diseases such as asthma and allergic rhinitis
- Eczema literally means “out boil” and refers to spongiosis, the process where microvesicles form and rupture, leaving erythema, edema, crusting, and oozing
- Pruritus is highly characteristic
 - Patient rub and scratch skin breakdown with oozing and crusting
 - Chronically this causes epidermal hyperplasia and hyperkeratosis
- 90% of patients colonize with *Staphylococcus aureus*, and are prone to episodes of superinfection

RISK FACTORS

Genetics

- Family history of atopy (asthma, allergic rhinitis) typical
- Mutation of the filaggrin protein, part of the epidermal barrier, is strongly associated

ETIOLOGY

Atopic dermatitis is caused by a deficit in epidermal integrity that allows foreign substances to enter and trigger immune responses.

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Rash, pruritus, and atopy traditionally prompt the diagnosis
- UK diagnostic criteria = pruritus and 3 of the following:
 - Flexural distribution
 - Atopic history (asthma or allergic rhinitis)
 - History of dry skin
 - Onset age < 2 yr
 - Objective signs of flexural dermatitis
- Additional findings:

- Cutaneous infections
- Itch when sweating
- Intolerance to wool and lipid solvents
- Triggered by stress

Physical-Exam

Dermatitis, located on areas of trauma or motion such as hands and feet and flexural areas

- Epidermal compromise:
 - Dry skin
 - Weeping
 - Oozing
 - Finally crusting
- Inflammation:
 - Maculopapular erythema
 - Edema
- Scratching leads to:
 - Excoriation
 - Cracking
 - Lichenification
 - Hyperkeratosis
- Additional findings:
 - Ichthyosis, palmar hyperlinearity, keratosis pilaris
 - Hand or foot dermatitis
 - Nipple eczema
 - Cheilitis
 - Dennie–Morgan infraorbital fold
 - Orbital darkening
 - Facial pallor or facial erythema
 - Pityriasis alba
 - Perifollicular accentuation
 - White dermographism or delayed blanch

Pediatric Considerations

- 70% of all cases begin during the 1st 5 yr of life
- Only 10% of cases start in adulthood
- 30% of children with atopic dermatitis develop asthma, 35% develop allergic rhinitis
- Infant distribution is classically extensor surfaces and head and face

ESSENTIAL WORKUP

History and physical exam

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Clinical diagnosis
- IgE commonly elevated but does not usually need to be tested

Diagnostic Procedures/Surgery

Generally reserved for settings outside of the ED:

- Radioallergosorbent test (RAST) sometimes used to identify allergic triggers
- Patch testing used if contact dermatitis is suspected

DIFFERENTIAL DIAGNOSIS

- Seborrheic dermatitis
- Neurodermatitis (lichen simplex chronicus)
- Allergic contact dermatitis
- Irritant dermatitis
- Psoriasis
- Dyshidrosis
- Ichthyosis
- Scabies



TREATMENT

ED TREATMENT/PROCEDURES

- Mild disease or disease of the head and neck:
 - Low-potency corticosteroids such as hydrocortisone 1–2.5%
 - Eucerin cream: Apply to affected areas BID
- Moderate or severe disease of the trunk and extremities:
 - Higher-potency corticosteroids such as triamcinolone 0.1% (moderate potency) or fluocinonide 0.05% ointment (high potency)
- Severe disease of the head and neck:
 - Topical calcineurin inhibitors such as pimecrolimus and tacrolimus
- 1st-generation antihistamines:
 - Diphenhydramine, hydroxyzine are used for relief of itching but are only weakly effective
- Behavioral interventions:
 - Avoid excessive bathing
 - Use of tepid water and mild soaps
 - Frequent use of emollients (Eucerin cream, Aquaphor ointment)
- Bacterial superinfection: Cephalexin, cefazolin:
 - Consider MRSA

MEDICATION

- Aquaphor ointment: Apply to affected areas BID
 - Contains lanolin alcohol
- Cephalexin: 500 mg (peds: 25–100 mg/kg/24h) PO q6h
- Diphenhydramine: 25–50 mg (peds: 5 mg/kg/24h) PO or IV q6h
- Eucerin cream: Apply to affected areas BID
 - Contains lanolin alcohol
- Fluocinonide 0.05% ointment: Apply to affected areas of body BID for the duration of the flare (high potency)
- Hydrocortisone 2.5% ointment: Apply to affected areas of body/face BID for the duration of the flare (low potency)
- Hydroxyzine: 25–100 mg (peds: 2 mg/kg/24h) PO q4–6h
- Pimecrolimus 1% cream: Apply to affected areas BID (peds: > 2 yr of age) for the duration of the flare
- Tacrolimus ointment: 0.1% (peds: > 2 yr of age: 0.03%) apply to affected areas BID for the duration of the flare
- Triamcinolone 0.1% ointment: Apply to affected areas of body BID for the duration of the flare (mid potency)

First Line

- Hydrocortisone 2.5% ointment: Apply to affected areas of body/face BID for the duration of the flare (low potency)
- Aquaphor ointment: Apply to affected areas BID

Second Line

- Triamcinolone 0.1% ointment: Apply to affected areas of body BID for the duration of the flare (mid potency)
 - Avoid the face and eyelids
- Fluocinonide 0.05% ointment: Apply to affected areas of body BID for the duration of the flare (high potency)
 - Avoid the face and eyelids
- Tacrolimus ointment: 0.1% (peds: > 2 yr of age: 0.03%) apply to affected areas BID for the duration of the flare
 - Can be used on the face
- Pimecrolimus 1% cream: Apply to affected areas BID (peds: > 2 yr of age) for the duration of the flare
 - Can be used on the face



FOLLOW-UP

DISPOSITION

Issues for Referral

Dermatology referral for problematic cases

FOLLOW-UP RECOMMENDATIONS

- Patients should be warned of adverse consequences of treatment:
 - High-potency steroids can cause thinning of the skin
 - Tacrolimus and pimecrolimus cause a stinging sensation for the 1st wk of therapy. Long term use can increase risk of cancer

PEARLS AND PITFALLS

- Consider secondary cellulitis, as 90% of patients with atopic dermatitis are eventually colonized with *S. aureus*
- Use tacrolimus and pimecrolimus for moderate to severe disease of the head and neck
- Consider in any patient with a severely pruritic rash
- Lotions have low lipid content and can cause drying
 - Heavy creams are preferred
- Do not use triamcinolone or fluocinonide on face or eyelids

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See Also (Topic, Algorithm, Electronic Media Element)

- Cellulitis
- CA-MRSA



CODES

ICD9

- 691.8 Other atopic dermatitis and related conditions
- 692.9 Contact dermatitis and other eczema, unspecified cause

ICD10

- L20.9 Atopic dermatitis, unspecified
- L20.82 Flexural eczema
- L30.9 Dermatitis, unspecified

EDEMA

Laura J. Macnow

BASICS

DESCRIPTION

- Clinically apparent accumulation of extravascular fluid due to a derangement in the balance of oncotic and hydrostatic forces:
 - Increase in venous/capillary hydrostatic pressure
 - Decrease in plasma oncotic pressure
 - Increase in interstitial oncotic pressure
 - Increase in capillary permeability
 - Increase in lymphatic pressure due to obstruction
 - Combination of these factors
- Generalized, as with CHF or nephrotic syndrome
- Localized, as with deep vein thrombosis
- Increased venous hydrostatic pressure or decreased oncotic pressure results in pitting edema
- Protein-rich extravasated fluid results in nonpitting edema
- In certain disorders, there is no clear relation to Starling forces:
 - Idiopathic (cyclic) edema:
 - Worsened with heat
 - More common in women
 - Not necessarily related to menses

ETIOLOGY

- Generalized:
 - Heart failure
 - Cor pulmonale
 - Cardiomyopathies
 - Constrictive pericarditis
 - Pulmonary HTN:
 - Sleep apnea
 - COPD
 - Acute glomerulonephritis
 - Renal failure
 - Medication related (often secondary to salt retention):
 - Steroids/estrogens/progestins
 - NSAIDs
 - Antihypertensives (especially vasodilators)
 - Lithium

- Cyclosporine
- Insulin
- Thiazolidinediones (glitazones)
- Growth hormone
- Interleukin-2
- MAOIs
- Pramipexole
- Docetaxel
- Minoxidil
- Acute withdrawal of diuretics
- Idiopathic (cyclic) edema
- Myxedema
- Cirrhosis
- Nephrotic syndrome
- Protein-losing enteropathy/malabsorption
- Starvation
- Pregnancy
- Localized:
 - Deep vein thrombosis
 - Venous insufficiency
 - Thrombophlebitis
 - Chronic lymphangitis
 - Cellulitis
 - Baker cyst
 - Vasculitis
 - Angioedema:
 - Allergic
 - Acquired
 - Hypothyroidism (myxedema)
 - Mechanical trauma
 - Thermal injuries
 - Radiation injuries
 - Chemical burns
 - Hemiplegia
 - Reflex sympathetic dystrophy
 - Compressive or invasive tumor
 - Postsurgical resection of lymphatics
 - Postirradiation
 - Filariasis



SIGNS AND SYMPTOMS

- Weight gain of several kilograms
- Discomfort in the affected areas
- Swelling
- Tenderness
- Pitting edema:
 - Increased venous hydrostatic pressure or decreased oncotic pressure
- Nonpitting edema:
 - Protein-rich extravasated fluid
- Generalized edema (anasarca):
 - Edema is most prominent in dependent areas:
 - Feet
 - Sacrum
 - Bilateral lower extremities
 - Face/periorbital (especially in the morning)
 - Cardiac:
 - Dyspnea
 - Orthopnea
 - Paroxysmal nocturnal dyspnea
 - Increased jugular venous pressure
 - Rales
 - S3 gallop
 - Renal:
 - Anorexia
 - Puffy eyelids
 - Frothy urine
 - Oliguria
 - Dark urine
 - Hematuria
 - HTN
 - Hepatic:
 - Jaundice
 - Spider angiomas
 - Palmar erythema
 - Gynecomastia
 - Testicular atrophy
 - Ascites
 - Myxedema:
 - Pretibial nonpitting edema
 - Dry waxy swelling of skin and SC tissues
 - Periorbital most common (puffy eyes)
 - Nondependent areas
 - Fatigue

- Cold intolerance
- Weight gain
- Constipation
- Slowed deep-tendon reflex relaxation
- Idiopathic:
 - Diurnal weight gain/loss
- Localized:
 - Chronic venous insufficiency:
 - Chronic pitting
 - Skin discoloration (hemosiderin deposits)
 - Dermatitis/ulceration
 - Varicose veins
 - History of trauma:
 - Mechanical, thermal, radiation
 - Infectious/inflammatory:
 - Chills
 - Fever
 - Erythema
 - Increased warmth
 - Allergic:
 - Pruritus
 - Hives
 - Involvement of the lips and the oral mucosa

Pregnancy Considerations

- Common secondary to hormonally mediated fluid retention
- When involving hands and face, may be early sign of preeclampsia
- Dependent edema:
 - Usually in late pregnancy
 - From impedance of venous return
- Diuretics contraindicated

ESSENTIAL WORKUP

Diagnostic studies should be directed by the underlying etiology suggested by the history and physical exam.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Cardiac etiology suspected:
 - BNP or NT-proBNP
- Deep vein thrombosis suspected:
 - d-dimer (for patients with low clinical probability to rule out DVT)

- Renal etiology suspected:
 - Electrolytes
 - BUN and creatinine
 - Urinalysis
 - Urine electrolytes and protein
 - Serum lipids
- Hepatic etiology suspected:
 - Serum albumin
 - Liver function tests
 - Prothrombin time and partial thromboplastin time
- Myxedema suspected:
 - Thyroid function tests

Imaging

- Cardiac etiology suspected:
 - EKG
 - CXR
 - ECG
- Localized edema to an extremity:
 - US (duplex scanning) or contrast venography
- High suspicion for abdominal or pelvic malignancy:
 - Abdominal/pelvic CT

DIFFERENTIAL DIAGNOSIS

- Cellulitis
- Contact dermatitis
- Diffuse SC infiltrative process
- Lymphedema
- Obesity



TREATMENT

INITIAL STABILIZATION/THERAPY

See “ED Treatment.”

ED TREATMENT/PROCEDURES

- Treatment should be directed toward the underlying cause.
- Diuretics are usually indicated in cases of generalized edema but are not required emergently.
- Diuretics may be deleterious in patients with cirrhosis and ascites, as rapid fluid shifts may precipitate hepatorenal syndrome.

MEDICATION

- Amiloride: 5–10 mg PO daily
- Captopril: 6.25–100 mg PO TID (max. 450 mg/d)
- Furosemide: 20–80 mg IV/PO QID (max. 600 mg/d)
- Hydrochlorothiazide: 25–100 mg PO BID
- Spironolactone: 25–200 mg PO BID



FOLLOW-UP

DISPOSITION

Admission Criteria

- Base the decision to admit the patient on the underlying etiology.
- Concomitant cardiovascular or pulmonary compromise
- Inability to ambulate without adequate home support
- Hypoxia

Discharge Criteria

- Patient should be advised to decrease salt intake.
- Elastic support stockings
- Elevation of involved limbs

Issues for Referral

- Patients > 45 yr with chronic edema, or whose symptoms suggest a cardiopulmonary etiology require follow-up EKG.
- Patients with pulmonary HTN of unknown cause should be referred for a sleep study to evaluate for sleep apnea.
- A negative US in a patient at high risk for DVT requires urgent repeat study in 5–7 days.

FOLLOW-UP RECOMMENDATIONS

Patients with chronic edema may follow-up with primary care doctor for continued workup and treatment.

PEARLS AND PITFALLS

- Classify edema as generalized vs. localized, pitting vs. nonpitting.
- Pitting edema is caused by “protein-poor” extravasated fluid (by increased hydrostatic pressure or decreased oncotic pressure).
- Nonpitting edema is caused by “protein-rich” extravasated fluids (lymphedema or increased capillary permeability).
- Generalized or bilateral leg edema necessitates workup of systemic disease.

- Acute unilateral leg edema requires evaluation for DVT.
- Consider preeclampsia in pregnant patients.

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See Also (Topic, Algorithm, Electronic Media Element)

- Congestive Heart Failure
- Cor Pulmonale
- Deep Vein Thrombosis
- Angioedema
- Cirrhosis
- Venous Insufficiency
- Nephritic Syndrome
- Nephrotic Syndrome



CODES

ICD9

- 782.3 Edema
- 992.7 Heat edema
- 995.1 Angioneurotic edema, not elsewhere classified

ICD10

- R60.9 Edema, unspecified
- T67.7XXA Heat edema, initial encounter
- T78.3XXA Angioneurotic edema, initial encounter

EHRlichiosis

Roger M. Barkin • Jonathan A. Edlow

BASICS

DESCRIPTION

- Tick-borne human infection presenting as a nonspecific febrile illness
- Several forms of ehrlichiosis exist; 2 predominate in North America
 - Human monocytic ehrlichiosis (HME), 1st described in 1987:
 - Vector tick: *Amblyomma americanum* (lone star tick)
 - Geographic range: Central, southern, and mid-Atlantic states, with range expanding to parts of New England
 - Human granulocytic ehrlichiosis or human granulocytic anaplasmosis (HGE or HGA), 1st described in 1994:
 - Vector tick: *Ixodes scapularis* (deer tick)
 - Geographic range: East Coast, mid-Central States, and Pacific Northwest (same areas as Lyme disease which is more common in US than HME)
- All are tick borne but have different vectors and geographic ranges. Other species have been reported, but at present HME and HGE are the important ehrlichial human pathogens.

ETIOLOGY

- 2 distinct species of obligate intracellular organisms
- The taxonomy of these pathogens has changed in recent years as more DNA and ribosomal RNA data become available.
- HME is caused by the organism *Ehrlichia chaffeensis*.
- HGE/HGA is caused by *Anaplasma phagocytophila* (a new name as of 2002).
- The vasculitis found in Rocky Mountain spotted fever (RMSF) is usually not present.
- A 3rd type may also be encountered, caused by *Ehrlichia ewingii*, which has the tick vector of the lone star tick. Clinically similar to HME.
- Compared with RMSF, older individuals are usually affected, commonly >40 yr of age.

DIAGNOSIS

SIGNS AND SYMPTOMS

- Signs and symptoms of HME and HGE/HGA are similar.
- Many patients who are infected undergo asymptomatic seroconversion.
- The spectrum reported may overrepresent the more severely affected patients.

- With any tick-borne infection, patients can be coinfecting by more than 1 pathogen from the same tick bite:
 - May have a complicated presentation of 2 different diseases
- 1/4 of children have severe disease.

History

- The season and other epidemiologic factors are important in diagnosing tick-borne diseases:
 - Most commonly present from April to October
 - Variability is likely owing to changes in weather patterns from year to year and from region to region.
- Symptom onset from 1–2 wk (median 9–10 days) following the tick bite:
 - Bite of the larger lone star tick is more likely to be recalled by the patient than that of the smaller deer tick.
- Abrupt onset of:
 - Fever
 - Chills
 - Headache
 - Myalgias
 - Malaise
- Rash:
 - HME (35–60% of cases)
 - HGE or HGA (~5–10% of cases)
 - Often delayed and may be variable
- Symptoms may relate to complications of ehrlichiosis, such as:
 - ARDS
 - Renal failure
 - Hypotension and shock
 - Rhabdomyolysis
 - GI disturbances
 - CNS or peripheral nervous system (PNS) involvement, such as encephalopathy and meningitis as well as seizures
 - DIC
 - Immunocompromised patients have more severe complications.

Physical-Exam

- Fever
- Rash:
 - May be macular, maculopapular, or petechial
 - May be absent during 1st wk of illness
 - Usually involves trunk and spares hands and feet
- Lymphadenopathy

- Hepatosplenomegaly
- Neurologic findings:
 - Abnormal mental status
 - Meningismus
 - Nystagmus
- Pulmonary findings (rales, rhonchi) depending on pulmonary complications

Pediatric Considerations

- Fever, headache, and rash present in 48%
- Lymphadenopathy in 45%

ALERT

- Ehrlichiosis is a potentially fatal tick-borne illness that is usually diagnosed clinically.
- Consider this diagnosis in all patients with nonspecific febrile illnesses, especially during the warm months of the year, and definitely if there is a history of tick bite.
- The Centers for Disease Control and Prevention (CDC) define the illness as fever with 1 or more of the following: Headache, myalgia, anemia, leukopenia, thrombocytopenia, or elevation of serum transaminase; + serologic evidence of 4-fold change in IgG specific antibody by IFA or detection of specific target by PCR assay, demonstration of antigen on biopsy/autopsy sample, or isolation of organism in cell culture.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC:
 - Leukopenia
 - Thrombocytopenia
 - Anemia
- Hepatic transaminases:
 - Often elevated 2–6 times normal
- Indirect immunofluorescence antibody test, specific for HME and HGA
 - Usual test available
 - Threshold for a positive test is usually made by the individual lab testing the serum.
 - 94–99% sensitive when 2nd sample obtained over 14 days from onset of illness
- Wright stain of peripheral blood:
 - Morula may be seen:
 - Small intracytoplasmic ehrlichial DNA inclusion bodies
 - Diagnostic
 - Sensitivity of seeing morulae depend on who is looking, for how long,

and the immunologic competence of the patient.

- Found more commonly in HGE/HGA (~50%) than in HME (~10–15%)
- Culture and PCR for HEM and HGA
 - Not routinely available
- Antibody titer tests:
 - Not available in real time
- Lumbar puncture
 - Pleocytosis with predominance of lymphocytes and increased total protein

Imaging

- Head CT for encephalopathy
- CXR for fever/dyspnea

DIFFERENTIAL DIAGNOSIS

- Most tick-borne illnesses:
 - RMSF
 - Lyme disease
 - Babesiosis
- Many viral and bacterial infections and numerous other infectious diseases, especially early in their course, can initially present with an undifferentiated febrile illness similar to ehrlichiosis.
- Mononucleosis
- Thrombotic thrombocytopenia purpura
- Hematologic malignancy
- Cholangitis
- Pneumonia



TREATMENT

INITIAL STABILIZATION/THERAPY

ABCs

ED TREATMENT/PROCEDURES

- Initiate antibiotics:
 - Doxycycline:
 - Drug of choice
 - Children who are affected should also receive doxycycline. 14 days of treatment does not appear to cause significant discoloration of permanent teeth. The risks and benefits in children <9 yr old should be specifically discussed with parents.
 - Treatment should be continued for at least 3 days past defervescence

for a min. total course of 7 days. Severe or complicated disease requires a longer course.

– Rifampin for:

- Pregnant patients
 - Allergy to doxycycline
 - Mildly affected children < 9 yr of age
 - Patients who are pregnant, allergic to doxycycline, or mildly affected can be given rifampin for 7–10 days.
- Initiate therapy for other tick-borne diseases that may have been cotransmitted.

MEDICATION

Doxycycline:

- Adults: 100 mg IV/PO q12h for 10 days or for 3–5 days after defervescence.
- Children (severely affected): 4 mg/kg q12h IV/PO up to max. of adult dose; older children can be dosed as adult.

Pediatric Considerations

Despite the fact that doxycycline is generally contraindicated in patients < 9 yr old, it is the drug of choice in young children who are severely affected by ehrlichiosis. In less affected children, rifampin has been used successfully.

Pregnancy Considerations

Rifampin can be used to treat pregnant women with ehrlichiosis. When life-threatening disease is present, doxycycline may be considered.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Significant comorbidities/severely affected
- Cannot take PO antibiotics
- Immunosuppressed patients
- *E. chaffeensis* (HME) has a case fatality rate up to 3%.

Discharge Criteria

- Healthy appearing
- Symptoms typically last 1–2 wk; recovery occurring without sequelae.
- Long-term neurologic complications have been reported.

Issues for Referral

Severe disease or presence of complications

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Lyme Disease
- Rocky Mountain Spotted Fever
- Tick-borne Diseases



CODES

ICD9

- 082.40 Ehrlichiosis, unspecified
- 082.41 Ehrlichiosis chafeensis [E. chafeensis]
- 082.49 Other ehrlichiosis

ICD10

- A77.40 Ehrlichiosis, unspecified
- A77.41 Ehrlichiosis chafeensis [E. chafeensis]
- A77.49 Other ehrlichiosis

ELBOW INJURIES

Christian M. Sloane

BASICS

DESCRIPTION

Bony Injuries

- Supracondylar fracture:
 - Most common in children
 - Peak ages 5–10 yr, rarely occurs > 15 yr
 - Extension type (98%): Fall on outstretched *hand* (FOOSH) with fully extended or hyperextended arm:
 - Type 1: Minimal or no displacement
 - Type 2: Slightly displaced fracture; posterior cortex intact
 - Type 3: Totally displaced fracture; posterior cortex broken
 - Flexion type: Blow directly to flexed elbow:
 - Type 1: Minimal or no displacement
 - Type 2: Slightly displaced fracture; anterior cortex intact
 - Type 3: Totally displaced fracture; anterior cortex broken
- Radial head fracture:
 - Usually indirect mechanism (e.g., FOOSH)
 - Radial head driven into capitellum

Soft Tissue Injuries

- Elbow dislocation:
 - 2nd only to shoulder as most dislocated joint
 - Most are posterior.
- Medial/lateral epicondylitis:
 - Overuse injuries usually related to rotary motion at elbow
 - Involving attachment points of hand and wrist flexor/extensor groups to elbow
 - Plumbers, carpenters, tennis players, golfers
 - Pain made worse by resisted contraction of particular muscle groups

Pediatric Considerations

- Subluxed radial head (nursemaid's elbow)
- 20% of all upper extremity injuries in children
- Peak age 1–4 yr; occurs more frequently in females than males
- Sudden longitudinal pull on forearm with forearm pronated

ETIOLOGY

- Mechanism aids in determining expected injury.
- Trauma predominates.
- Most elbow injuries caused by indirect trauma are transmitted through bones of forearm (e.g., FOOSH)
- Direct blows account for very few fractures or dislocations.

DIAGNOSIS

SIGNS AND SYMPTOMS

How patient carries arm may give clues to diagnosis.

Bony Injuries

Supracondylar fracture:

- Flexion type:
 - Patient supports injured forearm with other arm and elbow in 90° flexion.
 - Loss of olecranon prominence
- Extension type:
 - Patient holds arm at side in S-type configuration.

Soft Tissue Injuries

- Elbow dislocations:
 - Posterior: Abnormal prominence of olecranon
 - Anterior: Loss of olecranon prominence
- Radial head subluxation:
 - Elbow slightly flexed and forearm pronated, resists moving arm at elbow
- Medial/lateral epicondylitis:
 - Gradual onset of dull ache over inner/outer aspect of elbow referred to forearm
 - Pain increases with grasping and twisting motions.

ESSENTIAL WORKUP

- Radiographs
- Assess wrist and shoulder for associated injury.
- Evaluate neurovascular status of limb.
- Assess skin integrity.
- Examine for compartment syndrome, which is more common in supracondylar fractures.

ALERT

- Injuries to ipsilateral upper limb, particularly fractures to midshaft humerus and distal forearm, are common.
- Evaluate for associated neurovascular injuries (up to 20%).

DIAGNOSIS TESTS & INTERPRETATION

Lab

None specific for elbow injuries

Imaging

- Not usually necessary if overuse injury suspected
- Routine anteroposterior (AP) and lateral; add oblique for assessment of subtle injuries to radial head/distal humerus.
- Fat pad sign:
 - Seen with intra-articular injuries
 - Normally, anterior fat pad is a narrow radiolucent strip anterior to humerus.
 - Posterior fat pad is normally *not* visible.
 - *Anterior fat pad sign* indicates joint effusion/injury when raised and becomes more perpendicular to anterior humeral cortex (sail sign).
 - *Posterior fat pad sign* indicates effusion/injury:
 - In adults, posterior fat pad sign without other obvious fracture implies radial head fracture.
 - In children, it implies supracondylar fracture.

Pediatric Considerations

- Fractures in children often occur through unossified cartilage, making radiographic interpretation confusing.
- A line drawn down the anterior surface of humerus should always bisect the capitellum in lateral view.
- If any bony relationships appear questionable on radiographs, obtain comparison view of uninvolved elbow.
- Suspect nonaccidental trauma if history does not fit injury.
- Ossification centers: 1st appear:
 - Capitellum: 3–6 mo
 - Radial head: 3–5 yr
 - Medial epicondyle: 5–7 yr
 - Trochlea: 9–10 yr
 - Olecranon: 9–10 yr
 - Lateral epicondyle: 9–13 yr

DIFFERENTIAL DIAGNOSIS

- Sprain/strain
- Effusion
- Contusion
- Bursitis

- Arthritis



TREATMENT

PRE HOSPITAL

Appropriate splinting

INITIAL STABILIZATION/THERAPY

Immobilization to prevent further injury before taking radiographs is essential.

ED TREATMENT/PROCEDURES

- Orthopedic consultation is recommended for all but nondisplaced, stable fractures, which can generally be splinted with 24–48 hr orthopedic follow-up.
- Fractures generally requiring orthopedic consultation:
 - _ Transcondylar, intercondylar, condylar, epicondylar fractures
 - _ Fractures involving articular surfaces such as capitellum or trochlea
- Supracondylar fractures:
 - _ Type 1 can be handled by ED physician with 24–48 hr orthopedic follow-up.
 - _ Elbow may be flexed and splinted with posterior splint.
 - _ Types 2 and 3 require immediate orthopedic consult.
 - _ Reduce these in ED when fracture is associated with vascular compromise.
- Anterior dislocation:
 - _ Reduce immediately if vascular structures compromised.
 - _ Then flex to 90° and place posterior splint.
- Posterior dislocation:
 - _ Reduce immediately if vascular structures compromised.
 - _ Then flex to 90° and place posterior splint.
- Radial head fracture:
 - _ Minimally displaced fractures may be aspirated to remove hemarthrosis; instill bupivacaine (Marcaine) and immobilize.
 - _ Other types should have orthopedic consult.
- Radial head subluxation:
 - _ In 1 continuous motion, supinate and flex elbow while placing slight pressure on radial head.
 - _ Hyperpronation technique is possibly more effective—while grasping the patient's elbow the wrist is hyperpronated until a palpable click is felt.
 - _ Often will feel click with reduction
 - _ If exam suggests fracture but radiograph is negative, splint and have patient follow up in 24–48 hr for re-evaluation.
- Medial/lateral epicondylitis:
 - _ Severe cases can be splinted.
 - _ Rest, heat, anti-inflammatory agents

ALERT

- Neurovascular injuries to numerous structures that pass about the elbow, including anterior interosseous nerve, ulnar and radial nerves, brachial artery
- Volkmann ischemic contracture is compartment syndrome of forearm.

MEDICATION

- Conscious sedation is often required to achieve reductions; see Conscious Sedation.
- Ibuprofen: 600–800 mg (peds: 5–10 mg/kg) PO TID
- Naprosyn: 250–500 mg (peds: 10–20 mg/kg) PO BID
- Tylenol with codeine no. 3: 1 or 2 tabs (peds: 0.5–1 mg/kg codeine) PO q4–6h; Do not exceed acetaminophen 4 g/24h
- Morphine sulfate: 0.1 mg/kg IV q2–6h
- Hydromorphone 5 mg/Acetaminophen 300 mg
- Acetaminophen do not exceed 4 g/24h
- Vicodin: 1–2 tabs PO q4–6h



FOLLOW-UP

DISPOSITION

Admission Criteria

- Vascular injuries, open fractures
- Fractures requiring operative reduction or internal fixation
- Admit all patients with extensive swelling or ecchymosis for overnight observation and elevation to monitor for and decrease risk for compartment syndrome.

Discharge Criteria

- Stable fractures or reduced dislocations with none of the above features
- Splint and arrange orthopedic follow-up in 24–48 hr.
- Uncomplicated soft tissue injuries

PEARLS AND PITFALLS

- Failure to appreciate that a posterior fat pad sign is abnormal.
- Always check for neurovascular injury with injuries about the elbow, especially with dislocations, pre- and postreduction.
- Always educate parents of a child with a supracondylar fracture about the signs and symptoms of compartment syndrome.

ADDITIONAL READING

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- Chasm RM, Swencki SA. Pediatric orthopedic emergencies. *Emerg Med Clin North Am.* 2010;28(4):907–926.
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CODES

ICD9

- 812.41 Closed supracondylar fracture of humerus
- 813.05 Closed fracture of head of radius
- 959.3 Elbow, forearm, and wrist injury

ICD10

- S42.414A Nondisp simple suprcndl fx w/o intrcndl fx r humerus, init
- S52.126A Nondisp fx of head of unsp radius, init for clos fx
- S59.909A Unspecified injury of unspecified elbow, initial encounter

ELECTRICAL INJURY

Marilyn M. Hallock



BASICS

DESCRIPTION

- Electricity is the flow of electrons through a conductor, across a gradient, from high to low concentration
- Nature and severity of electrical injuries depend on the voltage, current strength and type, resistance to flow, and duration of contact
- Ohm law: Voltage (V) = current (I) × resistance (R):
 - Voltage is directly proportional to current and is inversely proportional to resistance.
 - High-voltage (> 600 V) and low-voltage sources:
 - Telephone lines: 65 V
 - Household general circuit: 110 V
 - Electrical range or dryer: 220 V
 - Household power lines: 220 V
 - Subway 3rd rail: 600 V
 - Residential trunk line: 7,620 V
 - Industrial electrical power line: 100,000 V
 - Household devices can contain a transformer stepping up a seemingly low-voltage source to high voltage:
 - Microwave, television, computer
 - Resistance (R) is determined by the current's pathway through the body:
 - Nerves, muscles, blood vessels have low resistance and are better electrical conductors than are bone, tendon, fat
 - Water and sweat on skin decrease resistance; calloused skin increases resistance
 - More resistance means less flow, and more conversion to heat
 - Current is measured in amperes (I) and is a measure of the amount of energy flowing through an object:
 - “Let go” current is the max. current a person can grasp and release before muscle tetany inhibits letting go
 - Household general circuit: 15–30 A
 - Tingling sensation/perception: 0.2–2 mA
 - Pain: 1–4 mA
 - Average child “let go” current: 3–5 mA
 - Adult “let go” current: 6–9 mA; higher for men than women
 - Skeletal muscle tetany current: 16–20 mA
 - Respiratory muscle paralysis: 20–50 mA

- Ventricular fibrillation: 50–120 mA
- Alternating current (AC):
 - Electron flow rhythmically reverses direction:
 - Homes and offices in US use standard 60 Hz
 - Can produce continuous tetanic muscle contraction, loss of voluntary control of muscles, prolonged contact
 - More dangerous than direct current (DC)
 - More likely to result in ventricular fibrillation at household current level:
 - Stimulation can continue through T-wave period of cardiac cycle
- DC:
 - Continuous electron flow in 1 direction
 - Defibrillators and pacemakers, industrial sources
 - Large, single muscle spasm tends to throw victim from source:
 - Increased risk of traumatic blunt injuries
 - Shorter duration of exposure
 - More likely to result in asystole
- Trimodal distribution of electrical injuries:
 - Toddlers (household outlets and cords)
 - Teenagers (risk-taking behavior)
 - Adults (work-related injuries)

ETIOLOGY

Types of electrical injury:

- Direct contact causing tissue destruction:
 - Electrothermal burn may cause skin or deep tissue coagulation necrosis
 - Minor visible injuries may be misleading for extensive deep tissue injury
 - Location of damage is point of contact with source and point of contact with ground
- Flame:
 - Burns from burning clothing or other substances
- Electrical arc indirect contact:
 - Burns from the heat of a high-voltage arc (a flash burn) that passes electricity through air
 - May cause thermal and flame burns
 - Flash burns usually result in superficial partial-thickness burns
- Primary electrical phenomena:
 - Cardiac arrhythmias
 - Muscle contractions and tetany
- Secondary injury from trauma:
 - Supraphysiologic muscle contraction
 - Fall or being thrown

SIGNS AND SYMPTOMS

- **Head/neck/ENT:**
 - Common entry site for high-voltage injuries:
 - Facial and corneal burns
 - Perforated tympanic membranes
 - Cataracts and optic nerve atrophy may present initially, or delayed 4–6 mo
 - Intraocular hemorrhage, uveitis
 - Cervical spine injury
- **Cardiovascular:**
 - Cardiac arrest, asystole, and ventricular fibrillation are leading causes of death
 - Other arrhythmias and EKG findings: Sinus tachycardia, atrial fibrillation, premature ventricular contractions, transient ST-elevation, reversible QT-prolongation:
 - Sometimes delayed up to 12 hr
 - Usually resolve spontaneously
 - Myocardial damage occurs rarely:
 - Generally epicardial, not transmural
 - Damage does not follow distribution of coronary arteries
 - EKG will not show standard injury patterns
- **Respiratory:**
 - Brain injury causing respiratory center inhibition
 - Tetanic contraction/paralysis of chest wall/diaphragm muscles:
 - May cause respiratory arrest
 - Postcardiac arrest, respiratory arrest
 - Traumatic lung injury
 - Lung tissue itself appears resistant to electrical injury, probably owing to air content.
- **Neurologic:**
 - Respiratory arrest
 - Amnesia, transient confusion
 - Loss of consciousness, altered mental status, seizures, coma
 - Spinal cord injury:
 - May result from blunt trauma or DC effects (hand-to-hand flow)
 - Localized paresis up to/including quadriplegia
 - Long-term neurologic complications:
 - Seizures, peripheral nerve damage, spinal cord syndromes, psychiatric problems
- **Vascular:**

- Muscle necrosis and compartment syndromes
- Thrombosis in slow-moving venous system owing to coagulation
- Intimal injury in fast-moving arterial system may lead to acute or delayed arterial malfunction.
- Renal failure secondary to myoglobinuria
- Skeletal system/orthopedics:
 - Supraphysiologic tetanic muscle contractions from electrostimulation
 - Classically described injuries:
 - Vertebral column fracture
 - Posterior shoulder dislocation
 - Femoral neck fracture
- Dermatologic:
 - Contact/ground wounds: Hands, feet, and head most common and most severe sites
 - “Kissing” burns from current exit and re-entry on flexor surfaces

Pediatric Considerations

Mouth burn most common < 4 yr; sucking/biting on household electrical cord:

- Cosmetic deformity risk if commissure involved
- Delayed bleeding (3–5 days) from labial artery when eschar separates
- Risk of damage to developing dentition

Pregnancy Considerations

Fetus much less resistant to electrical shock than mother:

- Obstetric consult or referral for all pregnant patients regardless of symptoms:
 - Risk of placental abruption or threatened miscarriage
 - Fetal monitoring if > 20 wk gestation

History

- Determine whether exposure was high or low voltage, the duration and location of contact, or concomitant trauma
- If unwitnessed respiratory arrest or ventricular fibrillation in patient, consider electrical injury

Physical-Exam

Search the skin for entry/exit wounds and kiss/arch wounds at flexor surfaces

ESSENTIAL WORKUP

- Urinalysis for myoglobin
- EKG and cardiac enzymes for high-voltage victims, and low-voltage victims with cardiorespiratory complaints
- Cardiac monitoring indications:

- Cardiac arrest
- Loss of consciousness
- Chest pain
- Hypoxia
- Abnormal EKG
- Dysrhythmia in pre-hospital or ED setting
- History of cardiac disease
- Significant risk factors for coronary artery disease
- Suspicion of conductive injury
- Concomitant injury severe enough to warrant admission
- Prolonged monitoring is probably unnecessary in asymptomatic patients with normal EKG, no dysrhythmias, and exposure to < 240 V

DIAGNOSIS TESTS & NTERPRETATION

Lab

- For most exposures to household current, no testing is indicated:
 - Low-voltage burns can still cause dysrhythmias, seizures, and other complications if contact is near the chest or head
- Urinalysis for myoglobinuria
- Creatinine kinase, electrolytes, BUN, creatinine:
 - Positive urine myoglobin and/or high-voltage exposure
 - Provides baseline renal function, possible presence of hyperkalemia and metabolic acidosis
- Cardiac markers in:
 - Abnormal EKG or dysrhythmia
 - High-voltage exposures or low-voltage victims with cardiorespiratory complaints

Imaging

Dictated by clinical indications

DIFFERENTIAL DIAGNOSIS

- Thermal burns from electrical arcing flash burn vs. deep electrothermal injury
- Instability owing to traumatic injuries vs. electrical burns



TREATMENT

PRE HOSPITAL

- Secure scene; turn off power source for high-voltage incident
- Assume traumatic injury in unstable or unconscious patient:
 - Spinal immobilization

- Standard basic life support/advanced cardiac life support care
- Early CPR in postelectric shock arrest may allow time for heart to restart
- Splint fractures and dislocations
- Cover burns with clean, dry dressings

ALERT

Care must be exercised at scene to ensure that rescuers do not contact live electrical sources

INITIAL STABILIZATION/THERAPY

- ABCs
- Local wound care for thermal burns
- Immobilize/reduce fractures and dislocations

ED TREATMENT/PROCEDURES

- IV fluid resuscitation:
 - Larger fluid volumes may be required owing to extensive 3rd spacing in injured muscle.
 - Rapid administration to reach urine output of 1 mL/kg/hr
 - Foley catheter
- Evaluate for myoglobinuria and prevent renal failure:
 - Maintain good urine output
 - IV sodium bicarbonate increases solubility of myoglobin in urine
 - Consider furosemide/mannitol
 - Monitor renal function
- Tetanus prophylaxis
- Pain control as required

MEDICATION

- Bicarbonate: 1 ampule (50 mEq) IV, then add 2 ampules to 1 L of D₅W to maintain urine pH >7.45
- Furosemide: 0.5 mg/kg IV
- Mannitol: 25 g (ped: 0.25–0.5 mg/kg) IV bolus, then 12.5 mg/kg/h IV titrated to urine flow > 1 mL/kg/h



FOLLOW-UP

DISPOSITION

Admission Criteria

- Documented loss of consciousness
- Dysrhythmias, abnormal EKG, or evidence of myocardial damage

- Suspicion of deep tissue damage
- Myoglobinuria or acidosis
- Burn criteria for admit or transfer to burn center
- Traumatic injuries requiring admission
- Pregnant patients > 20 wk gestation

Discharge Criteria

- Minor, low-voltage injury (< 240 V) with no associated injuries, normal physical exam, and asymptomatic
- Cutaneous burns or mild persistent symptoms with normal EKG and no urinary heme pigment
- Stable in ED after period of observation
- Discharge 1st-trimester patient with threatened miscarriage instructions
- Pediatric patients with isolated oral burns and close adult care

Issues for Referral

- Burn wound care
- Persistence of current symptoms or new delayed symptoms:
 - Neurology for delayed weakness, paresthesias
- Obstetrics for pregnant patients
- Dental or reconstructive surgery for pediatric oral burns

FOLLOW-UP RECOMMENDATIONS

Ophthalmology for delayed cataracts in significant electrical current injuries

PEARLS AND PITFALLS

- Prolonged cardiac monitoring is probably unnecessary in asymptomatic patients with normal EKG, no dysrhythmias, and exposure to < 240 V
- With significant electrical burn injuries, administer enough IV fluid to maintain adequate urine output and to stabilize the vital signs:
 - Extensive 3rd spacing may occur

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- [Burns](#)
- [Lightning Injury](#)
- [Rhabdomyolysis](#)



CODES

ICD9

994.8 Electrocutation and nonfatal effects of electric current

ICD10

[T75.4XXA Electrocutation, initial encounter](#)

ENCEPHALITIS

Mary Saunders

BASICS

DESCRIPTION

- Acute inflammation of the brain
- 20,000 cases in US annually
- Mortality: 10%
- Inflammatory reaction occurs within brain parenchyma with destruction of neurons, parenchymal edema, and petechial hemorrhages
- Route of CNS infection usually hematogenous
 - Respiratory or GI tract
 - Blood transfusion
 - Organ transplant
- Neural migration occurs with rabies, herpes simplex virus (HSV), and varicella zoster virus (VZV) encephalitis

ETIOLOGY

- Viral is most common
- Noninfectious
 - Autoimmune, paraneoplastic, collagen vascular disease
- 50% of cases have no identifiable cause

Specific Viruses

- HSV:
 - 10–20% of all encephalitides
 - Primary or reactivation
 - Early treatment improves prognosis
- Arbovirus:
 - 10–15% of all encephalitides
 - Zoonotic transmission (mosquitoes, ticks) in warm months
 - Eastern equine causes fulminant encephalitis:
 - Tropism for the hippocampus
 - Abrupt onset of headache, fever, vomiting progressing to coma
 - Western equine occurs mostly in the western 2/3 of US:
 - Often preceded by nonspecific upper respiratory/GI tract symptoms
 - Japanese—most prevalent arboviral encephalitis worldwide:
 - Indolent course of fever, headache, myalgias, and fatigue followed by confusion, delirium, masklike facies, and parkinsonism, seizures, brainstem dysfunction, coma, and death

- **Flavivirus:**
 - West Nile virus—increased incidence in North America:
 - Found in mosquitoes and birds
 - Febrile illness, often with rash
 - Headache
 - Lymphadenopathy
 - Polyarthropathy
 - Increased morbidity/mortality in elderly patients
 - Flaccid paralysis can lead to respiratory failure with 50% mortality
- **Enteroviral:**
 - Occurs mainly in children < 10 yr old
 - Relatively benign course with little or no long-term sequelae
- **Measles encephalitis:**
 - Occurs several days to 2–3 wk after primary infection and rash, or after years of latent infection
 - Abrupt onset and rapid progression to coma
 - Seizures common (50–60%)
 - Postimmunization incidence of 1 per 1 million vaccinated
- **HIV encephalitis:**
 - Lower CD4 counts predispose to encephalitis
 - Typical features include motor spasticity and dementia
 - Involvement of white matter with extensive neural degeneration
- **Rhabdovirus: Rabies**
 - CNS infection in the absence of systemic infection

Nonviral

- *Mycoplasma pneumoniae*
- *Toxoplasma gondii*
- *Rickettsia rickettsii*
- *Mycobacterium tuberculosis*
- *Borrelia burgdorferi*
- *Bartonella henselae*

Immunocompromised/HIV Patients

- Histoplasma
- *Cryptococcus neoformans*
- VZV
- *Listeria monocytogenes*
- Cytomegalovirus (CMV)
- *T. gondii*
- Human herpesvirus type 6 (HHV-6)

Autoimmune

- Anti-LGI1 encephalitis
- Anti-N-methyl-D-aspartate receptor (anti-NMDAR) encephalitis

DIAGNOSIS

SIGNS AND SYMPTOMS

- Often begins with a preceding flulike illness over a few days:
 - _ Mild headache, fever, sore throat, reduced appetite, myalgias
- Altered level of consciousness, drowsiness, coma
- Impaired cognitive ability and personality change, hallucinations, psychosis
- Restlessness, agitation, irritability, delirium
- Rash:
 - _ Lyme disease
 - _ Rocky Mountain spotted fever
 - _ Varicella
 - _ HSV
- Seizures
- Fever, headache, vomiting, possible meningismus
- Focal neurologic deficits, tremor, ataxia, cranial nerve palsies (more common than meningitis)
- Papilledema on funduscopy
 - _ Autonomic dysfunction can lead to hypotension and cardiac arrhythmias
- Clinical picture varies from mild headache and mild cognitive/emotional lability to severe agitation, seizures, coma, permanent neurologic sequelae, and death
- Clinical course of symptoms may be slow moving or rapidly progressive

History

Arboviruses (eastern equine, western equine, St. Louis, and West Nile virus) cause disease when mosquitoes are active, whereas HSV can occur at any time

Physical-Exam

- Patients with encephalitis have an altered mental status ranging from subtle deficits to complete unresponsiveness
- Other findings reflect neurologic involvement

ESSENTIAL WORKUP

- Lumbar puncture
- Cell count/chemistry:
 - _ Elevated WBC, predominantly lymphocytes
 - _ Elevated protein
 - _ Glucose (normal in viral disease)

- Gram stain with or without India ink for suspected/confirmed HIV
- Viral and bacterial cultures (fungi if indicated by history)
- Antigen assays for:
 - HSV
 - Cryptococcus
 - Toxoplasmosis
 - Other viral antigen and antibody assays if available (enterovirus, adenovirus, CMV, mumps, varicella zoster)

DIAGNOSIS TESTS & NTERPRETATION

Lab

- CBC:
 - WBC usually elevated; however, a normal WBC does not exclude infection
- Electrolytes, glucose, BUN, creatinine
- Bacterial and viral blood cultures
- Liver function tests, ammonia level if hepatic failure suspected
- Carboxyhemoglobin level if CO poisoning suspected
- Toxicology screen if ingestion suspected in differential
- Polymerase chain reaction (PCR):
 - Confirm viral nucleic acids in CSF
 - HSV, varicella, enteroviruses, others
 - West Nile virus IgM serology

Imaging

- CT scan:
 - To rule out trauma, hemorrhagic conditions, and mass lesions
 - Cerebral edema may be the only finding consistent with encephalitis
 - HSV may show parenchymal hemorrhagic areas of the frontal and temporal lobes, along with edema
- MRI:
 - Hypodense temporal lobes in HSV

Diagnostic Procedures/Surgery

EEG may be useful in the presence of proven or suspected seizures

DIFFERENTIAL DIAGNOSIS

- Meningitis
- Brain abscess
- Sepsis
- Stroke (hemorrhagic or ischemic)
- Head injury
- Subarachnoid hemorrhage

- Encephalopathy (hepatic, uremic)
- Epilepsy
- Acute disseminated encephalomyelitis (ADEM)
- Metabolic:
 - Electrolyte abnormalities (Na^+ , K^+ , Cl^- , Ca^{2+} , Mg^{2+} , phosphate)
 - Hypoglycemia
 - Hyperglycemic nonketotic coma
- Neoplastic
- Drugs/toxins
- Carbon monoxide (CO) inhalation



TREATMENT

PRE HOSPITAL

Stabilize. Treat seizures

INITIAL STABILIZATION/THERAPY

- ABCs:
 - Intubate patients who are obtunded/comatose/absent gag reflex
- Naloxone, thiamine, glucose (or Accu-Chek) for altered mental status
- For signs of raised intracranial pressure on funduscopy or CT:
 - Hyperventilate to PCO_2 of 25–30 mm Hg
 - Administer mannitol
 - Neurosurgical consult for suspected hydrocephalus
- Run IV saline at KVO or half maintenance to avoid cerebral edema

ED TREATMENT/PROCEDURES

- Seizure control:
 - Abort with lorazepam or diazepam
 - Initiate antiseizure medication (fosphenytoin or phenobarbital) if more than 1 seizure has occurred
- No specific treatment for most viral encephalitides:
 - Steroid use controversial
- Treat HSV encephalitis with acyclovir IV:
 - Initiate if considered likely based on clinical grounds, CT, and CSF findings.
- Initiate ganciclovir and foscarnet for suspected immunocompromised-related infections (CMV, HHV-6).
- Administer antibiotic to cover for meningitis if diagnosis uncertain, especially when rash present (e.g., meningococemia, rickettsia)

MEDICATION

- Acyclovir: 10 mg/kg IV div. q8h, max. 30 mg/kg/d (peds: 20 mg/kg IV div. q8h up

to age 12 yr)

- Lorazepam: 2–4 mg per dose slow IV (peds: 0.05–0.1 mg/kg) per dose
- Diazepam: 5 mg IV per dose (peds: 0.1–0.2 mg/kg IV or 0.2–0.5 mg/kg per rectum)
- Fosphenytoin: Loading dose 20 mg/kg IV to a max. 1 g
- Ganciclovir: 5 mg/kg IV div. q12h
- Foscarnet: 90 mg/kg IV div. q12h or 60 mg/kg IV q8h
- Mannitol: 0.5–1 g/kg of a 20% solution to run IV over 20–30 min
- Phenobarbital: Load 15–20 mg/kg to 300–800 mg IV at 25 mg/min. Monitor respirations



FOLLOW-UP

DISPOSITION

Admission Criteria

All patients

PEARLS AND PITFALLS

Empiric treatment for HSV-1 infection with acyclovir should always be initiated as soon as possible if the patient has encephalitis without apparent explanation to decrease morbidity/mortality

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Meningitis



CODES

ICD9

- 049.9 Unspecified non-arthropod-borne viral diseases of central nervous system
- 058.29 Other human herpesvirus encephalitis
- 323.9 Unspecified causes of encephalitis, myelitis, and encephalomyelitis

ICD10

- A86 Unspecified viral encephalitis
- B00.4 Herpesviral encephalitis
- G04.90 Encephalitis and encephalomyelitis, unspecified

ENDOCARDITIS

Michael S. Murphy



BASICS

DESCRIPTION

An inflammation of the endothelial surface of the heart

- Various cardiac structures may be involved:
 - _ Native heart valves (most common)
 - _ Prosthetic valves
 - _ Interventricular septum
 - _ Chordae tendineae
 - _ Mural endocardium
 - _ Intracardiac devices
- Characterized by a vegetation (a thrombus with superimposed microorganisms)
 - _ Bacterial colonization of the initially sterile vegetation composed of fibrin and platelets
 - _ Bacterial growth enlarges the vegetation, further impeding blood flow and inciting inflammation.
 - _ Propagation of the infection through systemic emboli
- Almost always secondary to bacterial infection
- Rare noninfectious causes
 - _ Nonbacterial thrombotic endocarditis or marantic endocarditis
 - Often due to a hypercoagulable state
 - Small sterile vegetations
 - _ Libman–Sacks endocarditis
 - Complications of lupus erythematosus
 - Due to the deposition of immune complexes that cause an inflammatory reaction
 - Small vegetations

EPIDEMIOLOGY

- More common in men (ratios from 3.2 to 9.1)
 - _ M: 8.6–12.7 cases/100,000 person-yr
 - _ F: 1.4–6.7 cases/100,000 person-yr
- Risk factors:
 - _ Older patients
 - _ Poor dental hygiene
 - _ Comorbidities
 - Rheumatic heart disease
 - Prosthetic valve

- Hemodialysis
- Diabetes
- IV drug abuse (IVDA):
 - Greater risk than rheumatic heart disease or prosthetic valves
 - Predilection for right-sided heart valves
- Septic embolization
 - Cerebral complications
 - Cerebral embolism
 - Intracranial hemorrhage
 - Cerebral abscess
 - Extracerebral embolic events
 - Pulmonary
 - Splenic
 - Renal
 - Mycotic aneurysms (aorta, renal artery, splenic artery, hepatic artery, mesenteric arteries, etc.)
 - Hepatic
 - Coronary
- Risk factor for recurrent endocarditis:
 - Structural heart disease serves as common vegetative site due to altered intracardiac flow:
 - Mitral valve prolapse
 - Aortic valve dysfunction
 - Congenital heart disorders in the pediatric populations:
 - Tetralogy of Fallot
 - Aortic stenosis
 - Patent ductus arteriosus
 - Ventricular septal defects
 - Aortic coarctation
 - Prosthetic valves
 - Indwelling catheters
 - Any mechanical device may serve as a portal of entry or attachment for microorganisms.

ETIOLOGY

- Major categories:
 - Bacterial endocarditis
 - Prosthetic valve endocarditis
 - Nonbacterial thrombotic endocarditis:
 - Malignancy
 - Uremia
 - Burns
 - Systemic lupus erythematosus

- Common organisms:
 - *Staphylococcus aureus* (most common pathogen):
 - Seen in all populations, especially IVDA and toxic illness
 - Sometimes metastatic
 - *Streptococcus viridans*:
 - Found in oropharynx, common agent in native valve endocarditis
 - *Streptococcus bovis*:
 - Common association with colonic polyps or GI malignancy
 - *Streptococcus pneumoniae*:
 - Causes rapid valvular destruction, abscess, and CHF
 - Risk factor: Alcoholism
 - *Staphylococcus epidermidis*
 - Enterococci:
 - Seen in young women and old men following instrumentation or infection
 - Candida and Aspergillus:
 - Found in IVDA, prosthetic valves, or immunocompromised patients
 - HACEK (Haemophilus sp.)
 - Culture-negative endocarditis (Q fever, psittacosis, Bartonella, brucellosis)

DIAGNOSIS

SIGNS AND SYMPTOMS

- Fever:
 - Present in 86% of patients
 - May be absent in certain settings:
 - Elderly
 - CHF
 - Severe debility
 - Chronic renal failure
 - Flulike illness
 - Chills
 - Sweats
 - Rigors
 - Malaise
- Head, eyes, ears, nose, and throat:
 - Retinal hemorrhages or Roth spots
- Respiratory:
 - Dyspnea
 - Cough
 - Heart failure
- Cardiac:

- A new or changing murmur in 80–85% of patients
- Abdominal:
 - Abdominal or back pain
 - Splenomegaly (15–50%)
- Extremities:
 - Myalgias
 - Arthralgias
 - Digital clubbing
- Neurologic:
 - Altered mental status
 - Septic embolization (stroke or mycotic aneurysm)
- Skin:
 - Cutaneous vasculitic lesions:
 - Mucosal and conjunctival petechiae
 - Splinter hemorrhages
 - Osler nodes: Erythematous, painful tender nodules
 - Janeway lesions: Erythematous or hemorrhagic, macular or nodular lesions, a few millimeters in diameter on the hands and feet

History

- Fever duration and pattern
- Risk factors:
 - Prior cardiac disease
 - Source of bacteremia:
 - Indwelling intravascular catheters
 - IV drug use
 - Poor dental hygiene

Physical-Exam

- Heart and lung exam:
 - New cardiac regurgitant murmur
 - Heart failure
- Assess for splenomegaly.
- Assess for septic emboli:
 - Fundi, skin, nail beds
 - Careful neurologic exam for small focal deficits

ESSENTIAL WORKUP

- Identify risk factors for endocarditis in patients with fever of unknown etiology.
- Blood cultures
- ECG is needed to confirm the diagnosis.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC:
 - Anemia (sometimes hemolytic)
 - Leukocytosis (with granulocytosis and bandemia)
- Blood cultures:
 - Multiple sets (3 sets over a time period) should be obtained before antibiotic administration:
 - 5–10% with endocarditis have false-negative cultures
 - Consider culture of catheter device
- Elevated sedimentation rate and C-reactive protein (lacks specificity)
- Urinalysis:
 - Microscopic hematuria

Imaging

- CXR:
 - CHF
 - Septic pulmonary emboli, which may be seen in right-sided endocarditis
- EKG
 - Arrhythmia, new heart block
- Echocardiogram
 - Acute valvular pathology
 - Abscess
 - Vegetations
 - Transesophageal echo provides greater sensitivity.
- CT scan
 - May provide comprehensive information and valvular abnormalities

DIFFERENTIAL DIAGNOSIS

- Rheumatic fever
- Atrial myxoma
- Acute pericarditis
- MI
- Aortic dissection with regurgitant valve
- Thrombotic thrombocytopenic purpura
- Systemic lupus erythematosus
- Occult neoplasm with metastasis
- Septicemia
- Cotton fever



TREATMENT

INITIAL STABILIZATION/THERAPY

- Monitor for signs of heart failure.
- Operative repair if:
 - Severe valvular dysfunction causing failure
 - Unstable prosthesis
 - Perivalvular extension with intracardiac abscess
 - Antimicrobial therapy failure
 - Large or fungal vegetations
- Antibiotic therapy:
 - IV, bactericidal, and empiric, pending culture results
 - Native valve or congenital abnormality:
 - Penicillin G + nafcillin + gentamicin
 - Vancomycin + gentamicin
 - Prosthetic valve or history of IVDA:
 - Vancomycin + gentamicin + rifampin
 - Nafcillin + gentamicin + rifampin (if methicillin-resistant *S. aureus* [MRSA] is not suspected)
 - If MRSA vancomycin failure/intolerant consider daptomycin or quinupristin–dalfopristin
 - Vancomycin resistant
 - *Enterococcus faecium* consider quinupristin–dalfopristin
 - Enterococcal: Penicillin G + gentamicin; vancomycin + gentamicin
 - Enterococcal (gentamicin resistant): Penicillin G + streptomycin
 - Fungal:
 - Amphotericin B
 - HACEK:
 - Ceftriaxone

MEDICATION

- Amphotericin B:
 - Test dose 0.1 mg/kg up to 1 mg slow IV
 - Wait 2–4 hr.
 - If tolerated, begin 0.25 mg/kg IV and advance to 0.6 mg/kg IV QID
- Ceftriaxone: 2 g/d IV (peds: 100 mg/kg/24h)
- Daptomycin: 4 mg/kg/d U IV
- Gentamicin: 1 mg/kg IV q8h (peds: 3 mg/kg/24h in 3 equally div. doses)
- Nafcillin: 2 g IV q4h
- Penicillin G: 4 million IU IV q4h (peds: 300,000 U/kg/d div. into 4 equal doses)
- Quinupristin–dalfopristin: 7.5 mg/kg IV q8h (peds: 7.5 mg/kg/12h)
- Rifampin: 600 mg PO QID
- Streptomycin: 15 mg/kg/24h IV/IM in 2 equally div. doses (peds: 20 mg/kg/24h IV in 2 equally div. doses)
- Vancomycin: 15 mg/kg IV q12h (peds: 40 mg/kg/24h in 2–3 equally div. doses)



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients with risk factors who exhibit pathologic criteria or clinical findings
- All IV drug users with fever
- Admit patients with cardiovascular instability to an intensive care unit/monitored setting.

Discharge Criteria

None

FOLLOW-UP RECOMMENDATIONS

- Expected course:
 - Most patients will defervesce within 1 wk.
- Complications:
 - Cardiac: CHF, valve abscess, pericarditis, fistula
 - Neurologic: Embolic stroke, abscess, hemorrhage
 - Embolization: CNS, pulmonary, ischemic extremities
 - Mycotic aneurysms: Cerebral or systemic
 - Renal: Infarction, nephritis, abscess
 - Metastatic abscess: Kidney, spleen, tissue

PEARLS AND PITFALLS

- Fever, new or changing murmur
- 50% of cases occur in patients with no known history of valve disease
- Recent health care exposure/device consider as risk factor
- Common complications; watch for stroke, embolization, heart failure, intracardiac abscess
- Admit IV drug abusers presenting with fever to rule out endocarditis.
- Empiric therapy for acutely ill after 2–3 sets of blood cultures from separate venipuncture sites.

ADDITIONAL READING

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- Hoen B, Duval X. Clinical practice. Infective endocarditis. *N Engl J Med.* 2013;368(15):1425–1433.
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**CODES****ICD9**

- 421.0 Acute and subacute bacterial endocarditis
- 424.90 Endocarditis, valve unspecified, unspecified cause
- 996.61 Infection and inflammatory reaction due to cardiac device, implant, and graft

ICD10

- I33.0 Acute and subacute infective endocarditis
- I38 Endocarditis, valve unspecified
- T82.6XXA Infect/inflm reaction due to cardiac valve prosthesis, init

ENDOMETRIOSIS

Francis L. Counselman



BASICS

DESCRIPTION

- Presence of endometrial tissue and glands outside uterus
- An estrogen-dependent chronic inflammatory disease
- Affects 6–10% of women of reproductive age and 50–60% of women/teenage girls with pelvic pain
- Endometrial tissue found anywhere in pelvic cavity, on ovaries, uterine ligament (due to retrograde menstruation) and distant sites, including bowel and lungs

ETIOLOGY

- Unknown

Pediatric Considerations

Not prior to menarche

RISK FACTORS

- Anatomic obstruction of menstrual outflow
- Early menarche
- Short menstrual cycles
- Genetic component suggested by twin and family studies



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Dysmenorrhea (50–90%)
- Deep pelvic pain
- Dyspareunia
- Dysfunctional uterine bleeding
- Lower abdominal pain
- Nausea, abdominal distention
- Infertility (30–50%)

Physical-Exam

- Focal pain or tenderness on pelvic exam
- Tenderness along uterosacral ligament

- Retroverted uterus
- Rectovaginal nodularity
- Pelvic mass
- Physical exam can vary depending on location of endometrial tissue
- Catamenial pneumothorax occurs during menses due to pleural endometriosis

ESSENTIAL WORKUP

- Pregnancy test
- GC/chlamydia testing
- Other tests as directed by history and physical exam
- Rarely diagnosed in ED

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Pregnancy test
- GC/chlamydia testing
- Hematocrit if bleeding
- Type and screen if significant bleeding
- Other labs as directed by history and physical exam

Imaging

- Ultrasound (11% sensitivity)
- Doppler ultrasound
- CT scan (15% sensitivity)
- MRI (69% sensitivity; 75% specificity)
- Typically not helpful in ED

Diagnostic Procedures/Surgery

Laparoscopy usually required for definitive diagnosis

DIFFERENTIAL DIAGNOSIS

- Appendicitis
- Dysfunctional uterine bleeding
- Ectopic pregnancy
- Inflammatory bowel disease
- Irritable bowel disease
- Menstrual cramps/mittelschmerz
- Ovarian cyst
- Ovarian torsion
- Pelvic inflammatory disease
- Tubo-ovarian abscess



TREATMENT

PRE HOSPITAL

- Stabilize as needed.
- Pain control as necessary

INITIAL STABILIZATION/THERAPY

- Treat hypotension or tachycardia from blood loss with isotonic IV fluids
- May need to transfuse packed red blood cells (PRBCs) if significant bleeding

ED TREATMENT/PROCEDURES

- Analgesia
- Oral contraceptive (i.e., medroxyprogesterone acetate) or gonadotropin-releasing hormone agonist (i.e., leuprolide acetate) in consultation with gynecologist or primary care physician
- Gynecology consultation for significant bleeding, pain, or serious complication

MEDICATION

- Ibuprofen: 400–800 mg PO q6–8h (max. 3.2 g/d)
- Acetaminophen: 325–650 mg PO q4–6h (max. 4 g/d)
- Ketorolac: 15–30 mg IV or 30–60 mg IM
- Morphine: 4–8 mg IM/IV or equivalent analgesic

First Line

- Ibuprofen: 400–800 mg PO q6–8h (max. 3.2 g/d)
- Acetaminophen: 325–650 mg PO q4–6h (max. 4 g/d)
- Ketorolac: 15–30 mg IV or 30–60 mg IM



FOLLOW-UP

DISPOSITION

Admission Criteria

- Intractable pain
- Significant bleeding
- Unclear diagnosis
- Need for further workup and treatment
- Peritoneal signs

Discharge Criteria

Most patients with suspected endometriosis can be discharged with pain control and gynecology referral

FOLLOW-UP RECOMMENDATIONS

Suspected cases of endometriosis should be referred to a gynecologist for evaluation and treatment

PEARLS AND PITFALLS

- Occurs in 6–10% of women of reproductive age
- Endometriosis frequently causes cyclical pelvic pain
- Rarely diagnosed initially in ED; delay between symptom onset and diagnosis frequently years
- Rule out other emergency medical conditions and treat symptoms as needed
- Endometriosis is a chronic condition that necessitates outpatient monitoring by a gynecologist or primary care physician

ADDITIONAL READING

- Cirilli AR, Cipot SJ. Emergency evaluation and management of vaginal bleeding in the nonpregnant patient. *Emerg Med Clin North Am.* 2012;30:991–1006.
- Giudice LC. Clinical practice. Endometriosis. *N Eng J Med.* 2010;362:2389–2398.
- McLeod BS, Retzliff MG. Epidemiology of endometriosis: An assessment of risk factors. *Clin Obstet Gynecol.* 2010;53:389–396.
- Vercellini P, Crosignani P, Somigliana E, et al. “Waiting for Godot”: A commonsense approach to the medical treatment of endometriosis. *Hum Reprod.* 2011;26:3–13.



CODES

ICD9

- 617.1 Endometriosis of ovary
- 617.3 Endometriosis of pelvic peritoneum
- 617.9 Endometriosis, site unspecified

ICD10

- N80.1 Endometriosis of ovary
- N80.3 Endometriosis of pelvic peritoneum
- N80.9 Endometriosis, unspecified

EPIDIDYMITIS/ORCHITIS

Matthew D. Cook • Kevin R. Weaver

BASICS

DESCRIPTION

Epididymitis

- **Definition:** Inflammation or infection of the epididymis
- Rare in prepubertal boys
- **Pathogenesis:**
 - Initial stages:
 - Cellular inflammation begins in vas deferens, descends to epididymis
 - Acute phase:
 - Epididymis is swollen and indurated in upper and lower poles.
 - Spermatic cord thickened
 - Testis may become edematous owing to passive congestion or inflammation.
 - Resolution:
 - May be complete without sequelae
 - Peritubular fibrosis may develop, occluding ductules.
- **Complications:**
 - 2/3 of men have atrophy due to partial vascular thrombosis of testicular artery.
 - Abscess and infarction rare (5%)
 - Incidence of infertility with unilateral epididymitis unknown:
 - 50% with bilateral epididymitis

Orchitis

- **Definition:** Inflammation or infection of the testicle:
 - Usually from direct extension of the same process within the epididymis
 - Isolated testicular infection is rare:
 - Can result from hematogenous spread of bacteria or following mumps infection
- **Categories:**
 - Pyogenic bacterial orchitis secondary to bacterial involvement of epididymis
 - Viral orchitis:
 - Most commonly due to mumps
 - Rare in prepubertal boys; occurs in 20–30% of postpubertal boys with mumps.
 - Occurs 4–6 days after parotitis but can occur without parotitis.
 - Unilateral in 70% of patients

- Usually resolution in 6–10 days
- 30–50% of testes involved have residual atrophy; rarely affects fertility
- Granulomatous orchitis:
 - Syphilis
 - Mycobacterium and fungal diseases
 - Usually occurs in immunocompromised host

ETIOLOGY

Epididymitis

- Children:
 - Most common in children < 1 yr or between the ages of 12–15 yr
 - Etiology identified in only 25% of prepubertal boys
 - Coliform or pseudomonal UTI
 - Sexually transmitted diseases rare in prepubertal males
 - Associated with predisposing abnormalities of lower urinary tract
- Young men, age < 35 yr:
 - Usually sexually transmitted
 - *Chlamydia trachomatis* (28–88%) with severe inflammation with minimal destruction
 - *Neisseria gonorrhoea* (3–28%)
 - Coliform bacteria (7–24%):
 - Highly destructive with tendency for abscess
 - Coliform bacteria more common in insertive partners in anal intercourse
 - *Ureaplasma urealyticum* (sole organism in only 6% of cases)
- Older men, age > 35 yr:
 - Commonly associated with underlying urologic pathology (benign prostatic hypertrophy, prostate cancer, strictures)
 - May have acute or chronic bacterial prostatitis
 - Coliform bacteria more common (23–67%), especially after instrumentation
 - *C. trachomatis* (8–80%)
 - Klebsiella and Pseudomonas species
 - *N. gonorrhoea* (15%)
 - Gram-positive cocci
- Drug related:
 - Amiodarone-induced epididymitis:
 - Usually with amiodarone levels > therapeutic levels
- Granulomatous:
 - Etiology maybe related to mycobacterial, syphilis, or fungal infections:
 - *Mycobacterium tuberculosis* is the most common cause of granulomatous disease affecting the epididymis

- Suspect in HIV patients
- Urine cultures often negative for *M. tuberculosis*
- Vasculitis:
 - Polyarteritis nodosa
 - Behçet disease
 - Henoch–Schönlein purpura

Orchitis

- Pyogenic bacterial orchitis:
 - *Escherichia coli*
 - *Klebsiella pneumoniae*
 - *Pseudomonas aeruginosa*
 - Staphylococci
 - Streptococci
- Viral orchitis:
 - Mumps:
 - 20% may develop epididymo-orchitis.
 - Rarely associated with live-attenuated mumps vaccine
- Coxsackie A and lymphocytic choriomeningitis virus
- Granulomatous orchitis: Syphilis, mycobacterial and fungal diseases:
 - Suspect in HIV patients
- Fungal orchitis:
 - Blastomycosis in endemic regions
 - Invasive candidal infections in immunosuppressed hosts
- Post-traumatic orchitis: Inflammation

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Gradual onset of mild to moderate testicular or scrotal pain, usually unilateral
- Progressive scrotal swelling
- Dysuria (30%):
 - Recent UTI
 - History of abnormal bladder function
- Urethral discharge:
 - Of patients with gonococcal epididymitis, 21–30% did not complain of urethral discharge.
 - No demonstrable urethral discharge in 50%
- Fever (14–28%)
- Recent urethral instrumentation or catheterization

Physical-Exam

- Tenderness in groin, lower abdomen, or scrotum
- Scrotal skin commonly erythematous and warm
- Early:
 - May feel swollen, indurated epididymis
- Later:
 - May not be able to distinguish epididymis from testis
 - Spermatic cord may be edematous.
- Intact cremasteric reflex
- Prehn sign:
 - Pain relief with testicular elevation
 - Commonly observed but not specific
- Coexistent prostatitis is rare (8%).
- Pyogenic bacterial orchitis:
 - Patients usually are acutely ill.
 - Fever
 - Intense discomfort, swelling of testicle
 - Often reactive hydrocele

ESSENTIAL WORKUP

- Must differentiate from testicular torsion
- Early consultation with urologist if strong suspicion of testicular torsion

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC:
 - Often leukocytosis in the range of 10,000–30,000/mm³
- Urinalysis and culture:
 - Positive leukocyte esterase on first-void urine or >10 WBC per high-power field on first-void urine sediment
 - 15–50% of patients with epididymo-orchitis have pyuria.
 - 24% of patients have positive urine bacterial cultures.
- Urethral swab (50–73% have demonstrable urethritis despite minority of symptoms)
 - Gram stain and culture or DNA amplification for *C. trachomatis*/*N. gonorrhoea*
 - Avoid bladder emptying within 2 hr of tests (lowers sensitivity).
 - Especially for postpubertal and sexually active
- Blood culture if systemically ill

Imaging

- US: Color Doppler imaging:

- 82–100% sensitivity, 100% specificity in detecting testicular torsion or decreased blood flow
- Epididymo-orchitis:
 - Hyperemia
 - Increased vascularity and blood flow
- Advantages:
 - Can evaluate for epididymitis or other causes of scrotal pain
 - 70% sensitivity, 88% specificity for epididymitis
- Disadvantages:
 - Highly examiner dependent
 - Difficult in infants or children
- Testicular scintigraphy:
 - Radionuclide study to assess perfusion
 - 90–100% sensitivity, 89–97% specificity in detecting testicular torsion
 - Inflammatory processes have increased flow and uptake.
 - Not routinely available at many institutions

Diagnostic Procedures/Surgery

Surgical exploration indications:

- Scrotal abscess
- If torsion cannot be excluded
- Suspected or proved ischemia caused by severe epididymitis
- Patient with solitary testicle
- Scrotal fixation: Indicates severe inflammation and potential suppuration

DIFFERENTIAL DIAGNOSIS

- Testicular torsion
- Testicular tumor
- Torsion of testicular appendages
- Trauma to scrotum
- Acute hernia
- Acute hydrocele



TREATMENT

PRE HOSPITAL

- IV access
- IV fluids, especially if systemically ill

INITIAL STABILIZATION/THERAPY

- IV access
- IV fluids, especially if systemically ill

ED TREATMENT/PROCEDURES

- **Antibiotics:**
 - Cover for chlamydial and gonococcal etiologies if adult or presumed sexually transmitted
 - Cover for coliform etiology:
 - Child, or adult > 35 yr of age
 - Insertive partner in anal intercourse
 - Presumed nonsexually transmitted
- Bed rest, scrotal support, ice packs
- Analgesics and anti-inflammatories

MEDICATION

- Age < 35 yr or sexually active postpubertal males:
 - Ceftriaxone 250 mg IM once + doxycycline 100 mg PO BID for 10 days:
 - May substitute azithromycin 1 g PO once for doxycycline if tetracycline allergy
 - Quinolones no longer recommended if suspect *N. gonorrhoea*
- Age > 35 yr or insertive partners in anal intercourse or negative culture/DNA amplification for *C. trachomatis*/*N. gonorrhoea* or allergy to cephalosporins/tetracyclines:
 - Ofloxacin 300 mg PO BID or levofloxacin 500 mg/d PO for 10 days

Pediatric Considerations

- Bacterial epididymitis is uncommon in prepubertal boys and antibiotic regimens are not well established.
- If concurrent UTI:
 - TMP–SMX: 4 mg/kg TMP and 20 mg/kg SMX BID for 10 days
- Avoid quinolones and tetracyclines in children



FOLLOW-UP

DISPOSITION

Admission Criteria

- Surgical indications present
- Older age group if it is the only way to ensure appropriate workup:
 - Many will have underlying urologic pathology.
- Systemically ill, fever, nausea, vomiting
- Scrotal abscess
- Intractable pain

Discharge Criteria

- Fails to meet admission criteria
- Patient with good follow-up
- Able to take oral antibiotics

Issues for Referral

- Children need workup for urologic abnormalities:
 - Voiding cystourethrography, renal US
- If bacteriuria present, exam of lower tract with cystoscopy after treatment completed

FOLLOW-UP RECOMMENDATIONS

- Failure to improve within 3 days of commencing antibiotics warrants urologic evaluation.
- Persistence of symptoms after full antibiotic course warrants search for other causes of epididymitis:
 - TB or fungal epididymitis, scrotal abscess, tumor, infarction.
- Sexual partners of patients with suspected or confirmed *C. trachomatis*/*N. gonorrhoea* should be tested/treated.
- Children need urology consult for evaluation of structural urogenital abnormalities.

PEARLS AND PITFALLS

- Testicular torsion should be ruled out in all cases of new-onset testicular pain.
- Epididymitis usually due to STD in sexually active men < 35 yr
- Epididymitis usually due to coliform bacteria in men > 35 yr
- Antibiotic treatment is started immediately and empirically based on clinical picture.

ADDITIONAL READING

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- Tracy CR, Steers WD, Costabile R. Diagnosis and management of epididymitis. *Urol Clin North Am*. 2008;35(1):101–108.
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See Also (Topic, Algorithm, Electronic Media Element)

- Gonococcal Disease
- Prostatitis
- Testicular Torsion
- Urethritis



CODES

ICD9

- 604.90 Orchitis and epididymitis, unspecified
- 604.91 Orchitis and epididymitis in diseases classified elsewhere
- 604.99 Other orchitis, epididymitis, and epididymo-orchitis, without mention of abscess

ICD10

- N45.1 Epididymitis
- N45.2 Orchitis
- N45.3 Epididymo-orchitis

EPIDURAL ABSCESS

Richard S. Krause

BASICS

DESCRIPTION

- A rare pyogenic infection of the spinal epidural space
 - 2–25/100,000 admissions
- Most common in thoracic spine, followed by lumbar and cervical

ETIOLOGY

- Focus of infection is present followed by either hematogenous spread (~50%) or direct extension
 - No focus identified in ~1/3
- Most common source is skin infection:
 - Any pyogenic infection may be source
- *Staphylococcus aureus* accounts for >50% of cases:
 - Many are MRSA
 - Streptococcus is 2nd most common
- *Haemophilus influenzae*, gram-negative bacilli, mycobacteria, anaerobic, coagulase-negative Staphylococcus, fungal, and mixed infections also occur
- Complication of epidural catheter or spinal surgery
- Unusual complication of lumbar puncture (usually follows multiple attempts)

Pediatric Considerations

- Children present similar to adults with back pain, fever, and neurologic signs as well as nonspecific systemic symptoms
- Infants may exhibit only fever, irritability, and associated meningitis
- Sphincter disturbance is frequently seen
- Usually secondary to hematogenous spread
- Location and bacteriology similar to adults

DIAGNOSIS

SIGNS AND SYMPTOMS

- Fever and severe back pain represent “red flag” for potentially serious condition:
 - If pain is radicular or there is neurologic disturbance, likelihood of epidural abscess is increased
- Classic presentation:
 - Severe, progressive back pain (often radicular)
 - Fever

- Neurologic deficit:
 - Weakness or paralysis
 - Sensory level
 - Sphincter disturbance
 - May present with signs and symptoms of sepsis without prominent back pain
- Occurs at all ages including infants:
 - Peak is at ages 60–70 yr
- Most patients have predisposing condition:
 - IV drug abuse (IVDA)
 - Diabetes
 - Malignancy
 - Chronic steroids
 - Chronic alcoholism
 - Instrumentation or spinal surgery
 - Indwelling vascular catheter
- May occur in the absence of identifiable predisposing factors

History

- Back pain
- Fever
- Neurologic deficit:
 - Weakness
 - Paresthesias
 - Incontinence

Physical-Exam

- Fever
- Localized spinal tenderness and/or erythema
- Neurologic deficit
- Evidence of IV drug use or other predisposing factors

ESSENTIAL WORKUP

- History should include predisposing conditions when this diagnosis is suspected
- Physical exam for source of infection, localized spinal tenderness, and neurologic findings:
 - Decreased sphincter tone
 - Saddle anesthesia
 - Lower extremity weakness
- Postvoid residual or sonography
- Younger adults should have < 50 mL postvoid residual urine:
 - Older adults may have residual of 100 mL
- Erythrocyte sedimentation rate (ESR) as below

- MRI with and without gadolinium contrast is the diagnostic test of choice:
 - CT with IV contrast or myelography if MR not available
 - Suspected epidural abscess is an emergency and requires emergent imaging

DIAGNOSIS TESTS & INTERPRETATION

Lab

- ESR is almost always elevated (~100%), but is nonspecific:
 - Normal ESR makes diagnosis much less likely
- C-reactive protein nearly always elevated
- Blood cultures often positive (~60%)
- Leukocytosis with left shift is common (~70%)
- CSF often abnormal, but nondiagnostic; routine lumbar puncture should be avoided when epidural abscess is primarily suspected (may cause meningitis)

Imaging

- MRI is at least 90% sensitive:
 - Shows high-intensity lesion on T2 imaging
- CT with IV contrast if MR or myelography not available
- Myelography and CT myelography are also sensitive, but risk dissemination
- Plain films are often abnormal but nonspecific

DIFFERENTIAL DIAGNOSIS

- Diagnosis is difficult owing to rarity of condition and nonspecific symptoms:
 - Multiple physician encounters commonly precede diagnosis
 - Most common initial diagnosis is benign musculoskeletal pathology: Muscular or ligamentous pain, degenerative arthritis, compression fracture, discogenic pain
- Back pain with fever, systemic signs, and symptoms:
 - Vertebral osteomyelitis
 - Spinal tumor (usually there is a known primary)
 - Meningitis (cervical epidural abscess may mimic, but bacterial meningitis usually associated with abnormal mental status)
 - Discitis (usually postinstrumentation)
 - Pyelonephritis
- Back pain with neurologic signs and symptoms:
 - Cord compression
 - Cord ischemia
 - Disc herniation

Pediatric Considerations

Fever and back pain should be urgently investigated with MRI when epidural abscess is suspected



TREATMENT

PRE HOSPITAL

Spinal immobilization if trauma suspected or other cause of fracture suspected

INITIAL STABILIZATION/THERAPY

Broad-spectrum parenteral antibiotics early

- Must include coverage for *S. aureus*, Streptococci, and gram-negative rods
- Vancomycin (for possible MRSA) and a 3rd-generation cephalosporin (for gram-negative coverage) are appropriate initial antibiotics
- Cover *Pseudomonas* if IVDA
- 1 suggested regimen includes vancomycin, ceftazidime, and metronidazole (for anaerobes)

ED TREATMENT/PROCEDURES

- Urgent imaging essential when diagnosis is considered
- Delay in treatment is associated with poorer outcome
- If unable to localize lesion on physical exam, consider imaging entire spine
- Urgent neurosurgical consultation or transfer for definitive therapy (surgical decompression) after diagnosis and antibiotic administration:
 - Conservative treatment (prolonged [6 wk] antibiotic therapy) may be successful

MEDICATION

- Ceftazidime: 2 g (peds: 50 mg/kg)
- Vancomycin: 15 mg/kg IV loading dose (peds: 10–15 mg/kg q6–8h) q12h
- Metronidazole: 500 mg IV

First Line

- Ceftazidime
- Vancomycin
- Metronidazole if anaerobes suspected



FOLLOW-UP

DISPOSITION

Admission Criteria

Patients with epidural abscess should be admitted; MRI is needed emergently; transfer patient if necessary

Discharge Criteria

Patients with definite or strongly suspected epidural abscess should not be discharged

Issues for Referral

Patients with spinal epidural abscess require admission to facility with neurosurgical capability:

- Transfer usually indicated if a neurosurgeon or MRI is unavailable:
 - Administer antibiotics and obtain blood cultures (positive in ~ 60%) prior to antibiotics unless this results in a delay

PEARLS AND PITFALLS

- Successfully treated epidural abscess may reoccur, especially in the setting of decreased immunity
- Patients with Staphylococcal bacteremia and back pain or neurologic signs/symptoms should be investigated for epidural abscess
- Failure to order images that include the involved area:
 - Careful physical exam for areas of spinal tenderness and level of neurologic deficit may help avoid this pitfall:
 - Consider both thoracic and lumbar imaging for a problem suspected in the mid back and cervical and thoracic imaging for upper back and neck pathology
 - If unable to localize lesion on physical exam, consider imaging entire spine

ADDITIONAL READING

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- Davis DP, Salazar A, Chan TC, et al. Prospective evaluation of a clinical decision guideline to diagnose spinal epidural abscess in patients who present to the emergency department with spine pain. *J Neurosurg Spine.* 2011;14:765–770.
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CODES

ICD9

324.1 Intraspinal abscess

ICD10

G06.1 Intraspinal abscess and granuloma

EPIDURAL HEMATOMA

Stephen R. Hayden

BASICS

DESCRIPTION

- Direct skull trauma
- Inward bending of calvarium causes bleeding when dura separates from skull:
 - Middle meningeal artery is involved in bleed > 50% of time.
 - Meningeal vein is involved in 1/3.
- Skull fracture is associated in 75% of cases, less commonly in children.
- > 50% have epidural hematoma (EDH) as isolated head injury:
 - Most commonly associated with subdural hematoma (SDH) and cerebral contusion
- Classic CT finding is lenticular, unilateral convexity, usually in temporal region.
- It usually does not cross suture lines, but may cross midline.

ETIOLOGY

- Accounts for 1.5% of traumatic brain injury (TBI)
- Male/female incidence is 3:1.
- Peak incidence is 2nd–3rd decade of life.
- Motor vehicle accidents (MVAs), assault, and falls are most common causes:
 - Of all blunt mechanisms, assault has highest association with intracranial injury requiring neurosurgical intervention.
- Uncommon in very young (< 5 yr) or elderly patients
- Mortality is 12% and is related to preoperative condition.

Pediatric Considerations

- Head injury is the most common cause of death and acquired disability in childhood.
- Falls, pedestrian-struck bicycle accidents are most common causes:
 - Most severe head injuries in children are from MVA.
 - Always consider possibility of nonaccidental trauma.
- < 50% have altered level of consciousness (LOC):
 - If EDH in differential diagnosis (DD), CT should be obtained.
- Bleeding is more likely to be venous.
- Good outcome in 95% of children < 5 yr

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Altered or deteriorating LOC
- LOC: 85% will have at some point in course:
 - Only 11–30% will have a lucid interval.
- Nausea and vomiting: 40%

Pediatric Considerations

- Many times the only clinical sign is drop in hematocrit (Hct) of 40% in infants.
- Bulging fontanel with vomiting, seizures, or lethargy also suggests EDH in infants.
- <50% of children have LOC at time of injury.
- Posterior fossa lesions are seen more commonly in children.

Physical-Exam

- Pupillary dilation: 20–40%:
 - Usually on same side as lesion (90%)
- Hemiparesis > 1/3:
 - Usually on opposite side from lesion (80%)

ESSENTIAL WORKUP

Head imaging, as below

DIAGNOSIS TESTS & INTERPRETATION

Lab

- ABG, CBC, chemistry, PT/PTT
- Blood ETOH and drug screen as appropriate

Imaging

- Noncontrast CT of head:
 - Admission perfusion CT may help predict prognosis.
 - Lenticular, biconvex hematoma with smooth borders may be seen.
 - Mixed density lesion may indicate active bleeding.
 - Most commonly seen in temporal parietal region
- Plain films may show skull fractures:
 - CT with bone windows is more often used.
- Spine series
- Further workup of trauma as indicated

Pediatric Considerations

US may be used for diagnosis in infants with open fontanels.

DIFFERENTIAL DIAGNOSIS

- History of recent head trauma lends itself to the diagnosis:

- Trauma may be minor in infants and toddlers.
- Consider other diagnosis:
 - SDH
 - Cerebral concussion/contusion
 - Intracerebral bleed
 - Diffuse axonal injury
 - Subdural hygroma
 - Shaken baby syndrome
 - Toxic, metabolic, or infectious causes



TREATMENT

PRE HOSPITAL

- Head-injured patients have 25% improved mortality when triaged to regional trauma centers.
- Spinal immobilization is essential.
- Ensure adequate oxygenation throughout transport:
 - Intubation and airway protection may be necessary.

INITIAL STABILIZATION/THERAPY

- Prevent hypoxia and hypotension:
 - Rapid-sequence intubation for signs of deterioration or increased intracranial pressure (ICP)
 - Controlled ventilation to PCO_2 of 35–40 mm Hg
 - Avoid hyperventilation unless signs of brain herniation are present.
 - Avoid induction agents, which may increase ICP (e.g., ketamine).
- Elevate head of bed 20° – 30° after adequate fluid resuscitation.
- Perform rapid neurologic assessment:
 - Glasgow coma scale (GCS) score:
 - 14–15; minor head injury
 - 9–13; moderate head injury
 - < 8; severe:
 - Reflexes; pupils, corneal, gag, brainstem reflexes
- Secondary survey will reveal coexisting injury in >50%.

ED TREATMENT/PROCEDURES

- Early surgical intervention (< 4 hr) in comatose patients with EDH improves meaningful survival:
 - Burr hole is placed at fracture site or side with ipsilateral pupillary dilation.
 - Rapid craniectomy is occasionally performed if bleeding is not controlled at site of burr hole.
- Nonsurgical intervention in asymptomatic patients is associated with high rate of

deterioration; > 30% require surgical intervention.

- Maintain euvoemia with isotonic fluids.
- Continuous end tidal CO₂ monitoring:
 - Arterial line placement for close monitoring of MAP, PO₂, PCO₂
 - Foley catheter to monitor input/output (I/O) status
- Control ICP:
 - Prevent pain, posturing, and increased respiratory effort:
 - Sedation with benzodiazepines
 - Neuromuscular blockade with vecuronium or rocuronium in intubated patients
 - Etomidate is a good induction agent.
 - Barbiturate coma should be initiated for refractory increased ICP in neurosurgical ICU.
 - Mannitol may be used once euvolemic:
 - Shown to increase MAP greater than coronary perfusion pressure (CPP) and cerebral blood flow (CBF), as well as decrease ICP
 - Keep osmolality between 295 and 310.
 - Use furosemide (Lasix) as adjunct only if no risk of hypovolemia.
- Treat HTN:
 - Labetalol or hydralazine
- Treat hyperglycemia if present:
 - Associated with increased lactic acidosis and mortality in patients with TBI
- Treat and prevent seizures:
 - Diazepam and Dilantin
- Not considered helpful:
 - Steroids
 - Antibiotic prophylaxis
 - Hyperventilation in the absence of herniation
 - Fluid restriction
 - Calcium channel blockers
- Factors associated with poor outcome:
 - Age > 40 yr
 - Increased admission base deficit
 - Large hematoma with rapid expansion
 - Increased midline shift
 - Lower admission GCS or unconsciousness at presentation
 - Postoperative ICP > 3
 - Prolonged anisocoria
 - Associated brain injuries or concomitant trauma injuries

Pediatric Considerations

Hemodynamically significant blood loss can result from scalp lacerations and subgaleal

hematomas: Direct pressure and control of bleeding is indicated.

MEDICATION

- Diazepam: 5–10 mg (peds: 0.1–0.2 mg/kg) IV
- Dilantin: Adult/peds: Load 18 mg/kg at 25–50 mg/min
- Etomidate: 0.3 mg/kg IV
- Fentanyl: 2–4 U μ g/kg IV
- Furosemide (Lasix): Adults/peds: 0.5 mg/kg IV
- Hydralazine: 10 mg/h IV (peds: 0.1–0.5 mg/kg IV) q3–4h PRN
- Labetalol: 15–30 mg/h IV (peds: 0.4–1 mg/kg/h IV continuous infusion; max. 3 mg/kg/h)
- Levetiracetam: 1,500 mg IV/PO q12h
- Lidocaine: As preinduction agent, 1.5 mg/kg IV
- Mannitol: Adults/peds: 0.25–1 g/kg IV q4h
- Midazolam: 1–2 mg (peds: 0.15 mg/kg IV \times 1) IV q10min PRN
- Pentobarbital: 1–5 mg IV q6h
- Prothrombin complex concentrate 50 U/ kg IV
- Rocuronium: 1 mg/kg IV
- Thiopental: As induction agent, 20 mg/kg IV

Pediatric Considerations

- Hypertonic saline has been shown to be beneficial in some pediatric studies (1.7–3%).
- NaCl 3%: 2–6 mL/kg IV. Infusion 0.1–1 mL/kg/h



FOLLOW-UP

DISPOSITION

Admission Criteria

- All patients with CT abnormality or altered LOC should be admitted to ICU setting with frequent neurologic assessment.
- Patients should have repeated CT exam in 12–24 hr or if clinical deterioration occurs.
- Patients at increased risk of deterioration include those with rapid bleeds, associated skull fracture, or lower GCS or neurologic deficits.

Discharge Criteria

Admission is necessary for all patients with EDH.

ADDITIONAL READING

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CODES

ICD9

- 852.40 Extradural hemorrhage following injury without mention of open intracranial wound, unspecified state of consciousness
- 852.41 Extradural hemorrhage following injury without mention of open intracranial wound, with no loss of consciousness
- 852.46 Extradural hemorrhage following injury without mention of open intracranial wound, with loss of consciousness of unspecified duration

ICD10

- S06.4X0A Epidural hemorrhage w/o loss of consciousness, init encntr
- S06.4X7A Epidur hemor w LOC w death d/t brain injury bf consc, init
- S06.4X9A Epidural hemorrhage w LOC of unsp duration, init

EPIGLOTTITIS, ADULT

Jonathan Fisher • Colby Redfield



BASICS

DESCRIPTION

- Rapidly progressive inflammation of the epiglottis and surrounding tissues leading to airway compromise
- May be more indolent in adults than pediatrics; rapid progression to total airway occlusion still seen in adults
- Although the incidence of pediatric epiglottitis has been decreasing, the incidence in adults is increasing
- Inflammation of supraglottic structures:
 - Epiglottis:
 - Edema is the primary airway concern
 - May be primary or secondary from adjacent structures
 - Vallecula
 - Arytenoids
- Incidence is 1–4:100,000 adults per year and rising
- More common in men: 3:1
- Adult mortality rate is 7% (<1% in children)
- Most common in 5th decade of life
- Immunocompromised patients may be particularly fulminant, with minimally associated symptoms and unusual pathogens, such as *Candida* and *Pseudomonas aeruginosa*
- Complications:
 - Total airway obstruction
 - Retropharyngeal abscess
 - Acute respiratory distress syndrome
 - Pneumonia
 - Empyema

ETIOLOGY

- Infectious causes:
 - *Haemophilus influenzae B*, also type A and nontypeable strains
 - *Haemophilus parainfluenzae*
 - *Streptococcus pneumoniae*
 - *Staphylococcus aureus*
 - Group A Streptococcus
 - *Neisseria meningitis*
 - Herpes simplex

- Cytomegalovirus
- *P. aeruginosa*
- Numerous other uncommon agents
- Physical agents:
 - Chemical and thermal burns
 - Toxic or illicit drug inhalation
- Trauma, instrumentation

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- General:
 - Fever
- Upper respiratory tract infection symptoms
- Prodrome absent in significant number of cases
- Head, eyes, ears, nose, throat:
 - Dysphagia
 - Muffled voice
 - Voice change:
 - “Hot potato” voice
 - Hoarseness
 - Foreign body sensation in throat
 - Drooling
 - Associated tonsillar, peritonsillar, uvular findings
- Respiratory:
 - Subjective sense of obstructed airway
 - Short of breath

Physical-Exam

- General:
 - Fever
 - Toxic appearing
 - Sitting up in “tripod” stance
- Head, eyes, ears, nose, throat:
 - “Cherry red” epiglottitis is classic, may be pale and edematous in up to 50%
 - Hyoid/thyroid cartilage tender to gentle palpation
 - Tracheal rock: Pain with movement of the larynx from side to side
 - Lymphadenopathy
- Respiratory:
 - Stridor

- Sudden loss of airway
- Respiratory distress with accessory muscle use

ALERT

Patients with respiratory distress are at high risk for rapid progression to complete airway obstruction. Surgical airway management may be required.

ESSENTIAL WORKUP

If significant respiratory distress:

- Avoid invasive diagnostic procedures
- Manage empirically with antibiotics and control of airway prior to further diagnostic evaluation

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC with differential
- Blood cultures
- Cultures of pharynx:
 - Only if no signs of respiratory distress

Imaging

- In patients with moderate to severe respiratory distress, the airway should be managed prior to imaging
- Portable lateral soft tissue x-ray:
 - Epiglottic “thumb” sign:
 - Thickening of the epiglottis
 - “Vallecula” sign:
 - The vallecula is normally well-delineated, deep, and roughly parallel to the pharyngotracheal air column
 - Absence of a deep and well-defined vallecula, approaching the level of the hyoid bone
 - Swelling of the arytenoids and aryepiglottic folds
 - Prevertebral soft tissue swelling
 - Significant false-negative with imaging
 - If suspected with negative film results, rule out with indirect visualization
- CT:
 - Indicated when a laryngoscopic evaluation cannot be performed or if coexistent soft tissue complications are suspected

Diagnostic Procedures/Surgery

- Avoid prior to airway management if any signs of respiratory distress are present, including stridor

- Nasopharyngoscopy (mini-fiberoptic scope)
- Indirect laryngoscopy

DIFFERENTIAL DIAGNOSIS

- Croup
- Airway foreign body
- Anaphylaxis
- Paradoxical vocal cord dysfunction
- Angioedema
- Laryngitis
- Pharyngitis
- Oropharyngeal abscess (peritonsillar or retropharyngeal)
- Bacterial tracheitis
- Congenital anomaly
- Meningitis



TREATMENT

PRE HOSPITAL

- Transport patients in position of comfort
- Supplemental oxygen as tolerated; avoid increasing anxiety
- Intubation indicated only if patient is in severe respiratory distress:
 - Likely difficult airway and significant chance of exacerbating compromise with laryngoscopy attempts
- Inhaled agents, racemic epinephrine, and β -agonists have no demonstrated value.

INITIAL STABILIZATION/THERAPY

- ABCs
- Be prepared with all equipment on hand for definitive airway management, including a surgical airway, from presentation until diagnosis is ruled out or transport to intensive care setting
- Exam of the airway can trigger airway obstruction
- Orotracheal intubation in patients with signs of obstruction or significant respiratory distress:
 - Respiratory distress/airway failure may develop precipitously
 - Consider ear-nose-throat/surgical consult if patient's condition permits for possible difficult/surgical airway
- Needle jet insufflation may be a life-saving temporizing measure if a surgical airway is not immediately attainable with failed intubation

ED TREATMENT/PROCEDURES

- Humidified oxygen support
- IV access, hydration as indicated

- Begin antibiotic coverage empirically
- Corticosteroids are controversial

MEDICATION

First Line

- Cefotaxime: 2 g IV q8h
- Ceftriaxone: 2 g IV q24h

Second Line

- Ampicillin/sulbactam: 3 g IV initially, then 200–300 mg/kg/d in 4 div. doses + vancomycin 1 g IV q12h
- Trimethoprim–sulfamethoxazole: 320 mg IV initially, then 4–5 mg/kg IV q12h
- Consider adding increased coverage against *S. aureus*:
 - Nafcillin: 150–200 mg/kg IV per day in 4 div. doses
 - Clindamycin: 600–900 mg IV q8h
- Rifampin prophylaxis:
 - Adults: 600 mg/d PO for 4 days
 - > 1 mo of age: 20 mg/kg/d PO for 4 days
 - < 1 mo of age: 10 mg/kg/d PO for 4 days



FOLLOW-UP

DISPOSITION

Admission Criteria

Any patient with a suspected or confirmed diagnosis of epiglottitis should be admitted to an ICU setting for IV antibiotics and airway management

Discharge Criteria

- Patients should not be discharged unless the diagnosis has been ruled out by visualization of the supraglottic structures by a physician familiar with physical appearance of the disease
- Close contacts should receive prophylactic treatment with rifampin

Issues for Referral

ENT consultation should be obtained

PEARLS AND PITFALLS

- Failure to manage the airway in a timely manner
- Avoid any unnecessary intervention until airway is secured

- Mortality is 7% in adults with epiglottitis

ADDITIONAL READING

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CODES

ICD9

- 464.3 Acute epiglottitis
- 464.30 Acute epiglottitis without mention of obstruction
- 464.31 Acute epiglottitis with obstruction

ICD10

- J05.1 Acute epiglottitis
- J05.10 Acute epiglottitis without obstruction
- J05.11 Acute epiglottitis with obstruction

EPIGLOTTITIS, PEDIATRIC

Beverly Bauman



BASICS

DESCRIPTION

- Inflammation of the epiglottis and surrounding supraglottic region, which is potentially life threatening due to progressive airway obstruction
- Children are at greater risk of upper airway obstruction owing to:
 - Decreased cross-sectional area of the upper airway (resistance is proportional to the inverse of the radius to the 4th power)
 - Loose attachment of mucosal surface and increased vascularity of mucosa allows for edema
 - Dynamic collapse of the airway
- A precipitous decline in the incidence of childhood epiglottitis since the introduction of the *Haemophilus influenzae* vaccination has occurred, although vaccine failure may result in rare cases among children who have been immunized
- In the post-Hib vaccine era, the mean age for this disease has increased, and it is now more commonly seen in adolescents and adults than in toddlers or young school-aged children.
- May occur throughout the year

ALERT

All patients with suspected epiglottitis require intensive monitoring and intervention. Rapid progression of airway obstruction may occur.

ETIOLOGY

- Infection:
 - *H. influenzae* type B
 - *Streptococcus pneumoniae*
 - Group A β -hemolytic
 - Streptococcus
 - *Staphylococcus aureus*
 - Viruses
 - Less common infections include Klebsiella, Pseudomonas, Candida
- Caustic
- Thermal
- Traumatic
- Post-transplant lymphoproliferative disorder
- Hereditary angioedema

SIGNS AND SYMPTOMS

History

- Usually fulminant presentation without prodromal illness
- General:
 - Irritability, throat pain (often described as patient's worse sore throat), fever, noisy breathing
 - Progressive toxicity and respiratory distress
 - Adults have often been previously seen by a physician 1 or more times before diagnosis is made. Adults may present with the “worst sore throat of my life.”

Physical-Exam

- General:
 - Toxic appearing
 - High fever is typical.
 - Rapid onset and progression
- Throat:
 - Drooling
 - Dysphagia
 - Muffled “hot potato” voice
 - Older patients often have very painful throat.
- Respiratory:
 - Rapidly progressive respiratory distress (dyspnea in only 1/3 of adults)
 - Children usually prefer to sit upright, leaning forward with open mouth (“tripod sniffing position”) to maximize air entry.
 - Subtle stridor that may progress to severe stridor (stridor in only 10% of adults)
- Complications:
 - Airway obstruction is the most severe complication.
 - Epiglottic abscess
 - Associated pneumonia and atelectasis

ESSENTIAL WORKUP

- Epiglottitis is a clinical diagnosis.
- Indirect laryngoscopy or any attempts to directly visualize the epiglottis are not indicated in children with suspected epiglottitis unless performed in a controlled environment. (In adolescents or adults, use of fiberoptic nasopharyngoscope may be indicated for patients without impending airway obstruction.)
- If infection is suspected, obtain cultures of the epiglottis during laryngoscopy after

airway is secure.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Avoid lab tests until airway is controlled.
- Throat cultures after control of airway
- Blood cultures after airway is secure:
 - Often positive if *H. influenzae* is the pathogen

Imaging

- Radiographs of the soft tissue lateral neck:
 - Usually not necessary to make the diagnosis
 - Creates additional risk by delaying stabilization of the airway, promoting airway obstruction by agitating the patient, and often removing the child from the ED to an uncontrolled environment. Children should never go unaccompanied to radiology. Personnel and equipment to control airway must always be available.
- Variable findings:
 - Normal
 - Swelling of the epiglottis (“thumbprint sign”) and often supraglottic region
 - Ballooned hypopharynx
 - Obliteration of vallecula
 - EW/C3W (epiglottic width to 3rd cervical vertebral body width) ratio of >0.5

Diagnostic Procedures/Surgery

Laryngoscopy:

- In a controlled environment whenever possible
- Cultures of the epiglottis during laryngoscopy after the airway is secured may help identify pathogens and direct treatment.
- Epiglottis will appear swollen, inflamed, reddened.

DIFFERENTIAL DIAGNOSIS

- Other infectious processes:
 - Bacterial tracheitis
 - Retropharyngeal abscess
 - Peritonsillar abscess
 - Croup (laryngotracheobronchitis)—primarily in younger children, but there is a significant overlap in the ages of presentation.
 - Pertussis
 - Mononucleosis
 - Ludwig angina

– Diphtheria

- Anaphylactic reaction with angioedema
- Hereditary angioedema
- Foreign body in upper airway
- Laryngeal trauma
- Laryngospasm
- Inhalation or aspiration of toxins (e.g., hydrocarbons)
- Airway burns (have been related to crack cocaine)
- Hyperventilation
- CNS disorders



TREATMENT

PRE HOSPITAL

Degree and mode of intervention must reflect degree of obstruction, time and means of transport, capability of care providers, etc. Consult and notify receiving hospital.

INITIAL STABILIZATION/THERAPY

- Airway management if patient is in extremis
- Bag-valve-mask ventilation with 100% O₂ with cricoid pressure often provides adequate ventilation and time to prepare for intubation and move to a controlled setting such as the operating room.
- Oral intubation:
 - Use an endotracheal tube (ETT) size that is 1 or 2 sizes smaller than indicated by age or length.
 - Direct compression of the anterior neck in the glottic region may help visualize air bubbles at the opening of the swollen glottis.
 - Instruments used for difficult airways may be adjunctive devices.
- If oral intubation fails:
 - Emergent cricothyrotomy or needle cricothyrotomy if age older than 10–12 yr
 - Needle cricothyrotomy if age younger than 10–12 yr

ED TREATMENT/PROCEDURES

- 100% O₂ as tolerated by patient
- Allow child to remain in position of comfort and do not force child to lie down, which may worsen airway obstruction.
- Although not proven, racemic epinephrine or L-epinephrine by nebulizer may temporize symptoms while plans for a definitive airway are rapidly arranged. It must be done with caution to avoid agitating the child.
- Avoid procedures that agitate the child such as IV access and blood draws.
- Empiric invasive airway management may be indicated:

- Patients with rapidly progressive respiratory difficulty, tachypnea, worsening throat pain, tachycardia, or hypoxemia
- Patients at high risk of acute obstruction (e.g., children with immunodeficiency disorders)
- Intubate in operating room or controlled environment by most skilled person.
- Use inhalational anesthesia before intubation.
- Have appropriate ETTs of various diameters of available to accommodate the inflamed supraglottic region.
- Surgical backup is required in case intubation is not possible; then emergency tracheotomy or cricothyrotomy can be performed.
- Equipment for intubation and for a surgical airway or needle cricothyrotomy must be available at the bedside.
- Administer IV antibiotics: 2nd- or 3rd-generation cephalosporins are active against β -lactamase-producing *H. influenzae*.
- Steroids are controversial but frequently administered, particularly in patients with chemical or thermal epiglottitis.

MEDICATION

First Line

- Ampicillin/sulbactam: 200–300 mg/kg/24h q6h IV
- Cefotaxime: 150 mg/kg/24h q6–8h IV
- Ceftriaxone: 100 mg/kg/24h q12h IV

Second Line

- Ampicillin: 100–200 mg/kg/24h q6h IV given with chloramphenicol
- Chloramphenicol: 75–100 mg/kg/24h q6h IV
- Meropenem: 120 mg/kg/24h q8h max. dose 6 g/24h
- Decadron: 0.6 mg/kg/d (max. 10 mg) IV.
 - Steroid use is controversial
- Epinephrine, racemic: 0.05 mL/kg (max. 0.5 mL) q30min in 2.5 mL normal saline (NS) via nebulizer
- L-epinephrine, 1:1,000: 0.5 mL/kg (max. 5 mL) q30min via nebulizer
- Rifampin for household contact prophylaxis: 20 mg/kg (max. 600 mg) daily for 4 days
- If hereditary angioedema is the suspected cause of epiglottitis, C1 esterase inhibitor concentrate (alternatively, if C1-INH is not available, consider fresh frozen plasma). Expert consultation recommended



FOLLOW-UP

DISPOSITION

Admission Criteria

Patients with suspected or proven epiglottitis should be admitted to ICU after stabilization of airway and administration of antibiotics and fluids.

Discharge Criteria

- Rifampin prophylaxis may be indicated for close contacts of *H. influenzae* epiglottitis. If the household has children younger than 12 mo of age or children who are unimmunized, incompletely immunized, or immunosuppressed, prophylaxis is indicated for nonpregnant household contacts. Child care center contacts should receive prophylaxis when 2 or more cases of Hib invasive disease have occurred within 60 days.
- All cases of invasive *H. influenzae* disease should be reported to the local or state public health department.

Issues for Referral

Critical care or pulmonary consult on all patients

PEARLS AND PITFALLS

True airway emergency. Patient must be monitored and accompanied at all times by someone with airway stabilization capabilities.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Bacterial Tracheitis
- Croup

- Epiglottitis, Adult



CODES

ICD9

- 464.3 Acute epiglottitis
- 464.30 Acute epiglottitis without mention of obstruction
- 464.31 Acute epiglottitis with obstruction

ICD10

- J05.1 Acute epiglottitis
- J05.10 Acute epiglottitis without obstruction
- J05.11 Acute epiglottitis with obstruction

EPIPHYSEAL INJURIES

Neha P. Raukar • Daniel L. Savitt

BASICS

DESCRIPTION

- Fractures through the physis accounts for 21–30% of pediatric long bone fractures with 30% of these leading to a growth disturbance:
 - Most frequently seen in the distal radius and ulna, distal tibia and fibula, and the phalanges
 - More common than ligamentous injury in children:
 - Tensile strength of pediatric bone is less than adjacent ligaments.
 - Physis is the weakest part of pediatric bone.
 - Similar injury in an adult usually causes a sprain.
- Most common during peak growth:
 - Females: Age 9–12
 - Males: Age 12–15
 - Much less common in infancy and early childhood because epiphysis is not ossified and acts as a shock absorber
- Twice as common in males because female bones mature earlier
- Salter–Harris (SH) classification (introduced in 1963, simplest and most commonly used classification system):
 - Type I:
 - Fracture line confined to physis
 - Complete epiphyseal separation from metaphysis through the physis
 - If periosteum remains intact, epiphysis will not displace.
 - Clinical diagnosis made with focal tenderness over the physis
 - Most common example is SCFE.
 - Growth disturbance is rare.
 - Type II:
 - Accounts for ~ 80% of physeal fracture patterns
 - Fracture propagates along physis, and fragment from metaphysis accompanies the displaced epiphysis (Thurston–Holland sign)
 - Periosteum torn opposite metaphyseal fragment
 - Growth is rarely disturbed.
 - Type III:
 - Rare
 - Fracture through a portion of physis extending through the epiphysis
 - Distal tibia most commonly affected
 - If displaced, requires reduction to maintain anatomic alignment
 - Growth disturbance may occur despite anatomic reduction because

blood supply can be affected.

- Type IV:
 - Fracture originates at articular surface.
 - Extends through physis and into metaphysis
 - Distal humerus most commonly affected
 - Also has Thurston–Holland fragment
 - Anatomic reduction essential and displaced fractures require ORIF
 - Growth arrest is common even with optimal treatment.
- Type V:
 - Results from severe crush injury to physis
 - No immediately visible radiographic alteration so almost impossible to diagnose initially
 - Compression forces lead to physeal injuries and inevitable growth disturbances.
 - Often found in retrospect
- Ogden modified the SH system to include injuries to the surrounding anatomy—periosteum, perichondrium, and zone of Ranvier:
 - Ogden Type VI: Involves the peripheral perichondrium including the zone of Ranvier
 - Ogden Type VII: Involves epiphysis only
- Peterson classification system, 1994:
 - Result of a 10 yr retrospective study
 - Showed that 16% of physeal injuries could not be classified by the SH system
 - Includes 2 different fracture patterns:
 - Peterson Type I—transverse fracture through the metaphysis with 1 or more longitudinal extensions into the physis (this is similar to SH II except most of the energy is transmitted through the metaphysis, leading to a fracture, and not the physis; there is very little growth plate disturbance, this was actually the most common fracture pattern found)
 - Peterson Type VI—a part of the epiphysis, physis, and metaphysis are missing due to an open injury, classically by a lawnmower. Severe growth disturbance.
 - Peterson Types II–V are similar to the SH II–V.

ETIOLOGY

- Competitive and recreational injuries
- Traumatic injuries
- Child abuse
- Extreme cold
- Radiation injury
- Genetic, neurologic, and metabolic disease

SIGNS AND SYMPTOMS

History

- Most commonly occurs after a fall
- Extreme cold and radiation can injure the physeal plate.

Physical-Exam

- Focal tenderness
- Swelling
- Limited mobility
- If lower extremity involved, patient may be nonweight bearing
- Joint laxity:
 - _ Can be due to physeal injury and not ligamentous injury

ESSENTIAL WORKUP

- Radiographs to classify the extent of the injury
- Assess pulses and capillary filling distal to injury.
- Evaluate distal motor and sensory function.
- Verify integrity of skin overlying injury.
- Address and manage coexisting injuries.

DIAGNOSIS TESTS & INTERPRETATION

Imaging

- Plain radiography of injured extremity:
 - _ Type I fractures:
 - Usually normal
 - May appreciate a slightly separated physis or an associated joint effusion
 - Consider comparison views of contralateral joint to detect small defects.
 - Callus may be present on follow-up films.
 - _ Types II–IV: Films diagnostic of fracture
 - _ Type V:
 - Initial film often normal
 - Subsequent radiographs may reveal premature bone arrest.
- Ultrasound can be helpful in infants whose cartilage has not ossified.
- CT scan: Helpful in assessing orientation of comminuted fragments
- MRI:
 - _ Most accurate in the acute phase of injury

- Can identify physal arrest lines
- Recommended if diagnosis remains equivocal and identification of a specific fracture would alter management

DIFFERENTIAL DIAGNOSIS

- Strain
- Sprain
- Contusion



TREATMENT

PRE HOSPITAL

- Immobilize limb in position found if no compromise in vascular status
- Apply ice or cold packs to injury.
- Assess injured extremity for neurologic and vascular function.
- Consider concomitant injuries.

INITIAL STABILIZATION/THERAPY

- Analgesia
- Apply sterile dressings to open wounds.
- Control bleeding of open wounds.

ED TREATMENT/PROCEDURES

- Reduction/alignment required in displaced fractures:
 - Need to achieve anatomic alignment
- Vascular or neurologic compromise distal to injury requires immediate intervention.
- Immobilization of all suspected or radiographically confirmed physal injuries:
 - Splint must immobilize joint proximal and distal to injury in anatomic alignment and neutral position.
 - Limit activity of the injured limb.
- Open fractures:
 - IV antibiotics for *Staphylococcus aureus*, group A streptococcus, and potential anaerobes depending on mechanism and after cultures are obtained
 - Copious irrigation with saline
 - Sterile dressing
 - Orthopedic consultation
- Consultation:
 - Open fractures
 - Type II with displacement and Types III and higher

MEDICATION

First Line

Pain management:

- Fentanyl: 2–3 µg/kg IV; transmucosal lollipops 5–15 µg/kg, max. 400 mg; contraindicated if <10 kg
- Morphine: 0.1 mg/kg IV/IM

If open:

- Cefazolin: 25–50 mg/kg/d IV/IM q6–8h
- Penicillin G: 100,000–300,000 U/kg/24 h IM, or IV in 4–6 div. doses—has better strep and corynebacterium coverage—for farm injuries
- Gentamicin: 5–7.5 mg/kg/d—for obviously contaminated injuries



FOLLOW-UP

DISPOSITION

Admission Criteria

- Open fractures
- Open surgical reduction required
- Consider with Type III and IV fractures

Discharge Criteria

- Low-grade fractures and fractures with higher grade if follow-up is definite
- Splint
- Analgesics
- Ice packs
- Elevation of affected limb
- Orthopedic follow-up within 1 wk

Issues for Referral

All injuries involving the physis should follow-up with a musculoskeletal specialist.

FOLLOW-UP RECOMMENDATIONS

Usually necessary, especially with higher-grade injuries, to monitor limb length:

- Involves periodic physical exam and radiographic evaluation

PEARLS AND PITFALLS

- Long-term complications:
 - Limb length discrepancy if entire growth plate affected
 - Angulation if only a part of the physis is affected
- In patients with suspected SH fracture and negative radiograph, immobilization with follow-up in a few days is appropriate.

ADDITIONAL READING

- Rathjen KE, Birch JG. Physeal injuries and growth disturbances. In: Beaty JH, Kasser JR, eds. *Rockwood & Wilkins' Fractures in Children*. 6th ed. Philadelphia, PA: Lippincott Williams and Wilkins; 2006:11.
- Rodríguez-Merchán EC. Pediatric skeletal trauma: A review and historical perspective. *Clin Orthop Relat Res*. 2005;432:8–13.
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CODES

ICD9

- 812.09 Other closed fracture of upper end of humerus
- 813.42 Other closed fractures of distal end of radius (alone)
- 813.43 Closed fracture of distal end of ulna (alone)

ICD10

- S49.009A Unsp physeal fx upper end of humerus, unsp arm, init
- S59.009A Unsp physeal fracture of lower end of ulna, unsp arm, init
- S59.209A Unsp physeal fracture of lower end of radius, unsp arm, init

EPISTAXIS

Richard E. Wolfe • Christopher M. McCarthy II

BASICS

DESCRIPTION

- Nosebleeds are a common emergency presentation that is usually minor and self-limited but rarely may be life threatening:
 - _ Lifetime incidence of ~60%:
 - The incidence decreases with age, with most cases seen in children <10 yr.
 - Male > female
 - Severe bleeds requiring surgical intervention are more common in patients >50 yr.
 - Occurs more frequently with low humidity during the winter, in northern climates, and at high altitude
- The nasal cavity is supplied with blood vessels originating from both the internal and external carotid arteries.
- Location of the hemorrhage determines therapy:
 - _ Anterior epistaxis (90% of cases): Bleeding can be visualized in anterior nose.
 - Most commonly bleeding is located at Kiesselbach plexus, an anastomotic network of vessels on the anteroinferior nasal septum.
 - Rarely, bleeding is found on the posterior floor of the nasal cavity or the nasal septum.
- Posterior epistaxis (10% of cases): Bleeding source not within range of direct visualization.
 - _ Posterolateral branch of sphenopalatine artery

ETIOLOGY

- Idiopathic:
 - _ Dry nasal mucosa (low humidity)
- Nasal foreign body:
 - _ Children, mentally retarded patients, psychiatric patients
- Infection:
 - _ Rhinitis
 - _ Sinusitis
 - _ Nasal diphtheria
 - _ Nasal mucormycosis
- Allergic rhinitis
- Trauma:

- Nose picking
- Postoperative
- Facial trauma
- Barotrauma
- Environmental irritants:
 - Ammonia
 - Gasoline
 - Sulfuric acid
 - Glutaraldehyde
- Intranasal neoplasia: Most commonly Papilloma
- Coagulopathy:
 - Hemophilia A or B
 - Von Willebrand disease
 - Thrombocytopenia: Liver disease, leukemia, chemotherapy viral illness, or autoimmune disease.
 - Platelet dysfunction: Renal impairment or chronic alcohol consumption
- Drug induced:
 - Salicylates
 - NSAIDs
 - Heparin
 - Coumadin
- Hereditary hemorrhagic telangiectasia (Osler–Weber–Rendu disease)
- Atherosclerosis of nasal vasculature
- Endometriosis

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Laterality of the bleeding
- Intensity and amount of bleeding from the nares
- Recurrence of epistaxis and history of prior episodes
- Nasal obstruction and the duration of this symptom
- Complaints of vomiting or coughing blood
- Known tumors or coagulopathy
- Unusual bleeding or easy bruising suggests an underlying coagulopathy.
- Presence of systemic disease exacerbated by blood loss (coronary artery disease, chronic obstructive pulmonary disease)

Physical-Exam

- Evaluate vitals for hemorrhagic shock

- Careful exam for signs of coagulopathy:
 - Bruises
 - Petechiae and purpura
- Nasopharyngeal inspection:
 - Anesthetize nasopharynx prior to exam with cotton swab soaked in anesthetic and vasoactive agent.
- Attempt to identify bleeding source with nasal speculum
- Blood in mouth or oropharynx

ESSENTIAL WORKUP

- Assess stability: Airway compromise, hypovolemia.
- Determine source (anterior vs. posterior).
- Consider underlying coagulopathy.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Consider for severe bleeding or suspected coagulopathy:
 - CBC, type and cross-match, PT/INR, PTT, BUN

Diagnostic Procedures/Surgery

Direct visualization of nasal mucosa with nasal speculum:

- Pretreat with topical vasoconstricting agent and anesthetic.
- Ensure adequate lighting (i.e., headlamp) and suction.

DIFFERENTIAL DIAGNOSIS

- Hematemesis
- Hemoptysis

Pediatric Considerations

- Posterior epistaxis is rare in children; consider further workup for bleeding diatheses.
- Consider nasal foreign bodies or neoplasm, such as juvenile angiofibroma or papilloma.
- 4 wk of topical antiseptic ointment decreases incidence of recurrent epistaxis.



TREATMENT

PRE HOSPITAL

- Stable patients: Patient should bend forward at the waist; pinch nares closed, and spit out blood rather than swallow it.
- Unstable patients:

- Intubation, if airway is compromised
- IV access
- Crystalloid resuscitation, if signs of hypovolemia

INITIAL STABILIZATION/THERAPY

- Secure the airway in patients who are unconscious, have major facial trauma, or are otherwise at risk of obstruction or aspiration.
- Treat hypotension with crystalloids and blood products, if necessary, and ensure adequate IV access.

ED TREATMENT/PROCEDURES

- Universal precautions against blood/fluid contamination
- Anterior source:
 - Have patient apply direct pressure by pinching nares closed for 15 min. This may control bleeding and assist with visualization.
 - If bleeding persists, use bayonet forceps to place cotton pledgets soaked in vasoconstricting and anesthetic agents into affected nares.
 - If view is obstructed by blood have patient blow nose, or clear blood from view with irrigation and suction.
 - Visualize source of bleeding and cauterize limited area with silver nitrate.
 - Consider Gelfoam or Surgicel packing over cauterized site.
- Anterior nasal packing:
 - Indicated when cautery has failed to control bleeding
 - Associated with significant discomfort and infectious risk of sinusitis and toxic shock
 - Anterior nasal balloon:
 - Check the integrity of the balloons before insertion.
 - Cover with water-based lubricant or viscous lidocaine
 - Insert the device and inflate it slowly to avoid discomfort.
 - Use saline for the inflation if the balloon is to remain in place > a few hours.
 - Preformed nasal tampons:
 - Adequate anesthesia of the nasal passage should be ensured before placing the tampon.
 - Lubricate the tip of the sponge tampon with antibiotic ointment.
 - Insert it at a 45° angle ~1–2 cm into the nasal cavity.
 - Rotate the long axis of the tampon into a horizontal plane and push it firmly back into the nasal cavity.
 - If the pack does not fully expand from the blood, then use saline to complete the expansion.
 - Secure the drawstring to the cheek.
 - Petroleum-jelly–impregnated gauze:
 - Add an antibiotic ointment to the gauze.

- Ensure that a free end remains outside the nose.
- Place the gauze as far back as possible, starting on the floor of the nose.
- Repeat while securing the placed gauze with the speculum until the nose is fully packed.
- After anterior packing, persistent new bleeding may be a sign of inadequate packing or posterior source.
- Always treat patients with nasal packing with antibiotics to prevent sinusitis and prevent or limit *Staphylococcus aureus* infections that can lead to toxic shock syndrome (TSS).
- Posterior source:
 - Early endoscopic visualization and cautery of bleeding source may prevent need for posterior packing and admission.
 - Posterior packing may be accomplished with commercially available devices such as Nasostat or Epistat.
 - If commercial packs are unavailable, a Foley catheter may be directed into posterior nasopharynx until the tip visible in mouth. The balloon is then inflated and the catheter retracted until the balloon is lodged in the posterior nasopharynx. The catheter is then held in place by umbilical clamp.
 - Posterior pack should not be left in > 3 days due to infectious risk. Patient should be admitted and on telemetry while pack in place due to risk of vagal response.
- Complications of posterior packing:
 - Pressure necrosis of posterior oropharynx (do not overfill balloon)
 - Nasal trauma
 - Vagal response with reflex bradycardia
 - Aspiration
 - Infection/TSS
 - Hypoxia

MEDICATION

- Vasoactive solutions:
 - 4% cocaine
 - 1:1 mixture of 2% tetracaine and epinephrine (1:1,000)
 - 1:1 mixture of oxymetazoline 0.05% (Afrin) and lidocaine solution 4%
 - Phenylephrine (Neo-Synephrine)
- Antibiotics: For use while packing in place.
 - Amoxicillin–clavulanate potassium: 250 mg PO q8h
 - Cephalexin: 250 mg PO q6h
 - Clindamycin: 150 mg PO q6h
 - Trimethoprim–sulfamethoxazole: 160/800 mg PO q12h



FOLLOW-UP

DISPOSITION

Admission Criteria

- Severe blood loss requiring transfusion
- Severe coagulopathy that places the patient at risk of further blood loss
- Posterior nasal packing: Otolaryngology consult and admission for telemetry, supplemental oxygen, possible sedation, and observation; possible further surgical intervention (e.g., arterial ligation or embolization)
- Patients with anterior packing who do not have reliable follow up within 48 hr.

Discharge Criteria

Stable patients:

- Use Afrin nasal spray for 2 days.
- Lubricate nares with an antibiotic ointment.
- Humidify air.
- Avoid nose picking.
- All patients with nasal packing in place should be prescribed an antistaphylococcal antibiotic (amoxicillin–clavulanate, cephalexin, trimethoprim–sulfamethoxazole) for the duration that the packing remains in place for prevention of both acute sinusitis and TSS.

Issues for Referral

- Refer all patients with packing to a specialist within 48 hr.
- Patients with nonvisualized source, suspicious-appearing lesions, recurrent same-side bleeding, or nasal obstruction should be referred to an ORL specialist for an exam to rule out a neoplastic etiology or a foreign body.

FOLLOW-UP RECOMMENDATIONS

- Return to ED for bleeding not controlled by pressure, fever, difficulty breathing, or vomiting.
- Avoid any nose blowing for 12 hr after the bleeding stops.
- Avoid nose picking or putting anything into the nose.
- If the bleeding starts again, sit up and lean forward, pinch the soft part of the nose tightly for 10 min without letting go.
- Avoid lifting heavy objects or doing too much work right away.
- If there is no packing in the nose, put a small amount of petroleum jelly or antibiotic ointment inside the nostril 2 times a day for 4–5 days.
- Use a humidifier or vaporizer at home.

PEARLS AND PITFALLS

- Foreign bodies should be suspected in any unilateral nasal bleeding in small children, psychiatric patients, and patients with mental retardation.
- Avoid covering anterior nasal balloons with antibiotic ointment, as petroleum-based materials may cause a delayed rupture of the balloon.
- Avoid overinflating nasal balloons or placing a pack too tightly, as it can cause necrosis and eschars.
- Patients with packings should receive prophylactic antibiotics

ADDITIONAL READING

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CODES

ICD9

784.7 Epistaxis

ICD10

R04.0 Epistaxis

ERYSIPELAS

Irving Jacoby



BASICS

DESCRIPTION

- Superficial bacterial infection of the skin with prominent lymphatic involvement
- Leukocytosis is common
- Positive blood cultures in 3–5%

ETIOLOGY

- Group A β -hemolytic streptococcus is the causative organism (uncommonly, group C or G streptococci)
- Portals of entry:
 - Skin ulcers
 - Local trauma
 - Abrasions
 - Psoriatic or eczematous lesions
 - Fungal infections

Pediatric Considerations

- *Haemophilus influenzae* type b (HIB) causes facial cellulitis in children that may appear similar to erysipelas:
 - Should be considered in unimmunized children
 - Many will be bacteremic and require admission
 - Cefuroxime or other appropriate *H. influenzae* coverage is important
 - *H. influenzae* is much less common since widespread use of the HIB vaccine
- Group B streptococci can cause erysipelas in the newborn
- Can develop from infection of umbilical stump

Pregnancy Considerations

- Erythema of the breast in puerperal mastitis is often caused by Staphylococcus organisms, hence methicillin-resistant *S. aureus* (MRSA) should be covered
 - See Mastitis



DIAGNOSIS

SIGNS AND SYMPTOMS

- Most common sites of involvement are the face (5–20% of cases), lower legs (70–80% of cases), and ears
- Skin has an intense fiery red color, hence the name “Saint Anthony’s fire”

- Often bilateral on the face, but unilateral elsewhere
- Predilection for infants, children, and the elderly
- Systemic symptoms may include malaise, fever, chills, nausea, and vomiting
- Traumatic portal of entry on skin is not always apparent
- Rarely there may be an associated periorbital cellulitis or cavernous sinus involvement

History

- Facial erysipelas may follow a nasopharyngeal infection or trauma
- Predilection for areas of lymphatic obstruction:
 - _ Particularly in the upper extremity following radical mastectomy
 - _ Increased frequency after saphenous vein harvesting or stripping
 - _ May be a marker for previously undiagnosed lymphatic obstruction, or patients with congenital lymphedema (such as Milroy disease)
- 30% recurrence rate within 3 yr, owing to lymphatic obstruction caused by an episode of erysipelas

Physical-Exam

- Involved skin is:
 - _ Edematous
 - _ Indurated (peau d'orange)
 - _ Painful
 - _ Well-circumscribed plaque with sharp, clearly demarcated edges
- Classical butterfly rash on cheeks and across nose when affecting face
- Vesicles and bullae may be present in more serious infections

ESSENTIAL WORKUP

- The diagnosis is clinical:
 - _ Based on the characteristic skin findings and the clinical setting
- Needle-aspirate wound cultures are seldom positive and not indicated

DIAGNOSIS TESTS & NTERPRETATION

Lab

- Swabs of the skin are not indicated for culture, as they will show only skin organisms
- CBC with differential, and blood cultures should be performed in diabetics and other high-risk populations, or in patients with hypotension and those who require admission:
 - _ Blood cultures more likely to be positive in patients with lymphedema
- Check glucose in diabetics as infection may disrupt control
- Urinalysis: To check for proteinuria, hematuria, and red cell casts
 - _ Would suggest diagnosis of post-streptococcal glomerulonephritis (PSGN)

- If it occurs, usually around 2 wk after onset of skin infection
- Antistreptolysin O (ASL-O), anti-DNase B and streptolysin antibody serial titer changes are useful in diagnosing post-streptococcal immunologic entities such as rheumatic fever or glomerulonephritis,
 - Do not add anything to the diagnosis and management of uncomplicated erysipelas
 - Should not be routinely ordered unless there are already manifestations of such complications

Imaging

- There is no standard imaging for classical erysipelas
 - If deeper infection such as myositis is suspected, plain films of an extremity or CT scan may be performed to assess for the presence of gas
- Ultrasound may be useful to evaluate for an abscess if this is suspected, or in the leg to r/o deep vein thrombophlebitis DVT

DIFFERENTIAL DIAGNOSIS

- Abscess
- Acute bacterial sinusitis
- Allergic inflammation
- Cellulitis
- Contact dermatitis
- DVT
- Diffuse inflammatory carcinoma of the breast
- Familial mediterranean fever
- Herpes zoster, second division of cranial nerve V
- Impetigo
- Inflammatory dermatophytosis
- Mastitis
- Necrotizing fasciitis
- Periorbital cellulitis
- Systemic lupus erythematosus (SLE) with butterfly rash
- Streptococcal or staphylococcal TSS (sunburn-like rash)
- Venous stasis dermatitis
- Viral exanthem



TREATMENT

PRE HOSPITAL

Wearing gloves, followed by hand washing when managing patients, to decrease risk of transmission of streptococcal carriage

INITIAL STABILIZATION/THERAPY

Patients may be toxic and in need of intravenous fluid resuscitation or pressure support

ED TREATMENT/PROCEDURES

- Appropriate antibiotic therapy; treatment should be for 10 days:
 - Patients with extensive involvement should be admitted for parenteral antibiotic treatment
 - May switch to oral antibiotics when patient is stable and showing signs of response
- Mild cases: Patients can be discharged on oral therapy if nontoxic appearing, good compliance, and close follow-up can be ensured
- Penicillin is the drug of choice when symptoms are consistent with erysipelas
- If there is difficulty in distinguishing from cellulitis, staphylococcal coverage should be added:
 - Use penicillinase-resistant penicillin or 1st-generation cephalosporin
 - If in community with high incidence of MRSA, use vancomycin, or other anti-MRSA coverage
 - Reports of vancomycin-resistant Staphylococci are occurring
- Acetaminophen for fever
- Isolation while in hospital
 - Contagious

MEDICATION

OUTPATIENT

- Penicillin V: 500 mg PO q6h (peds: 25–50 mg/kg/d div. q6–8h) for 10 days.
- Amoxicillin: 500 mg PO q8h (peds: 50 mg/kg/d div. TID) for 10 days.
- Clindamycin: 300 mg PO QID (peds: 8–25 mg/kg/d suspension PO div. TID or QID) for 10 days.
- Dicloxacillin: 500 mg PO q6h (peds: 30–50 mg/kg/d PO div. q6h) for 10 days
- Erythromycin: 250–500 mg PO q6h (peds: 40 mg/kg/d PO in div. doses q6h) for 10 days
- Cephalexin: 500 mg PO q6h (peds: 40 mg/kg/d PO div. q8h) for 10 days
- Cefuroxime: 250–500 mg PO BID (peds: 30 mg/kg/d PO div. q12h) for 10 days.

INPATIENT

- Penicillin G: 2 million U q4h IV (peds: 25,000 U/kg IV q6h).
- Penicillin G, procaine: 600,000 U q12h IM
- Clindamycin: 600 mg q8h IV (peds: 20–40 mg/kg/d IV div. q8h)
- Vancomycin: 1 g IV q12h given over 1.5–2 hr to decrease risk of red man syndrome (peds: 10–15 mg/kg IV q6h)

First Line

- Oral or IV: Penicillin or 1st-generation cephalosporin
- Clindamycin for penicillin-allergic individuals

Second Line

Oral: Erythromycin



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients with extensive involvement, fever, toxic appearance, or in whom orbital or periorbital cellulitis is suspected
- Patients who live alone or are unable or unreliable to take oral medications will require admission for IV antibiotics
- Children more often require admission
 - Blood cultures
 - Intravenous antibiotics, including coverage for *H. influenzae*, should be initiated for patients who have not been immunized with HIB vaccine

Discharge Criteria

- Minimal facial involvement
- Nontoxic appearance
- Not immunosuppressed
- Able to tolerate and comply with oral therapy
- Adequate follow-up and supervision
- Diagnosis certain

Issues for Referral

- Refer to nephrologist for evaluation and treatment for PSGN if:
 - Hematuria, proteinuria, and red cell casts are noted on UA
 - Particularly in children between the ages of 5 and 15
- Infectious disease consultation for infection in immunocompromised patients who are at risk for unusual organisms

FOLLOW-UP RECOMMENDATIONS

- Use of pressure stocking on leg in the presence of lymphedema may reduce incidence of relapses
- Following erysipelas of legs, use of topical antifungal cream or ointment to treat underlying tinea pedis when present

PEARLS AND PITFALLS

- Failure to respond, or pain out of proportion to findings, might suggest deeper level of infection and require further workup to rule out necrotizing fasciitis, or

mixed aerobic/anaerobic necrotizing cellulitis

- Treatment of underlying lymphedema is associated with reduced incidence of relapses
- Presence of micropustules would suggest staphylococcal infection/cellulitis rather than erysipelas, and antibiotic coverage would need to be broader
- Presence of crepitus in skin should prompt search for alternate diagnosis
- Since infection is likely to have entered skin through traumatic skin break, remember to check for tetanus immunization status and update if necessary
- Consider prophylaxis for patients with frequent relapses

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Abscess
- Cellulitis
- MRSA, Community Acquired



CODES

ICD9

035 Erysipelas

ICD10

A46 Erysipelas

ERYTHEMA INFECTIOSUM

Benjamin S. Heavrin



BASICS

DESCRIPTION

- Characteristic viral exanthem also known as 5th disease:
 - 5th most common childhood rash historically described
 - Measles (1st), scarlet fever (2nd), rubella (3rd), Duke disease (4th), roseola (6th)
- Common symptoms: Viral prodrome followed by slapped-cheek rash and subsequent diffuse reticular rash +/- arthropathy
- Most common in school-aged children <14 yr
- Usually self-limited with lasting immunity
- Rare complications and chronic cases in patients with congenital anemias or immunosuppression
- Potential for severe complications to fetus if infection acquired during pregnancy
- Possible link to encephalopathy, epilepsy, meningitis, myocarditis, dilated cardiomyopathy, autoimmune hepatitis, HSP, ITP

ETIOLOGY

- Caused by human parvovirus B19, small SS-DNA virus:
 - Infects human erythroid progenitor cells, suppressing erythropoiesis
- Most common in late winter and spring
- Transmitted via respiratory droplets and blood products as well as vertical maternal-fetal transmission
- Incubation period 4–21 days
- Most contagious during the week PRIOR to rash onset
- Majority of adults have serologic evidence of prior infection



DIAGNOSIS

SIGNS AND SYMPTOMS

- “Slapped-cheek” appearance most common in young children
- Fever
- Malaise
- Delayed symptoms 4–14 days later:
 - Diffuse, pruritic, lacy rash (absent in most adults), most pronounced in extremities
 - Symmetric polyarthropathy, most common in middle-aged women:
 - Small joints involved in adolescents and adults

- Knees most commonly involved in children
- Secondary to immune-complex deposition
- However, most patients remain asymptomatic or only develop mild, nonspecific viral symptoms

History

- Mild constitutional symptoms (fever, headache, nasal congestion, nausea, sore throat)
- Contagious only until facial rash appears

Physical-Exam

- Stage 1:
 - “Slapped-cheek” rash of coalescent, warm, erythematous, edematous papules with circumoral pallor in young children
- Stage 2:
 - Nonspecific, diffuse, pruritic, maculopapular, reticular eruption
 - 4–21 days after facial rash, lasts up to 6 wk
 - More prominent on extremities
 - Usually spares palms and soles
- Stage 3:
 - Rash fades but recurs with exposure to sunlight, stress, exercise, and heat
- Usually complete resolution without scarring

ESSENTIAL WORKUP

Clinical diagnosis based on characteristic signs and symptoms.

DIAGNOSIS TESTS & INTERPRETATION

- Usually not necessary
- CBC and reticulocyte count if concern for aplastic crisis
- Confirm diagnosis if immunocompromised or pregnant:
 - Viral DNA PCR now available
 - IgM antibody confirms acute infection and persists for 2–3 mo
 - IgG presence confers lasting immunity
- In pregnancy, ultrasound to detect hydrops fetalis

DIFFERENTIAL DIAGNOSIS

- Allergic reaction
- Collagen vascular disease
- Coxsackie virus
- Drug eruptions
- Enterovirus
- Erysipelas
- Infectious mononucleosis

- Measles
- Nonspecific viral illness
- Rheumatoid arthritis
- Roseola
- Rubella
- Scarlet fever
- Sunburn



TREATMENT

Erythema infectiosum is usually self-limited and does not require treatment

PRE HOSPITAL

ABCs for severe cases and septic patients

INITIAL STABILIZATION/THERAPY

- ABCs, supplemental oxygen if indicated
- IVF with associated severe dehydration
- Severe anemia may also cause hypotension and hypoxia, transfuse PRBCs as indicated
- Pain control with acetaminophen, NSAIDs, or opiates as needed for severe arthropathy

ED TREATMENT/PROCEDURES

- No specific antiviral treatment or vaccine is available
- Send appropriate labs (CBC, reticulocytes, antibody testing) for severe cases
- Symptomatic treatment as needed:
 - _ IVF for severe dehydration
 - _ NSAIDs for arthropathy if no underlying renal insufficiency
 - _ Consider diphenhydramine for pruritus, caution parents about possible AMS
 - _ Antipyretics for fever
- PRBC transfusion for severe anemia
- ID consult: IVIG may have benefit for immunocompromised patients with chronic symptoms and red cell aplasia
- Hematology consult for severe cases
- Hospitalization and respiratory isolation for aplastic crisis

MEDICATION

- Acetaminophen: 500 mg (peds: 15 mg/kg/dose) PO q6h PRN fever for up to 5 days
 - _ Dose not to exceed 4 g/24h
- Diphenhydramine: 25 mg (peds: 1–2 mg/kg/dose) PO q6h PRN itching for up to 5 days
- Ibuprofen: 400 mg (peds: 10 mg/kg/dose) PO q8h PRN pain for up to 5 days
- IVIG only in consultation with ID specialist

DISPOSITION***Admission Criteria***

- Aplastic crisis or severe anemia
- Severely immunocompromised
- Hydrops fetalis
- Toxic appearance
- Severe arthritis

Discharge Criteria

- Nearly all patients
- Normal CBC, O₂ sat, and BP
- Patients are no longer contagious following appearance of facial rash and may return to day care, school, or work

Issues for Referral

- All patients without existing primary care physicians should be referred to a generalist for follow-up as needed
- Patients with hereditary anemias should be referred to hematology for follow-up in 1–2 days
- All immunocompromised patients require prompt subspecialty follow-up
- Pregnant patients with new infection should have immediate follow-up with OB/GYN for further monitoring and ultrasound

FOLLOW-UP RECOMMENDATIONS

- Pregnant women with new parvovirus B19 infection may need serial ultrasounds for 10–12 wk.
- Patients at risk for aplastic crisis should follow-up with the appropriate specialties 1–2 days after ED discharge for repeat CBC

PATIENT EDUCATION**Prevention:**

- No vaccine available
- Frequent handwashing helps prevent spread
- No current recommendations to keep children out of school, since most children are no longer contagious by the time the diagnosis is made.
- Pregnant women may choose to stay away from a workplace outbreak, but no current official recommendation exists

COMPLICATIONS

- Transient aplastic crisis in patients with anemias: Sick cell disease, hereditary spherocytosis, thalassemia, iron-deficiency, or other conditions with shortened red cell lifespan:
 - Usually full recovery within 2 wk
- Persistent infection with severe anemia if immunocompromised and unable to mount antibody response, especially with HIV
- Arthritis or hypersensitivity dermatitis in adults:
 - May have transient rheumatoid factor positivity, but no true association with rheumatoid arthritis and no joint destruction
- Association with papular, purpuric gloves, and socks syndrome in adults:
 - Symmetric, painful progressive rash and edema of hands and feet
 - Erythema progresses to petechiae, purpura, and occasionally bullae
 - This syndrome is also associated with many other viruses and drugs
- Extremely rare – hepatosplenomegaly, heart failure, CVA, thrombocytopenia, leukopenia

Pregnancy Considerations

- Risk of hydrops fetalis in pregnancy
- 60% of pregnant women are susceptible to new infection
- 30% risk of transplacental infection with new maternal infection
- Affects fetal liver (main site of erythropoiesis), leading to anemia, CHF, myocarditis, IUGR
- 2–6% risk of fetal loss, highest in 2nd trimester

PEARLS AND PITFALLS

- Parvovirus B19 is usually a self-limited, mild illness.
- Common symptoms include “slapped-cheeks” rash with subsequent diffuse lacy rash and arthropathy
- Patients are no longer contagious when the rash appears and aplastic crisis resolves
- Evaluate all patients with history of hereditary or iron-deficiency anemia for aplastic crisis
- Evaluate all patients with history of immunosuppression for chronic infection with persistent anemia
- Confirm diagnosis in all pregnant patients. If no proven immunity, monitor for fetal complications and refer for follow-up

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- Weir E. Parvovirus B19 infection: Fifth disease and more. *CMAJ*. 2005;172:743.



CODES

ICD9

057.0 Erythema infectiosum (fifth disease)

ICD10

B08.3 Erythema infectiosum [fifth disease]

ERYTHEMA MULTIFORME

Gregory W. Hendey



BASICS

DESCRIPTION

- A rash caused by a hypersensitivity reaction:
 - May occur in response to various medications, infections, or other illness
- Erythema multiforme (EM) minor:
 - Typical target lesions
 - Edematous papules
 - Usually distributed peripherally
 - Benign, self-limited rash generally not associated with acute, serious illness
- EM major
 - Also called bullous EM
 - Target lesions
 - Edematous papules
 - Also with peripheral distribution
 - Involves 1 or more mucous membranes
 - <10% total body surface area of epidermal detachment
- Differentiate from:
 - Stevens–Johnson syndrome (SJS):
 - Also <10% TBSA epidermal detachment
 - Often widespread blisters over trunk and face
 - Mucosal involvement
 - Toxic epidermal necrolysis (TEN)
 - >30% TBSA epidermal detachment
 - EM is now considered a different entity from SJS and TEN
- Most often affects children and young adults (>50% younger than 20 yr)
- Males are affected more often than females.

ETIOLOGY

- Hypersensitivity reaction, probably transient autoimmune defect
- *Herpes simplex virus* (HSV) is the most common precipitant (>70%).
- Other causes include:
 - Idiopathic
 - Medications
 - Penicillin
 - Sulfur based
 - Phenytoin
 - Barbiturates

- NSAIDs
- Vaccines
 - Diphtheria—tetanus
 - Hepatitis B
 - Smallpox
- Malignancy
- Infection
 - HIV
 - CMV
 - Hepatitis C
 - Mycoplasma infections

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Prodrôme: Infrequent systemic symptoms (mild fever/malaise), antecedent HSV in most cases (within 3 wk)
- Usually not associated with severe systemic illness

Physical-Exam

Characteristic rash:

- Lesions:
 - Symmetric dull red macules and papules
 - Evolve into round, well-demarcated target lesions with central clearing
 - *No epidermal necrosis* with EM minor
- *Multiforme* refers to the evolution of the rash through various stages at different times.
- Distribution:
 - Extremities
 - Dorsal hands and feet
 - Extensor surfaces
 - Elbows and knees.
- 1 of the few rashes that may involve palms and soles
- Spread: From extremities toward trunk
- Mucosal involvement: Minor blistering or erosions of 1 mucosal surface (lips/mouth)
- Duration: Usually 1–4 wk, but may become chronic or recurrent

ESSENTIAL WORKUP

Complete history and physical exam, with special attention to the skin, genitourinary

system, recent infectious symptoms, and recent medications

DIAGNOSIS TESTS & NTERPRETATION

Lab

No specific lab tests needed

Imaging

No specific imaging is helpful.

Diagnostic Procedures/Surgery

- Skin biopsy reveals mononuclear cell infiltrate around upper dermal blood vessels, without leukocytoclastic vasculitis and necrosis of epidermal keratinocytes.
- Biopsy is not necessary in most cases.

DIFFERENTIAL DIAGNOSIS

- Systemic lupus erythematosus
- Fixed drug eruption
- Pityriasis rosea
- Secondary syphilis
- Erythema migrans
- Urticaria
- SJS
- TEN
- Vasculitis
- Viral exanthem



TREATMENT

PRE HOSPITAL

Not contagious and does not require isolation or postexposure prophylaxis for exposed personnel

INITIAL STABILIZATION/THERAPY

Generally benign and self-limited, requiring no initial stabilization

ED TREATMENT/PROCEDURES

- Attempt to identify, treat, or remove underlying cause or precipitant.
- Symptomatic: Cool compresses, antipruritics

MEDICATION

- Antiviral agents:
 - _ Acute EM

- Treat within 48 hr of onset
- May not impact clinical course
 - Acyclovir: 400 mg PO TID
- Prevention of recurrent EM
 - Acyclovir 400 mg PO BID
 - Valacyclovir 500 mg PO BID
 - Famciclovir 250 mg PO BID
- Antipruritic agents:
 - Cetirizine (Zyrtec): 10 mg/d (peds: 2.5–5 mg) PO
 - Diphenhydramine: 25–50 mg (peds: 5 mg/kg/24h) PO q6–8h
 - Hydroxyzine: 25 mg PO q6–8h (peds: 2 mg/kg/24h div. q6–8h)
- Anesthetic for oral lesions
 - Magic mouthwash
- Oral corticosteroids:
 - Reserved for severe mucosal disease
 - Prednisone 40–60 mg PO QD tapered over 2–3 wk
- Medium-potency topical corticosteroids:
 - Triamcinolone 1% apply BID–QID
 - Do not use on face or eyelids
- Low-potency topical corticosteroids
 - For face or intertriginous regions
 - Hydrocortisone 1% apply BID–QID

First Line

- Topical corticosteroids (low to medium potency)
- Antipruritics

Second Line

- Antivirals
- Oral corticosteroids



FOLLOW-UP

DISPOSITION

Admission Criteria

- Admission is not needed unless required for another concurrent disorder.
- Unable to take PO fluids secondary to mucosal lesions

Discharge Criteria

EM is generally a benign disorder that does not require admission.

Issues for Referral

- Patients should be referred to a dermatologist if the diagnosis is uncertain or the rash is atypical or severe.
- Refer immediately to ophthalmologist if ocular involvement

FOLLOW-UP RECOMMENDATIONS

- Follow-up with primary care physician within 1 wk to assess:
 - Further evaluation of underlying conditions (infection, medications, malignancy, etc.)
 - Progression or resolution of rash
- Follow-up with a dermatologist within 1 wk if the diagnosis is uncertain.

PEARLS AND PITFALLS

- In patients with severe systemic illness, a more serious diagnosis should be considered, such as SJS or TEN.
- Most patients with EM have underlying HSV infection.
- Secondary syphilis may produce similar lesions on the palms and soles.
- Reassure patients that the rash of EM is benign and self-limited.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Herpes
- Stevens–Johnson Syndrome
- Toxic Epidermal Necrolysis



CODES

ICD9

- 695.10 Erythema multiforme, unspecified
- 695.13 Stevens-Johnson syndrome
- 695.15 Toxic epidermal necrolysis

ICD10

- L51.1 Stevens-Johnson syndrome
- L51.2 Toxic epidermal necrolysis [Lyell]
- L51.9 Erythema multiforme, unspecified

ERYTHEMA NODOSUM

Herbert G. Bivins

BASICS

DESCRIPTION

- Erythema nodosum (EN) is characterized by multiple symmetric, nonulcerative tender nodules on the extensor surface of the lower extremities, typically in young adults.
- Peak incidence in 3rd decade
- More common in women (4:1)
- Nodules are round with poorly demarcated edges and vary in size from 1 to 10 cm.
- Skin lesions are initially red, become progressively ecchymotic appearing as they resolve over 3–6 wk.
- Lesions are caused by inflammation of the septa between SC fat nodules (septal panniculitis).
- Spontaneous regression of lesions within 3–6 wk
- Major disease variants include:
 - EN migrans (usually mild unilateral disease with little or no systemic symptoms)
 - Chronic EN (lesions spread via extension, and associated systemic symptoms tend to be milder)

ETIOLOGY

- Immune-mediated response
- 30–50% of the time etiology is idiopathic
- Often a marker for underlying disease; specific etiologies include:
 - Drug reactions:
 - Oral contraceptives
 - Sulfonamides
 - Penicillins
 - Infections including:
 - *Streptococcal pharyngitis*
 - *Mycobacterium tuberculosis* (TB)
 - Atypical mycobacteria
 - Coccidioidomycosis
 - Hepatitis
 - Syphilis
 - Chlamydia
 - Rickettsia
 - Salmonella

- Campylobacter
- Yersinia
- Parasites
- Leprosy
- Systemic diseases:
 - Sarcoidosis
 - Inflammatory bowel disease
 - Behçet disease
 - Connective tissue disorders
- Malignancies such as lymphoma and leukemia
- Catscratch disease
- HIV infection
- Rarely can be caused by vaccines for hepatitis and TB (BCG)

Pediatric Considerations

Typically, EN begins 2–3 wk after onset of *S. pharyngitis*.

DIAGNOSIS

SIGNS AND SYMPTOMS

- Tender erythematous nodules symmetrically distributed on extensor surface of lower legs
- Lesions occasionally occur on fingers, hands, arms, calves, and thighs.
- In bedridden patients, dependent areas may be involved.
- Fever, malaise, leukocytosis, arthralgias, arthritis, and unilateral or bilateral hilar adenopathy with any form of the disease

History

- General symptoms may precede or accompany the rash:
 - Fever
 - General malaise
 - Polyarthralgias
- GI symptoms with EN may be a marker for:
 - Inflammatory bowel disease
 - Bacterial gastroenteritis
 - Pancreatitis
 - Behçet disease
 - A history of travel is important, as there are regional variations in etiology.

Physical-Exam

- A careful exam is important, as underlying etiology varies by region.
- Lesions are most common on the pretibial area but may occur on the thigh, upper

- extremities, neck and, rarely, the face.
- Absence of lesions on the lower extremities is atypical, as are ulcerated lesions.
- Lower-extremity edema may occur.
- Adenopathy should be evaluated.

ESSENTIAL WORKUP

Careful history and physical exam directed at detecting precipitating cause

DIAGNOSIS TESTS & NTERPRETATION

Lab

- CBC
- Throat culture/ASO titer
- ESR
- Appropriate chemistry tests
- Liver function tests
- Serologies for coccidioidomycosis in endemic regions
- TB skin testing in endemic regions

Imaging

CXR: Hilar adenopathy may be evidence of sarcoidosis, coccidioidomycosis, tuberculosis, or other fungal infections.

Diagnostic Procedures/Surgery

Definitive diagnosis made by deep elliptical biopsy and histopathologic evaluation (punch biopsy may be inadequate): Usually indicated for atypical cases or when TB is being considered

DIFFERENTIAL DIAGNOSIS

- EN migrans and chronic EN
- Any type of panniculitis can resemble EN.
- Differences can be determined histopathologically.
- Other disorders include:
 - Periarteritis nodosum
 - Migratory thrombophlebitis
 - Superficial varicose thrombophlebitis
 - Scleroderma
 - Systemic lupus erythematosus
 - α_1 -antitrypsin deficiency
 - Behçet syndrome
 - Lipodystrophies
 - Leukemic infiltration of fat
 - Panniculitis associated with steroid use, cold, and infection



TREATMENT

Pediatric Considerations

- Rare in children, *S. pharyngitis* is the most likely etiology.
- Cat scratch disease should be considered.

PRE HOSPITAL

Maintain universal precautions

INITIAL STABILIZATION/THERAPY

Airway, breathing, and circulation (ABCs); IV, oxygen, monitoring as appropriate

ED TREATMENT/PROCEDURES

- Treatment should be directed at underlying disease.
- Supportive therapies include rest and analgesics.
- Corticosteroids and potassium iodide may hasten resolution of symptoms.
- Systemic corticosteroids are contraindicated in the presence of certain underlying infections such as TB or coccidioidomycosis, which may disseminate with their use.
- Potassium iodide is contraindicated in hyperthyroid states.

MEDICATION

- Aspirin: 650 mg PO q4–6h PRN (peds: contraindicated)
 - _ Do not exceed 4 g/24 h
- Ibuprofen: 400–800 mg PO q8h (peds: 5–10 mg/kg PO q6h)
- Indomethacin: 25–50 mg PO q8h
- Potassium iodide/SSKI (used for resistant disease; contraindicated in hyperthyroidism): 900 mg PO daily for 3–4 wk
- Systemic corticosteroids (prednisone): 40 mg/d PO; duration determined by primary physician

First Line

- Rest/supportive care
- NSAIDs
- Treatment of underlying disease

Second Line

- Potassium iodide
- Steroids



FOLLOW-UP

DISPOSITION

Admission Criteria

Dictated by the severity of symptoms and the etiologic agent

Discharge Criteria

- Nontoxic patients, able to take PO fluids without difficulty
- Scheduled follow-up should be arranged.

Issues for Referral

- EN is usually self-limited and resolves in 3–6 wk.
- Atypical cases may need excisional biopsy.
- Steroid and potassium therapy needs primary physician monitoring.

FOLLOW-UP RECOMMENDATIONS

- Follow-up to assess for resolution with primary care physician or dermatologist.
- Evaluation of underlying etiology may require specialist referral.

PEARLS AND PITFALLS

- EN is usually idiopathic but may be the 1st sign of systemic disease.
- Lesions may recur if underlying disease is not treated.
- Atypical and chronic lesions may indicate an alternative diagnosis and need biopsy.
- Patients taking potassium or steroids need close follow-up.

ADDITIONAL READING

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CODES

ICD9

- 017.10 Erythema nodosum with hypersensitivity reaction in tuberculosis, unspecified

- 695.2 Erythema nodosum

ICD10

- A18.4 Tuberculosis of skin and subcutaneous tissue
- L52 Erythema nodosum

ESOPHAGEAL TRAUMA

Susan E. Dufel



BASICS

DESCRIPTION

- Adult esophagus is ~25–30 cm in length in close proximity to mediastinum with access to pleural space.
- It begins at hypopharynx posterior to larynx at level of cricoid cartilage.
- On either side of this slit are piriform recesses:
 - May be site for foreign body to lodge
- Sites of esophageal narrowing:
 - Cricopharyngeal muscle (upper esophageal sphincter)
 - Crossover of left main stem bronchus and aortic arch
 - Gastroesophageal junction (lower esophageal sphincter)
 - Areas of disease (cancer, webs, or Schatzki ring)
- Upper 3rd of esophagus is striated muscle:
 - Initiates swallowing
- Middle portion is mixture of striated and smooth.
- Distal portion is smooth muscle.
- It is a fixed structure, but can become displaced by other organs:
 - Goiter
 - Enlarged atria
 - Mediastinal masses

ETIOLOGY

Mechanism

- External forces or agents (30%):
 - Penetrating: Leading to tears:
 - Stab wounds
 - Missile wounds
 - Perforation:
 - Foreign bodies via direct penetration
 - Pressure necrosis
 - Chemical necrosis
 - Radiation necrosis from selective tissue ablation
 - Instrumentation
 - Blunt: Motor vehicle accident
- Internal forces or agents:
 - Caustic ingestions/burns:

- Acid pH < 2, alkali pH > 12 accidental or intentional
- Alkali (42%): Liquefaction necrosis causing burns, airway edema or compromise, perforation, chronic stricture, and cancer
- Acid (32%): Coagulation necrosis, thermal injury, and dehydration causing perforation, ulceration, and infection, more likely to perforate than alkali
- Chlorine bleach (26%): Mucosal edema, superficial erythema
- Infections:
 - Viruses (CMV, HPV, and HSV) or fungi in immunocompromised patients
- Drugs:
 - Less common but case series reported
 - Alendronate, Doxycycline, NSAIDs
 - Mycophenolate mofetil
 - May cause esophageal erosion or esophagitis
- Swallowed agents:
 - Food bolus impaction:
 - Coins, bones, buttons, marbles, pins, button batteries
 - Most common type is meat.
- In adults: Prisoners, psychiatric patients, intoxicated patients, or edentulous patients
- Iatrogenic (55%):
 - Perforation secondary to instrumentation, endoscopy most common cause
 - Nasotracheal intubation/nasogastric (NG) tube most common cause in emergency department
- Increased gastric pressure (15%):
 - Large pressure differences between thorax and intra-abdominal cavity:
 - May lead to lacerations or perforation
 - Mallory–Weiss syndrome:
 - Longitudinal tears in distal esophageal mucosa with bleeding
 - Boerhaave syndrome:
 - Spontaneous esophageal rupture
 - Full-thickness rupture of distal esophagus
 - Classically after alcohol or large meals and vomiting

Pediatric Considerations

- Foreign bodies
 - Accounts for 75–80% of swallowed foreign bodies:
 - Typically in infants ages 18–48 mo
 - Entrapment usually at upper esophageal sphincter
 - Perforations
 - Commonly iatrogenic with NG insertion, stricture dilation, and endotracheal intubation

- Caustic ingestions
 - More common in children <5 yr
 - Button batteries highly alkaline and need removal if lodged in esophagus within 4–6 hr
 - Packets of single use laundry/dishwasher detergents are prevalent with AAPCC issuing safety warning

DIAGNOSIS

SIGNS AND SYMPTOMS

General

- Dysphagia: Difficulty swallowing
- Odynophagia: Pain with swallowing
- Chest pain: Angina like, often pleuritic, severe, and unrelenting
- Hoarseness
- Dyspnea
- Tears or perforations:
 - Bleeding
 - Hematemesis
- Ingestions/foreign bodies:
 - Drooling or excessive salivation
 - Choking, gagging, vomiting, stridor, or wheezing
 - Inability of food or liquid to pass
- Caustic ingestions:
 - Oral pain
 - Abdominal pain
 - Vomiting
 - Drooling

History

- History of ingestions (type, time, amount)
- History of protracted vomiting
- History of inability to swallow after eating, foreign body sensation in throat
- History of penetrating trauma
- History of cancer therapy

Physical-Exam

- Tears or perforations:
 - SubQ air at base of neck
 - Hamman crunch:
 - Systolic crunching sound secondary to air in mediastinum

- Shock
- Septicemia
- Peritonitis
- Penetrating trauma:
 - Associated neck, chest, or abdominal injury with trauma:
 - Most commonly trachea
 - Associated with penetrating/blunt trauma
- Caustic ingestions:
 - Airway edema leading to stridor
 - Oral burns

ESSENTIAL WORKUP

High level of suspicion and early diagnosis are key:

- Mortality <5% for perforation if repaired within 24 hr; 75% if delayed
- Early endoscopy for caustic ingestions
- Chest/lateral neck radiograph

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC in cases of GI bleeding
- TXC for any extensive bleeding/OR candidate
- Coagulation studies
- Electrolytes for protracted vomiting or prolonged foreign body retention
- Arterial blood gas (ABG) for acid ingestions

Imaging

- CXR for foreign body or perforation:
 - Pneumomediastinum
 - Widened mediastinum
 - Pneumothorax
 - Pleural effusion
- Lateral cervical spine films for foreign body or perforation:
 - Retropharyngeal air or fluid
 - Cervical emphysema
- Fiberoptic nasopharyngoscopy for foreign body removal
- Esophagram for foreign bodies or suspected perforation:
 - 10–25% false-negative rate
 - Current recommendations for water-soluble contrast (Gastrografin) 1st if perforation likely
 - Barium may limit visibility for later endoscopy:
 - More irritating if extravasates into mediastinum
 - Water-soluble contrast provides better visibility:

- Less reaction if extravasates into mediastinum
- May cause chemical pneumonitis if aspirated
- Nonionic contrast may be safest but more expensive
- Endoscopy for suspected perforation, caustic ingestions, and esophageal foreign body removal
 - Severity of injury in caustic ingestions
 - 1st degree: Superficial mucosal damage, focal or diffuse, erythema, edema, mucosa sloughs without scar
 - 2nd degree: Mucosal and submucosal damage, ulcers and vesicles, granulation tissue and scar formation, stricture possible
 - 3rd degree: Transmural with deep ulcers, black discoloration, and wall perforation
- CT scanning with dilute oral contrast may be useful in diagnosis of perforations.

DIFFERENTIAL DIAGNOSIS

- Pulmonary:
 - Tracheal injury
 - Pneumothorax
- Cardiovascular:
 - Myocardial infarction
 - Aortic dissection
 - Spontaneous pneumomediastinum
- Other esophageal emergencies:
 - Peptic stricture
 - Esophageal neoplasm
 - Schatzki ring
 - Diverticula
 - Achalasia
 - Diffuse esophageal spasm
 - Nutcracker esophagus
 - Gastroesophageal reflux
 - Esophagitis
 - esophagitis esp. tetracycline



TREATMENT

PRE HOSPITAL

ALERT

- Chest pain should be presumed cardiac.
- Airway protection, frequent suctioning
- Intravenous crystalloid if patient is hypotensive, vomiting, or if hematemesis is

present

- Pain management
- Avoid neutralizing agents in caustic ingestions as that may worsen injury.
- Avoid copious amounts of oral fluids in caustic ingestions to prevent emesis.

INITIAL STABILIZATION/THERAPY

- Manage airway and resuscitate as needed
- Intravenous access, monitoring
- Early intubation for penetrating neck and chest wounds
- Frequent suctioning of copious secretions
- Fluid replacement

ED TREATMENT/PROCEDURES

- Foreign bodies/food impaction:
 - 80% pass, 20% need endoscopy, <1% need surgery
 - Glucagon may be tried: 1 mg IV and repeated in 20 min. Carbonated beverage in combo may be more effective
 - Nitroglycerin or nifedipine may be tried.
 - Diazepam may be of benefit in the upper (striated muscle) esophagus.
 - GI consultation and endoscopic extraction if not relieved
- Caustic ingestions:
 - Emesis/lavage contraindicated
 - Immediate decontamination with milk
 - Avoid neutralizing agents as they may cause exothermic reaction.
 - GI consultation for early endoscopy to provide prognostic information
 - No role for corticosteroids and may be harmful
- Tears/perforations:
 - Partial-thickness tears usually heal spontaneously.
 - GI consultation may be needed for diagnosis (endoscopy).
 - Perforation requires surgical consultation for thoracotomy and primary repair; some patients may be managed nonoperatively.
 - Broad-spectrum parenteral antibiotics for perforation

Pediatric Considerations

- Certain swallowed foreign bodies require GI consultation and endoscopic removal:
 - Sharp objects: Fish bones, straight pins, razor blades, pencil
 - Caustic objects: Button batteries
- Objects may pass on their own:
 - Coins, buttons, marbles
 - Open safety pins may pass spontaneously if blunt end forward.
- Consult pediatric GI specialist.

MEDICATION

- Foreign bodies/food impactions:
 - Glucagon: 1–2 mg (peds: 0.02–0.03 mg/kg) IV; may repeat once in 20 min
 - Nitroglycerin: 0.4 mg sublingually
 - Diazepam: 5–10 mg (peds: 1–2 mg) IV
- Perforation:
 - Cefoxitin: 1–2 g (peds: 100–160 mg/kg/24 h) IV q6–8h
 - Gentamicin: 1–1.7 mg/kg (peds: 1.5–2.5 mg/kg/24 h) IV q8h
 - Steroids not indicated in caustic ingestions



FOLLOW-UP

DISPOSITION

Admission Criteria

- Caustic ingestion
- Sharp foreign bodies
- Airway compromise
- Penetrating neck or chest trauma
- Evidence of sepsis, mediastinitis, or esophageal perforation
- Significant bleeding
- Inability to tolerate oral fluids

Discharge Criteria

- Self-limited bleeding from partial-thickness tear
- Foreign body or food impaction that has passed lower esophageal sphincter

PEARLS AND PITFALLS

Factors to predict outcomes in esophageal injuries:

- Time to diagnosis and definitive therapy: 24 hr decreases mortality by half.
- Location of injury: Cervical less than thoracic or abdominal
- Mechanism of injury: Spontaneous perforation has highest mortality 30–40%; iatrogenic 15–20%, and direct trauma 5–10%.

ADDITIONAL READING

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- Gander JW, Berdon WE, Cowles RA. Iatrogenic esophageal perforation in children. *Pediatr Surg Int*. 2009;25(5):395–401.
- Plott E, Jones D, McDermott D, et al. A state-of-the-art review of esophageal trauma: Where do we stand? *Dis Esophagus*. 2007;20:279–289.



See Also (Topic, Algorithm, Electronic Media Element)

- Boerhaave Syndrome
- Foreign Body, Caustic Ingestion, Esophageal
- Mallory–Weiss Syndrome



CODES

ICD9

- 862.22 Injury to esophagus without mention of open wound into cavity
- 862.32 Injury to esophagus with open wound into cavity
- 935.1 Foreign body in esophagus

ICD10

- S27.813A Laceration of esophagus (thoracic part), initial encounter
- S27.819A Unspecified injury of esophagus (thoracic part), initial encounter
- T18.108A Unsp foreign body in esophagus causing oth injury, initial encounter

ETHYLENE GLYCOL POISONING

Paul E. Stromberg • Kirk L. Cumpston

BASICS

DESCRIPTION

- Peak serum concentration in 1–4 hr
- Half-life, 2.5–4.5 hr
- <20% excreted unmetabolized by kidneys
- Pathophysiology:
 - Metabolized by hepatic alcohol dehydrogenase and aldehyde dehydrogenase ultimately to oxalic acid
 - Results in aldehyde and acid metabolites
 - Directly toxic to CNS, heart, and kidneys

ETIOLOGY

- Ethylene-glycol-containing products:
 - Antifreeze
 - Solvents
- Min. reported lethal dose is 30 mL of 100% ethylene glycol.

DIAGNOSIS

SIGNS AND SYMPTOMS

- Cardiovascular:
 - Tachycardia/bradycardia/other dysrhythmias
 - Hypertension/hypotension
- CNS:
 - Inebriation/irritability
 - Ataxia
 - Obtundation
 - Coma
 - Cerebral edema
 - Convulsions
 - Peripheral nervous system
 - Cranial nerve abnormalities
- GI:
 - Nausea/vomiting
 - Abdominal pain
- Pulmonary:
 - Hyperventilation/tachypnea/Kussmaul respiration

- Pulmonary edema
- Renal:
 - Acute renal failure
 - Crystalluria
- 3 stages (may overlap):
 - 1st stage: 1–12 hr after ingestion:
 - CNS depression
 - GI symptoms
 - Worsening acidosis
 - Coma
 - Convulsions
 - Cerebral edema
 - Tetany and myoclonus secondary to hypocalcemia
 - 2nd stage: 12–36 hr after ingestion:
 - Cardiopulmonary symptoms
 - When most deaths occur
 - 3rd stage: 36–72 hr after ingestion:
 - Oliguria
 - Flank pain
 - Acute renal failure

History

- Intentional or unintentional ethylene glycol ingestion
- No history but a patient with an unexplained high anion gap metabolic acidosis
- Elevated unexplained osmol gap

Physical-Exam

- Tachypnea
- Altered mental status

ESSENTIAL WORKUP

- History of all substances ingested
- Drawn simultaneously:
 - Arterial blood gas
 - Serum ethylene glycol, methanol, isopropyl alcohol, and ethanol serum concentration
 - Electrolytes, BUN/creatinine, glucose
 - Measured serum osmolality (by freezing point depression)
 - Serum calcium, phosphorus, magnesium

DIAGNOSIS TESTS & NTERPRETATION

Lab

- Determine the anion gap:
 - _ Anion gap = $(\text{Na}^+) - (\text{Cl}^- + \text{HCO}_3^-)$
 - _ Normal anion gap is 8–12.
- Determine osmol gap:
 - _ Osmol gap = measured osmolality – calculated osmolarity
 - _ Increased osmol gap: > 10
 - _ Calculated osmolarity = $2(\text{Na}^+) + \text{glucose}/18 + \text{BUN}/2.8 + \text{ethanol (mg/dL)}/4.6$
 - _ Calculated to screen for ethylene glycol ingestion because toxic alcohol serum concentration are not commonly available in timely manner from most clinical labs
 - _ Most useful early in course of ethylene glycol poisoning or with concurrent ethanol ingestion
 - _ With concurrent ethanol ingestion, osmol gap tends to be larger and acidosis tends to be less severe because relatively less ethylene glycol has been converted to acid-producing metabolites.
 - _ Normal osmol gap does not rule out ethylene glycol ingestion.
 - _ Late presentation after ethylene glycol ingestion may manifest itself with only an elevated anion gap without a significant osmol gap.
- Ethylene glycol, methanol, isopropyl alcohol serum concentration
- Ethanol serum concentration:
 - _ Measured to determine amount of ethanol bolus necessary to attain therapeutic serum concentration, and to determine coingestants
- Urinalysis:
 - _ Envelope-shaped oxalate crystals: Insensitive but specific finding.
 - _ Absence of urine calcium oxalate crystals does not rule out ethylene glycol exposure.
 - _ Ketones may be due to isopropyl alcohol ingestion, starvation, or diabetic ketoacidosis.

Diagnostic Procedures/Surgery

Wood lamp inspection of urine or gastric contents:

- Detects presence of fluorescein, a common antifreeze additive
- Insensitive and not specific marker of antifreeze ingestion
- Absence of urinary fluorescence does not rule out ethylene glycol exposure.

DIFFERENTIAL DIAGNOSIS

- Increased osmol gap:
 - _ Methanol
 - _ Ethanol
 - _ Diuretics (mannitol, glycerin, propylene glycol, sorbitol)
 - _ Isopropyl alcohol

- Ethylene glycol
- Acetone, ammonia
- Propylene glycol
- Elevated anion gap metabolic acidosis: *A CAT MUDPILES:*
 - Alcoholic ketoacidosis
 - Cyanide, CO, H₂S, others
 - Acetaminophen
 - Antiretrovirals (NRTI)
 - Toluene
 - Methanol, metformin
 - Uremia
 - Diabetic ketoacidosis
 - Paraldehyde, phenformin, propylene glycol
 - Iron, INH
 - Lactic acidosis
 - Ethylene glycol
 - Salicylate, acetylsalicylic acid (ASA; aspirin), starvation ketosis



TREATMENT

PRE HOSPITAL

- Bring containers of all possible substances ingested.
- Monitor airway and CNS depression.
- Dermal decontamination of an ethylene glycol chemical spill by removal of clothing and jewelry and irrigation with soap and water

INITIAL STABILIZATION/THERAPY

- ABCs
- Supplemental oxygen, cardiac monitor, secured IV line with 0.9% NS
- D₅₀W (or Accu-Check), naloxone, and thiamine for altered mental status

ED TREATMENT/PROCEDURES

- Prevent further ethylene glycol absorption:
 - Gastric lavage with nasogastric tube:
 - If < 1 hr since ingestion, if patient is in coma, or if history of large ingestion
 - Initial dose of activated charcoal for potential coingestants, but unlikely to help if only ethylene glycol:
 - Activated charcoal adsorbs ethylene glycol poorly.
- Prevent ethylene glycol conversion to toxic metabolites with fomepizole:
 - Fomepizole (4-MP, Antizol):
 - Initiate before ethylene glycol serum concentration returns, if

accidental ingestion greater than a sip or intentional ingestion
oraltered mental status associated with unexplained osmol gap or
elevated anion gap acidosis.

- Competitive inhibitor of alcohol dehydrogenase

– Disadvantages:

- Blurry vision
- Transient elevation of LFTs

– Advantages:

- Easy dosing
- No need for continuous infusion
- No inebriation/CNS depression
- No hypoglycemia, hyponatremia, or hyperosmolality
- Not necessary to check ethanol serum concentration
- Reduction in degree of nursing care and monitoring

● Ethanol therapy:

- 2nd choice antidote if fomepizole is not available
- Not FDA approved for treatment of ethylene glycol
- Initiate before ethylene glycol serum concentration returns, if potentially toxic ingestion is suspected.

– Ethanol: Greater affinity than ethylene glycol for alcohol dehydrogenase:

- Slows conversion to toxic metabolites

– Indications:

- History of accidental ethylene glycol ingestion of greater than a sip or intentional ethylene glycol ingestion
- Altered mental status associated with unexplained osmol gap or elevated anion gap metabolic acidosis

– Goal: Serum ethanol serum concentration of 100–150 mg/dL

- Continue ethanol therapy until ethylene glycol serum concentration is 25 mg/dL.

● Administer thiamine, pyridoxine, and magnesium:

- Cofactors in metabolism of ethylene glycol that may promote conversion to nontoxic metabolites.
- No human data supporting this theory

● Hemodialysis:

- Decreases elimination half-life of ethylene glycol and removes toxic metabolites
- Indications: Severe acidosis or osmol gap; persistent electrolyte or metabolic acidosis; renal insufficiency; pulmonary edema; cerebral edema; serum ethylene glycol serum concentration > 25 mg/dL
- Continue hemodialysis until ethylene glycol serum concentration approaches 25 mg/dL and metabolic acidosis resolves.

● Correct secondary disorders:

- Ensure adequate urine output via IV fluids.

- Sodium bicarbonate therapy for acidemia with pH < 7.1:
 - The goal is to maintain a serum pH in the normal range.
- Monitor/replace calcium:
 - Deposition of calcium into tissues can result in hypocalcemia.

Pregnancy Considerations

- Fomepizole is class C in pregnancy.
- Ethanol is not recommended in pregnancy. Class D/X

Pediatric Considerations

Ethanol can cause serious CNS depression and hypoglycemia when administered to children.

MEDICATION

- Activated charcoal: 1 g/kg PO
- Dextrose: D₅₀W 1 ampule: 50 mL or 25 g (peds: D₂₅W 2–4 mL/kg) IV
- Ethanol:
 - PO: 50% ethanol solution (100-proof liquor) via nasogastric tube:
 - Loading dose: 1.5 mL/kg
 - Maintenance dose: 0.2–0.4 mL/kg/h
 - Maintenance dose during hemodialysis: 0.4–0.7 mL/kg/h
 - IV: 10% ethanol in D₅W:
 - Loading dose: 8 mL/kg over 30–60 min
 - Maintenance infusion: 1–2 mL/kg/h
 - Maintenance infusion during hemodialysis: 2–4 mL/kg/h
- Fomepizole:
 - Loading dose: 15 mg/kg slow infusion over 30 min
 - Maintenance dose: 10 mg/kg q12h for 4 doses, then 15 mg/kg q12h until ethylene glycol serum concentration are reduced to < 25mg/dL
 - Dosing related to hemodialysis:
 - Do not administer dose at beginning of dialysis if last dose was < 6 hr previously.
 - Administer next dose if last dose was > 6 hr previously.
 - Dose q4h during dialysis.
 - If time between last dose and end of dialysis was < 1 hr from last dose, do not administer new dose.
 - If time between last dose and end of dialysis was 1–3 hr from last dose, administer 1/2 of next scheduled dose.
 - If time between last dose and end of dialysis was > 3 hr from last dose, administer next scheduled dose.
- Magnesium: 25–50 mg/kg IV 1 dose up to 2 g
- Naloxone: 2 mg (peds: 0.1 mg/kg) IV or IM initial dose

- Pyridoxine: 100 mg/d for 2 days
- Sodium bicarbonate: 1–2 mEq/kg in D₅W IV
- Thiamine: 100 mg (peds: 50 mg) IV or IM per day for 2 days



FOLLOW-UP

DISPOSITION

Admission Criteria

- All patients with significant ethylene glycol ingestion, even if initially asymptomatic
- ICU admission for seriously ill patients, metabolic acidosis, and renal failure
- Transfer to another facility if hemodialysis or fomepizole is indicated but not readily available.

Discharge Criteria

Asymptomatic patient with isolated ethylene glycol ingestion, if serum ethylene glycol serum concentration is undetectable and no metabolic acidosis

FOLLOW-UP RECOMMENDATIONS

Psychiatric referral for suicidal patients.

PEARLS AND PITFALLS

- An osmol gap <10 mmol/L does not rule out an ethylene glycol exposure.
- Administer fomepizole immediately and confirm exposure with a serum concentration for patients with an elevated anion gap and ethylene glycol exposure in the differential diagnosis.
- If you cannot confirm an ethylene glycol exposure, or do not have hemodialysis capabilities 24/7, or no antidote, transfer the patient to a facility which has all of the above capabilities.
- Not all patients will have an elevated osmol and anion gap. Early presenters will have an osmol gap only, and late presenters may have an anion gap only.
- Do not use the absence of urine crystals or fluorescence of the urine to rule out an ethylene glycol exposure.

ADDITIONAL READING

- Leikin J, Paloucek F. Ethylene glycol. Fomepizole. Alcohol. In: Leikin JB, Paloucek F, eds. *Leikin and Paloucek's Poisoning and Toxicology Handbook*. 4th ed. Boca Raton, FL: Lexi-Comp; 2008;989: 294–295, 794–795.
- Levine M, Curry SC, Ruha AM, et al. Ethylene glycol elimination kinetics and

outcomes in patients managed without hemodialysis. *Ann Emerg Med.* 2012;59:527–531.



CODES

ICD9

982.8 Toxic effect of other nonpetroleum-based solvents

ICD10

- T52.8X1A Toxic effect of organic solvents, accidental, init
- T52.8X2A Toxic effect of organic solvents, self-harm, init
- T52.8X4A Toxic effect of oth organic solvents, undetermined, init

EXTERNAL EAR CHONDRITIS/ABSCESS

Assaad J. Sayah

BASICS

DESCRIPTION

Inflammation and/or infection of the pinna

ETIOLOGY

- Mechanism:
 - Cartilage of the external ear is easily damaged due to:
 - Lack of overlying subcutaneous tissue
 - Relative avascularity
 - Exposed position
 - Chondritis:
 - Most commonly a secondary complication of otic trauma and burns
 - Onset is often insidious and may be delayed until apparent healing has occurred.
- Improper management may cause disfiguration of the pinna secondary to cartilage avascular necrosis:
 - Ranges from being a shriveled, cauliflower-like ear to complete loss of the external ear and possible stenosis of the auditory meatus.
- Causes:
 - Common causes of chondritis include:
 - Chemical or thermal burns
 - Frostbite
 - Hematoma formation
 - Trauma
 - Human/insect bites
 - Deep abrasions
 - External otitis
 - High piercing of the ear lobe especially with poor technique, hygiene, and aftercare.
 - Bacteria involved:
 - *Pseudomonas aeruginosa*
 - Staphylococcus
 - Proteus

DIAGNOSIS

SIGNS AND SYMPTOMS

- Initially a dull pain that increases in severity
- Fever
- Chills

History

- Ear trauma
- Ear piercing

Physical-Exam

- Pinna:
 - _ Painful
 - _ Exquisite tenderness
 - _ Erythematous
 - _ Warmth
 - _ Loss of contours caused by edema often with sparing of the lobule.
- Increase of the auriculocephalic angle
- Fluctuant areas develop with eventual breakdown and suppuration.

ESSENTIAL WORKUP

Clinical diagnosis:

- Typical physical findings in combination with aforementioned causes

DIAGNOSIS TESTS & INTERPRETATION

Lab

Only if systemic signs of infection:

- CBC
- Blood cultures
- Local cultures for chondritis and abscess drainage

DIFFERENTIAL DIAGNOSIS

- Allergic reaction
- Mastoiditis
- Dermatitis
- Hematoma



TREATMENT

ED TREATMENT/PROCEDURES

General postinjury preventive measures:

- Prevention of chondritis is of utmost importance:
 - _ Difficult management and disfiguring potential

- Avoid pressure to the injured ear.
- Minimize active débridement of eschars and crusts.
- Gentle washing twice daily with antibacterial soap and water followed by complete drying and application of topical antibiotics
- Keep hair away from the ear.
- Oral antibiotics for minor cases of early ear-lobe inflammation
- Parenteral antibiotics and early surgical drainage for patients with chondritis

MEDICATION

- Ciprofloxacin: 500 mg PO BID (adult)
- Cephalexin: 500 mg (peds: 50 mg/kg/d) PO QID
- Dicloxacillin: 500 mg (peds: 25 mg/kg/d) PO QID
- IV antibiotics for severe infection
- Apply topical antibiotics when there is a break in skin barrier.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Edema, erythema, and significant ear tenderness
- Toxic patient with fever and chills
- Immunocompromised patient

Discharge Criteria

Stable patient without systemic signs with close ear, nose, and throat (ENT) follow-up

Issues for Referral

ENT consult:

- For chondritis, abscess, and necrosis of the involved cartilage
- Early surgical drainage for chondritis and abscess

PEARLS AND PITFALLS

Aggressive early management may prevent gross ear deformity:

- Antibiotic regimen should cover for *Pseudomonas*.

ADDITIONAL READING

- Fisher CG, Kacica MA, Bennett NM. Risk factors for cartilage infections of the ear. *Am J Prev Med.* 2005;29(3):204–209.
- Guss J, Ruckenstein MJ. Infections of the external ear. In: Cummings CW, Flint PW, Haughey BH, et al., eds. *Otolaryngology: Head & Neck Surgery.* 5th ed.

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- Rowshan HH, Keith K, Baur D, et al. Pseudomonas aeruginosa infection of the auricular cartilage caused by “high ear piercing”: A case report and review of the literature. *J Oral Maxillofac Surg.* 2008;66(3):543–546.
- Van Wijk MP, Kummer JA, Kon M. Ear piercing techniques and their effect on cartilage, a histologic study. *J Plast Reconstr Aesthet Surg.* 2008;61(suppl 1):S104–S109.



CODES

ICD9

- 380.03 Chondritis of pinna
- 380.10 Infective otitis externa, unspecified

ICD10

- H60.00 Abscess of external ear, unspecified ear
- H61.033 Chondritis of external ear, bilateral
- H61.039 Chondritis of external ear, unspecified ear

EXTREMITY TRAUMA, PENETRATING

Gary M. Vilke

BASICS

DESCRIPTION

Penetrating injury to extremity

ETIOLOGY

- Stab or puncture
- Gunshot
- Laceration
- Bite
- High-pressure injection injury

DIAGNOSIS

SIGNS AND SYMPTOMS

- Entry and exit wound (if present), lacerations
- High-muzzle-velocity gunshot wounds:
 - _ Produce shock wave that results in significant tissue injury
 - _ Often exit wound demonstrates more tissue damage than entrance wound.
- Vascular injury:
 - _ Arterial injury:
 - Decreased or absent distal pulse
 - Distal ischemic changes
 - Expanding hematoma
 - Bruit or thrill over injury
 - _ Presence of distal pulse does not exclude proximal vascular injury.
- Neurologic injury:
 - _ Paresthesias
 - _ Decreased or absent motor function
 - _ Diminished sensation distal to injury
- Musculoskeletal injury:
 - _ Visible deformity
 - _ Ligamentous laxity in joints adjacent to injury suggests tendon injury.
 - _ Effusion in adjacent joint indicates fracture or ligamentous injury.
- Compartment syndrome:
 - _ Suggested by severe and constant pain over involved compartment
 - _ Pain on active and passive extension or flexion of distal extremity
 - _ Weakness, pain on palpation of compartment

- Hypesthesia of nerves in compartment
- Pulselessness and pallor are late findings.

History

- Mechanism of injury
- Age of wound
- Circumstances of wounding:
 - Assault
 - Self-inflicted wound
 - Domestic violence
- Comorbid conditions:
 - Immunosuppression or diabetes
 - Valvular heart disease
 - Asplenia
 - Peripheral vascular disease

Physical-Exam

- Note location, length, depth, and shape of primary wound and exit wound, if present.
- Vascular injury:
 - Compare distal pulses by palpation and with Doppler study.
 - Assess capillary refill:
 - Abnormal if > 2 sec
 - Ankle-brachial index (ABI):
 - Take BP in calf and arm (involved extremity).
 - Systolic pressure difference of > 10 mm Hg suggests vascular injury.
 - Expanding hematoma, bruit, or thrill over injury also indicates vascular injury.
- Neurologic injury:
 - Assess distal motor function and sensory function:
 - 2-point discrimination
 - Light touch
 - Proprioception
- Musculoskeletal injury:
 - Note associated crush, tendon, or ligamentous injury and bony deformity.
 - Examine adjacent joints for range of motion.
 - Assess for compartment syndrome.
- Explore wound for foreign body (FB).

ESSENTIAL WORKUP

- Physical exam
- Imaging if findings suggestive of bony injury or possible FB

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Culture of acute wounds is not indicated.
- Wounds with signs of infection may be cultured to guide antibiotic choice.

Imaging

- Radiograph to evaluate for radiopaque FB or underlying fracture:
 - _ Min. AP and lateral views
- Radiolucent FBs may be located by US, fluoroscopy, or CT.

Diagnostic Procedures/Surgery

Arteriogram is indicated when vascular injury is suspected and immediate vascular surgery not required.

DIFFERENTIAL DIAGNOSIS

Any medical condition that presents with findings consistent with extremity trauma or a wound



TREATMENT

PRE HOSPITAL

Cautions:

- Control hemorrhage with direct pressure over site.
- Elevate extremity.
- Evaluate neurovascular status.
- Leave impaled objects in place and stabilize in current position.
- Pain control

INITIAL STABILIZATION/THERAPY

- Manage airway and resuscitate as indicated.
- Expose wound completely and remove constricting clothing or jewelry.
- Control hemorrhage with direct pressure.
- Blind clamping within wound and prolonged tourniquet use are not recommended.

ED TREATMENT/PROCEDURES

- Pain control
- Complete neurologic assessment before local anesthesia
- Prolonged soaking of wounds, particularly with cytotoxic agents, is *not* recommended.
- Remove any visible debris and débride devitalized tissue.
- Most important is copious high-pressure irrigation with saline.

- Tetanus prophylaxis
- Stab wounds and gunshot wounds should receive single dose of cefazolin in ED.
- Immobilize extremity if there is suspicion of significant vascular injury, tendon injury, fracture, or joint violation.
- Loss of pulse or distal ischemia requires emergent surgery:
 - Do not delay surgical management for arteriogram.
- Lacerations may be closed if they have been adequately cleaned, have minimal tissue loss, and are seen within 6–8 hr of injury:
 - Delayed primary closure is an alternative for older or contaminated wounds.
- Puncture or gunshot wounds should *not* be closed primarily.
- Special considerations:
 - Plantar puncture wounds:
 - Examine wound carefully under bright light.
 - Remove any foreign material.
 - Clean wound carefully.
 - Coring wound is controversial and should be reserved for removal of devitalized tissue or imbedded debris:
 - Probing or high-pressure irrigation of puncture wound will only force particulate matter further into wound.
 - Prophylactic antibiotics are not recommended (unless patient is diabetic or immunocompromised or if the wound is highly contaminated or delayed in presentation).
 - If not treated with aggressive debridement, can lead to osteomyelitis
 - High-pressure injuries of hand:
 - Orthopedic evaluation in ED is essential because wounds that appear trivial on surface may have product track up tendon sheaths into more proximal aspects of hand.
 - Some paints and other products are radiopaque, and plain radiographs may demonstrate extent of spread.
 - Soft tissue FBs:
 - Small inert FB in wound, including bullets, not easily retrievable and not in close proximity to joint, tendon, vessel, or nerve can be left in place with close follow-up.
 - FB in hands and feet should be referred to specialist as they often migrate and become or remain symptomatic.
 - Organic materials (thorns, wood, spines, clothing) should be removed as they are very reactive.

MEDICATION

- Tetanus prophylaxis: TDap 0.5 mL IM (TD only if > 65 yr
- Wounds > 12 hr old, especially of hands and lower extremities, crush wounds with devitalized tissue, contaminated wounds

First Line

- Cefazolin: 1 g IV/IM (peds: 20–40 mg/kg IV/IM single dose in ED)
- Cephalexin: 500 mg PO (peds: 25–50 mg/kg/d) QID for 7 days *or*
 - Amoxicillin/clavulanate: 875/125 mg PO (peds: 25 mg/kg/d) BID for 7 days
- Erythromycin: 333 mg PO TID (peds: 40 mg/kg/d q6h for 7 days)
- Contaminated wounds in patients with pre-existing valvular heart disease:
 - Cefazolin: 1 g IV/IM, then cephalexin 500 mg PO QID for 7 days
- Plantar through shoe at risk for *Pseudomonas*:
 - Ciprofloxacin 500 mg BID for 7–10 days *or*
 - Levofloxacin 500 mg QD for 7–10 days

Second Line

If penicillin allergic:

- EES: 800 mg PO, then 400 mg PO q6h for 7 days *or*
- Clindamycin: 300 mg PO q6–8h for 7 days



FOLLOW-UP

DISPOSITION

Admission Criteria

- Emergent surgical consultation and admission are required for any penetrating wounds with potential for vascular compromise, associated compartment syndrome, and joint penetration.
- High-muzzle-velocity penetrating gunshot wounds
- Diabetic or immunocompromised patients with contaminated wounds

Discharge Criteria

Penetrating extremity injuries not requiring surgical intervention may be discharged after appropriate wound care with instructions to elevate extremity, keep wound clean, and to return for recheck in 24–48 hr or for any signs of infection.

Issues for Referral

- Plantar puncture wounds: Close follow-up is necessary to assess for infection from unseen FB.
- Delayed primary closure is alternative for older or contaminated wounds.
- Wounds at high risk for infection should have close 1–2 day follow-up.

FOLLOW-UP RECOMMENDATIONS

Return to the ED for increasing pain, numbness, tingling, redness, swelling drainage, fevers or other changes in clinical presentation.

PEARLS AND PITFALLS

- Presence of distal pulse does not exclude proximal vascular injury.
- High-pressure injuries of hand may have wounds that appear trivial on surface but track up tendon sheaths into more proximal aspects of hand.
- Plantar surface puncture wounds through shoes or socks have relatively high risk of retained foreign material – patients should be told of this possibility.
- Post-puncture wound infections failing to respond to antibiotics should be suspected of having retained FB.

ADDITIONAL READING

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- Newton EJ, Love J. Acute complications of extremity trauma. *Emerg Med Clin North Am.* 2007;25(3):751–761.



See Also (Topic, Algorithm, Electronic Media Element)

- Bite, Animal
- Compartment Syndrome
- Ring/Constricting Band Removal



CODES

ICD9

- 884.0 Multiple and unspecified open wound of upper limb, without mention of complication
- 894.0 Multiple and unspecified open wound of lower limb, without mention of complication
- 928.9 Crushing injury of unspecified site of lower limb

ICD10

- S41.139A Puncture wound w/o foreign body of unsp upper arm, init
- S81.839A Puncture wound w/o foreign body, unsp lower leg, init encntr
- S87.80XA Crushing injury of unspecified lower leg, initial encounter

FACIAL FRACTURES

David W. Munter

BASICS

DESCRIPTION

- Typically blunt trauma from motor vehicle accidents, direct blows including assaults, or falls.
- Consider physical assault and domestic violence, especially in women and children.
- Open fractures common.
- Many facial fractures are complex and are not easily classified.

ETIOLOGY

- Le Fort fractures involve the maxilla and are classified as:
 - Le Fort I: Transverse fracture of maxilla below nose but above teeth through lateral wall of maxillary sinus to lateral pterygoid plate.
 - Le Fort II: Pyramidal fracture from nasal and ethmoid bones through zygomaticomaxillary suture and maxilla, often involving maxillary sinuses and infraorbital rims.
 - Le Fort III: Craniofacial disjunction with elongated, flattened face owing to fractures through frontozygomatic suture, orbit, base of nose, and ethmoid bone.
 - Le Fort IV: Includes frontal bone in addition to Le Fort III.
 - A patient may have different level Le Fort fractures on each side of the face.
- Zygomatic arch fractures often occur in 2 or 3 places and can involve the orbit and maxilla (tripod fracture).
- Inner plate frontal sinus fractures are associated with CSF leaks and ocular injuries.
- Orbital fractures most commonly involve the orbital floor (blow-out fracture), and are commonly associated with ocular injuries but can involve the medial and lateral orbital walls.

Geriatric Considerations

- Falls most common cause.
- Zygoma most common bone fractured.
- Beware of associated cervical and intracranial injuries.

Pediatric Considerations

- Maxillofacial fractures rarely seen in children younger than 6 yr; suspect nonaccidental trauma.

- Falls and motor vehicle accidents account for most cases.
- Over 50% have severe associated injuries, high incidence of associated head injury.
- Fractures of the orbit are the most common facial fracture in children (excluding nose)

DIAGNOSIS

SIGNS AND SYMPTOMS

- Most post-traumatic deformities of the face represent underlying fractures.
- Pain, swelling, ecchymosis, and deformity.
- CSF rhinorrhea, facial hemorrhage, epistaxis, raccoon eyes.
- Facial anesthesia with nerve entrapment or injury.
- Associated injuries; tooth, mandible, eye, tear duct, skull, and neck.
- Bluish fluid-filled sac overlying nasal septum is a septal hematoma and is critical to detect.

History

- Mechanism of injury.
- Associated injuries.

Physical-Exam

- Immediately assess airway.
- Most important:
 - _ Palpate entire face for tenderness, step-offs, depressions, and crepitus.
 - _ Check for mandibular injuries or malocclusion.
 - _ Nasal speculum exam for septal hematoma or CSF leak.
 - _ Assess for areas of facial anesthesia.
 - _ Careful eye exam including funduscopic exam; obtain a visual acuity; assess for telecanthus (intercanthal width $> 30\text{--}35$ mm), upward dysconjugate gaze (indicative of ocular muscle entrapment in an orbital floor blow-out fracture).
- Le Fort fractures are assessed by placing thumb and index finger of 1 hand on the bridge of the nose and pulling upper teeth with other hand:
 - _ Le Fort I: Movement of hard palate and maxillary dentition only (your hand on the nose will not feel movement).
 - _ Le Fort II: Movement of hard palate, maxillary dentition, and nose (your hand on the nose will feel movement).
 - _ Le Fort III: Movement of entire midface.

Pediatric Considerations

- Sedation may be needed to perform an adequate exam.

ESSENTIAL WORKUP

- After airway is secured, other injuries take precedence.
- Radiologic studies in all cases of suspected facial fractures.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Indicated for evaluation of associated injuries or if needed for preoperative reasons.

Imaging

- Facial bone CT scanning with reconstructions is the imaging modality of choice for suspected facial injuries.
- Plain films such as a Waters view are less helpful.
 - May show fractures, asymmetry, or blood in the sinuses, or the classic teardrop opacity in the maxillary sinus representing an orbital floor blow-out fracture.
- Jug-handle views (submental vertex) may visualize zygomatic arch fractures.

DIFFERENTIAL DIAGNOSIS

- Nasal fracture.
- Zygoma fractures (arch or tripod fracture).
- Le Fort fracture.
- Skull fractures including frontal sinus fractures and cribriform plate fractures.
- Nasofrontoethmoid complex fractures.
- Mandibular fractures.
- Orbital fracture including blow-out fracture
- Associated injuries to teeth, neck, and brain.
- Contusions or lacerations without underlying fractures.



TREATMENT

PRE HOSPITAL

ALERT

- Airway control takes precedence:
 - Attempt chin lift, jaw thrust, and suctioning first.
 - Underlying injuries may make these attempts as well as use of bag/valve/mask (BVM) device unsuccessful.
 - Severe facial fractures may preclude oral intubation.
 - Nasotracheal intubation contraindicated in massive facial or nasal trauma.
 - Cricothyroidotomy performed if intubation using rapid-sequence induction

(RSI) cannot be performed.

- If associated injuries are present, protect cervical spine.

INITIAL STABILIZATION/THERAPY

- Aggressively manage airway if not patent, patient requires airway protection, or ongoing swelling or bleeding threatens airway. RSI is initial airway management of choice in facial injuries; use etomidate or midazolam and vecuronium, rocuronium, or succinylcholine for RSI.
- Surgical airway (cricothyroidotomy or needle cricothyroidotomy) may be required if RSI is unsuccessful.
- Nasotracheal intubation is contraindicated in most facial fractures.
- Protect cervical spine until clinically or radiographically cleared.
- Once airway is secure, other major injuries take precedence over facial injuries.
- Bleeding may be difficult to control and may require posterior packing if direct pressure does not work.

ED TREATMENT/PROCEDURES

- Consult ear, nose, throat specialist; plastic surgery; or oral surgery for complex fractures, including all Le Fort fractures, and neurosurgery for frontal sinus fractures involving the posterior table.
- Antibiotics (cefazolin or clindamycin in penicillin-allergic patients) for open fractures and CSF leak.
- Tetanus prophylaxis.
- Parenteral pain medication (morphine or fentanyl).
- A septal hematoma must be drained in the ED:
 - Anesthetize, aspirate with an 18G–20G needle, and pack both nares with Vaseline gauze.
 - Discharge on amoxicillin or erythromycin with recheck in 24 hr by ear, nose, and throat specialist.
- Nondisplaced zygomatic fractures can be discharged with analgesics (acetaminophen or ibuprofen); refer displaced zygoma and tripod fractures that are otherwise stable for outpatient reduction in 2–3 days after swelling is reduced.
- Overlying lacerations with simple fractures can be sutured in the emergency department; if patient is discharged, treat with amoxicillin or azithromycin.
- Patients discharged with facial fractures with blood in the sinus should be treated with amoxicillin or azithromycin.

Pediatric Considerations

- Surgical cricothyroidotomy should not be performed in children younger than 8 yr:
 - Needle cricothyroidotomy with jet ventilation may be performed.
- Children are at high risk of associated injuries.
- Repair of facial fractures should not be delayed more than 3–4 days (rapid healing of facial fractures and the risk of malunion and cosmetic deformity).

MEDICATION

- Acetaminophen: 500 mg (peds: 10–15 mg/kg, do not exceed 5 doses/24 h) PO q4–6h, do not exceed 4 g/24 h
- Amoxicillin: 250 mg (peds: 40–80 mg/kg/24 h) PO q8h
- Azithromycin: 500 mg PO day 1 followed by 250 mg PO days 2–4 (peds: 10 mg/kg PO day 1 followed by 5 mg/kg days 2–4)
- Cefazolin: 1 g (peds: 50–100 mg/kg/24 h) IV or IM
- Clindamycin: 600–900 mg (peds: 25–40 mg/kg/24 h) PO q8h
- Diazepam: 5–10 mg (peds: 0.1–0.2 mg/kg) IV
- Etomidate: 0.2–0.3 mg/kg (peds: 0.2–0.3 mg/kg) IV (not recommended in children <10 yr)
- Fentanyl: 2–10 µg/kg (peds: 2–3 µg/kg) IV
- Ibuprofen: 600–800 mg (peds: 20–40 mg/kg/24 h) PO TID–QID
- Ketamine: 1–2 mg/kg (peds: 1–2 mg/kg) IV
- Midazolam: 2–5 mg (peds: 0.02–0.05 mg/kg per dose, max. dose 0.4 mg/kg total and not >10 mg) IV over 2–3 min
- Morphine sulfate: 0.1–0.2 mg/kg (peds: 0.1–0.2 mg/kg) IV q1–4h titrated
- Rocuronium: 0.6–1.2 mg/kg (peds: 0.6 mg/kg) IV
- Succinylcholine: 1–1.5 mg/kg (peds: 1–2 mg/kg) IV
- Vecuronium: 0.1–0.3 mg/kg (peds: 0.1–0.3 mg/kg) IV



FOLLOW-UP

DISPOSITION

Admission Criteria

- Significant associated trauma.
- Airway compromise.
- Le Fort II and III fractures.
- CSF leak.
- Posterior table frontal sinus fractures.
- Most open fractures, excluding simple nasal fractures with lacerations.

Discharge Criteria

- No evidence of significant head, neck, or other injuries.
- Closed fractures of the zygoma, orbit, sinus, or anterior table of the frontal sinus with appropriate follow-up in 24–36 hr.
- Septal hematomas that have been drained in the emergency department require follow-up in 24 hr.
- Refer displaced zygoma and tripod fractures that are otherwise stable for outpatient reduction in 2–3 days after swelling is reduced.

Issues for Referral

- ENT, plastic surgery, or neurosurgery may all handle facial fractures, actual referral depends on practice patterns at your institution. If there is no CSF leak or involvement of the posterior table of the frontal sinus, it is reasonable to initially consult ENT.

PEARLS AND PITFALLS

- Facial fractures and injuries can be very dramatic in appearance.
 - Airway management always takes precedence. Avoid nasotracheal intubation.
 - After the airway is secured as necessary, evaluation of other injuries takes precedence—do not miss life-threatening injuries.
 - Cervical spine.
 - Pulmonary or thoracic.
 - Intra-abdominal injuries.
- Have a low threshold for obtaining facial bone CT for evaluation of facial injuries.
- Facial fractures are frequently associated with ocular injuries. Perform a thorough eye exam.
- Always assess for a nasal septal hematoma.
- Missing teeth must be accounted for, obtain a CXR to rule out aspiration.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Blow-out Fracture
- Mandibular Fracture
- Nasal Fracture
- Rapid Sequence Intubation



CODES

ICD9

- 802.4 Closed fracture of malar and maxillary bones
- 802.6 Closed fracture of orbital floor (blow-out)
- 802.8 Closed fracture of other facial bones

ICD10

- S02.3XXA Fracture of orbital floor, init encntr for closed fracture
- S02.92XA Unsp fracture of facial bones, init for clos fx
- S02.401A Maxillary fracture, unsp, init encntr for closed fracture

FAILURE TO THRIVE

Roger M. Barkin

BASICS

DESCRIPTION

- Not a single disease, but a description of a group of symptoms
- Inadequate physical growth:
 - Usually diagnosed earlier than age 2 yr
- Broadly divided into:
 - Organic (underlying medical condition)
 - Nonorganic (no underlying medical condition)
- Found in all socioeconomic groups
- Poverty increases risk of failure to thrive (FTT)
- May result in long-term growth, behavioral, and developmental difficulties, particularly in children who fail to thrive in the first few months of life

ETIOLOGY

Many diseases with unique causes resulting in 1 or more of:

- Inadequate caloric intake
- Inadequate caloric absorption, malabsorption
- Excessive caloric expenditure
- These may be secondary to underlying chronic disease

DIAGNOSIS

SIGNS AND SYMPTOMS

- No universally accepted definition
- Failure to achieve or maintain a growth rate appropriate for age
- Weight less than 2 standard deviations below normal for age (corrected for prematurity) and sex
- Weight that crosses downward through 2 major percentiles (major percentiles are 5th, 10th, 25th, 50th, 75th, and 90th percentiles) on standard growth chart (see Additional Reading below)
- There is an associated change in the velocity of growth of 1 or more growth parameters. Any of the 3 routinely monitored growth parameters may be impaired initially:
 - Weight loss initially followed by impaired growth in length/height and finally head circumference usually caused with caloric inadequacy.
 - Primary length/height fall-off often associated with endocrinology problem
 - Impairment in growth of head circumference commonly caused by CNS

primary condition.

- Although the pattern is usually one of slow decrease in growth velocity, an abrupt change may occur, usually indicative of an organic origin.
- Can manifest as:
 - Reduced muscle mass
 - Loss of subcutaneous fat
 - Alopecia
 - Dermatitis
 - Chronic disease
 - Marasmus
 - Kwashiorkor
 - Associated endocrinologic findings
 - Abnormal neurologic exam and development
 - Decreased immunologic function and increased risk of infection

History

- Detailed feeding history:
 - Breast-feeding:
 - Prior breast-feeding experience
 - Frequency of feedings
 - Length of feedings
 - Family support for breast-feeding
 - Formula:
 - Type of formula (milk, soy, elemental, preemie)
 - How formula is prepared (ready to feed, powder, liquid concentrate)
 - Frequency of feedings
 - Volume per feeding
 - Solid foods
 - Vomiting associated with feeds
 - Urine and stool output:
 - Blood in stool
- Gestational history:
 - Maternal medical complications
 - Drug or alcohol use
- Birth history:
 - Complications, intrauterine growth retardation, prematurity
 - Birth weight
 - Congenital anomalies
 - Intrauterine exposures/infections
- Developmental history:
 - Achievement of appropriate milestones
 - Child's perceived temperament
- Psychosocial history:

- Family composition
- Family/social support
- Stresses
- Maternal depression
- Abuse or neglect

Physical-Exam

- Weight, length/height, head circumference:
 - Plotted on appropriate growth chart:
 - Include as many prior growth points as possible
- Dysmorphic features:
 - Cardiac disorders
 - Pulmonary disorders
 - GI disorders
- Skin exam to include signs of child abuse

ESSENTIAL WORKUP

- Detailed history and physical exam
- Growth parameters plotted on appropriate growth charts
- Observation of family–child interaction
- Direct observation of feeding
- CBC, CRP, electrolytes, urinalysis and urine culture, and if indicated, lead level

DIAGNOSIS TESTS & NTERPRETATION

Lab

- CBC:
 - Anemia
 - Infection
 - Leukemia/malignancy
 - Lead level
 - Lead poisoning
- Chemistry panel (electrolytes, BUN, creatinine, glucose, liver function, protein, albumin, calcium, phosphate, magnesium):
 - Hydration and acidosis
 - Metabolic and endocrinologic disorders including thyroid disease. Often checking the routine newborn screening (NBS) is useful
 - Diabetes mellitus
 - Renal disease
 - Blood gas analysis
 - Renal tubular acidosis
 - Inborn errors of metabolism
- Urinalysis with culture:

- Renal disease
- Infection
- HIV
- Stool studies including occult blood, culture, and ova and parasites

Imaging

- CXR:
 - TB
 - Pneumonia
 - Cardiomegaly
- Bone age

Diagnostic Procedures/Surgery

- pH probe:
 - Gastroesophageal reflux
- Sweat chloride test:
 - Cystic fibrosis (may be part of NBS)
- Tuberculin skin testing

DIFFERENTIAL DIAGNOSIS

- Organic causes:
 - GI:
 - Malabsorption syndromes
 - Celiac disease
 - Cystic fibrosis
 - Food allergy
 - Inflammatory bowel disease
 - Hepatobiliary disease
 - Hepatitis
 - Cirrhosis
 - Biliary atresia
 - Obstructive disease
 - Pyloric stenosis
 - Malrotation
 - Hirschsprung disease
 - Pancreatitis
 - Short gut syndrome
 - Gastroesophageal reflux
 - Vitamin deficiencies
 - Cardiac:
 - Congenital heart disease
 - Cyanotic
 - Congestive

- Acquired heart disease
- Pulmonary:
 - Bronchopulmonary dysplasia
 - Obstructive sleep apnea
 - Chronic lung disease
 - Cystic fibrosis
- Hematologic/oncologic:
 - Iron-deficiency anemia
 - Thalassemia
 - Lead poisoning
 - Leukemia
- Renal:
 - Chronic renal insufficiency
 - Renal tubular acidosis
 - Recurrent UTIs
- Neurologic/CNS:
 - Hydrocephalus
 - Hypertonia/hypotonia
 - Generalized weakness (i.e., spinal muscular atrophy)
 - Oromotor dysfunction
- Immunologic:
 - AIDS
- Endocrine:
 - Diabetes mellitus
 - Thyroid/parathyroid disease
 - Adrenal disease
 - Growth hormone deficiency
 - Hypopituitarism
 - Hypophosphatemic rickets
- Infectious:
 - TB
 - Parasite
 - UTI
- Genetic/congenital:
 - Fetal alcohol syndrome
 - Smith–Lemli–Opitz syndrome
 - Cleft lip/palate
 - Inborn errors of metabolism
 - Many genetic syndromes can contribute.
- Toxic
- Nonorganic causes:
 - Parent–child dysfunction:
 - Mother–infant bonding problems

- Maternal mental illness/substance abuse
- Inexperienced mother
- Breast-feeding difficulties
- Improper formula preparation
- Inadequate availability of formula
- Chaotic family environment
- Child abuse or neglect
- Munchhausen syndrome by proxy



TREATMENT

INITIAL STABILIZATION/THERAPY

- Check for hypoglycemia
- Fluid resuscitation when dehydrated
- Supportive/nonjudgmental environment

ED TREATMENT/PROCEDURES

- Recognize/identify child with FTT
- Rule out organic abnormalities:
 - Organic causes may have specific treatments.
- Social services consult
- Breast-feeding consult:
 - Advise on appropriate feeding.

MEDICATION

Dependent on underlying cause



FOLLOW-UP

DISPOSITION

Admission Criteria

- Organic cause requiring medical admission
- Nonorganic causes to observe caregiver–child interaction
- Nonorganic causes to observe weight while monitoring oral intake. This is particularly appropriate in children < 3–6 mo of age because of the potential impact upon cognitive development
- Suspected child abuse/neglect
- Severe dehydration, malnutrition, or electrolyte imbalance

Discharge Criteria

- Case appropriately managed by primary care physician

- Follow-up is adequate to provide close monitoring of intake and growth.

Issues for Referral

Subspecialty referral depending on cause

ADDITIONAL READING

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- Shah MD. Failure to thrive in children. *J Clin Gastroenterol.* 2002;35(5):371–374.



CODES

ICD9

- 779.34 Failure to thrive in newborn
- 783.41 Failure to thrive

ICD10

- P92.6 Failure to thrive in newborn
- R62.51 Failure to thrive (child)

FATIGUE

Matthew B. Mostofi

BASICS

DESCRIPTION

- A subjective state of overwhelming, sustained exhaustion and decreased capacity for physical and mental work that is not relieved by rest
- Fatigue occurs with or without objective findings on physical exam.
- Fatigue is a common complaint in people with and without systemic disease, which makes this complaint a challenge to practicing physicians.

ETIOLOGY

- The specific mechanisms of fatigue are unknown.
- Hematologic:
 - Anemia
 - Leukemia
- Endocrine:
 - Thyroid disorders
 - Adrenal insufficiency
 - Diabetes
 - Pregnancy
- Malignancy:
 - Paraneoplastic syndromes
- Psychiatric:
 - Chronic pain
 - Emotional distress
 - Depression
 - Eating disorders
 - Chemical dependency
 - Withdrawal syndromes
- Sleep disorders:
 - Insomnia
 - Sleep apnea
- Cardiac and pulmonary disorders
- Infections acute and chronic
- Rheumatic and autoimmune disorders
- Nutritional deficiencies including electrolyte abnormalities
- Physical inactivity and deconditioning
- Medications
- Chronic fatigue syndrome:

- Symptom complex defined by the CDC
- Severe chronic fatigue lasting >6 mo
- Not explained by any medical or psychiatric diagnosis
- Presence of 4 or more of the following 8 symptoms:
 - Headache
 - Arthralgias
 - Sleep disturbances
 - Lymphadenopathy
 - Exercise intolerance
 - Myalgias
 - Impaired memory/concentration
 - Sore throat

DIAGNOSIS

SIGNS AND SYMPTOMS

- Fatigue is a subjective complaint of exhaustion or tired sensation that interferes with normal activities of life, and symptoms do not resolve with sleep.
- There are no specific signs of fatigue, but frequently physical signs may hint at the underlying cause of complaint.

History

- Onset, pattern, duration of fatigue
- Associated symptoms: Fever, night sweats, weakness, dyspnea, weight loss/gain, sleep patterns
- Past medical and surgical history
- Psychiatric history: Emotional and mental stressors, depression
- Social history: Alcohol, drug use, major life events
- Medications
- Full review of systems

Physical-Exam

- A complete physical exam should be focused on trying to identify an underlying cause for patient's symptoms. No physical findings are specific to fatigue.
- A partial list of physical exam findings which may suggest an underlying cause include:
 - Vital signs
 - HEENT
 - Pupils for evidence of toxidrome
 - Sclera for icterus in liver disease
 - Conjunctiva pale in anemia
 - Thyroid for enlargement, pain, or nodule that would suggest

dysfunction

- Heart: Murmurs or S3 may suggest LV dysfunction.
- Lung: Abnormal AP diameter or breath sounds may suggest chronic or acute lung disease.
- Abdomen: Tenderness or masses should be investigated.
- Skin: Rash may suggest infectious or autoimmune disease, lack of turgor may suggest dehydration, hyperpigmentation in Addison disease.
- Neurologic: True weakness or areflexia may suggest neuromuscular disorder, all new focal weakness should be investigated.
- Musculoskeletal: Indwelling IV lines or dialysis catheters should prompt investigation of electrolyte abnormality or occult bacteremia.

ESSENTIAL WORKUP

- Because fatigue is a subjective complaint, the essential workup is directed at identification of an underlying cause.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Lab evaluation should be directed by findings of history and physical exam.
- CBC:
 - Screen for anemia or leukemia.
- Serum glucose:
 - Both hyperglycemia and hypoglycemia can present with fatigue.
- Pregnancy test
- Electrolytes with BUN/creatinine
- Thyroid-stimulating hormone:
 - Screen for hypothyroidism.
- Urine drug screen

Imaging

Imaging/special test: Special tests are reserved for evaluation of abnormal physical exam findings or history suggesting further evaluation.

Diagnostic Procedures/Surgery

Any diagnostic procedures considered should be reserved for evaluation of abnormal physical exam findings or history suggesting further evaluation.

DIFFERENTIAL DIAGNOSIS

- Infection:
 - Bacteremia
 - Urosepsis
 - Pneumonia

- Viral syndromes
- Abscess
- Epstein–Barr virus, monospot
- Cytomegalovirus
- HIV
- Human herpesvirus 6
- Immunologic/connective tissue:
 - Rheumatologic (rheumatoid arthritis, systemic lupus erythematosus, juvenile rheumatoid arthritis)
 - Osteoarthritis
 - Fibromyalgia
 - Myasthenia gravis
 - Lambert–Eaton syndrome
- Neoplastic:
 - Solid or hematologic cancers
- Metabolic:
 - Electrolyte abnormalities
 - Mitochondrial diseases
 - Bromism
- Hematologic:
 - Anemia
 - Hypovolemia
 - Hemoglobinopathy
- Endocrine:
 - Hyperthyroid or hypothyroid
 - Adrenal insufficiency
 - Diabetes
 - Hypoglycemia
- Neurologic:
 - Multiple sclerosis
 - Cerebrovascular accident
 - Amyotrophic lateral sclerosis
- Cardiovascular:
 - MI
 - Cardiomyopathy
 - CHF
- Pulmonary:
 - Pneumonia
 - Chronic obstructive pulmonary disease
 - Asthma
 - Sleep apnea
- GI:
 - Reflux

- Peptic ulcer disease
- Liver disease
- Autonomic dysfunction
- Lifestyle:
 - Excessive or insufficient exercise
 - Obesity
- Psychiatric:
 - Major depression
 - Anxiety
 - Grief
 - Stress
- Medication related:
 - Drug interactions
 - Commonly caused by BP, cardiovascular, psychiatric, and narcotic medications
- Dehydration



TREATMENT

PRE HOSPITAL

Evaluate vital signs:

- Collect relevant information that could help psychosocial evaluation.

INITIAL STABILIZATION/THERAPY

- ABCs
- Administer supplemental oxygen for hypoxia.
- IV fluid bolus for signs of dehydration

ED TREATMENT/PROCEDURES

- Treatment should be directed to correction of the underlying cause of fatigue:
 - Identify and treat any infectious process.
 - Correct metabolic and hematologic disturbances.
 - Diagnose progressive neurologic disease and acute psychiatric crisis.
 - Initiate workup for endocrine and neoplastic disease.
 - Stop any offending medications or toxins.
- Most cases will not have identifiable cause, so reassurance and close follow-up is required.
- Recommend appropriate diet, exercise regimen, and consistent sleep cycles.

MEDICATION

First Line

Medication should be reserved for treatment of the underlying cause of symptoms.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Underlying disease requiring IV medication or monitoring
- Failure to thrive as outpatient
- Unable to provide for self

Discharge Criteria

- Able to care for self
- Serious disturbances have been excluded.
- Adequate follow-up is arranged.

Issues for Referral

Most patients who are evaluated for fatigue in the ED should be referred:

- When the cause of a patient's fatigue symptoms have been clearly identified, referral should be directed to the appropriate specialist.
- When the cause of a patient's fatigue symptoms are not clearly identified, a primary care referral is indicated.

PEARLS AND PITFALLS

- Fatigue is a subjective symptom complex, and a complete history and physical exam are needed.
- Beware of patients with unreliable history and physical exam. The elderly, children, intoxicated, and those with decreased mental ability may all have life-threatening disease and present with a complaint of fatigue.

ADDITIONAL READING

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department with non-specific complaints: The Basel Non-specific Complaints (BANC) study. *Acad Emerg Med.* 2010;17(3):284–292.



CODES

ICD9

- 729.1 Myalgia and myositis, unspecified
- 780.71 Chronic fatigue syndrome
- 780.79 Other malaise and fatigue

ICD10

- M79.1 Myalgia
- R53.82 Chronic fatigue, unspecified
- R53.83 Other fatigue

FEEDING PROBLEMS, PEDIATRIC

Richard Gabor • Niels K. Rathlev



BASICS

DESCRIPTION

- Problems may present in 1 or several of the components of “feeding”:
 - Getting food into oral cavity: Appetite, food-seeking behavior, ingestion
 - Swallowing food: Oral and pharyngeal phases
 - Ingestion and absorption: Esophageal swallowing, GI phase
- Acute feeding problems may be a component of acute systemic disease:
 - Infection, bowel obstruction
- Chronic feeding problems may result from underlying neuromuscular, cardiovascular, or behavioral issues:
 - Cerebral palsy, prematurity, congenital heart disease, chronic neglect
- Minor feeding difficulties reported in 25–50% of normal children:
 - Mainly colic, vomiting, slow feeding, and refusal to eat
- More severe problems observed in 40–70% of infants born prematurely or children with chronic medical conditions.

ETIOLOGY

- Several distinct areas of pathology—but overlap is common
- Structural abnormalities:
 - Naso-oropharynx:
 - Cleft lip/palate
 - Choanal atresia
 - Micrognathia and/or Pierre Robin sequence
 - Macroglossia
 - Tonsillar hypertrophy
 - Retropharyngeal mass or abscess
 - Larynx and trachea:
 - Laryngeal cleft or cyst
 - Subglottic stenosis
 - Laryngo- or tracheomalacia
 - Tracheoesophageal fistula
 - Esophagus:
 - Esophageal strictures, stenosis, or web
 - Tracheoesophageal compression from vascular ring/sling
 - Esophageal mass or tumor
 - Foreign body
- Neurologic conditions:

- Cerebral palsy
- Muscular dystrophies
- Mitochondrial disorders
- Arnold–Chiari malformation
- Myasthenia gravis
- Brainstem injury
- Pervasive developmental disorder (autism spectrum disorders)
- Infant botulism
- Brainstem glioma
- Polymyositis/dermatomyositis
- Prematurity
- Immune disorders:
 - Allergy
 - Eosinophilic esophagitis
 - Celiac disease
- Congenital heart disease:
 - Precorrection: Fatigue, respiratory compromise, increased metabolic needs
 - Postcorrection: Any/all of the above, recurrent laryngeal nerve injury
- Chronic aspiration
- Conditioned dysphagia:
 - Gastroesophageal reflux (GER)
 - Prolonged tube or parenteral feeding early in life
- Metabolic disorders:
 - Hypothyroidism
 - Inborn errors of metabolism
- Acute illness or event:
 - Sepsis
 - Pharyngitis
 - Intussusception
 - Malrotation
 - Shaken baby syndrome
- Behavioral issues:
 - Poor environmental stimulation
 - Dysfunctional feeder–child interaction
 - Selective food refusal
 - Rumination
 - Phobias
 - Conditioned emotional reactions
 - Depression
 - Poverty (inadequate food available)

SIGNS AND SYMPTOMS

Common presentations:

- Caregiver concerns regarding feeding or postfeeding behavior
- Poor weight gain/failure to thrive
- Recurrent or chronic respiratory illness

History

- Onset of problem
- Length of meals (often prolonged)
- Food refusal/oral aversion
- Independent feeding (if >8 mo):
 - Neuromuscular problems decrease ability to get food to the mouth
- Failure to thrive/poor weight gain
- Recurrent pneumonia/respiratory distress:
 - Most aspiration episodes are silent in infants
 - Recurrent pneumonia or wheezing may be primary symptoms of chronic aspiration
 - Chronic lung disease
- Recurrent vomiting or gagging:
 - If yes, when
- Diarrhea, rectal bleeding
- Onset of irritability or lethargy during feeding, colic
- Duration of feeding highly variable, especially in breast-fed infants—for all ages, feeding times > 30 min on a regular basis is cause for concern:
 - Full-term healthy infant usually has 2–3 oz of formula every 2–3 hr.
 - Breast-fed baby eats 10–20 min on each breast every 2–3 hr.
 - As child gets older, duration and frequency may decrease.
 - 1 mo old normally eats 4 oz every 4 hr.

Physical-Exam

- Vital signs, including oximetry
- Weight, length, head circumference:
 - Comparison with prior measurements; plotting growth curve
 - Slow velocity of growth
 - Impaired nutritional status. Severe cases may show emaciation, weakness, apathy.
- General physical exam—especially note:
 - Affect and social responsiveness
 - Dysmorphism (facial asymmetry, tongue and jaw size, etc.)
 - ENT—oropharyngeal inflammation, infection, or anatomic abnormality
 - Cardiovascular status (murmur, tachycardia, tachypnea, retractions)
 - Pulmonary—tachypnea, color change, evidence of aspiration

- Abdominal exam—bowel sounds, distension, tenderness, masses
- Neurologic—tone, coordination, alertness
- Skin: Allergic rash or atopy:
 - Loss of subcutaneous fluid or fat is often most apparent around the eyes, which will appear “sunken” in most dehydrated or malnourished infants
 - Edema, however, may occur with protein deficiency (kwashiorkor).
- Observation of feeding: Neuromuscular tone, posture, position; patient motivation; oral structure and function; efficiency of oral intake:
 - Ability to handle oral secretions
 - Pace of feeding
 - Noisy airway sounds after swallowing
 - Gagging, coughing, or emesis during feeding
 - Respiratory distress with feeding
 - Oximetry during feeding may be helpful
 - Onset of fatigue or irritability
 - Duration of feeding

ESSENTIAL WORKUP

- A well-hydrated, comfortable child with a normal physical exam and recent history of good weight gain may not need any ED workup beyond assuring good follow-up.
- Children who show evidence of distress, dehydration, discomfort, respiratory distress, or poor weight gain require further evaluation.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Initial assessment if child failing to thrive or appears malnourished:
 - CBC, urinalysis, electrolytes, BUN, glucose, erythrocyte sedimentation rate (ESR) and/or CRP, thyroid functions, LFTs, total protein, and albumin
- Cultures of blood, urine, if concern of infection—CSF analysis and culture if concern for meningitis
- Serum NH₃, urine for organic acids, and blood for inborn errors or metabolism if concern for metabolic disorders

Imaging

- CXR if suspected cardiopulmonary concerns
- EKG if cardiac disease suspected
- Referral or admission for ultrasound and other imaging studies as indicated. Fiberoptic or videofluoroscopic evaluation of swallowing may be needed.
- MRI if concerns for brainstem, skull base, or spinal problems

Diagnostic Procedures/Surgery

- May need a multidisciplinary evaluation involving speech pathologist, pediatrician, and potentially an otolaryngologist.
- Surgical correction of specific pathology

DIFFERENTIAL DIAGNOSIS

Feeding disorder encompasses symptoms observed as a final pathway for many disorders.

Specific clues to the etiology may include:

- Prolonged feeding, fatigue:
 - Consider cardiac disease.
- Recurrent pneumonias:
 - Consider chronic aspiration.
- Stridor with feeds:
 - Consider glottic or subglottic anomalies.
- Suck–swallow–breathing coordination:
 - Consider nasal congestion, choanal atresia.
- Vomiting, diarrhea, abdominal pain, colic:
 - Consider allergy or GER.



TREATMENT

PRE HOSPITAL

- Assess vital signs and hydration; resuscitate as necessary.
- Assess for and treat hypoglycemia.

INITIAL STABILIZATION/THERAPY

- Cardiovascular/respiratory/fluid resuscitation as needed
- Assess for and treat hypoglycemia if suspected.

ALERT

- Certain inborn errors of metabolism (glycogen storage diseases) can cause profound hypoglycemia if unable to take PO feeds—if known or suspected, IV dextrose should be started immediately
- Bilious vomiting in a young infant may be a sign of malrotation with volvulus causing intestinal ischemia—this requires emergent surgical consultation.

ED TREATMENT/PROCEDURES

- Treat dehydration if present:
 - Oral rehydration if practical
 - IV if PO contraindicated, not tolerated, or impractical
- Ondansetron for acute vomiting
- Treat respiratory distress if present:

- Nasal suction to clear secretions prior to feeding may be very helpful in young infants with URI/bronchiolitis symptoms
- Oxygen and other interventions as needed
- Treat infection if suspected.

ALERT

Patients with severe malnutrition are at risk for sepsis AND may have blunted physiologic responses—a high index of suspicion for infection is warranted in severely malnourished patients.

MEDICATION

Ondansetron: 0.1 mg/kg IV or PO q8h PRN nausea or vomiting—min. oral dose 2 mg, max. dose 4 mg:

- Monitor if patient at risk of QT prolongation
- For short-term use (2–3 doses) in patients >6 mo.
- Review FDA black box warning re QT prolongation



FOLLOW-UP

DISPOSITION

Admission Criteria

- Suspected systemic infection
- Inability to maintain hydration
- Sustained hypoxia during feeding
- Significant failure to thrive:
 - Particularly in infants <3 mo
- Decompensated cardiopulmonary disease
- Symptomatic anemia or endocrine dysfunction
- Negligent or overwhelmed caretaker

Discharge Criteria

- Demonstrated ability to tolerate oral feedings
- Weight gain if failure to thrive
- Reliable caretaker and follow-up

Issues for Referral

- Specific referrals based on source of problem
- For complex or chronic feeding problems, a multidisciplinary approach is often needed.
- Chronic disease process may interfere with feeding AND increase caloric needs:
 - Nonoral nutrition such as percutaneous endoscopic gastrostomy (PEG) tubes

are often needed to address these issues.

FOLLOW-UP RECOMMENDATIONS

- When available, a primary provider is the most important resource for follow-up.
- In the case of complex problems, a multidisciplinary approach is often needed—the primary provider is often in the best position to coordinate this.

PEARLS AND PITFALLS

- Successful feeding in infants requires coordinated, effective interaction of complex physiologic, developmental, and environmental factors.
- The factors are interdependent—disruption of 1 often leads to disruption of others:
 - Premature infant gavage fed for immature suck–swallow coordination, misses critical period for developing this reflex—develops aversion to oral stimulus because of recurrent noxious stimuli.
- Feeding problems of recent, acute onset are likely to have a single identifiable cause:
 - Gastroenteritis, pyloric stenosis, pharyngitis, sepsis
- In an infant with upper respiratory symptoms the answer may be as simple as vigorously suctioning the nose to effectively clear it immediately before feeding
- More chronic, long-term problems are more likely to have multifactorial and/or subtle causes:
 - Feeding is an essential part of the parent–child interaction:
 - Dysfunctional interaction may be the cause of or a response to a feeding problem.
- Chronic feeding issues of medical origin may result in continued behavioral feeding difficulties even after the medical problem is corrected.
- Swallowing disorders and aspiration are frequently occult.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Failure to Thrive
- Feeding Tube Complications
- Inborn Errors of Metabolism

- Intussusception
- Irritable Infant
- Malrotation
- Pyloric Stenosis
- Vomiting, Pediatric



CODES

ICD9

- 779.31 Feeding problems in newborn
- 783.0 Anorexia
- 783.3 Feeding difficulties and mismanagement

ICD10

- P92.9 Feeding problem of newborn, unspecified
- R63.0 Anorexia
- R63.3 Feeding difficulties

FEEDING TUBE COMPLICATIONS

Colleen N. Hickey • Jennifer L. Kolodchak



BASICS

DESCRIPTION

- Extubation:
 - Accidental or intentional
 - More common with nasoenteric tubes compared with percutaneous endoscopic gastrostomy (PEG) tubes, gastrostomy tubes (G tubes), or jejunostomy tubes (J tubes)
- Occlusion:
 - Small diameter:
 - Most common with nasoenteric tubes
 - Pill fragments
 - Inadequate flushing
 - Physical incompatibilities between formula and medications:
 - Adherence of formula residue to inner wall
 - Essential to rule out malposition, fracture, and dislodgment
- Peristomal wound infections:
 - Risk factors:
 - Malnutrition
 - Stomal leak
 - Local irritation
 - Poor wound care
 - Immunosuppression
 - Diabetes mellitus
 - Poor wound healing
 - Obesity
 - Excessive traction on tube:
 - Leads to delayed maturation of gastrocutaneous tract
 - Increases stomal leakage
- Stoma leak:
 - Problematic with distal obstruction (mechanical or dysmotility); more common with high gastric residual
 - Excessive tube motion
- Aspiration pneumonia:
 - At risk:
 - Impaired cough/gag reflex
 - Delayed gastric emptying from ileus
 - Obstruction

- Gastroparesis
- Gastroesophageal reflux (frequent with large nasoenteric tube)
- Diarrhea:
 - _ Medication induced:
 - Antibiotics
 - Promotility agents
 - _ Overgrowth of *Clostridium difficile*, other bacteria, or *Candida*
 - _ High osmolar formula
- Feeding intolerance:
 - _ High residual suggests GI motility dysfunction
 - _ Delivery is too rapid
 - _ High osmolarity formula
 - _ Lactose or fat intolerance
 - _ Low serum albumin
- Uncommon complications:
 - _ Abdominal wall hematoma
 - _ Fistulas:
 - Hepatogastric
 - Gastrocolic
 - Colocutaneous
 - _ Perforation (usually with placement)
 - _ Pressure sores/ulcerations
 - _ GI bleeding
 - Esophagitis, gastric pressure ulcers, concomitant PUD
 - _ Gastric outlet obstruction
 - Partial or complete obstruction at the pylorus or duodenum by part of tube or Foley catheter balloon used for temporary replacement
 - _ Buried bumper syndrome
 - Rare but potentially serious
 - Bumper becomes lodged between the gastric wall and skin due to gastric ulceration from excessive tension
 - _ Bowel volvulus around PEG tube

Pediatric Considerations

Increased risk of aspiration:

- Delayed gastric emptying
- Immaturity of lower esophageal sphincter

DIAGNOSIS

SIGNS AND SYMPTOMS

- Extubation:

- Tube removed from source
- Occlusion:
 - Unable to pass liquid through tube
- Tube migration:
 - Distal displacement of PEG tube
 - Obstruction at or distal to pylorus
 - Dumping syndrome
 - Ischemia
 - Intussusception
 - Evidence of distal prolapse on external tube (if marked)
- Peristomal wound infections:
 - Cellulitis
 - Abscess formation
 - Necrotizing fasciitis
- Stoma leak:
 - Leakage of feedings/GI tract contents around stoma
- Aspiration pneumonia:
 - Cough
 - Dyspnea
 - Hypoxia
 - Food particulate in pulmonary secretions
 - Fever
- Misplacement of nasoenteric tube in pulmonary tree:
 - Pneumothorax
 - Hydrothorax
 - Pleural effusion
 - Bronchopleural fistula
 - Pneumonia
- Diarrhea:
 - Frequent loose stools
 - Dehydration
- Intolerance to enteral nutrition:
 - High residuals
 - Associated with increased risk of aspiration

ESSENTIAL WORKUP

- Carefully examine the tube site and position of feeding tube within wound
- For suspected tube migration, obtain a water-soluble contrast radiography of the tube to establish the tube position within the abdomen/stomach/intestine

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Peristomal wound infections:
 - CBC for significant infections
 - Blood culture if systemically ill
- Aspiration pneumonia:
 - Pulse oximetry or arterial blood gas
 - CBC
 - Electrolytes, BUN/creatinine, glucose
 - Blood and sputum culture
- Diarrhea:
 - Stool for white blood cells/culture/*C. difficile* toxin
- GI bleeding
 - Serial CBC

Imaging

- CXR:
 - Nasoenteric tube position
 - Aspiration pneumonia
- Water-soluble contrast radiography for suspected tube migration

Diagnostic Procedures/Surgery

Endoscopy to evaluate for tube migration



TREATMENT

PRE HOSPITAL

ALERT

If extubation of tube has occurred, transport tube with patient to facilitate easier replacement

INITIAL STABILIZATION/THERAPY

- ABCs
- IV fluid resuscitation for dehydration/sepsis

ED TREATMENT/PROCEDURES

Extubation

- Nasoenteric tube:
 - Replace in emergency department
 - Confirm position by radiograph before use
- PEG tube and gastrojejunal (G-J) tube:
 - Takes 4–6 wk for gastrocutaneous tract/fistula to mature

- Improper or aggressive attempt at tube replacement could lead to disruption of gastrocutaneous tract and subsequent peritonitis
- PEG tube in place >4 wk:
 - Replace in emergency department (may use a Foley catheter of equivalent size)
 - Confirm by water-soluble radiographic study
 - Secure catheter to abdominal wall to prevent distal migration
- PEG tube in place <4 wk:
 - Do not replace in ED
 - Risk of intraperitoneal placement
 - May need hospital admission and endoscopic tube replacement
- Surgical G tube or J tube:
 - Management similar to that for PEG tube
 - Early dislodgment within 1st 3 days requires emergency surgical consult and antibiotic coverage for peritonitis
 - May need endoscopic replacement if <4 wk old

Occlusion

- Attempt gentle irrigation with NS, water, carbonated soda, pancreatic enzymes
- If irrigation fails, replace tube
- Do not use meat tenderizer

Tube Migration

- If retraction of tube is possible and well tolerated:
 - Secure tube externally
 - Discharge home after brief trial of tube feeding
- If feeding is not tolerated, or if there are signs of persistent obstruction or peritonitis:
 - Admit with consult to appropriate service (surgical/GI).
- If external tube is cut (accidental or intentional) and the inner tube is within the abdomen:
 - Inner bumper usually passes through GI tract
 - Cases of obstruction, subsequent perforation, and peritonitis have been reported, especially in children

Peristomal Wound Infections

- Local wound care
- Antibiotics:
 - 1st-generation cephalosporin (cefazolin or cephalexin)
 - Ampicillin/sulbactam
 - Amoxicillin/clavulanic acid
 - Clindamycin (penicillin allergic)

- Outpatient management for milder cases
- More severe cases require surgical consult for possible drainage/debridement and inpatient care
- Prophylactic use of antibiotic (cefazolin) before tube placement decreases wound infection (3% vs. 18%)

Peristomal Leak

- Change from intermittent to continuous delivery
- Decrease rate of infusion
- Optimize nutritional status
- Relieve excess tension on tube
- Administer prokinetic agents (e.g., metoclopramide)
- Do NOT place larger tube
- Local care:
 - _ Keep site clean and dry
 - _ Barrier creams

Aspiration Pneumonia

- Stop enteral feeding
- Administer oxygen and broad-spectrum antibiotics
- Endotracheal intubation with mechanical ventilation for respiratory failure and airway protection when indicated
- Prevent by:
 - _ Elevation of head of bed
 - _ Monitoring gastric residual
 - _ Use of continuous infusion at graduated rate
 - _ Use of prokinetic agent

Diarrhea

- Manage cause
- Correct fluid and electrolyte imbalance
- Try isotonic, hypotonic, or fat- or lactose-free formulas
- High-fiber formula if above measures fail
- Antimotility agents:
 - _ Loperamide
 - _ Kaopectate
 - _ Cholestyramine

Formula Intolerance

Prokinetic agents promote gastric emptying

MEDICATION

- Amoxicillin/clavulanic acid (Augmentin): 500–875 mg (peds: 25–45 mg/kg/24 h) PO q12h
- Ampicillin/sulbactam: 1.5–3 g (peds: 100–200 mg/kg/24 h) IV q6h
- Cefazolin (Ancef, Kefzol): 500 mg–1 g (peds: 25–100 mg/kg/24 h) IV q6h
- Cephalexin (Keflex): 250–500 mg (peds: 25–50 mg/kg/24 h) PO q6h
- Cholestyramine: 2–4 g (peds: >6 yr 80 mg/kg q8h) PO q6–12h
- Clindamycin: 150–300 mg (peds: 5–10 mg/kg) IV q6h
- Kaopectate: 30 mL (peds: 3–6 yr old, 7.5 mL; 6–12 yr old, 15 mL) PO after each loose bowel movement up to 7 times per day
- Loperamide (Imodium): 4 mg initially, then 2 mg (peds: 1 mg q8h if 13–20 kg; 2 mg q12h if 20–30 kg; 2 mg q8h, if >30 kg not to exceed 9 mg/d) PO up to 16 mg/d
- Metoclopramide: 5–10 mg (peds: 0.1–0.2 mg/kg to max. 0.8 mg/kg/d) PO/IV/IM q6h (30 min before feeds and every night)



FOLLOW-UP

DISPOSITION

Admission Criteria

- PEG tube extubation within 1 wk of placement
- Surgical G tube or J tube extubation within 3 days of placement
- Significant peristomal wound infection
- Aspiration pneumonia
- Diarrhea associated with dehydration
- Active GI bleeding
- Peritonitis

Discharge Criteria

Successful replacement of extubated feeding tube

Issues for Referral

GI consult or surgical consult for feeding tube replacement when cannot be placed successfully in the emergency department

FOLLOW-UP RECOMMENDATIONS

Primary care or GI follow-up for recurrent feeding tube complications

PEARLS AND PITFALLS

- Radiography should be used to confirm placement of all feeding tubes
- Do not attempt replacement of a newly placed PEG tube, G tube, or J tube in the

ADDITIONAL READING

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- Stayner JL, Bhatnagar A, McGinn AN, et al. Feeding tube placement: Errors and complications. *Nutr Clin Pract*. 2012;27(6):738–748.



CODES

ICD9

- 536.40 Gastrostomy complication, unspecified
- 536.49 Other gastrostomy complications
- 996.79 Other complications due to other internal prosthetic device, implant, and graft

ICD10

- K94.20 Gastrostomy complication, unspecified
- T85.518A Breakdown (mechanical) of other gastrointestinal prosthetic devices, implants and grafts, initial encounter
- T85.528A Displacement of other gastrointestinal prosthetic devices, implants and grafts, initial encounter

FEMUR FRACTURE

Alexander D. Miller

BASICS

DESCRIPTION

Fractures classified according to:

- Location:
 - Proximal 3rd (subtrochanteric region)
 - Middle 3rd
 - Distal 3rd (distal metaphyseal–diaphyseal junction)
- Geometry:
 - Spiral
 - Transverse
 - Oblique
 - Segmental
- Extent of soft tissue injury:
 - Open
 - Closed
- There are 2 commonly accepted classification systems of femoral fractures: The AO/OTA and the Winquist and Hansen.
- Degree of comminution: Winquist and Hansen classification:
 - Grade I: Fracture with small fragment < 25% width of femoral shaft; stable lengthwise and rotationally
 - Grade II: Fracture with 25–50% width of femoral shaft; stable lengthwise; may or may not have rotational stability
 - Grade III: Fracture with > 50% width of femoral shaft; unstable lengthwise and rotationally
 - Grade IV: Circumferential loss of cortex; unstable lengthwise and rotationally

ETIOLOGY

- Usually requires major, high-energy trauma
- Patients are mostly young adults with high-energy injuries (motor vehicle accidents [MVAs], gunshot wounds [GSWs], falls):
 - Spiral fractures with falls from height
- Consider pathologic fracture if minor mechanism
- Can occasionally be due to stress fracture from repetitive activity
- Complications include compartment syndrome, fat embolism, adult respiratory distress syndrome (ARDS), hemorrhage.

Geriatric Considerations

- Atypical femur fractures have been associated with use of bisphosphonate medications.

Pediatric Considerations

- 70% of femoral fractures in children < 3 yr old are the result of nonaccidental trauma (NAT).
- Spiral fractures of the femur strongly suggest NAT.

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Thigh pain, deformity, swelling, shortening
- Patient unable to move hip or knee
- Commonly presents as multitrauma:
 - _ Chest, abdominal, pelvic, hip, knee injury, including dislocation

Physical-Exam

- Rarely open fracture, unless injury is due to penetrating trauma
- Patient may be hypotensive due to hemorrhage into the thigh.
- Patient may have impaired circulation in the distal leg due to vascular compromise, compartment syndrome.

ESSENTIAL WORKUP

- Radiographs (see Imaging)
- Assess distal pulses, palpate compartments, evaluate sensation and motor function.
- If pulses are not equal or palpable, bedside Doppler or angiography may be necessary.
- Search for associated injuries with multisystem trauma.
- In suspected NAT, obtain skeletal survey or bone scan.

DIAGNOSIS TESTS & INTERPRETATION

Lab

CBC, type and cross-match

Imaging

- AP pelvis, true lateral of the hip, AP and lateral views of the femur, complete knee series

- Baseline CXR, other films as indicated by trauma protocols

DIFFERENTIAL DIAGNOSIS

- Hip fracture or dislocation
- Knee fracture or dislocation
- Thigh contusion or hematoma



TREATMENT

PRE HOSPITAL

- Immobilization of the extremity and application of a traction splint can be important for tamponade of further blood loss into the thigh:
 - _ Backboard immobilization, rigid splinting, support of extremity for position of comfort
- Contraindications to traction:
 - _ Fractures close to the knee
 - _ Fracture or dislocation of the ipsilateral hip
 - _ Fractures of the pelvis
 - _ Fractures of the lower leg
- Do not attempt to reduce open fractures in the field; cover open wounds with sterile dressings.
- Monitor closely for development of hemorrhagic shock, as thigh can contain 4–6 U of blood.

INITIAL STABILIZATION/THERAPY

- Airway, chest, abdominal injuries take precedence.
- Monitor BP continuously for signs of hemorrhagic shock.

ED TREATMENT/PROCEDURES

- Maintain lower extremity stability.
- Remove splint and clothing.
- Pain control:
 - _ Isolated femur injuries: Parenteral analgesia
 - _ Multitrauma or pediatric patients: Femoral nerve block
- Orthopedic consultation necessary for all femur fractures:
 - _ Emergent if neurovascular compromise
 - _ Open fractures must go directly to the OR for irrigation and débridement.
- Antibiotics:
 - _ Fractures requiring surgery: Cefazolin if open fracture with laceration (clindamycin if allergic to cephalosporins). If extensive soft tissue damage or contamination: Consider gentamicin/tobramycin, tetanus.
 - _ If highly contaminated wound: Consider penicillin G to cover clostridial species.

- Femur fractures with diminished or absent distal pulses, an expanding hematoma, or a palpable pulsatile mass require immediate angiography or femoral artery exploration.
- Skeletal traction should be applied if the patient will not go to the OR immediately.

MEDICATION

- Antibiotics:
 - First line:
 - Cefazolin: 2 g IM/IV q6–8h (peds: 50–100 mg/kg IM/IV divided q6–8h max. 1 g)
 - Second line:
 - Clindamycin: 450–900 mg IM/IV q6–8h; max. dose: 4.8 g/d (peds: 20–40 mg/kg/d IM/IV in 3–4 divided doses)
- Moderate sedation:
 - Etomidate: 0.1–0.3 mg/kg IV once (not recommended for <12 yr)
 - Fentanyl: 50–100 µg IV over 1–2 min once (peds: >6 mo 1–2 µg/kg IV once)
 - Ketamine: Caution in adults due to potential for emergence reaction (peds: 0.2–1 mg/kg IV, 0.5–4 mg/kg IM once)
 - Midazolam: 0.07 mg/kg IM or 1 mg slow IV q2–3min up to 5 mg max. (peds: 0.25–0.5 mg/kg PO once to a max. of 20 mg PO; 6 mo–5 yr: 0.05–0.1 mg/kg IV titrate to max. of 0.6 mg/kg; 6–12 yr: 0.025–0.05 mg/kg IV titrate to max. of 0.4 mg/kg)
 - Propofol: 40 mg IV q10sec until induction; 5–60 µg/kg/min IV continuous infusion
- Pain control:
 - Hydromorphone: 0.5–2 mg IM/SC/slow IV q4–6h PRN; titrate for pain control (peds: 0.015 mg/kg per dose IV q4–6h PRN)
 - Morphine: 2–10 mg IV q4h; titrate for pain control (peds: 0.1 mg/kg IV q4h; titrate for pain control to max. 15 mg/dose)

Pediatric Considerations

- Assess markers for NAT:
 - Delay in presentation
 - History of mechanism inconsistent with the injury
 - Isolated trauma to the thigh, associated burns, bruises, or linear abrasions
- Assess for dislocation of the femoral capital epiphysis.
- Depending on the age of the patient and the fracture type, pediatric femoral fractures may not require operative treatment.



FOLLOW-UP

DISPOSITION

Admission Criteria

- All femur fractures must be admitted except as noted below in Discharge Criteria.
- Any suspicion of NAT in children

Discharge Criteria

In certain rare circumstances of pathologic fracture or femur fractures in patients who are not ambulatory and would not undergo operative fixation, discharge can be considered in consultation with orthopedics if adequate pain control can be achieved and proper follow-up ensured.

FOLLOW-UP RECOMMENDATIONS

Follow-up will likely be determined by operating surgeon based on clinical course.

PEARLS AND PITFALLS

- Due to the high-energy mechanism required to incur a femoral fracture, other associated traumatic injuries must be ruled out.
- Document neurovascular function on initial assessment and frequently reassess.
- Depending on the age of the patient and the fracture type, pediatric femoral fractures may not require operative treatment.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Hip Injury



CODES

ICD9

- 820.22 Closed fracture of subtrochanteric section of neck of femur
- 821.00 Closed fracture of unspecified part of femur
- 821.01 Closed fracture of shaft of femur

ICD10

- S72.26XA Nondisplaced subtrochanteric fracture of unsp femur, init
- S72.90XA Unsp fracture of unsp femur, init enctr for closed fracture
- S72.309A Unsp fracture of shaft of unsp femur, init for clos fx

FEVER, ADULT

Matthew L. Wong



BASICS

DESCRIPTION

- Fever is an elevation of core body temperature caused by an increase in the body's thermoregulatory set point.
- Prostaglandin E2 (PGE2) synthesis in the anterior hypothalamus controls the thermostat, and is the target of antipyretics.
- Core temperature is regulated to $37^{\circ}\text{C} \pm 2^{\circ}\text{C}$.
- Autonomic discharge from hypothalamus can raise core temperature through shivering and dermal vasoconstriction.
- Normal circadian variation in core temperature occurs with nadir in early morning and peaks in late afternoon.
- Fever is not synonymous with hyperthermia or hyperpyrexia.
- Hyperthermia is an elevated temperature with normal thermostat set point; caused by excessive endogenous heat production or endogenous production (e.g., malignant hyperthermia or heat stroke).
- Hyperpyrexia is extreme fever $> 41.5^{\circ}\text{C}$ usually from CNS hemorrhages.
- Both exogenous and endogenous factors can raise the body's set thermoregulatory point:
 - Endogenous pyrogens include PGE2, IL-1, IL-6, TNF, IFN- γ .
 - Exogenous pyrogens include lipopolysaccharide (LPS) endotoxin and other TLR ligands, and toxic shock syndrome toxin (TSST-1) and other MHC II ligands.
- Patients on anticytokine medications or glucocorticoids have impaired fever response.
- Fever of unknown origin (FUO):
 - Fever $> 38.3^{\circ}\text{C}$ for at least 3 wk as an outpatient and 3 days of inpatient evaluation or 3 outpatient visits without determining etiology.

ETIOLOGY

- Infectious processes:
 - CNS, chest and lung, gastrointestinal, genitourinary, skin, soft tissue and bone, vascular and endocardial
 - Iatrogenic: Catheters, implants, hardware, recent surgical sites.
- 1° CNS processes such as CVA, trauma, seizures
- Neoplastic fevers
- Drug fever:
 - Most drugs can cause elevated temperatures by a wide variety of

mechanisms

- Toxidromes (e.g., adrenergic, anticholinergic, dopaminergic, salicylate overdose, serotonin toxicity)
- Hypersensitivity:
 - Allergic reaction
 - Serum sickness
- Jarisch–Herxheimer reaction
- Local phlebitis from irritant drugs
- Severe withdrawal:
 - Alcohol
 - Benzodiazepines
- Systemic rheumatologic and inflammatory diseases (e.g., familial Mediterranean fever, rheumatoid arthritis, sarcoidosis, systemic lupus erythematosus, temporal arteritis)
- Endocrine:
 - Hyperthyroidism, pheochromocytoma
- Miscellaneous:
 - Alcoholic cirrhosis
 - Acute inhalation exposures (e.g., metal fume fever)
 - Cotton fever:
 - Febrile reaction from an injected contaminant when IV drug abusers strain drug through cotton
 - Sickle cell disease
 - Hemolytic anemia
 - Pulmonary embolus
- Common causes of FUO:
 - Infectious:
 - Abdominal and pelvic abscesses
 - Cardiac (endocarditis, pericarditis)
 - Cat scratch disease
 - Cytomegalovirus
 - Epstein–Barr virus
 - TB (miliary, renal, or meningitic)
 - Typhoid enteric fevers
 - Visceral leishmaniasis
 - Neoplastic:
 - Colon adenocarcinoma
 - Hepatocellular carcinoma and metastases
 - Myeloproliferative disorders
 - Leukemia and lymphoma
 - Renal cell carcinoma

SIGNS AND SYMPTOMS**History**

- Chills, shivering, and rigors:
 - Rigors may suggest bacteremia
- Weight loss:
 - Suggestive of neoplastic, chronic infectious, or endocrine disorders
- Night sweats:
 - Suggestive of neoplastic, chronic inflammatory disease, or TB
- Specific fever patterns:
 - Daily morning temperature spikes:
 - Miliary TB, typhoid fever, polyarteritis nodosa
 - Relapsing fevers: Febrile episode with alternating afebrile intervals:
 - Seen in malaria, *Borrelia* infections, rat-bite fever, and lymphoma
 - Remittent fever: Temperature falls daily but does not return to normal:
 - Seen in TB and viral diseases
 - Intermittent fevers: Exaggerated circadian rhythm:
 - Seen in systemic infections, malignancy, and drug fever
 - Double quotidian fever:
 - Common pattern of 2 temperature spikes in 24 hr
 - In FUO, consider miliary TB, visceral leishmaniasis, and malarial infections
- High-risk features:
 - Anticytokine therapy (e.g., TNF- α monoclonal antibodies, calcineurin inhibitors)
 - Glucocorticoid use
 - Immunosuppressed states
 - Incomplete vaccination status
 - IV drug use
 - Pregnancy and peripartum patients
 - Rash
 - Recent chemotherapy
 - Recent travel
 - Splenectomy

Physical-Exam

- Elevated core temperature:
 - Temperature $>38^{\circ}\text{C}$ (100.4°F) rectally or 37.5°C (99.5°F) orally
 - Lower thresholds in patients older than 65 yr, as the febrile response is not as strong

- Diaphoresis:
 - Absence of diaphoresis with severe hyperthermia suggests anticholinergic poisoning or heat stroke.
- Tachycardia:
 - For each degree of elevation in temperature in Fahrenheit, there should be a 10 bpm increase in pulse.
 - Relative bradycardia (Faget sign):
 - Associated with malaria, typhoid fever, CNS disorders, lymphoma, drug fever, brucellosis, ornithosis, Legionnaire disease, Lyme disease, and factitious fevers
- Muscle rigidity, clonus, and hyper-reflexia:
 - Associated with specific toxidromes and medical conditions
- Changes in mental status:
 - Toxic–metabolic encephalopathy vs. primary CNS disorder
- Rash:
 - Lesion type, distribution, and progression can offer important clues to diagnosis.
 - Petechia, purpura, vesicles, mucosal, or palm and sole involvement require special note
- Signs of hyperthyroidism:
 - Goiter
 - Exophthalmos

ESSENTIAL WORKUP

- Core temperature is most acutely measured rectally.
- Careful history and physical exam (PE) necessary to determine need for further diagnostic testing:
 - History should elicit any sick contacts, previous infections, occupational exposures, recent travel, medications, animal or tick exposure, and immunization status.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC:
 - Important in determining neutropenia in patients with risk factors
 - Neutrophilia and bandemia suggestive of bacterial infection
 - Lymphocytosis suggestive of typhoid, TB, brucellosis, and viral disease
 - Atypical lymphocytosis seen in mononucleosis, cytomegalovirus, HIV, rubella, varicella, measles, and viral hepatitis
 - Monocytosis suggestive of TB, brucellosis, viral illness, and lymphoma
- Lactate:
 - Initial and repeat measurements useful for screening for sepsis, risk

stratification, and management decisions

- Urinalysis and urine culture
- Blood cultures:
 - Obtain for all systemically ill patients, and patients at risk for bacteremia
- Thick and thin blood smears and malaria antigen testing in at-risk individuals for parasitic and intraerythrocytic infections
- Stool culture and *Clostridium difficile* assay for suspected individuals.
- Heterophile antibody testing in select patients.
- Erythrocyte sedimentation rate and C-reactive protein generally not useful:
 - Very high values suggestive of endocarditis, osteomyelitis, TB, and rheumatologic conditions.

Geriatric Considerations

- Decreased immunocompetence, increased risk of systemic spread, increased exposure to health care settings, may have comorbid conditions.
- If institutionalized consider the infectious implications of multiple potential sick contacts.

Imaging

- CXR:
 - In patients with PE finding of cardiopulmonary disease and patients with unclear fever source
- CT or MRI may be indicated if lumbar puncture or osteomyelitis is considered, respectively.

DIFFERENTIAL DIAGNOSIS

- The differential diagnosis is very broad as listed above, but is generally categorized as infectious vs. noninfectious, and by immunocompetency.



TREATMENT

PRE HOSPITAL

- No specific field interventions required
- Monitoring and IV access should be obtained in the field for unstable patients or patients with altered mental status.

INITIAL STABILIZATION/THERAPY

- ABCs for unstable patients.
- Initiate early broad-spectrum antibiotics for patients with suspected sepsis or unstable vital signs, particularly those who are at high risk for serious bacterial infection.

ED TREATMENT/PROCEDURES

- Antipyretics:
 - Generally either acetaminophen or NSAIDs
 - Inhibit the cyclooxygenase enzyme, thereby blocking synthesis of prostaglandins.
- Empiric antibiotics for neutropenic patients:
 - Combination therapy:
 - Extended spectrum β -lactam (ceftazidime, piperacillin) with an aminoglycoside
 - Monotherapy:
 - Cefepime
 - Ceftazidime
 - Imipenem
- Empiric antibiotics for asplenic patients for encapsulated bacteria
- Empiric antiviral therapy for patients with encephalitis and potential disseminated viral infections (e.g., recent organ or bone marrow transplant patients, AIDS patients)
- External cooling mechanism rarely indicated

MEDICATION

- Antipyretics:
 - Acetaminophen: 650–1,000 mg PO/PR q4–6h; do not exceed 4 g/24h
 - Aspirin: 650 mg PO q4h; do not exceed 4 g/24h
 - Ibuprofen: 800 mg PO q6h
- Antibiotics:
 - Cefepime: 2 g IV q12
 - Ceftazidime: 2 g IV q8
 - Gentamicin or tobramycin (D): 2 mg/kg IV load then 1.7 mg/kg q8h + piperacillin/tazobactam (B) 3.375 g IV q4h or ticarcillin/clavulanate (B) 3.1 g IV q4h
 - Imipenem/cilastatin: 500–1,000 mg IV q8h
 - Meropenem (B): 1 g IV q8h
 - Ciprofloxacin: 750 mg PO BID + amoxicillin/clavulanate (B) 875 mg PO BID
- Antivirals:
 - Herpes simplex virus and varicella-zoster virus (VZV):
 - Acyclovir 10–15 mg/kg IV q8h
 - Influenza A and B:
 - Oseltamivir 75 mg PO q12h



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients with unstable vital signs require ICU admission.
- When identified, the underlying source of the fever usually determines the disposition.
- Certain high-risk groups who have fever without an identifiable source:
 - Neutropenic patients
 - Immunosuppressed or immunocompromised patients
 - Asplenic patients
 - IV drug abusers
- Lower thresholds for admission in patients older than 60 yr and diabetics

Discharge Criteria

Immunocompetent patients with stable vital signs and an identified source of fever or a high suspicion of a nonthreatening viral infection may be safely discharged.

Issues for Referral

The suspected etiology of the fever determines the referral to a primary care physician or a specialist.

FOLLOW-UP RECOMMENDATIONS

Appropriate outpatient treatment and follow-up for further outpatient assessment of the suspected etiology.

PEARLS AND PITFALLS

- Screening lactates for sepsis.
- Early, empiric, and broad-spectrum antibiotic coverage for all septic patients.
- Consider all potential sources of infection.
- Careful consideration for the immunosuppressed, elderly, and IV drug users.

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CODES

ICD9

- 780.60 Fever, unspecified
- 780.61 Fever presenting with conditions classified elsewhere

ICD10

- R50.2 Drug induced fever
- R50.9 Fever, unspecified
- R50.81 Fever presenting with conditions classified elsewhere

FEVER, PEDIATRIC

Nathan W. Mick • David A. Peak



BASICS

DESCRIPTION

- Fever is defined as a temperature of 38°C (100.4°F) rectally:
 - Oral and tympanic temperatures are generally 0.6°C–1°C lower.
- Tympanic temperatures are not accurate in children younger than 6 mo.
- Axillary temperatures are generally unreliable.
- Children who are afebrile but have a reliable history of documented fever should be considered to be febrile to the degree reported.

ETIOLOGY

- Bacteremia (*Haemophilus influenzae* type B, *Streptococcus pneumoniae*), viral illness, often accompanied by exanthem (varicella, roseola, rubella), coxsackievirus (hand-foot-and-mouth disease), abscess:
 - *H. influenzae* type B and *S. pneumoniae* vaccines have reduced incidence of Haemophilus and pneumococcal disease
- CNS: Meningitis, encephalitis
- Head, eyes, ears, neck, and throat (HEENT): Otitis media, facial cellulitis, orbital/periorbital cellulitis, pharyngitis (group A β -hemolytic streptococcus, herpangina, adenovirus pharyngoconjunctival fever), viral gingivostomatitis (herpes and coxsackievirus), cervical adenitis, sinusitis, mastoiditis, conjunctivitis, peritonsillar/retropharyngeal abscess
- Respiratory: Croup (paramyxovirus), epiglottitis, bronchiolitis (respiratory syncytial virus [RSV]), pneumonia, empyema, influenza
- Cardiovascular: Purulent pericarditis, endocarditis, myocarditis
- Genitourinary (GU): Cystitis, pyelonephritis
- GI: Bacterial diarrhea, intussusception, appendicitis, hepatitis
- Extremity: Osteomyelitis, septic arthritis, cellulitis
- Miscellaneous: Herpes simplex virus infection in the neonate, Kawasaki disease, vaccine (DPT) reaction, heat exhaustion/stroke, factitious, familial dysautonomia, thyrotoxicosis, collagen vascular disease, vasculitis, rheumatic fever, malignancy, drug induced, overbundling (uncommon, recheck 15 min after unbundling)



DIAGNOSIS

SIGNS AND SYMPTOMS

- Clinical appearance must be evaluated. Airway, breathing, and circulation (especially dehydration with impaired perfusion/color) need specific evaluation.

- Toxicity associated with lethargy, delayed capillary refill, hypoventilation/hyperventilation, weak cry, decreased PO intake; purpuric or petechial rash, and/or hypotonia. Initial observation is crucial in this evaluation.
- Tachycardia or tachypnea may be the only finding in children with serious underlying condition.
- Fever with a temperature $>38^{\circ}\text{C}$ can raise a child's heart rate by 10 bpm for each degree Fahrenheit.
- Temperature $>40^{\circ}\text{C}$ have been associated with an elevated bacteremia rate in children <24 mo.
- Altered mental status:
 - Lethargy presenting with decreased level of consciousness
 - Irritability
 - Impaired interaction with environment, parents, physician, toys
- Physical exam (PE) to search for underlying condition
- Tachypnea and low oximetry are the most sensitive signs for pneumonia. Also useful are rales, hypoxemia, cough >10 days, and fever >5 days.
- Risk factors for occult UTI include female sex, uncircumcised boys, fever without source, and fever $>39^{\circ}\text{C}$.
- Febrile seizures
- Temperatures $>42^{\circ}\text{C}$ often have a noninfectious cause.
- Serious infection may occur in the absence of fever.
- Antipyretics may change findings without impacting underlying disease. This may be useful in evaluation of patient, esp. with respect to mental status
- $\sim 20\%$ of children will have fever without definable source after history and PE.

ESSENTIAL WORKUP

- Oxygen saturation as mandatory 5th vital sign
- Resuscitate as appropriate.
- Determine duration of illness, degree, pattern and height of fever, use of antipyretics, past medical history, drug allergies, immunization status and history, recent medications/antibiotics, birth history if younger than 6 mo of age, exposures, feeding, activity, urine/bowel habits, travel history, and relevant review of systems.
- Search for underlying condition.
- Initiate antipyretic therapy.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC with differential
- Urinalysis (UA) and culture in all male infants younger than 6 mo, uncircumcised male infants younger than 12 mo, and females younger than 2 yr. Urines for culture should be obtained by catheterization or suprapubic techniques.

- Blood culture:
 - The development of automated blood culture systems has led to more rapid detection of bacterial pathogens.
- CSF for cell counts, Gram stain, culture, protein, and glucose for toxic children and those 0–28/30 days of age; consider for nontoxic-appearing children 28–90 days of age as well as older ones in whom meningitis must be excluded.
- Stool for WBCs and culture when diarrhea present and suggestion of bacterial process
- C-reactive protein (CRP) elevation is commonly found and provides confirmatory data related to the presence of infection. The sedimentation rate (ESR) is also an adjunctive measure.
- Procalcitonin is being used in some settings as additional confirmatory information.

Imaging

- CXR to exclude pneumonia if patient tachypneic or hypoxic
- Other studies as indicated to evaluate for specific underlying infection

DIFFERENTIAL DIAGNOSIS

See “Etiology.”



TREATMENT

PRE HOSPITAL

- Resuscitate as appropriate.
- Begin cooling with antipyretics.

INITIAL STABILIZATION/THERAPY

- Treat any life-threatening conditions.
- Antipyretic therapy
- Evaporative cooling techniques, such as sponge bath, have minimal role.

ED TREATMENT/PROCEDURES

- Focal infections require evaluation and treatment.
- Toxic children require prompt septic workup and appropriate antibiotics.
- All potential life-threatening conditions must be excluded before treating a minor acute illness, which is more common.
- Infants 0–28 days old need a full sepsis workup: CBC, UA, cultures (blood, urine, CSF), lumbar puncture. A CXR should be obtained if there is suspicion for pneumonia:
 - Antibiotics: Ampicillin and either gentamicin or cefotaxime; consider acyclovir for infants at risk for HSV

- Admit
- Well-appearing infants 29–90 days old need workup, selective antibiotic use (ceftriaxone), and re-evaluation within 24 hr:
 - *H. influenzae* type B and *S. pneumoniae* incidence has declined significantly with widespread vaccination.
 - It is currently reasonable to perform CBC, UA, blood culture, and urine culture with selective lumbar puncture, coupled with ceftriaxone IM in low-risk patients (see definition under Disposition) if re-evaluation in 24 hr is ensured. Well-appearing infants 60–90 days of age may be managed without LP or antibiotics selectively.
 - While lumbar puncture is optional in this setting, treatment with empiric antibiotics (ceftriaxone) without lumbar puncture may compromise subsequent re-evaluation.
 - Presence of RSV or influenza in this age group decreases but does not eliminate the risk of bacteremia and meningitis, but the rate of UTI is still appreciable.
- Children 3 mo–3 yr of age are evaluated selectively; those with recognizable viral syndrome (croup, stomatitis, varicella, bronchiolitis) generally do not require workup unless there is toxicity; antibiotic use is individualized for specific identifiable infections and pending appropriate cultures:
 - Well-appearing children with a temperature $> 39^{\circ}\text{C}$ and no identifiable infection should prompt a UA and culture in all male infants younger than 6 mo, uncircumcised male infants younger than 12 mo, and females younger than 2 yr. Urine for culture should be obtained by catheterization or suprapubic techniques
 - Obtaining blood work or performing a lumbar puncture on a child 6 mo–3 yr of age is a clinical decision. Mandatory lumbar puncture in this age group based solely on the presence of fever has not been shown to be cost-effective and is not routinely recommended
 - Children 3–6 mo of age who are incompletely immunized and have WBC $> 15,000/\text{mm}^3$ and no identifiable infection may benefit from empiric antibiotics until preliminary blood cultures are available because of the risk of bacteremia.
 - Widespread immunization for *Pneumococcus* and *H. influenzae* have decreased the incidence of invasive infections by these bacteria.
- Immunocompromised children need aggressive evaluation, as do children with fever and petechiae/purpura or sickle cell disease.
- If methicillin-resistant *S. aureus* is considered, clindamycin or trimethoprim-sulfamethoxazole may be useful.
- Patients with underlying malignancy, central venous catheters, or ventricular peritoneal shunts may have few findings other than fever.

MEDICATION

First Line

- Cefotaxime: 100–150 mg/kg/d IV divided q8h
- Ceftriaxone: 50–100 mg/kg/d IV/IM divided q12h
- Vancomycin: 40–60 mg/kg/d IV divided q6–8h if *S. pneumoniae* suspected until sensitivities defined
- Ampicillin: 150 mg/kg/d IV divided q4–6h
- Gentamicin: 5 mg/kg/d IV divided q8–12h

Second Line

- Acetaminophen: 15 mg/kg per dose PO/PR (per rectum) q4–6h; do not exceed 5 doses/24 h
- Ibuprofen: 10 mg/kg per dose PO q6–8h
- Specific antibiotics for identified or specific conditions



FOLLOW-UP

DISPOSITION

Admission Criteria

- All toxic patients
- Infants 0–28 days of age with temperature $> 38^{\circ}\text{C}$
- Nontoxic infants 29–90 days of age with temperature $> 38^{\circ}\text{C}$ who do not meet low-risk criteria (see definition under Discharge Criteria)
- Patients with fever and petechiae/purpura are generally admitted unless there is a specific nonlife-threatening cause.
- Immunocompromised children
- Poor compliance or follow-up

Discharge Criteria

- Infants 29–90 days of age meeting low-risk criteria:
 - No prior hospitalizations, chronic illness, antibiotic therapy, prematurity
 - Reliable, mature parents with home phone, available transport, thermometer, and living in relative proximity to ED
 - No evidence of focal infection (except otitis media); nontoxic appearing; normal activity, perfusion, and hydration with age-appropriate vital signs
 - Normal WBC ($5\text{--}15,000/\text{mm}^3$), urine (negative Gram stain of unspun urine or leukocyte esterase or < 5 WBC/high power field [HPF]), stool (< 5 WBC/HPF) if performed, and CSF (< 8 WBC/ mm^3 and negative Gram stain) if performed
- Infants 3–36 mo of age who are nontoxic and previously healthy with good follow-up:

– Antipyretics

- Follow-up by phone in 12–24 hr and re-evaluate in 24–48 hr with parental instructions to return if concerns develop or patient worsens.

FOLLOW-UP RECOMMENDATIONS

Patients discharged with fever require close follow-up, usually by their primary care provider and guidelines of when to return with any change or worsening of signs or symptoms.

PEARLS AND PITFALLS

- Fever is the most common presenting complaint in children. It may reflect a life-threatening condition.
- Children under 28 days of age are generally treated empirically, pending culture results.
- Older children need close follow-up and specific discharge instructions.
- Subtle findings such as tachycardia, tachypnea, or altered mental status may be indicative of significant underlying infection.

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CODES

ICD9

- 780.60 Fever, unspecified
- 780.61 Fever presenting with conditions classified elsewhere

ICD10

- R50.9 Fever, unspecified
- R50.81 Fever presenting with conditions classified elsewhere

FIBROCYSTIC BREAST CHANGES

Ryan E. Christensen • David A. Pearson

BASICS

DESCRIPTION

- Generalized term for benign breast changes that are poorly defined
- No longer considered a pathologic disease process as they are found in the majority of healthy women
- Most common of all benign breast conditions
- Changes include:
 - Benign cysts
 - Breast pain (mastalgia or mastodynia), which may or may not be cyclic
 - Diffuse and focal nodularity
 - Palpable fibroadenomas
 - Nipple discharge—may be green or brownish, though usually nonbloody
- Spontaneous, persistent discharge warrants further evaluation
- Occurs in ~60% of women
- Symptoms of pain and tenderness become progressively worse until menopause
- Pain is often most prominent during the premenstrual phase and improves with the onset of menses
 - Breast pain alone is a rare symptom of cancer and accounts for only 0.2–2% of cases
- Synonyms: Adenosis, benign breast disease, cystic mastitis, cystic disease of the breast, fibroadenosis, fibrocystic disease, mammary dysplasia.

ETIOLOGY

- Mechanism of development not well understood
- Likely an enhanced or exaggerated reaction of breast tissue to cyclic levels of female reproductive hormones:
 - May be caused by imbalance of the estrogen to progesterone ratio
 - May occur secondary to increased daily prolactin production
- Most common in women 30–50 yr old
- Pain is most likely caused by rapid expansion of simple cysts
- Symptoms may continue into menopause secondary to hormone replacement therapy
- Incidence is decreased in women taking oral contraceptives
- Risk factors include:
 - Family history of fibrocystic changes
 - Oral contraceptives
 - Hormone replacement therapy

- Increasing age
- Diet (high fat intake; caffeine/methylxanthines)



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Mastalgia and tenderness:
 - Persistent or intermittent
 - Often occurs during premenstrual phase of normal menstrual cycle
 - Usually bilateral
 - Pain may radiate to shoulders and upper arms
- Lumpiness, nodularity:
 - May be localized or generalized
- Increased engorgement and breast density:
 - Breasts described as being dull and heavy
 - Caused by fluctuations in the size of the cystic areas
- Spontaneous or expressible nipple discharge
- Abnormal nipple sensations, including pruritis
- Family history of cysts is common

Physical-Exam

- Palpate the 4 breast quadrants while patient is sitting and then while lying down
- Note any changes from normal, including overlying erythema and warmth that may suggest an alternative diagnosis
- Examine for regional nodes (axillary, clavicular, etc.)
- Fibrocystic changes feel doughy with vague nodularity
- Nodules are typically discrete and mobile
- Usually more marked in the superior and lateral quadrants
- Small groups of cysts often described as palpating a “plate full of peas”
- Large cysts have consistency of a balloon filled with water
- Breast exam is most sensitive 7–9 days after 1st day of menses when the breasts are least congested

DIAGNOSIS TESTS & INTERPRETATION

Lab

- A detailed lab evaluation is usually not necessary in the emergency department
- Prolactin and thyroid-stimulating hormone may be helpful if galactorrhea is present

Imaging

- Ultrasound if < 30 yr old
- Mammography if > 30 yr old
- Ultrasound:
 - Can differentiate cystic from solid masses
 - Benign cysts:
 - Typically demonstrate a uniform outer margin without asymmetry or irregular thickness of the cyst wall
 - Have no central echoes
 - Posterior wall enhancement is normal
 - Can assist in aspiration of deep and nonpalpable cysts
 - Also used to conservatively follow cyst size
 - Performed at a specialized breast center with trained techs and interpreting radiologists
- Mammography:
 - Benign changes may falsely appear malignant
 - Difficult to interpret in women < 30 yr old due to breast tissue density
 - Should be performed either before aspiration or 7–10 days afterward to avoid artifact

Diagnostic Procedures/Surgery

- Fine needle aspiration:
 - Usually performed by a specialist
 - May be performed therapeutically for symptomatic or large masses
 - Allows differentiation between cystic and solid masses
 - Obtain cytology studies to evaluate for malignancy
- Excisional biopsy:
 - Performed by surgeon
 - Indicated for solid lumps that are not proven benign

DIFFERENTIAL DIAGNOSIS

- Benign breast masses:
 - Breast abscess
 - Duct ectasia
 - Mastitis
 - Simple fibroadenomas
 - Solitary papillomas
- Malignant breast masses:
 - Atypical hyperplasia
 - Complex fibroadenomas
 - Diffuse papillomatosis
 - Ductal hyperplasia without atypia
 - Sclerosing adenosis



TREATMENT

ED TREATMENT/PROCEDURES

- Majority of women will not require any medical treatment
- Conservative therapy:
 - Support bra:
 - Reduces tension on supporting ligaments of the breast
 - May reduce inflammatory response and edema
 - Low dose diuretic for 2–3 days before onset of menses
 - NSAIDs
- Dietary changes are somewhat controversial:
 - Restricting dietary fat (to 25% of total calories) and eliminating caffeine
 - Increasing vitamin E and vitamin B₆
 - Herbal preparations such as primrose oil
- Hormonal therapy:
 - Should be initiated by PCP to enable follow-up during course of treatment
 - Oral contraceptives:
 - May decrease symptoms, particularly after 1 yr of use
 - Danazol (synthetic androgen) and Tamoxifen (partial estrogen antagonist):
 - Shown to be equally effective for treatment of severe cyclical mastalgia
 - Use may be limited by side effects (acne, hirsutism, weight gain, teratogenicity)
 - Bromocriptine (inhibits prolactin production):
 - Use also limited by side effects (headache, dizziness, nausea, constipation, weakness)
- Surgical intervention:
 - If a persistent nodule exists, excision is recommended regardless of findings on diagnostic imaging
 - If a large cyst recurs after aspiration on 2 occasions, it should be excised and sent for pathology

MEDICATION

- Bromocriptine: 2.5–5 mg/d BID
- Danazol: 100–400 mg/d BID for 6 mo
- Tamoxifen: 10–20 mg/d
- Oral contraceptive pills (vary)



FOLLOW-UP

DISPOSITION

Discharge Criteria

- Patients with fibrocystic changes are appropriate for discharge with outpatient follow-up
- Encourage patient to keep a breast pain record to determine whether pain is cyclic
- The importance of follow-up should be stressed to ensure patient health, disease prevention, and patient satisfaction
- Encourage regular breast self-exam, annual physical exams, and annual mammograms when appropriate

Issues for Referral

- Practically speaking, all breast masses evaluated in the ED need referral to a primary provider or specialized breast clinic
- Further investigation with imaging and possible biopsy are required for masses that persist throughout menses and are not cyclical
- Referral to a general surgeon may be required in certain cases where tissue biopsy is necessary

PEARLS AND PITFALLS

- Breast cancer may coexist with benign breast disease and fibrocystic changes:
 - _ Consider all cancer risk factors
 - _ Confirm follow-up plan
- Mastitis in a nonlactating patient should be treated as inflammatory carcinoma until proven otherwise
- Fibrocystic changes are usually bilateral; unilateral changes are suspicious for cancer
- Fear of breast cancer is high in all patients:
 - _ Give reassurance that fibrocystic breast changes are not cancerous
 - _ Have a low threshold for referral to a specialist

ADDITIONAL READING

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- Santen RJ, Mansel R. Benign breast disorders. *N Engl J Med*. 2005;353:275–285.



CODES

ICD9

- 610.1 Diffuse cystic mastopathy
- 610.2 Fibroadenosis of breast
- 610.9 Benign mammary dysplasia, unspecified

ICD10

- N60.19 Diffuse cystic mastopathy of unspecified breast
- N60.29 Fibroadenosis of unspecified breast
- N60.99 Unspecified benign mammary dysplasia of unspecified breast

FIBROMYALGIA

Michael P. Wilson • Austin Hopper



BASICS

DESCRIPTION

- Nonarticular, noninflammatory form of muscular and joint pain more common in females:
 - Widespread pain from stimuli that do not normally cause pain (allodynia)
 - > 11 diffuse tender points
 - Fatigue
 - Sleep disturbance
 - Muscle stiffness
 - Difficulties with attention, memory
 - Limited physical findings
- Not diagnosis of exclusion, may occur with other rheumatic diseases

ETIOLOGY

- Mechanism:
 - Painful symptoms believed to result from greater activation of pronociceptive (pain-causing) system relative to antinociceptive (pain-dampening) system in brain and spinal cord.
- Abnormalities identified as possible mechanism:
 - Increased substance P (facilitates pronociception)
 - Decreased biogenic amines (NE, serotonin, dopamine), which facilitate antinociception
 - Decreased gray matter in brain
 - Genetics: 1/3 of patients with fibromyalgia have a close relative who is affected:
 - Candidate genes include 5-HT_{2A}, serotonin transporter, D₄ receptor, others
 - Like many complex diseases, psychological factors play a role, with high incidence of psychiatric disorders.
 - In genetically predisposed individuals, likely starts as initial insult from age, trauma, illness, inflammation, etc.
 - Hypothalamus–pituitary–adrenal axis stress-response dysfunction has been indicated to precede development of fibromyalgia



DIAGNOSIS

SIGNS AND SYMPTOMS

Widespread pain reported above the waist, below the waist, on the left side of the body, and on the right side of the body along with axial skeleton pain:

- Pain reported for > 3 mo

History

- Generalized musculoskeletal pain and morning stiffness
- Weakness and fatigue
- Sleep disturbance
- Muscle spasms
- Persistent fatigue not relieved with rest (consider chronic fatigue syndrome)
- Numbness or tingling in the arms or legs
- Impaired concentration or memory
- Nausea, vomiting
- Abdominal pain or discomfort relieved with bowel movements (consider irritable bowel syndrome)
- Ear pain
- Sinus pressure (consider sinusitis)
- Jaw or face pain (consider TMJ disorder)
- Temple pain (consider temporal arteritis)
- Pelvic or bladder discomfort (consider interstitial cystitis)
- Tension or migraine headaches (consider causes of chronic headache)
- Irritation or itching at introitus (consider vulvodynia)

Physical-Exam

Exam findings usually limited

ESSENTIAL WORKUP

- History is key to diagnosis.
- In the ED, necessary only to distinguish between acute pain from trauma, injury, or new-onset medical conditions and chronic pain, which will require ongoing care and treatment.
- If a diagnosis of fibromyalgia is required, use classification criteria established by American College of Rheumatology (ACR) for fibromyalgia:
 - *Widespread* pain present for at least 3 mo defined as pain on both left and right side of body, above and below waist, and axial skeletal pain (cervical or anterior chest or thoracic spine or low back pain).
 - 11 of the 18 specific tender points on digital palpation with force of < 4 kg/cm (amount of pressure required to blanch thumbnail) known as the Widespread Pain Index (WPI)
 - The 9 *paired* (bilateral) tender points are located at the:
 - Occiput: Suboccipital muscle insertions
 - Low cervical: Anterior aspects of C5–C7 intertransverse spaces
 - Trapezius: Midpoint of upper border

- Supraspinatus: Above medial border of scapular spine
- 2nd rib: 2nd costochondral junction just lateral to the junctions on upper surfaces
- Lateral epicondyle: ~ 2 cm distal to epicondyles
- Gluteal: Upper outer quadrant of buttocks
- Greater trochanter: Posterior to trochanteric prominence
- Knee: Medial fat pad proximal to joint line
- OR diagnosis can be made with a WPI ≥ 7 and a Symptom Severity (SS) scale ≥ 5 OR WPI 3–6 and SS ≥ 9 .
- The SS scale (0–12) evaluates fatigue, waking unrefreshed, and cognitive symptoms
- The 3 symptoms are rated on the following scale for level of severity over the past week:
 - 0 = no problem
 - 1 = slight or mild problems, generally intermittent
 - 2 = moderate, considerable problems, often present and/or at a moderate level
 - 3 = severe: Pervasive, continuous, life-disturbing problems
- In addition, the SS scale rates somatic symptoms as follows:
 - 0 = no symptoms
 - 1 = few symptoms
 - 2 = moderate number of symptoms
 - 3 = great deal of symptoms
 - Somatic symptoms that might be considered: Muscle pain, irritable bowel syndrome, fatigue/tiredness, thinking or remembering problems, muscle weakness, headache, pain/cramps in the abdomen, numbness/tingling, dizziness, insomnia, depression, constipation, pain in the upper abdomen, nausea, nervousness, chest pain, blurred vision, fever, diarrhea, dry mouth, itching, wheezing, Raynaud phenomenon, hives/welts, ringing in ears, vomiting, heartburn, oral ulcers, loss of/change in taste, seizures, dry eyes, shortness of breath, loss of appetite, rash, sun sensitivity, hearing difficulties, easy bruising, hair loss, frequent urination, painful urination, and bladder spasms.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Required only for evaluation of alternative diagnoses or acute pain:
 - CBC
 - Blood chemistries
 - ESR
 - Muscle enzymes

- Thyroid function tests
- Urinalysis
- No specific lab abnormalities are characteristic of fibromyalgia.

Imaging

No specific radiographic abnormalities are characteristic.

Diagnostic Procedures/Surgery

Required only to evaluate causes of acute pain

DIFFERENTIAL DIAGNOSIS

- Myofascial pain syndrome (*trigger points* present, not *tender points*)
- Chronic fatigue syndrome
- Major depression
- Polymyalgia rheumatica
- Lyme disease
- Hypothyroidism
- Collagen vascular disease
- Electrolyte imbalance
- Myopathies (metabolic and drug induced)
- Osteomalacia
- Psychogenic rheumatism
- Eosinophilia–myalgia syndrome
- UTI
- Spondyloarthropathy
- Multiple chemical sensitivity
- Interstitial cystitis



TREATMENT

ED TREATMENT/PROCEDURES

- Patient education and reassurance:
 - Emphasize that fibromyalgia is not life-threatening and does not reduce life expectancy.
 - Disorder is chronic but not crippling or deforming.
 - Goal is to manage pain and improve functional disability.
- Patients will require ongoing care and should be referred to a primary physician or pain specialist.
- Pharmacologic therapy:
 - Pharmacotherapy for improving pain, relaxing muscles, and improving sleep quality has been most successful with CNS agents such as pregabalin or gabapentin.

- Opioids are not indicated for chronic pain and may actually worsen a patient’s long-term pain by acting as NDMA receptor agonists.
- Combinations of medications (e.g., amitriptyline and fluoxetine or amitriptyline and cyclobenzaprine) may be more beneficial than either medication alone.
- Tricyclic antidepressants (TCAs; amitriptyline, nortriptyline) likely superior to SSRIs.
- Serotonin norepinephrine reuptake inhibitors (duloxetine, milnacipran) may be more effective than SSRIs and better tolerated than TCAs.
- Tramadol is an adjunctive agent.
- Benzodiazepines (clonazepam) are of no benefit other than their role in sleep disturbances.
- NSAIDs and corticosteroids have not been shown to be effective.
- Steroids or local anesthetic (lidocaine) injection into tender points is controversial:
 - No studies available to prove efficacy

MEDICATION

- Acetaminophen: 650 mg PO q4h
- Amitriptyline: 25–50 mg PO at bedtime
- Cyclobenzaprine: 5–10 mg PO TID
- Duloxetine: 60 mg PO daily or BID
- Gabapentin: Start 300 mg PO TID, titrate upward beginning at 300 mg/d to max. of 1200 mg
- Milnacipran: Start at 12.5 mg/d, then titrate upward to max. of 50–100 mg PO BID
- Pregabalin: Start 50 mg PO TID, titrate upward to max. 450 mg/d PO in divided doses
- Tramadol: 300–400 mg/d PO



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients with serious underlying disease, intractable pain, or immunocompromised
- Patients with suicidal ideation

Discharge Criteria

Patients with uncomplicated fibromyalgia can be managed as outpatients.

FOLLOW-UP RECOMMENDATIONS

Lifestyle modifications:

- Physical exercise should be encouraged:
 - Exercise program should be gradual to avoid overexertion and discouragement.
 - Aerobic exercise is more beneficial than simple stretching.
 - Efficacy not maintained if exercise stops
- Good sleep pattern should also be discussed:
 - Establishing nightly ritual in preparation for sleep
 - Avoiding caffeine-containing beverages or foods in afternoon or evenings
- Encourage stress management and coping strategies.
- Participation in educational programs (e.g., cognitive-behavioral therapy):
 - Improvement is often sustained for months.

PEARLS AND PITFALLS

As fibromyalgia patients can develop acute symptoms, distinguishing between acute and chronic pain is critical.

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CODES

ICD9

729.1 Myalgia and myositis, unspecified

ICD10

M79.7 Fibromyalgia

FLAIL CHEST

Stephen L. Thornton

BASICS

DESCRIPTION

- Free-floating segment of chest wall:
 - 3 or more adjacent ribs are fractured in 2 or more places.
 - Rib fractures in conjunction with sternal fractures or costochondral separations
- The free-floating segment of chest wall paradoxically moves inward during inspiration and outward during expiration.
- The principal pathology associated with flail chest is the *associated pulmonary contusion*:
 - There is no alteration in ventilatory mechanics owing to the free-floating segment.

ETIOLOGY

- Blunt thoracic trauma
- Fall from a height
- Motor vehicle accident
- Assault
- Missile injury
- Ribs usually break at the point of impact or posterior angle:
 - Ribs 4–9 most prone to fracture.
 - Weakest point of ribs is 60° rotation from sternum.
- Transfer of kinetic energy to the lung parenchyma adjacent to the injury:
 - Disruption of the alveolocapillary membrane and development of pulmonary contusion
 - Arteriovenous shunting
 - Ventilation/perfusion mismatch
 - Hypoxemia
 - Respiratory failure may result.

Pediatric Considerations

- Relatively elastic chest wall makes rib fractures less common in children.
- Presence of rib fractures implies much higher energy absorption.

Geriatric Considerations

Much more susceptible to rib fractures:

- Described with low-energy mechanisms

- Complicated by osteoporosis



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Blunt thoracic trauma by any mechanism
- Mechanism as described by patient, parent, or pre-hospital personnel:
 - Seat belt usage
 - Steering wheel damage
 - Air bag deployment
- Localized chest wall pain increases with deep inspiration, coughing, moving
- Pleuritic chest pain
- Dyspnea
- Hemoptysis

Physical-Exam

- Flail chest paradoxically moves inward during inspiration and outward during expiration:
 - Can be missed due to muscle spasm and splinting respirations.
 - Inspection under tangential light may be useful.
- Multiple rib fractures:
 - Bony step-offs
 - Ecchymosis
 - Crepitus
 - Edema
 - Erythema and tenderness associated with:
 - Splinting respirations
 - Intercostal muscle spasm
 - Dyspnea, tachypnea
 - Onset may be insidious, increasing over time.
- Cyanosis, tachycardia, hypotension
- Auscultation with initially normal breath sounds progressing to wet rales or absent breath sounds

ESSENTIAL WORKUP

Diagnosis is initially made on clinical grounds and then supported by radiographs.

DIAGNOSIS TESTS & INTERPRETATION

Lab

Arterial blood gas analysis:

- May reveal hypoxemia
- Elevated alveolar–arterial gradient

Imaging

- Chest radiograph aids diagnosis and prognosis:
 - _ May reveal associated intrathoracic pathology:
 - Pneumothorax
 - Hemothorax
 - Pneumomediastinum
 - Pulmonary contusion
 - Widened mediastinal silhouette
 - _ Pulmonary contusion appears within 6–12 hr after injury:
 - Ranges from patchy alveolar infiltrates to frank consolidation
- Thoracic CT is useful in detecting associated intrathoracic injuries not identified on chest radiograph:
 - _ Thoracic CT found to show on average of 3 additional rib fractures compared with plain chest radiographs.

DIFFERENTIAL DIAGNOSIS

- Chest wall contusion or intercostal muscle strain
- Costochondral separation
- Sternal fracture and dislocation
- Radiographic differential diagnosis includes:
 - _ ARDS
 - _ Pulmonary laceration, infarction, or embolism
 - _ CHF
 - _ Pneumonia, abscess, other infectious processes
 - _ Noncardiogenic causes of pulmonary edema



TREATMENT

PRE HOSPITAL

- Positioning the patient with the injured side down can stabilize the involved chest wall:
 - _ Improve ventilation in noninjured hemithorax.
- Thoracic trauma with significant mechanism or combined with pre-existing pulmonary disease should be routed to the nearest trauma center.

INITIAL STABILIZATION/THERAPY

- Manage airway and resuscitate as indicated.
- IV line, O₂, continuous cardiac monitoring, and pulse oximetry

- Control airway:
 - Endotracheal intubation
 - Indicated for patients with severe hypoxemia ($\text{PaO}_2 < 60$ mm Hg on room air, < 80 mm Hg on 100% O_2)
 - Significant underlying lung disease
 - Impending respiratory failure

ED TREATMENT/PROCEDURES

- Maintain adequate oxygenation and ventilation.
- Monitor O_2 saturation and respiratory rate.
- In conscious and alert patients, O_2 administration via face mask is first-line therapy.
- If patient cannot maintain a $\text{PaO}_2 > 80$ mm Hg on high-flow oxygen, consider continuous positive airway pressure via mask or nasal bilevel positive airway pressure.
- Consider early endotracheal intubation and mechanical ventilation if the above fails:
 - Physiologic internal fixation of the flail segment
- External fixation or stabilization of the flail segment is not indicated.
- Adequate pain control is critical to maintaining adequate pulmonary function:
 - Avoid splinting, atelectasis, and pneumonia.
- Search for associated injuries and treat exacerbation of underlying lung disease.
- Intercostal nerve blocks with 0.5% bupivacaine are safe and effective when performed properly:
 - Provides 6–12 hr of pain relief
 - Perform intercostal nerve block posteriorly 2–3 fingerbreadths from the vertebral midline.
 - Inject 0.5–1 mL just under the inferior surface of the rib where the neurovascular bundle is located.
 - Aspirate 1st to be certain that the intercostal vessels have not been punctured.
- Prophylactic antibiotics are not indicated.

ALERT

Avoid overhydration:

- In the setting of pulmonary contusion, the need for IV crystalloid resuscitation must be weighed against the risk of increasing interstitial pulmonary edema.

MEDICATION

- Multiple acetaminophen/opioid analgesic combinations are available; see the alert below.
- Acetaminophen: 300 mg/codeine 30 mg (peds: 0.5–1 mg/kg codeine) PO q4–6h

- Acetaminophen: 500 mg/hydrocodone 5 mg PO q4–6h
- Acetaminophen: 750 mg/hydrocodone 7.5 mg PO q4–6h
- Acetaminophen: 325 mg/hydrocodone 10 mg PO q4–6h
- Acetaminophen: 325 mg/oxycodone 5 mg PO q6h
- Bupivacaine: 0.5% 0.5–1 mL per injection for intercostal nerve blocks
- Hydromorphone: 2–8 mg (peds: 0.03–0.08 mg/kg) PO q4–6h
- Hydromorphone: 1–4 mg (peds: 0.015 mg/kg) IV/IM/SC q4–6h
- Morphine sulfate: 0.05–0.1 mg/kg IV/IM/SC q2–6h
- Patient-controlled analgesia using fentanyl, hydromorphone, or morphine sulfate is effective.

ALERT

- Consider thoracic epidural analgesia for patients with intractable pain, oversedation, or hypoventilation secondary to opioid analgesics.
- NSAIDs discouraged due to the risk of GI bleeding.
- The dose of acetaminophen/opioid analgesic combinations is limited by the hepatic toxicity of acetaminophen.
- The max. acetaminophen dose is 1 g per dose and 4 g/d (peds: 15 mg/kg per dose, do not exceed 5 doses/24 hr)



FOLLOW-UP

DISPOSITION

Admission Criteria

All patients with flail chest should be admitted to critical care setting for close monitoring and adequate pain control.

Discharge Criteria

Patients found to have flail chest, with or without pulmonary contusion, should not be discharged.

PEARLS AND PITFALLS

- Early pain control is key.
- Beware of concomitant injuries such as pulmonary contusion and pneumothorax.
- Elderly patients have significantly poorer outcomes.

ADDITIONAL READING

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CODES

ICD9

807.4 Flail chest

ICD10

- S22.5XXA Flail chest, initial encounter for closed fracture
- S22.5XXB Flail chest, initial encounter for open fracture

FOOT FRACTURE

Stephen R. Hayden

BASICS

DESCRIPTION

Injury to tarsal bones or metatarsals including calcaneus, talus, navicular, cuboid, cuneiform, and metatarsals

ETIOLOGY

- Most common foot injuries are of the metatarsals and phalanges.
- The calcaneus is the most commonly fractured of the tarsal bones.
- Calcaneus fractures: Compression injury from sudden high-velocity impact to heel:
 - 75% are intra-articular; 50% have associated injuries:
 - 10% spine fractures
 - 25% with associated lower extremity trauma
 - 9% bilateral, 5% open
- Metatarsal fractures: Divided into stress fractures, twisting injuries, or direct trauma:
 - 1st metatarsal: Direct applied force
 - 2nd and 3rd metatarsals are most often involved in stress fractures and twisting injuries.
 - 5th metatarsal: Avulsion fracture (dancer's fracture) of proximal apophysis is the most common injury.
 - Jones fracture: Transverse fracture of the metaphyseal–diaphyseal junction of 5th metatarsal; results from twisting while foot inverted.
- Talus: Caused by dorsiflexion with axial load, common snowboarder's injury
- Navicular: Results from axial compression or stress fractures
- Cuboid and cuneiform fractures are rare and occur in conjunction with other injuries, often with tarsal–metatarsal injuries.
- Tarsal–metatarsal injuries (Lisfranc injuries) are high-energy injuries:
 - Axial load on plantar-flexed foot, or hindfoot fixed with forced foot eversion
 - Unstable forefoot on hindfoot
 - 20% go undiagnosed on initial visit.
 - 3 types: Convergent, divergent, and incongruent

Pediatric Considerations

- Metatarsal fractures account for 90% of foot fractures in children, usually from direct trauma:
 - Lesser metatarsal fractures (2–4) most common followed by base of 5th then base of 1st metatarsal.

- Physeal injury may occur with proximal 1st metatarsal fractures.
- Other common injuries include phalangeal fractures (17%) and navicular fractures (5%).
- Fractures of talus or calcaneus occur with distal tibia or fibula fractures (8%).
- Calcaneus fractures are less likely intra-articular. Less common to have associated spine fractures.



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- History of preceding trauma most common
- Stress fractures may present with increasing pain in the setting of repetitive activities.

Physical-Exam

- Ecchymosis, pain, swelling, or deformity of foot
- Pain with weight bearing
- Joint instability

ESSENTIAL WORKUP

- Physical exam of extremity is necessary to assess neurovascular status, skin integrity, gross swelling, deformity, or loss of function.
- Exam of spine is also essential in suspected calcaneus fractures, as there is a 10% incidence of coexistent injury.
- Anteroposterior/lateral and oblique views are necessary for all foot fractures.
- Complications:
 - Compartment syndrome most commonly presents as severe pain in a swollen foot:
 - Pressures > 35 mm Hg require opening of all major foot compartments.
 - May have hypesthesia of plantar foot
 - Weak toe flexion
 - Late findings include claw toe deformity.
 - Nonunion and avascular necrosis are common complications with talar neck fractures owing to distal blood supply.
 - Calcaneus fractures may be accompanied by sural nerve injury; test sensation along lateral aspect of foot.

DIAGNOSIS TESTS & INTERPRETATION

Imaging

Special views may be needed for some fractures:

- Lisfranc fractures may require stress views with weight bearing. They may require MRI to evaluate ligamentous stability. May require CT for evaluation of small fractures if clinically suspicious
- Fleck sign: Pathognomonic—avulsion of ligament from 2 MT base or medial cuneiform
- Talar fractures may require a 45° internal oblique view. May require CT.
- Midfoot fractures may require an external oblique foot view.
- Calcaneus fractures require an axial view and may require CT:
 - Bohler's angle $< 20^\circ$ suggests a compression fracture of calcaneus.
 - Lumbosacral spine films are necessary in all patients with calcaneus fractures.
- Stress fractures may require 2 wk to appear on plain films; bone scan or CT may be used to elucidate suspected fractures.

DIFFERENTIAL DIAGNOSIS

- Anterior effects of calcaneus and talar dome fractures can be misdiagnosed as ankle sprains.
- Foot contusions
- Freiberg disease: Osteochondrosis of 2nd metatarsal head may be mistaken for stress fracture.



TREATMENT

PRE HOSPITAL

- Ice bag should be placed on affected foot and foot and ankle immobilized.
- All patients suspected of calcaneus fracture should have spinal immobilization; often, mechanism is fall from height > 6 ft.

INITIAL STABILIZATION/THERAPY

Manage coexisting trauma as indicated.

ED TREATMENT/PROCEDURES

- Airway, breathing, and circulation management
- Assess for neurovascular compromise distal to fracture site.
- Dislocations must be reduced as quickly as possible with assessment of neurovascular status before and after procedure:
 - Procedural sedation usually required
- Immobilize, ice, and elevate in a bulky splint:
 - Application of circumferential cast should be delayed until swelling subsides.
- Crutches

- Pain management:
 - If large amount of swelling and pain with toe movement, suspect compartment syndrome.
 - Ultrasound-guided regional anesthesia may be used for reduction
- Orthopedic consult indicated early for displaced fractures:
 - Many injuries require repair within 6 hr of injury to prevent delay of open reduction with internal fixation for 6–10 days owing to swelling.

MEDICATION

- Cefazolin: 1 g IV/IM (peds: 25 mg/kg IV/IM)
- Diprivan: 40 mg IV q10s until sedation
- Etomidate: 0.1–0.2 mg/kg IV
- Fentanyl: 50–250 µg IV titrated (peds: 2 µg/kg IV)
- Hydromorphone 0.5–2 mg IV q2h (peds: 0.15 mg/kg IV q4–6h)
- Ibuprofen: 800 mg PO (peds: 10 mg/kg PO)
- Meperidine: 25–100 mg IV/IM titrated (peds: 1–1.75 mg/kg IV/IM)
- Methohexital: 1–1.5 mg/kg IV
- Morphine: 2–10 mg IV/IM titrated (peds: 0.1 mg/kg IV)



FOLLOW-UP

DISPOSITION

Admission Criteria

- Open fracture
- Evidence of compartment syndrome or neurovascular injury
- Open reduction internal fixation required immediately

Discharge Criteria

Most patients with metatarsal fractures can be discharged with orthopedic follow-up.

Issues for Referral

All open fractures, as well as all midfoot/Lisfranc injuries and displaced fractures that are not successfully reduced, should be seen in ED by an orthopedic specialist.

ADDITIONAL READING

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- Khan W, Oragui E, Akagha E. Common fractures and injuries of the ankle and foot: Functional anatomy, imaging, classification and management. *J Perioper Pract*. 2010;20(7):249–258.



CODES

ICD9

- 825.20 Closed fracture of unspecified bone(s) of foot [except toes]
- 825.25 Closed fracture of metatarsal bone(s)
- 825.29 Other closed fracture of tarsal and metatarsal bones

ICD10

- S92.209A Fracture of unsp tarsal bone(s) of unsp foot, init
- S92.309A Fracture of unsp metatarsal bone(s), unsp foot, init
- S92.909A Unsp fracture of unsp foot, init encntr for closed fracture

FOREARM FRACTURE, SHAFT/DISTAL

Stephen R. Hayden

BASICS

DESCRIPTION

- Forearm shaft fractures (single or paired) are often displaced by contraction of arm muscles; sometimes associated with concurrent dislocations:
 - *Galeazzi* fracture:
 - Distal radius fracture with distal radioulnar dislocation
 - *Monteggia* fracture:
 - Proximal ulnar fracture with dislocation of radial head
- Distal fractures include extension, flexion, and intra-articular classifications:
 - *Colles* fracture:
 - Hyperextension fracture of distal radius
 - Distal fragment displaced dorsally
 - Radial deviation
 - Often involves ulnar styloid and distal radioulnar joint
 - *Smith* fracture:
 - Hyperflexion fracture of distal radius
 - Distal fragment displaced volarly
 - *Barton* fracture:
 - Intra-articular fracture of dorsal rim of distal radius
 - Often associated with dislocation of carpal bones
 - *Hutchinson* fracture:
 - Intra-articular fracture of radial styloid

Pediatric Considerations

- Shaft fractures:
 - *Torus* fracture:
 - Compression (buckling) of cortex on 1 or both sides
 - *Greenstick* fracture:
 - Distraction of 1 side of cortex with opposite side intact
 - Plastic deformity:
 - Bowing of radius or ulna without apparent disruption of cortex
 - Multiple microfractures
- Distal fractures:
 - *Salter–Harris* type fractures (see Salter–Harris classification)

ETIOLOGY

- Direct blow to forearm

- Longitudinal compression load:
 - _ Fall on outstretched hand (FOOSH)
 - _ Horizontal force
- Excessive pronation, supination, hyperextension, or hyperflexion



DIAGNOSIS

SIGNS AND SYMPTOMS

- Deformity
- Pain, edema, erythema

History

- Associated events and concurrent injuries
- Past history of bone disease or old fractures
- History of repetitive stress of forearm movement
- Occupation
- Hand dominance

Physical-Exam

- Physical exam with special attention to skin integrity, deformity, and neurovascular status
- Forearm pain, crepitus, tenderness to palpation, deformity, shortening of forearm
- Forearm edema, ecchymosis, elbow or wrist joint effusions
- Abnormal mobility or loss of function at elbow/wrist/hand
- Neurologic abnormalities
- Vascular compromise

ALERT

Impending compartment syndrome

ESSENTIAL WORKUP

Suspected forearm fractures require anteroposterior (AP) and lateral radiographs, including joint above and joint below injury: Hand, wrist, and elbow.

DIAGNOSIS TESTS & INTERPRETATION

Lab

Preoperative labs as warranted

Imaging

Some intra-articular fractures may require CT imaging.

Diagnostic Procedures/Surgery

Compartment pressures should be measured for suspected compartment syndrome.

DIFFERENTIAL DIAGNOSIS

- Upper extremity muscle, ligamentous injury
- Elbow or wrist dislocations, including pediatric nursemaid's elbow
- Forearm contusions, hematomas
- Cellulitis, abscesses, soft tissue masses
- Forearm osteogenic tumors
- Osteomyelitis
- Upper extremity vascular or neurologic injuries
- Elbow or wrist arthritis, joint effusions
- Pediatric growth plates, nutrient vessels may be mistaken for fractures



TREATMENT

PRE HOSPITAL

- All suspected forearm fractures should be elevated, splinted, and immobilized, including elbow and wrist joints.
- All open fractures should be wrapped with sterile dressing before immobilization:
 - Do not reduce open fractures back under skin in the field.
 - In patients with isolated extremity trauma, analgesia may be administered.

ED TREATMENT/PROCEDURES

- Shaft fractures, nondisplaced:
 - Long-arm splint
 - Orthopedic referral
- Shaft fractures, displaced:
 - Orthopedic consultation
 - Often require open reduction, internal fixation
- Distal fractures, nondisplaced:
 - Forearm sugar-tong or AP splint
 - Orthopedic referral
- Distal fractures: *Colles/Smith*:
 - Simple, noncomminuted, extra-articular Colles and Smith fractures may be reduced in ED:
 - Splint (long-arm sugar-tong splint)
 - Sling
 - Referred to orthopedics
 - Complicated Colles and Smith fractures require orthopedic consultation.
- Distal fractures: *Barton/Hutchinson*:
 - Uncomplicated Barton and Hutchinson fractures

- Splint (AP or sugar-tong splint)
- Place in sling
- Referred to orthopedics
- Complicated fractures require orthopedic consultation.
- Open fractures:
 - Cover with sterile dressings.
 - IM/IV antibiotics
 - Tetanus immunization (if indicated)
 - Splint
 - Immediate orthopedic consultation
- Forearm fractures associated with compartment syndrome or neurovascular compromise require immediate orthopedic consultation.

Pediatric Considerations

- *Torus* and *Greenstick* fractures with $< 10^\circ$ of angulation may be treated with long-arm splint, sling, and orthopedic referral.
- *Plastic deformities* require orthopedic consultation:
 - Some minimally displaced plastic deformities may be placed in long-arm splint and sling.
- *Salter–Harris* type fractures require orthopedic consultation.

MEDICATION

- Acetaminophen: 325–1,000 mg PO q4h (peds: 10–15 mg/kg q4h PO)
- Antibiotics:
 - Open fractures require IM/IV antibiotics.
 - Cefazolin: 1–2 g IM/IV or equivalent 1st-generation cephalosporin; if contaminated, add an aminoglycoside
- Codeine: 15–60 mg PO/IM q4h (peds: > 2 yr, 0.5–1 mg/kg q4h PO/IM)
- Hydrocodone: 5–10 mg PO q4h
- Ibuprofen: 200–800 mg q4–8h (peds: > 6 mo, 5–10 mg/kg per dose q6h)
- Morphine sulfate: 2–10 mg IV/IM; titrate to pain (peds: 0.1 mg/kg per dose IV/IM)
- Tetanus: 0.5 mL IM every 10 yr



FOLLOW-UP

DISPOSITION

Admission Criteria

- Open fractures
- Fractures with compartment syndrome or neurovascular compromise
- Fractures needing immediate operative management or general anesthesia for

reduction

- Suspected nonaccidental trauma

Discharge Criteria

- Appropriate reduction and immobilization
- Arranged orthopedic follow-up
- Adequate pain control measures
- Cast/splint care discharge instructions provided and understood by patient
- Documentation of intact neurovascular function after ED treatment

Issues for Referral

All fractures (or suspected fractures) discharged from ED should be referred to orthopedic surgeon for close follow-up.

FOLLOW-UP RECOMMENDATIONS

All patients should be referred to an orthopedic surgeon or hand surgeon.

PEARLS AND PITFALLS

- Missed 2nd fracture
- Missed concurrent dislocation or subluxation
- Impending compartment syndrome

ADDITIONAL READING

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- Perron AD, Brady WJ. Evaluation and management of the high-risk orthopedic emergency. *Emerg Med Clin North Am*. 2003;21(1):159–204.



CODES

ICD9

- 813.23 Closed fracture of shaft of radius with ulna
- 813.44 Closed fracture of lower end of radius with ulna
- 813.80 Closed fracture of unspecified part of forearm

ICD10

- S52.90XA Unsp fracture of unsp forearm, init for clos fx
- S52.509A Unsp fracture of the lower end of unsp radius, init
- S52.609A Unsp fracture of lower end of unsp ulna, init for clos fx

FOREIGN BODY, EAR

Kathleen Nasci • Charles V. Pollack, Jr.



BASICS

DESCRIPTION

- Foreign bodies (FBs) lodged in the external auditory canal
- The external auditory canal:
 - Cartilaginous and bony passage lined with periosteum and skin
 - The periosteum is extremely sensitive, making removal a painful procedure:
 - In small children general anesthesia may be required to remove the object
 - FBs usually impact at the junction of the inner end of the cartilaginous portion of the canal or at the isthmus
 - Innervated by the facial, glossopharyngeal, vagus nerves
- Inanimate foreign objects are often associated with delayed presentations:
 - Children often delay reporting because of fear of punishment
 - Often the FB is an incidental finding in children during an ear exam
- Physical findings may change due to length of time the object is in the canal
- Children with cerumen impaction or those with pica are predisposed
- The location is often the right ear, due to the predominance of right handedness
- Children and psychiatric patients may insert anything sufficiently small to enter the external auditory canal.
- Ear FBs are most common in children < 8 yr
- Complications:
 - Canal laceration:
 - Usually caused by repeated attempts to remove a nongraspable object
 - Perforation of tympanic membrane:
 - More likely to result from removal procedure than the FB
 - Otitis externa
 - Malocclusion from erosion into the temporomandibular joint
 - Parapharyngeal abscess
 - Mastoiditis
 - Meningitis
 - Brain abscess
 - Insects may injure the tympanic membrane or canal by stinging, biting, or scratching
 - Button batteries can cause significant destruction due to the strong electrical currents and pressure necrosis
 - Typically, the most damage is caused by negative side of the battery
 - Damage to the facial nerve and ossicles have been reported

- Symptoms usually resolve within a few days after FB removal

ETIOLOGY

- Children:
 - Stones
 - Small beads
 - Paper
 - Toys
 - Seeds and popcorn kernels
 - Beans and other food and organic materials
 - Button batteries:
 - Higher risk for necrosis than other FBs
- Competent adults:
 - Cotton-swab tips
 - Earplugs
 - Insects:
 - Cockroach most common in US
 - Hidden illicit drugs



DIAGNOSIS

SIGNS AND SYMPTOMS

- Decreased hearing
- Excessive crying in infants
- Unilateral ear pain
- Fullness
- Loud noises
- Buzzing sound (with live insects)
- Nausea
- Dizziness
- Ipsilateral tearing
- Purulent discharge from the external ear
- Itching
- Bleeding

History

- Travel or camping history or poor living conditions suggests insects in the external ear canal
- Inquire about previous attempts to remove the FB and any trauma associated with these attempts

Physical-Exam

Otoscopic exam should be performed before and after removal of the FB:

- Identify type of FB to determine removal procedure:
 - Button battery
 - Live insect
 - Vegetable
 - Inanimate object
 - Size
 - Risk of swelling when exposed to water
- Perform a bilateral exam; especially important in children and psychiatric patients, and prevent overlooking a quiescent FB in the contralateral ear
- Attempt to visualize tympanic membrane to assess for rupture
- Assess for otitis externa
- Assess for retained fragments after the removal
- Always exam the nonaffected ear and nostrils for additional FBs
- Significant pain, vertigo, or ataxia, nystagmus, hearing loss, otorrhea, or facial nerve paralysis are concerning signs and an otolaryngologist consultation should be considered

ESSENTIAL WORKUP

Careful otoscopic exam:

- Minimize pain
- Gain the patient's trust
- Identify the FB before attempting removal

DIAGNOSIS TESTS & INTERPRETATION

Lab

None indicated

Imaging

CT scan if infectious or erosive sequelae are suspected

Diagnostic Procedures/Surgery

Otomicroscope:

- May be used when standard ED techniques fail or the equipment is available to emergency medical staff

DIFFERENTIAL DIAGNOSIS

- Cerumen impaction
- Granuloma
- Hematoma
- Injury
- Otitis externa

- Perforated tympanic membrane
- Residual otitis externa after self-extraction of the FB
- Tumor



TREATMENT

PRE HOSPITAL

- Cautions:
 - _ Severe ear pain, sensation of movement, and loud, buzzing sound:
 - Typical signs of a live insect in external auditory canal
 - Instill warm lidocaine or mineral oil into affected ear to kill insect
- Controversies:
 - _ Attempts at removal in the field are not indicated:
 - Lack of appropriate equipment
 - Prior failed attempts may make future attempts more difficult

INITIAL STABILIZATION/THERAPY

For a patient in distress because of a live insect:

- Drown or immobilize insect before any removal attempts
- Instill warm solution into the external auditory canal:
 - _ 2% lidocaine solution
 - _ Ether
 - _ Alcohol
 - _ Mineral oil
- Cold fluids should not be used so as to avoid a caloric response

ED TREATMENT/PROCEDURES

- Prepare the equipment and the patient:
 - _ Strong light source
 - _ Otoscope or operating microscope
 - _ Achieve proper head immobilization
 - _ Retract the pinna of the ear in a posterosuperior direction to straighten the canal
- Analgesia:
 - _ Lidocaine instillation for topical anesthesia:
 - Liquid 1–2% solution is preferred to viscous lidocaine.
 - Lidocaine injection of the 4 quadrants of the canal using a tuberculin syringe through the otoscope
 - 1–2% lidocaine, with or without epinephrine
- Procedural sedation:
 - _ Indicated for children and uncooperative adults
 - _ Use before attempts, as unsuccessful efforts may produce bleeding, edema,

- or injury to the tympanic membrane
- Ketamine for children
- Benzodiazepines for older patients
- Consider fentanyl if analgesia is indicated during removal
- Options for removal:
 - Water irrigation:
 - Perform careful visualization
 - Place an Angiocath catheter adjacent to, or preferably distal to, the FB
 - Inject warm water or sterile saline through catheter via a syringe
 - Backwash the FB out
 - Never attempt removal by irrigation when the FB is a button battery
 - Use of instruments to dislodge the FB:
 - Alligator forceps removal
 - Cupped forceps: Numbers 3, 5, and 7 suction tips, preferably with Frazier suction cups
 - Cerumen loops
 - Right-angle blunt hooks
 - Suction catheters:
 - Best used for small objects
 - Fogarty catheter:
 - Carefully pass beyond the FB and inflate and withdraw; this approach puts the tympanic membrane at particular risk of inadvertent injury
 - Cyanoacrylate glue on the tip of a blunt probe:
 - Place on the FB for 10 sec, and then pull
 - May contaminate the ear with glue, and this technique has been associated with tympanic membrane rupture
 - Acetone:
 - Used to dissolve Styrofoam FBs or loosen superglue
 - Otomicroscopy:
 - Usually performed in the OR although reports of use in the ED have been positive
- Vegetable matter:
 - Avoid irrigation of FBs that will swell when exposed to water
 - Attempt removal with instrument
 - Forceps usually work with graspable objects
 - Be certain to delineate clearly between FB and inflamed external auditory canal tissue
- Nonvegetable inanimate FB removal:
 - If easily grasped, attempt removal with forceps
 - If not accessible, attempt removal with irrigation
- Polished or smooth object extraction:
 - Visualize

- Direct suction
- Blunt right-angled probe: Pass beyond the FB; rotate 90°; remove it with the FB
- Fogarty catheter
- Cyanoacrylate glue
- Insect removal:
 - Kill insect by rapidly instilling alcohol, 2% lidocaine (Xylocaine), or mineral oil into the ear
 - Once killed, remove with forceps or by irrigation
 - Re-examine to ensure that all insect parts are removed
- Sharp objects:
 - Remove with operating microscope
 - Consider otolaryngologic referral if there is evidence of trauma or if patient is uncooperative

MEDICATION

First Line

- Fentanyl: 1 µg/kg IV
- Ketamine: 1–2 mg/kg IV or 4 mg/kg IM
- Midazolam: 1 mg IV slowly q2–3min up to 5 mg (peds: 6 mo–5 yr, 0.05–0.1 mg/kg, titrate to max. of 0.6 mg/kg; 6–12 yr, 0.025–0.05 mg/kg, titrate to max. of 0.4 mg/kg)

Second Line

- Cortisporin otic: 4 gtt in ear QID
- Amoxicillin: 500 mg PO (peds: 80–90 mg/kg/24 h) PO TID for 7–10 days.
- Augmentin: 875 mg (peds: 90 mg/kg/24 h) PO BID for 7–10 days.
- Fill ear canal 5× per day with a combination of antibiotic and steroid otic solution for 5–7 days if there is suspected infection or abrasion



FOLLOW-UP

DISPOSITION

Admission Criteria

Hospital admission if the FB is a button battery that cannot be removed

Discharge Criteria

- FB removed
- Inability to remove a FB that will not cause rapid tissue necrosis
- Oral antibiotics (amoxicillin or Augmentin) should be initiated in cases with

tympanic membrane perforation

Issues for Referral

Follow-up with ENT specialist as an outpatient:

- Inability to remove a FB
- Immunocompromised patients with signs of otitis externa

FOLLOW-UP RECOMMENDATIONS

- Patient should be instructed not to place any objects in ear
- A short course of analgesics after traumatic FB removal
- Otitis externa:
 - Topical antimicrobial such as Cortisporin suspension
- Immunocompromised patients may require oral antibiotics
- Perforated tympanic membrane:
 - Prophylaxis with antibiotics
 - Follow-up with ENT specialist
- Avoid submersion in water until follow-up if trauma or infection present

PEARLS AND PITFALLS

- Use procedural sedation with uncooperative patients or when a difficult removal is anticipated
- Irrigation in patients with button batteries in the ear should never be performed as the electrical current or battery contents can cause liquefaction tissue necrosis.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- [Tympanic Membrane Perforation](#)
- [Procedural Sedation](#)



CODES

ICD9

931 Foreign body in ear

ICD10

- [T16.1XXA Foreign body in right ear, initial encounter](#)
- [T16.2XXA Foreign body in left ear, initial encounter](#)
- [T16.9XXA Foreign body in ear, unspecified ear, initial encounter](#)

FOREIGN BODY, ESOPHAGEAL

Joanna W. Davidson



BASICS

DESCRIPTION

- Esophageal foreign bodies (FBs) typically lodge at 3 sites of physiologic constriction:
 - Cricopharyngeal muscle—63%, most common (C6)
 - Gastroesophageal junction—20% (T11)
 - Aortic arch—10% (T4)
- 90% of ingested FBs pass spontaneously.
- 10–20% are removed endoscopically, and 1% or less require surgery.

ETIOLOGY

- Most common adult and adolescent FBs are food boluses and bones
- Increased risk:
 - Edentulous adults
 - Intoxicated patients
 - Patients with underlying esophageal disease: Schatzki B-rings or peptic strictures are most common

Pediatric Considerations

- 80% of FB ingestions occur in pediatric age group, peak ages 6 mo–6 yr, particularly younger than 2 yr.
- Coins are most common:
 - Most common: 80% of esophageal FBs
- 2 additional areas of constriction: Thoracic inlet (T1) and tracheal bifurcation (T6)



DIAGNOSIS

SIGNS AND SYMPTOMS

- Acute ingestion:
 - Dysphagia
 - Odynophagia
 - Drooling
 - Retching/self-induced vomiting
 - Choking
 - Gagging
 - Blood-stained saliva
- Chronically retained FB:

- Respiratory symptoms predominate (paraesophageal tissue swelling compromises adjacent trachea):
 - Cough
 - Stridor
 - Hoarseness
- Chest pain
- Site of FB sensation usually corresponds to esophageal level of FB
- Esophageal perforation
- 15–35% if ingest sharp object:
 - Redness
 - Swelling
 - Crepitus in the neck
 - Peritonitis
- <20% asymptomatic

Pediatric Considerations

Signs/symptoms:

- Refusal to eat
- Stridor
- Upper respiratory tract infection
- Neck/throat pain

History

- Adults:
 - Usually provide unequivocal history
 - 80% present within 1st 24 hr
 - 5% will present with airway obstruction (cafe coronary)
- Children:
 - 50% asymptomatic
 - History can be unclear if unwitnessed ingestion is not witnessed
 - Drooling, refusal to eat, unexplained gagging, cough, wheeze, choking
 - More likely than adults to have respiratory symptoms

ESSENTIAL WORKUP

- History about object ingested: Type, when, and how
- Physical exam focused by degree of distress exhibited:
 - Esophagus:
 - Obstruction—saliva pooling, aspiration
 - Perforation—crepitus, pain, pleurisy
 - Hemorrhage
 - Oropharynx:
 - Red, irritated throat
 - Palatal abrasions

- Lung:
 - Stridor and wheezing
- Abdomen:
 - Peritonitis or bowel obstruction

DIAGNOSIS TESTS & INTERPRETATION

Imaging

- Biplane chest radiograph including all of neck for FB localization:
 - Food boluses usually do not need radiographs.
 - Esophageal FBs often align themselves in coronal plane.
 - Esophageal perforation is noted by air in retropharyngeal space, in soft tissues of neck, or by pneumomediastinum.
- Ingested, impacted bones visible on plain film < 50%
- CT scan replacing esophageal contrast studies for nonradiopaque FBs:
 - Radiolucent objects include small pieces of glass, bone fragments, aluminum, plastic, pieces of wood
 - Visualizes perforation or infection
- Endoscopy is a method of choice for localizing and managing most esophageal FBs
 - Ability to inspect surrounding esophageal mucosa for pathology
 - Diagnostic and therapeutic

DIFFERENTIAL DIAGNOSIS

- Globus hystericus phenomenon (“lump in throat”)
- Esophageal mucosal irritation
- Esophagitis
- Croup
- Epiglottitis
- Upper respiratory tract infection
- Retropharyngeal abscess



TREATMENT

PRE HOSPITAL

Cautions:

- Airway maintenance and prevention of aspiration paramount
- Oxygen for patients in distress
- Place patient in whatever position gives most comfort.
- Ipecac and cathartics contraindicated

INITIAL STABILIZATION/THERAPY

- Airway, breathing, and circulation management 1st priority

- Prevent aspiration

ED TREATMENT/PROCEDURES

- Direct laryngoscopy or fiberoptic scope may allow removal of very proximal objects
- Urgent endoscopy recommended:
 - Ingestion of sharp or elongated objects
 - > 6 cm long
 - > 2.5 cm
 - Irregular/sharp edges (toothpicks, soda can tabs)
 - Ingestion of multiple FBs; especially magnets
 - Evidence of perforation
 - Coin at level of cricopharyngeus muscle in a child
 - Airway compromise
 - Presence of FB for > 24 hr
 - Food bolus with complete obstruction
 - Observation can be considered
 - Asymptomatic patients with coins or smooth objects (not button batteries) in distal esophagus:
 - Observe up to 24 hr after ingestion to see whether it will pass into stomach.
 - Objects that reach stomach and are shorter than 5 cm and < 2 cm in diameter usually pass through GI tract without difficulty, but daily radiographs are still recommended.
 - Danger of perforation increases after 24 hr.
 - Removal options:
 - IV glucagon:
 - Decreases lower esophageal sphincter tone without interfering with esophageal contractions
 - Falling out of favor for endoscopy
 - Less effective if underlying Schatzki ring or stricture
 - Permits distal food boluses to pass into the stomach
 - For impactions < 24 hr duration
 - Fluoroscopically guided Foley catheter extraction:
 - Successful and safe in experienced hands
 - Foley catheter (10F–16F) placed nasally, passed into esophagus, tip and balloon pushed beyond FB under fluoroscopic control
 - Foley balloon inflated with contrast and catheter slowly withdrawn
 - Contraindicated in chronic ingestions, uncooperative patients, sharp-pointed objects
 - Foley catheters or dilator (bougienage) may also be used to

push distal FB into stomach

- Endoscopy:
 - Preferred method to remove acute or chronic FBs
 - 98% effective
 - Always used with impactions of long duration (>2–4 days) because of associated esophageal irritation/edema
 - General endotracheal anesthesia needed in difficult cases: Infants, psychiatric patients, difficult FB
 - Risk of complications increases after 24 hr, ideal to be done within 6–12 hr
- Surgical intervention:
 - Reserved for patients in whom FB cannot be removed by other methods
 - ~1–2% of all patients
 - Toothpicks and bones common objects
- Specific ingestions
 - Impacted food bolus obstructing esophagus:
 - Emergent removal indicated
 - Proteolytic enzymes (papain) not recommended because of esophageal perforation, hypernatremia, and aspiration complications
- Button batteries:
 - Extract emergently
 - Batteries frequently leak: Potassium hydroxide and mercuric oxide are the most toxic constituents.
 - Alkali produced from external flow of current can cause liquefaction necrosis.
 - Full-thickness mucosal burns can occur within 4–6 hr (combination of chemical, electrical, pressure injuries).
 - Battery in stomach will usually pass without difficulty; batteries remaining in stomach for >3–4 days should be removed.
 - Once past duodenal sweep, 85% are passed within 72 hr.
- Narcotic/amphetamine packets:
 - Body packing seen in regions of high drug traffic
 - Packets usually seen on radiographs
 - Rupture or leakage of contents can be fatal.
- Magnets/“Bucky Balls”:
 - Opposing magnets attract bringing sections of stomach/bowel together creating obstruction
 - Early GI consult for removal vs. laparotomy

MEDICATION

Glucagon: 1–2 mg IV push after test dose to determine hypersensitivity



DISPOSITION

Admission Criteria

- Seriously ill patients and those with complications such as esophageal perforation, migration of FB through esophageal wall, significant bleeding
- Airway compromise
- Symptomatic patients in whom attempts to remove FB are unsuccessful

Discharge Criteria

- Asymptomatic patients in whom FB has been removed or passed distal to esophagus
- Asymptomatic patients with distal esophageal smooth FBs need re-exam within 12–24 hr to ascertain whether spontaneous passage into stomach has occurred.

Issues for Referral

GI consult for sharp or pointed esophageal FBs, those obstructed in upper or mid esophagus and battery button FBs.

FOLLOW-UP RECOMMENDATIONS

GI referral for patients with suspected underlying etiology for esophageal obstruction

PEARLS AND PITFALLS

- Perform radiographs to locate radiopaque FBs.
- Maintain a high suspicion for esophageal perforation.

ADDITIONAL READING

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CODES

ICD9

935.1 Foreign body in esophagus

ICD10

- T18.108A Unsp foreign body in esophagus causing oth injury, init
- T18.128A Food in esophagus causing other injury, initial encounter

FOREIGN BODY, NASAL

Bradley E. Efune • David A. Pearson



BASICS

DESCRIPTION

- Object impacted in the nasal cavity
- Most common site of foreign body insertion in children
- Type of foreign body limited only by nostril size
- Population at risk:
 - Children between 2–6 yr most common
 - Mental retardation
 - Psychiatric illness
- Causes of worsening impaction and difficulties with removal:
 - Organic material may expand if moistened
 - Mucosal swelling over time
- Complications:
 - Sinusitis is the most common complication
 - Foreign bodies may migrate into the sinuses
 - Septal perforation
 - Bronchial aspiration
 - High risk of complications with button batteries:
 - Ischemic mucosa
 - Turbinate or septal damage
 - Saddle-nose deformity

ETIOLOGY

- Food
- Beans
- Seeds
- Beads
- Rocks
- Paper
- Pieces of toys
- Sponge pieces
- Vegetable matter
- Insects and live worms
- Button batteries:
 - High risk of complications compared with other foreign bodies (tissue necrosis, septal perforation, saddle nose); require rapid removal
- Magnets:

- Used to mimic nasal piercing
- Often imbedded in nasal tissue, leading to difficult removal
- May cause intestinal perforation if swallowed, especially newer high-powered neodymium magnets
- Glass fragments

DIAGNOSIS

SIGNS AND SYMPTOMS

- Most nasal foreign bodies are asymptomatic.
- Unilateral nasal obstruction
- Nasal pain
- Difficulties with nasal breathing
- Nasal discharge:
 - Acute or chronic
 - Unilateral
 - Foul smelling
 - Halitosis
- Sinus discomfort
- Persistent epistaxis
- Local inflammation
- Septal perforation
- Ingestion or aspiration of foreign body

History

- Child witnessed putting object into nose
- Foreign body noticed by parent or caretaker
- Many children are reluctant to admit to placing a foreign body for fear of adult disapproval
- Delayed presentation:
 - When placement of the object is unwitnessed, the child may present weeks after with nasal discharge and bleeding
 - Often misdiagnosed at this stage as sinusitis

ESSENTIAL WORKUP

Visualization of the foreign body in the nostril: Always check both nostrils

DIAGNOSIS TESTS & INTERPRETATION

Imaging

- Fiberoptic visualization if foreign body cannot be visualized on rhinoscopy
- Sinus films if present for extended period:
 - Symptom persistence despite removal of the foreign body and antibiotics

- May need chest or abdomen films for aspiration/ingestion

DIFFERENTIAL DIAGNOSIS

- Sinusitis
- Swollen inferior turbinate:
 - May be mistaken for a pink bead
- Rhinitis
- Nasal polyp
- Benign tumors:
 - Hemangioma most common
- Malignant tumors:
 - Lymphoma
 - Rhabdomyosarcoma
 - Nasopharyngeal carcinoma
 - Esthesioneuroblastoma (also known as an olfactory neuroblastoma)
- Congenital masses:
 - Dermoid
 - Encephalocele
 - Glioma
 - Teratoma
- Retropharyngeal abscess
- Traumatic dislocation of nasal bones or septum
- Nasal deformity:
 - Usually associated with cleft palate
- Nasopharyngeal stenosis
- Rhinitis medicamentosa:
 - Rebound nasal mucosal edema caused by extended use of topical decongestants



TREATMENT

PRE HOSPITAL

- Cautions:
 - Transport in sitting position:
 - To avoid posterior displacement and possible aspiration of foreign body
- Avoid interventions that upset the child.
 - Forceful negative inspiration from crying may lead to aspiration

ED TREATMENT/PROCEDURES

- Topical vasoconstrictors:
 - Presence of mucosal edema, or bleeding secondary to removal attempts

- Nebulized epinephrine
- Cocaine: 4%
- Oxymetazoline: 0.05%
- Phenylephrine: 0.125–0.5%
- Positive pressure for children:
 - Occlude contralateral nostril
 - Upright sitting position if possible
 - Positive pressure applied to mouth only (best done by parents)
 - Deliver brisk puff as child begins to inhale
 - Parent may tell the child he or she will be given a “big kiss.”
 - Placement of 4 × 4 gauze pads on caregiver’s cheek
 - Foreign body dislodges onto cheek of the provider or into room
 - Repeated as necessary
 - Can use straw in older children to create pressure without mouth to mouth
 - Alternatively, deliver puff with a bag-mask over the mouth and O₂ at 10–15 L/min.
 - Alternatively, into contralateral nostril male–male adapter on oxygen tubing, deliver wall oxygen at 10–15 L/min.
 - Risk of barotrauma with sustained, unmodulated positive pressure
- Hooked probe, alligator forceps:
 - Anterior foreign bodies that are easily grasped
 - Headlamp, nasal speculum facilitate use
 - Risk of further posterior displacement
- Suction catheter:
 - Best for round, smooth objects
 - Optimal retrieval with suction catheter
 - Suction tip placed against the object
 - Suction turned up to 100–140 mm Hg
 - Catheter and object withdrawn
- Cyanoacrylate tissue glue:
 - Film of glue applied to cut end of hollow plastic swab handle
 - Apply against object for 60 sec, and then withdraw
 - Caution with nontissue cyanoacrylate glues; tissue irritation
- Balloon catheters:
 - Used primarily when instrumentation fails
 - 5F or 6F Foley or Fogarty balloon catheter lubricated with 2% lidocaine jelly
 - Advance catheter past object
 - Following inflation with 2–3 mL of air, gently withdraw catheter
- Magnet for removal of metal foreign body described; limited experience
- Snare technique:
 - 24G wire made into a loop with a hemostat

- Useful when size of object known
- Thin wire can slip through swollen tissue, behind object, allowing it to be pulled free

MEDICATION

- Cocaine: 4% solution, 2 drops affected nares
- Lidocaine: 4% solution, 2 drops affected nares
- Oxymetazoline: 0.05%, 2–3 drops/sprays affected nares
- Phenylephrine: 0.125–0.5%, 2–3 sprays affected nares
- Procedural sedation may be necessary



FOLLOW-UP

DISPOSITION

Admission Criteria

Referral for ambulatory surgical removal:

- Foreign body cannot be recovered in ED
- Removal under general anesthesia is required

Discharge Criteria

- Ensure that there is no airway compromise
- Return if bleeding, infection (nasal discharge)
- If a button battery was removed, monitor for delayed sequelae as outpatient:
 - Ischemic mucosa
 - Turbinate or septal damage
 - Saddle-nose deformity

Issues for Referral

- Follow up with otolaryngologist if:
 - Removal unsuccessful in ED
 - Concern for nasal mucosa injury

FOLLOW-UP RECOMMENDATIONS

- Return to the ED immediately if:
 - Coughing, wheezing, noisy, or difficult breathing
 - Vomiting, gagging, choking, drooling, neck or throat pain, or inability to swallow
- Parents should be instructed to seek medical care for the following:
 - Fever
 - Headache or facial pain
 - Persistent epistaxis

- Persistent drainage of nasal fluid

PEARLS AND PITFALLS

- Consider nasal foreign bodies in children 2–6 yr presenting with what appears to be sinusitis
- Parents are best suited to perform positive-pressure removal to avoid frightening the child
 - Often successful, with little/no sedation
 - Can make other techniques more likely to succeed, even if it fails
- Mix equal parts Lidocaine 4% with oxymetazoline to deliver simultaneously

ADDITIONAL READING

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CODES

ICD9

932 Foreign body in nose

ICD10

T17.1XXA Foreign body in nostril, initial encounter

FOREIGN BODY, RECTAL

Joanna W. Davidson



BASICS

DESCRIPTION

- Self-insertion (autoeroticism):
 - Phallic substitutes inserted by patient or partner
 - Usually men aged 20–40 yr, with male to female ratio 20:1
- Ingested objects lodged in rectum:
 - Chicken bones
 - Fish bones
 - Toothpick
- Iatrogenic accidental:
 - Thermometer
 - Enema tips
 - Foreign bodies (FBs) used to aid in removal of feces
- Assault:
 - Knife or pipe forcibly inserted
 - Incidence of perforation is very high.
- Concealment:
 - Body packing, “mules” illegally transporting drugs



DIAGNOSIS

SIGNS AND SYMPTOMS

- Complaint of rectal FB
- Rectal fullness
- Rectal pain
- Perirectal abscess (with imbedded bones/toothpick)
- FB on rectal exam:
 - High-lying FBs are located proximal to rectosigmoid junction and are not palpable on rectal exam.
 - Low-lying FBs are usually located in rectal ampulla and are palpable on rectal exam.
- Some patients may not be forthcoming with history
- Can present with vague symptoms of abdominal pain or obstruction
- Can present as bowel perforation with full peritonitis
- Often late presentation hours or days after placement, following repeated failed attempts at removal
- Rectal Organ Injury Scale (proposed by American Association for the Surgery of

Trauma):

- Grade I—Hematoma: Contusion or hematoma without devascularization:
 - Most injuries due to rectal FB are Grade I
- Grade II—Laceration 50% circumference
- Grade III—Laceration >50% circumference
- Grade IV—Full-thickness laceration with extension into perineum
- Grade V—Devascularized segment

ESSENTIAL WORKUP

- Identify number, type, and duration of FBs and mechanism of insertion.
- Physical exam with emphasis on abdominal and rectal exam
 - Classified as high-riding vs. low-riding based on relationship to rectosigmoid junction
- Biplane radiographic films to confirm number and size of FBs
- Serious injury more common with assault

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC:
 - For bleeding or peritonitis
- Urinalysis:
 - For urethral/bladder injuries

Imaging

- Plain radiograph:
 - Consider doing kidneys, ureters, and bladder (KUB) radiograph prior to rectal exam to rule out objects harmful to examiner.
 - Define and locate FB.
 - Assess for complications of retained FB including bowel perforation and obstruction.
 - May be used serially to follow descent of FB
- CT scan of abdomen/pelvis:
 - To exclude perforation or abscess formation

DIFFERENTIAL DIAGNOSIS

- Pseudo-FB:
 - Patients insist there is FB when radiograph, rectal exams, and proctoscopy results are normal.
- Perirectal abscess
- Hemorrhoid



TREATMENT

PRE HOSPITAL

Cautions:

- Patient has usually tried to remove FB and failed.
- Further attempts at extraction will not work and could cause perforation.

INITIAL STABILIZATION/THERAPY

- Perforation with peritonitis and sepsis:
 - _ 0.9% NS IV fluid 500 mL bolus
 - _ Broad-spectrum antibiotics (anaerobic and gram-negative aerobes):
 - Cefoxitin, cefotetan, ticarcillin–clavulanate, ampicillin–sulbactam, imipenem, meropenem, ertapenem, *or*
 - Metronidazole/clindamycin _+ aminoglycoside/3rd-generation cephalosporin/fluoroquinolone/aztreonam
 - _ Urgent surgical consult
- Advanced trauma life support (ATLS) with evidence of other trauma

ED TREATMENT/PROCEDURES

- Appropriate sedation and analgesia is important to overcome spasm, rectal edema.
- Avoid enemas or suppositories.
- Low-lying small rectal FBs that are not fragile or sharp are candidates for ED removal:
 - _ Firmly hold bimanually or with forceps
 - _ Remove with gentle but firm continuous traction to overcome anal sphincter.
 - _ Colonic mucosa tightly adherent to distal end of FB creates vacuum and impedes withdrawal of object:
 - Passage of Foley catheter beyond object with insufflation of air breaks vacuum and permits retrieval.
 - _ Awake and cooperative patients can facilitate transanal extraction with valsalva.
 - _ May use instruments to assist with extraction: Obstetrical forceps, tenaculum, ring forceps, vacuum extractor
 - _ 60% of rectal FBs may be removed transanally in the ED under proper sedation.
 - _ Following extraction, anorectum must be thoroughly evaluated to rule out occult injury.
- High-lying rectal FBs:
 - _ Not immediately accessible through rectum
 - _ Usually require surgical or GI consult

- Attempt may be made to position object into low-lying position with gentle abdominal pressure
- Avoid blind transanal removal
- Direct visualization with lubricated operating anoscope (after blockage of sphincter and pudendal nerve with local anesthesia)
- Admission and observation for spontaneous descent (with serial radiographs)
- Laparotomy may be necessary as last resort if other methods fail, or if patient has evidence of perforation.
- Consider surgical or GI consult for other complicated rectal FBs:
 - Larger objects
 - Objects that have remained > 24 hr with resulting edema
 - Objects with sharp edges
 - Proctoscopy/sigmoidoscopy after extraction to examine colonic mucosa
- Body packers:
 - Ruptured packets of concealed illicit drugs can cause systemic toxicity, bowel necrosis, and death.
 - Sharp instruments should not be used for retrieval, and other instruments should be used with extreme caution.

MEDICATION

- Ampicillin–sulbactam (Unasyn): 3g IV q6h (peds: 100–200 mg/kg/d div. q6h)
- Ceftriaxone (Rocephin): 1–2 g IV q12h (peds: 50–75 mg/kg IV daily)
- Ciprofloxacin (Cipro): 400 mg IV q8–12h
- Clindamycin: 600–900 mg (peds: 20–40 mg/kg/24h) IV q8h
- Levofloxacin (Levoquin): 500 mg IV q24h
- Metronidazole: 15 mg/kg IV once, then 7.5 mg/kg IV q6h
- Piperacillin–tazobactam (Zosyn): 3.75 g IV q6h or 4.5 g IV q8h (peds: 240–400 mg/kg/d div. q6–8h)

Pediatric Considerations

- Removal under general anesthesia for children who are too young to cooperate
- It is probably child abuse if FB other than enema tips or thermometer is present.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Failed extraction in ED requires surgical removal in the operating room.
- Evidence of mucosal tear on proctoscopy should be observed for 24 hr (no antibiotic indicated).

- Symptom of rectal pain associated with removal of sharp FB indicates possibility of small perforation with developing abscess and requires exam under anesthesia.

Discharge Criteria

- Reliable patient with atraumatic insertion and removal of rectal FB
- Instruct to return for rectal pain, abdominal pain, fever, or massive rectal bleeding.

Issues for Referral

GI or surgery consult if unable to remove FB in ED

FOLLOW-UP RECOMMENDATIONS

Flexible sigmoidoscopy or rigid proctoscopy to evaluate for mucosal injury following retrieval of rectal FB regardless of method used is recommended.

PEARLS AND PITFALLS

- Passage of Foley catheter beyond object with insufflation of air breaks vacuum and permits retrieval.
- Provide adequate sedation/analgesia when attempting FB removal in the ED.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Rectal Trauma



CODES

ICD9

937 Foreign body in anus and rectum

ICD10

T18.5XXA Foreign body in anus and rectum, initial encounter

FOURNIER GANGRENE

Gary M. Vilke



BASICS

DESCRIPTION

- Inadequate hygiene leads to scrotal skin maceration and excoriation:
 - Portal of entry for bacteria in tissue
- Once skin barrier is broken, polymicrobial flora spread along *fascial planes* of perineum.
- Colles fascia fuses with urogenital diaphragm, slowing propagation posteriorly and laterally.
- Anteriorly, Buck and Scarpa fascia are continuous, allowing rapid extension to anterior abdominal wall and laterally along fascia lata.
- Testes and urethra are usually spared.
- 3 anatomic origins account for most cases:
 - Lower urinary tract (40%): Urethral strictures, indwelling catheters
 - Penile or scrotal (30%): Condom catheters, hydradenitis, balanitis
 - Anorectal (30%): Fistulas, perirectal infections, hemorrhoids
- Rarely, intra-abdominal sources such as perforating appendicitis, diverticulitis, or pancreatitis have produced Fournier gangrene by dependent contiguous spread.

ETIOLOGY

- Infection by polymicrobial flora (mixed aerobic and anaerobic organisms)
- Mixed bacteria exert synergistic tissue-destructive effect.
- End arterial thrombosis in subcutaneous tissues produces anaerobic environment.
- Bacterial toxins and tissue necrosis factors may contribute to clinical presentation.
- Risk factors:
 - Trauma
 - Diabetes
 - Alcoholism
 - Other immunocompromised states
 - Morbid obesity
 - Abdominal surgery



DIAGNOSIS

SIGNS AND SYMPTOMS

- Rapidly progressive necrotizing infection of *perineum* involving subcutaneous and fascial tissues and often muscle layers:
 - Usually seen in diabetics or immunocompromised patients

- Sources of infection may be flora from genitourinary, rectal, or penile/scrotal regions.

Pediatric Considerations

- Though unusual in children, > 50 cases have been described.
- Most often are complications of burns, circumcision, balanitis, severe diaper rashes, or insect bites
- Organisms are more frequently *Staphylococcus* or *Streptococcus*.
- Pediatric patients have more local disease and are less toxic.

History

- Duration of symptoms:
 - _ Fevers or chills
 - _ Pain is out of proportion to exam in early phases, but eventually dead tissue becomes insensate.
 - _ Nausea and vomiting
 - _ Urinary infection symptoms
- Rapidity with which symptoms are progressing
- Identify if diabetic or immunocompromised
- Lethargy and inappropriate indifference to the illness are common.

Physical-Exam

- Patients are often toxic in appearance with nausea, vomiting, fever, chills, and pain.
- Careful exam of the genitalia and perirectal region
- Assess for skin findings:
 - _ Bronze or violaceous discoloration of skin
 - _ Thin brown watery discharge
 - _ Ulceration, bullous vesicles
 - _ Crepitance, SC air
 - _ Frank necrosis and eschar formation

ESSENTIAL WORKUP

- Fournier gangrene is a clinical diagnosis.
- History and physical exam with special attention to perineum
- Evaluate for signs of sepsis.
- Early surgical consultation for emergent débridement is essential.
- Other workup directed toward relevant comorbid factors such as diabetes or immunocompromised status

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Other than Gram stain of tissue and associated drainage, there are no specific lab tests that are diagnostic of Fournier gangrene.
- Urinalysis should be performed.
- Leukocytosis, anemia, electrolyte imbalances, acidosis, and renal failure are common.
- Disseminated intravascular coagulation (DIC) may be present; PT, PTT, fibrin-split products, and fibrinogen levels help identify.
- If patient is suspected of or known to have diabetes, glucose, electrolytes, and serum ketones to evaluate for diabetes and diabetic ketoacidosis (DKA)
- Culture of blood, urine, and tissue (when available)

Imaging

- Plain films of the pelvis may reveal subcutaneous emphysema and ileus.
- CT scanning helps if intra-abdominal or ischiorectal source is suspected.
- US may be useful in differentiating from other causes of acute scrotum.

Diagnostic Procedures/Surgery

Retrograde urethrography, anoscopy, proctosigmoidoscopy, and barium enemas may be helpful to localize anatomic sources of infection.

DIFFERENTIAL DIAGNOSIS

- Epididymitis/orchitis
- Insect and human bites
- Perirectal infections
- Scrotal abscess/inguinal abscess
- Scrotal cellulitis
- Testicular torsion
- Tinea cruris



TREATMENT

PRE HOSPITAL

Patients may be hypotensive from septic shock and require aggressive fluid resuscitation and vasopressor support.

INITIAL STABILIZATION/THERAPY

- Manage airway and resuscitate as indicated.
- Central venous access, aggressive fluid resuscitation, and pressure support as indicated:
 - Avoid femoral access, femoral venipuncture, and lower extremity venous access
- Early goal-directed therapy if septic

- Foley catheter placement or suprapubic access if indicated

ED TREATMENT/PROCEDURES

- Empiric broad-spectrum antibiotics
- Early emergent aggressive surgical débridement
- Adjunctive hyperbaric oxygen therapy coordinated with surgical care
- Treat dehydration and correct electrolytes.
- Blood products as needed for DIC or anemia; oxygen debt can be minimized by keeping hematocrit > 30%.
- Tetanus prophylaxis as indicated

Pediatric Considerations

- More conservative surgical approach
- Adequate staphylococcal coverage

MEDICATION

- Antibiotic regimens:
 - Multidrug regimen:
 - Ampicillin: 2 g IV q6h (peds: 50 mg/kg) *and*
 - Clindamycin: 900 mg IV q8h (peds: 10 mg/kg) *and*
 - Gentamicin: 5 mg/kg daily load IV q8h
 - Ciprofloxacin: 500 mg IV *and*
 - Clindamycin: 900mg IV initial ED dose
 - Single-drug regimens (peds: Safety not established)
 - Ampicillin/sulbactam: 3 g IV initial ED dose
 - Imipenem: 1 g IV initial ED dose
 - Piperacillin/tazobactam: 3.375 g IV initial ED dose
 - Ticarcillin/clavulanate: 3.1 g IV initial ED dose
- Cover for possible MRSA with Vancomycin 1 g IV initial ED dose
- Blood products as indicated
- Dopamine or dobutamine IV drips starting at 5 µg/kg/min titrating to effect if hypotensive after aggressive hydration
- Insulin adjusted to control glucose and acidosis



FOLLOW-UP

DISPOSITION

Admission Criteria

- All patients with Fournier gangrene require admission and surgical ICU care.
- Mortality estimates of 3–38% emphasize need for early aggressive care.
- Consider early transfer to facility capable of providing adjunctive hyperbaric

oxygen therapy if stable for transport.

Discharge Criteria

No patients with Fournier gangrene should be discharged.

PEARLS AND PITFALLS

- Failure to perform a careful genital exam, particularly in a pediatric patient
- Failure to initiate antibiotics in a timely manner

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Cellulitis
- Urinary Tract Infection, Adult



CODES

ICD9

608.83 Vascular disorders of male genital organs

ICD10

N49.3 Fournier gangrene

FRACTURE, OPEN

Christy Rosa Mohler

BASICS

DESCRIPTION

- Continuity between skin violation and fracture site, ranging from a puncture wound to grossly exposed bone
- Surgical urgency, as delays in care increase risk of infection and rate of complications
- Predisposition to complications in certain patients:
 - Massive soft tissue damage
 - Severe wound contamination
 - Compromised vascularity
 - Fracture instability
 - Compromised host (diabetes, vascular disease)

ETIOLOGY

Open fractures typically result from significant blunt force or penetrating trauma.

DIAGNOSIS

SIGNS AND SYMPTOMS

- Deformity with nearby violation in skin integrity
- Neurovascular compromise may occur.
- Additional traumatic injuries are frequently present.

History

Significant trauma

Physical-Exam

- Complete neurologic and vascular exam
- Examine thoroughly for other traumatic injuries.

ESSENTIAL WORKUP

- Plain radiographs including joints above and below the affected area
- Guided workup based on mechanism and evidence of other traumatic injuries

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC, chemistry panel, coagulation studies for large-bone (femur, pelvis) fractures or multiple-trauma victims
- Type and screen or type and cross-match for potential of significant blood loss.
- Predébridement and postdébridement cultures have limited value and are not recommended.

Imaging

Doppler or angiography if vascular damage is suspected:

- Posterior knee dislocation
- Ischemic extremity
- Massive soft tissue injury in high-risk areas

Diagnostic Procedures/Surgery

- Measurement of compartment pressures if concern for compartment syndrome
- Consider arthrogram by intra-articular injection of saline or methylene blue if joint involvement is suspected.
- Angiography if noninvasive techniques are inadequate for ruling out vascular compromise

DIFFERENTIAL DIAGNOSIS

Noncontiguous laceration/abrasion



TREATMENT

PRE HOSPITAL

- Moist, sterile dressings over open wounds
- Immobilize joints above and below fracture.
- Control bleeding with local compression.
- Consider tourniquet for traumatic amputations or uncontrollable hemorrhage.
- Longitudinal traction of involved extremity if distal pulses absent

INITIAL STABILIZATION/THERAPY

- Management of ABCs.
- Gentle reduction and immobilization of fracture

ED TREATMENT/PROCEDURES

- Intravenous access
- Keep patient NPO
- Tetanus vaccination, if needed
- Antibiotics reduce the incidence of early infection in open fractures and should be given early in the ED course.
- Minimize number of times dressing is removed to avoid secondary contamination.

- Examine limb regularly for compartment syndrome and neurovascular status.
- Certain large joint open fracture/dislocations should be reduced emergently in the ED (ankle, elbow, knee)
- Urgent orthopedic consultation for formal irrigation, débridement, and possible operative fixation.
- Vascular surgery consultation for injuries with potential vascular damage

MEDICATION

- Cefazolin: 1–2 g (peds: 20 mg/kg IM/IV)
- Add gentamicin: 1.5–2 mg/kg IV for more extensive injuries and highly contaminated wounds (peds: 2–2.5 mg/kg IV)
- Add penicillin G: 4–5 million U IV in farmyard injuries, vascular injuries, and in wounds at risk of contamination with *Clostridium* (peds: 50,000 U/kg IV)
- Tetanus booster: 0.5 mL IM
- Tetanus immunoglobulin: 250 IU IM if not previously immunized against tetanus
- Morphine sulfate: 2–10 mg (peds: 0.05–0.1 mg/kg per dose IV or equivalent analgesic)

Pediatric Considerations

DTaP booster for children <7 yr of age



FOLLOW-UP

DISPOSITION

Admission Criteria

Most patients will be admitted for irrigation, débridement, IV antibiotics, and possibly operative fixation.

Discharge Criteria

Simple open fractures may be washed out and immobilized in the ED after consultation with an orthopedic surgeon. The patient should be discharged with oral antibiotics.

Issues for Referral

Most open fractures will require emergent orthopedic consultation and may require trauma team evaluation for other injuries.

FOLLOW-UP RECOMMENDATIONS

Patients discharged from the emergency department should be followed up with an orthopedic surgeon in 1–2 days.

PEARLS AND PITFALLS

- Open fractures are surgical urgencies requiring prompt orthopedic consultation.
- 40–70% of patients with open fractures have other traumatic injuries.
- Prompt and thorough ED assessment and treatment can significantly decrease morbidity in patients with open fractures.

ADDITIONAL READING

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CODES

ICD9

- 818.1 Ill-defined open fractures of upper limb
- 827.1 Other, multiple and ill-defined fractures of lower limb, open
- 829.1 Fracture of unspecified bone, open

ICD10

- S52.90XB Unsp fracture of unsp forearm, init for opn fx type I/2
- S82.90XB Unsp fracture of unsp lower leg, init for opn fx type I/2
- T14.8 Other injury of unspecified body region

FRACTURES, PEDIATRIC

Adam Z. Barkin



BASICS

DESCRIPTION

- 20% of pediatric patients with acute traumatic injuries will have a fracture
- Boys have fractures more commonly than girls
- Anatomy:
 - Diaphysis: Pysis to pysis; bone shaft
 - Epiphysis: Cartilaginous center at or near end of bone that is site of bone growth
 - Epiphyseal (growth) plate: Radiolucent line between epiphysis and metaphysis; cartilaginous
 - Metaphysis: Region of rapidly growing trabecular bone underlying base of cartilaginous growth plate; between diaphysis and epiphysis
 - Most long bones are ossified by the end of puberty
- Bones are highly resilient, elastic, and springy
- Allow for fractures not seen in adults:
 - Greenstick fracture:
 - Incomplete fracture through cortex on opposite side of impact
 - Torus (buckle) fracture:
 - Usually at junction of metaphysis and diaphysis
 - Compression of bone of 1 cortex
 - Plastic deformity:
 - Bowing without disruption of cortex
 - Fractures involving the physis
- Cartilaginous growth plates are potential areas of injury.
- Ligaments more resistant to injury than growth plates
- Salter–Harris classification:
 - Risk of growth disturbance increases from type I to type V.
 - Type I:
 - Separation of epiphysis from metaphysis without displacement or injury to the growth plate
 - Tenderness and pain at point of growth plate
 - Radiograph typically normal
 - Growth disturbance is rare.
 - Type II:
 - Metaphyseal fracture extending to physis
 - Most common
 - Growth disturbance is rare.

- Type III:
 - Intra-articular fracture extending through the epiphysis into the physis
 - Most common site is distal tibial epiphysis.
 - Growth disturbance possible
- Type IV:
 - Epiphyseal, physeal, and metaphyseal fracture
 - Lateral condyle of humerus is the most common site.
 - Growth disturbance highly likely
- Type V:
 - Crush injury to epiphyseal plate, producing growth arrest
 - Usually occurs in joints that move in only 1 plane such as knee
- Fractures often accompany dislocations.
- Nonaccidental trauma (NAT) if history inconsistent with findings

ETIOLOGY

- Mechanism is useful in defining the potential and type of injury
- Obesity and rapid growth spurts are risk factors.
- NAT:
 - Any fracture in a child younger than 1 yr of age in whom history is not consistent with injury
 - Metaphyseal “corner” fractures are pathognomonic.
 - Posterior rib fractures
 - Spiral femur fracture
 - Fractures at different stages of healing
 - Skull fractures crossing suture lines, especially in children younger than 1 yr
 - Unusual behavior in child or parent



DIAGNOSIS

SIGNS AND SYMPTOMS

- Decreased limb movement, unwilling to use
- Swelling
- Tenderness
- Deformity
- Ecchymosis
- Crepitus
- Limp
- Abnormal neurovascular status of extremity
- Compartment syndrome:
 - Severe pain, especially in forearm, calf, foot
 - Pain with passive stretching of fingers or toes
 - Sensory deficit in the distal extremity

- Cool extremity
- Pulseless extremity
- Open fracture may be obvious or subtle (collection of blood with fat globules under skin)

History

- Mechanism of injury:
 - Velocity of car, bike, etc.
 - Height of fall
- Neurologic compromise
- Events surrounding injury
- Other injuries

Physical-Exam

- Thorough secondary survey looking for deformities, bruising, other injuries
- Assess neurovascular status:
 - Motor/sensation
 - Distal pulses
 - Capillary refill
- Range of motion of all joints involved
- Exclude concurrent injuries
- Ensure that history is consistent with injury

ESSENTIAL WORKUP

- Prompt immobilization
- Imaging as below

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Required only if concomitant injuries, surgery anticipated, or multiple/major bone involvement
- CBC, ESR if infection suspected

Imaging

- Anteroposterior (AP), lateral, and oblique radiographs as necessary, including the joint above and below the fracture
- Comparison views may be useful if growth plates are involved.
- Follow-up radiographs at 7–10 days may be required to exclude avascular necrosis or Salter I fractures.
- Bone scan/CT/MRI may be useful to exclude fractures if plain radiographs are unhelpful or to evaluate for infection.

Diagnostic Procedures/Surgery

Arthrocentesis if infection is suspected

DIFFERENTIAL DIAGNOSIS

- Sprain or strain
- Contusion
- Infection
- Tumor
- Neurologic deficits
- Subtle dislocations such as radial head subluxation (nursemaid's elbow)
- NAT



TREATMENT

PRE HOSPITAL

Immobilization

INITIAL STABILIZATION/THERAPY

- Resuscitation for concurrent injuries
- Immobilization

ED TREATMENT/PROCEDURES

- Management of life-threatening concurrent injuries
- Pain control
- Dislocations require immediate assessment and attention to neurovascular compromise:
 - Mechanism helps in understanding the direction of the force required to reduce.
- Alignment is essential, particularly when fracture involves a joint surface.
- Appropriate reporting of NAT

Salter-Harris Fractures

- Type I and type II fractures require immobilization and orthopedic follow-up.
- Type II distal femur fractures, type III, and type IV require urgent orthopedic consultation for anatomic reduction.
- Type V fractures require immobilization and consultation.
- Anatomic reduction does not eliminate possibility of growth disturbance.

Clavicle Fracture

- Figure-of-8 splint or sling for comfort
- Distal 3rd clavicle fractures should be referred with initial sling and swathe or shoulder immobilizer.

Elbow Fracture

- > 50% are supracondylar
 - 10–15% have neural injury
- May present with only posterior effusion on lateral radiograph
- Orthopedic consultation because of potential neurovascular complications
- Brachial artery injury, median nerve injury possible
- Volar compartment syndrome of forearm (results in Volkmann contracture)
- Epiphyseal injury with long-term growth abnormalities

Distal Radius and Ulna Fractures

- Most common site of pediatric fracture: Distal radius
- Reduce angulated fractures > 15°
- Pronator fat pad along volar radius may indicate occult fracture
- Colles fracture:
 - Reduce by traction in the line of deformity to disimpact the fragments, followed by pressure on the dorsal aspect of the distal fragment and volar aspect of the proximal fragment.
 - Correct radial deviation.
 - Immobilize wrist and elbow (sugar-tong splint)
 - Orthopedic consultation
- Torus fracture (incomplete fracture; buckling or angulation on the compression side of the bone only):
 - Most often in distal forearm
- Greenstick fracture (incomplete fracture of diaphysis of long bone with fracture on tension side of cortex):
 - Immobilize.
 - Reduction if angulation > 30° in infants, > 15° in children

Tibia or Fibula Fracture

- Isolated fibular fractures: Short-leg walking cast
- Nondisplaced tibial fracture: Long-leg posterior splint, nonweight bearing
- Displaced tibial fracture and complex fractures require consultation.
- Toddler's fractures:
 - Nondisplaced, oblique, distal tibia fracture
 - May need tangential view radiograph or bone scan to diagnose
 - Splint if suspect and repeat radiograph in 7–10 days.
- May apply Ottawa Ankle Rules to children

Slipped Capital Femoral Epiphysis

- Disruption through capital femoral epiphysis
- Need AP and frog-leg x-rays
- Overweight adolescent boys

- May have referred pain to knee, thigh, or groin
- Nonweight bearing with prompt orthopedic follow-up
- Often bilateral

Femur Fracture

- Most common long-bone fracture

Stress Fractures

- Increasingly common
- Insidious onset
- Vague, achy pain
- Usually associated with rigorous activity
- Treatment:
 - _ Selective bracing
 - _ Activity modification

Open Fractures

- Irrigate and dress with moist saline gauze
- Immobilize
- Cefazolin if only small laceration and minimal contamination
- Gentamicin if moderate contamination, high-energy injury, or significant soft tissue injury
- Consider penicillin if concern for clostridia infection (farm injury, fecal or soil contamination)
- Small wounds with minimal soft tissue injury may be treated with oral antibiotics and immobilization in consultation with orthopedist

Child with Limp

- Careful exam and review of systems for signs of rheumatologic disease, infection, or malignancy
 - _ Pediatric patients with leukemia may present with limp as their initial complaint
- CBC, ESR, CRP, arthrocentesis may be indicated
- Transient synovitis vs. septic hip
 - _ More likely septic if:
 - Fever
 - Elevated ESR/CRP
 - WBC elevation
 - Refusal to bear weight

MEDICATION

- Acetaminophen: 10–15 mg/kg PO(PR (per rectum) q4–6h; Do not exceed 5

doses/24 h

- Cefazolin: 25–100 mg/kg daily IM/IV q8h
- Gentamicin: 2.5 mg/kg IV/IM q8h or 6.5–7.5 mg/kg IV/IM q24h
- Hematoma block: 1% lidocaine without epinephrine (max. 3–5 mg/kg)
- Ibuprofen: 10 mg/kg PO q6–8h (first-line treatment)
- Morphine: 0.05–0.2 mg/kg SC/IM/IV q2–4h



FOLLOW-UP

DISPOSITION

Admission Criteria

- NAT (or per social services)
- Open fracture
- Potential neurovascular compromise/compartment syndrome:
 - Condylar or supracondylar humerus fracture
 - Femoral shaft

Discharge Criteria

- Uncomplicated fracture: No concurrent injury or neurovascular/compartment compromise
- Follow-up arranged and parents understand injury and management

Issues for Referral

All Salter–Harris fractures should have orthopedic follow-up.

PEARLS AND PITFALLS

- History is essential in evaluation of NAT
- Undress patient fully especially if suspicion for NAT
- Have a low threshold to splint and/or consult orthopedist
- Pain control is essential and often underdosed.
- Distal radius is often associated with other fractures: Ulna, elbow, carpal bones

ADDITIONAL READING

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- Chasm RM, Swencki SA. Pediatric orthopedic emergencies. *Emerg Med Clin North Am*. 2010;28:907–926.
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See Also (Topic, Algorithm, Electronic Media Element)

- Conscious Sedation
- C-spine Fractures, Pediatric
- Fractures, Open
- Nursemaid's Elbow
- Shoulder Dislocation
- Slipped Capital Femoral Epiphysis



CODES

ICD9

- 803.00 Other closed skull fracture without mention of intracranial injury, unspecified state of consciousness
- 807.00 Closed fracture of rib(s), unspecified
- 829.0 Fracture of unspecified bone, closed

ICD10

- S02.91XA Unsp fracture of skull, init encntr for closed fracture
- S22.39XA Fracture of one rib, unsp side, init for clos fx
- T14.8 Other injury of unspecified body region

FROSTBITE

Joseph M. Weber

BASICS

DESCRIPTION

- Tissue damage caused by cold temperature exposure
- Mechanism:
 - Tissue damage results from:
 - Direct cell damage: Intracellular ice crystal formation
 - Indirect cell damage: Extracellular ice crystal formation leads to intracellular dehydration and enzymatic disruption.
 - Reperfusion injury: Occurs upon rewarming. Fluid rich in inflammatory mediators (prostaglandin and thromboxane) extravasates through damaged endothelium promoting vasoconstriction and platelet aggregation.
 - Clear blisters form from extracellular exudation of fluid.
 - Hemorrhagic blisters occur when deeper subdermal vessels are disrupted, indicating more severe tissue injury.
 - The end result is arterial thrombosis, ischemia, and ultimately, necrosis.
 - Devitalized tissue demarcates as the injury evolves over weeks to months, hence the phrase “frostbite in January, amputate in July.”

ETIOLOGY

- Cold exposure: Duration of exposure, wind chill, humidity, and wet skin and clothing all increase the likelihood of frostbite.
- Predisposing factors:
 - Extremes of age
 - Altered mental status (intoxication or psychiatric illness)
 - Poor circulatory status

DIAGNOSIS

SIGNS AND SYMPTOMS

- Extremities (fingers, toes) and head (ears, nose) most commonly affected.
- After rewarming frostbite can be classified; however, initial classification often fails to provide an accurate prognosis and does not alter initial management.
- Superficial frostbite:
 - Only skin structures involved. Usually no tissue loss.
 - 1st degree: Erythema and edema with stinging, burning, and throbbing. No

- blisters or necrosis.
- 2nd degree: Significant edema, clear blister formation. Numbness common.
- Deep frostbite:
 - Tissue loss inevitable.
 - 3rd degree: Involves subcutaneous tissue. Hemorrhagic blister formation due to subdermal venous plexus injury:
 - Initially insensate, injuries develop severe pain/burning on rewarming.
 - 4th degree: Involves muscle, tendon, and bone. Initially mottled, deep red, or cyanotic.
 - Unfavorable prognostic indicators include: Hemorrhagic blisters, persistent cyanosis, mottling, anesthesia, and reduced mobility after rewarming.
 - Devitalized tissue demarcates as the injury evolves over weeks to months forming skin necrosis and dry black eschar.

ESSENTIAL WORKUP

- Diagnosis is based on the clinical presentation. Wound description should include skin color and temperature, blister formation and color, and soft tissue consistency.
- A neurologic and vascular exam should include pulses (by Doppler if necessary), cap refill, and 2-point discrimination.
- Look for underlying factors contributing to cold exposure and comorbid conditions requiring emergency management:
 - Hypothermia
 - Trauma
 - Hypoglycemia
 - Cardiac or neurologic problems
 - Intoxication/overdose
 - Compartment syndrome

DIAGNOSIS TESTS & INTERPRETATION

Lab

- None indicated in mild cases
- For deep frostbite:
 - CBC
 - Electrolytes, BUN/creatinine, glucose
 - Urinalysis/CK for evidence for myoglobinuria
- Cultures and Gram stains from open areas when infection suspected

Imaging

Technetium-99 scintigraphy or MRA:

- May be helpful in early identification of salvageable vs. unsalvageable tissue

- Permits earlier decision about amputation

Diagnostic Procedures/Surgery

Method to create a warm water bath in the ED:

- Whirlpool hydrotherapy ideal, however, most EDs do not have
- Mix hot and cold tap water from a standard hospital sink in a large basin
- Use a thermometer to keep temperature between 40°C and 42°C.
- The water will cool quickly: Intermittently add warm water or replace the water to keep the temperature in the proper range.
- Warmer temperatures can cause thermal injury while cooler temperatures delay thawing and decrease tissue survival.

DIFFERENTIAL DIAGNOSIS

- Frostnip:
 - Superficial, reversible ice crystal formation without tissue destruction
 - Transient numbness and paresthesia resolve after dry rewarming.
- Trench (immersion) foot:
 - Exposure to wet cold for prolonged periods
 - Neurovascular damage without ice crystal formation
 - Pallor, mottling, paresthesias, pulselessness, paralysis, and numbness
 - May be difficult to distinguish from post-thaw phase of frostbite
 - Hyperemia with dry rewarming may last up to 6 wk.
- Chilblains:
 - Chronic repeated exposure to dry cold
 - Localized erythema, cyanosis, plaques, and vesicles
 - Recurrent episodes common in patients with underlying vasculitis
 - Symptomatic treatment, dry rewarming



TREATMENT

PRE HOSPITAL

- Protect and immobilize frostbitten area during transport
- Remove restrictive or wet garments
- Avoid dry rewarming of the frostbitten limb if there is a likelihood of refreezing injury during transport.
- If evacuation will be delayed and suitable facilities are available, field rewarming in warm (40°C–42°C) water can be attempted.
- Rubbing, manipulating the limb, or applying snow while it is still frozen is contraindicated.

ALERT

- Hypothermia:

- Common in frostbite victims
- In the severely hypothermic patient, avoid rough handling to minimize risk of cardiac dysrhythmias.

INITIAL STABILIZATION/THERAPY

- ABCs management
- Identify and correct hypothermia.
- IV fluid volume expansion with 0.9% NS for severe frostbite
- Protect frostbitten areas from excessive handling during resuscitation.

ED TREATMENT/PROCEDURES

- If the injury is <24 hr old and has not yet been rewarmed:
 - Initiate rapid rewarming of the frostbitten extremity in a 40–42°C water bath for 15–30 min.
 - Stop treatment when the limb is warm, red, and pliable.
 - Monitor water temperature closely to prevent thermal injury.
- Analgesia: IV morphine
- NSAIDs (e.g., ibuprofen) to combat the effects of prostaglandins on skin necrosis.
- Aloe vera topical cream:
 - Recommended for all intact blisters
 - Combats the arachidonic acid cascade
 - Avoid preparations containing alcohol, scent, salicylates, all of which interfere with aloe effectiveness.
- Blister débridement or aspiration:
 - Indicated for clear blebs:
 - Removes thromboxane and prostaglandins
 - Contraindicated for hemorrhagic blebs:
 - Exposes deeper structures to dehydration and infection
- Tetanus prophylaxis
- Antibacterial prophylaxis:
 - Consider during the hyperemic recovery phase (at least 2–3 days) in severely frostbitten areas
 - Against *Streptococci*, *Staphylococci*, and *Pseudomonas* species (cephalosporin, penicillinase-resistant penicillin, quinolone)
 - Topical antibacterial agents interfere with the use of aloe vera cream and should be considered a 2nd-line approach.
- Elevation and splinting of frostbitten area
- Change dressing 2–4 times daily.
- Avoid vasoconstrictive agents (including tobacco).
- Adjunctive treatments include:
 - Thrombolytic therapy (<24 hr of cold exposure):
 - Both intra-arterial and systemic tPA may improve tissue salvage rates.
 - Consult with plastic/burn surgeon before treatment.

- Vasodilator therapy:
 - Pentoxifylline—limited data
 - Iloprost—limited data and availability

MEDICATION

- Aloe vera: Topical cream (70% concentration) q6h
- Cephalexin (cephalosporin): 500 mg (peds: 25–50 mg/kg/24h q6h) PO QID
- Ciprofloxacin (quinolone): 500 mg PO BID
- Dicloxacillin (penicillinase-resistant penicillin): 500 mg (peds: 12.5–25 mg/kg/24h q6h) PO QID
- Ibuprofen (NSAID): 800 mg (peds: 40 mg/kg/24h q6–8h) PO TID
- Morphine sulfate: 0.1–0.2 mg/kg (peds: 0.1 mg/kg) IV or IM PRN (titrate to patient response)



FOLLOW-UP

DISPOSITION

Admission Criteria

- All but the most superficial cases should be admitted.
- Lower admission threshold where risk of refreezing exists.
- Immersion (trench) foot patients may be discharged only if an environment that allows for proper treatment can be provided.

Discharge Criteria

Minimal superficial injury, all others should be admitted.

Issues for Referral

General, burn, plastic, or hand surgeon should be consulted in all but the most superficial of cases.

FOLLOW-UP RECOMMENDATIONS

All discharged patients should be referred to a general, burn, plastic, or hand surgeon.

PEARLS AND PITFALLS

Pitfalls:

- Allowing freeze, thaw, refreeze cycle to occur
- Failure to keep warm water bath between 40°C and 42°C during rewarming
- Failure to address hypothermia or other systemic illness
- Failure to consider compartment syndrome in a pulseless frostbitten extremity

ADDITIONAL READING

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- Wolfson AB, ed. *Harwood-Nuss' Emergency Medicine*. 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2010:1599–1603.



See Also (Topic, Algorithm, Electronic Media Element)

Hypothermia



CODES

ICD9

- 991.0 Frostbite of face
- 991.1 Frostbite of hand
- 991.3 Frostbite of other and unspecified sites

ICD10

- T33.09XA Superficial frostbite of other part of head, init encntr
- T33.90XA Superficial frostbite of unspecified sites, init encntr
- T33.539A Superficial frostbite of unspecified finger(s), init encntr

GALLSTONE ILEUS

Joanna W. Davidson



BASICS

DESCRIPTION

- Mechanical intestinal obstruction secondary to impaction of a gallstone within bowel lumen
- Stone is usually > 2.5 cm
- 1–3% of all intestinal obstructions
- Most cases occur in patients > 65
- Female > male (5:1)
- Mortality 15–18%

ETIOLOGY

- Chronic gallbladder inflammation causes adhesions between gallbladder and adjacent bowel wall
- Cholecystocolonic fistula develops, permitting stone passage into intestine:
 - Duodenum is the most common site of fistula formation, followed by colon
 - Gastric fistulas are possible but rare
- Site of impaction
 - Terminal ileum most common (54–65%)
 - Narrowest part of small intestine at level of ileocecal valve
 - Jejunum (27%)
 - Duodenum (1–3%)
 - Gastric outlet obstruction caused by duodenal impaction referred to as *Bouveret syndrome*
 - Large bowel obstruction is rare



DIAGNOSIS

SIGNS AND SYMPTOMS

- “Tumbling” abdominal discomfort:
 - Episodic abdominal pain as stone lodges and dislodges throughout the intestines.
 - Complete impaction leads to severe, often acute abdominal pain.
- Nausea
- Vomiting:
 - Can be bilious or feculent
- Obstipation
- Abdominal distention and tympany

- Abdominal tenderness:
 - Peritoneal findings develop late in the course of disease
- Abnormal bowel sounds

History

- Only 50–60% of patients have a history of biliary colic or gallstone disease.
- Gallstone ileus has been associated with cardiovascular disease, diabetes, and obesity.

Physical-Exam

- Abdominal exam for:
 - Abdominal distension/tenderness
- Jaundice may occur

ESSENTIAL WORKUP

Evaluate for intestinal obstruction.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Electrolytes, BUN/creatinine, glucose since decreased oral intake and vomiting leads to electrolyte abnormality
- Liver function panel and bilirubin may be elevated
- Amylase:
 - Elevated in late obstructions
- CBC/hematocrit:
 - Hemoconcentration secondary to dehydration
- Elevated WBC nonspecific

Imaging

- Flat and upright abdominal radiographs:
 - Multiple air–fluid levels and distended bowel consistent with bowel obstruction
 - Rigler triad: 2 of 3 pathognomonic (present in 30–50%):
 - Air in the biliary tree (pneumobilia)
 - Partial or complete bowel obstruction
 - Ectopic stone visualized within the intestinal tract
- CXR:
 - Evaluate for pneumoperitoneum
- Abdominal CT scan:
 - Test of choice
 - Can directly visualize and localize stone within intestinal lumen
- Abdominal US:

- Can identify pneumobilia and gallstones, but lower yield in locating obstructing stone

DIFFERENTIAL DIAGNOSIS

- Paralytic ileus
- Extrinsic bowel obstruction:
 - Adhesions
 - Volvulus
 - Hernia
 - Intussusception
- GI malignancy
- Diverticulitis
- Bezoar
- Inflammatory bowel disease
- Pseudo-obstruction
- Cholecystitis
- Ascending cholangitis
- Pancreatitis



TREATMENT

PRE HOSPITAL

Establish IV access

INITIAL STABILIZATION/THERAPY

IV fluid resuscitation

ED TREATMENT/PROCEDURES

- Nasogastric suction to decompress the stomach and intestine
- Nothing PO
- Electrolyte replacement
- Monitor urine output
- Analgesics
- Broad-spectrum antibiotics to cover bowel flora:
 - Piperacillin/tazobactam
 - Ampicillin/sulbactam
 - Ticarcillin/clavulanate
 - Alternatives include imipenem, meropenem, 3rd-generation cephalosporin + metronidazole.
- Surgical consultation

MEDICATION

- Ampicillin/sulbactam: 3 g IV q6h (peds: 100–200 mg/kg/24 h)

- Piperacillin/tazobactam: 3.375 g IV q6h (peds: 240–400 mg/kg/24 h)
- Ticarcillin/clavulanate: 3.1 g IV q4–6h



FOLLOW-UP

DISPOSITION

Admission Criteria

- Admit all patients with gallstone ileus
- Surgical evaluation for emergent operative intervention

Discharge Criteria

None

FOLLOW-UP RECOMMENDATIONS

Surgical consultation in ED for evaluation and operative intervention

PEARLS AND PITFALLS

- Gallstone ileus is a mechanical intestinal obstruction rather than a true ileus.
- Emergent surgical consultation is required for definitive management.
- High mortality rates stem from delay in diagnosis and patient comorbidities.
- Suspect gallstone ileus in elderly patients, especially women, with signs/symptoms of bowel obstruction and no previous surgical history.
- Only 10% of ectopic gallstones can be visualized on plain radiographs. CT imaging is more sensitive and specific for detecting intraluminal stones.
- Only 1/2 of the patients have a previous history of biliary colic or gallstone disease.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- [Cholecystitis](#)
- [Cholelithiasis](#)



CODES

ICD9

560.31 Gallstone ileus

ICD10

[K56.3 Gallstone ileus](#)

GANGRENE

Stephen R. Hayden



BASICS

DESCRIPTION

- Gas gangrene or clostridial myonecrosis
- An acute, rapidly progressive, gas-forming necrotizing infection of muscle and subcutaneous tissue
- Can be seen in post-traumatic or postoperative situations
- Progressive invasion and destruction of healthy muscle tissue

ETIOLOGY

- Clostridial organisms:
 - _ Facultative anaerobic, spore-forming, gram-positive bacillus
 - _ Produces a number of toxins; the most prevalent and lethal is α -toxin.
- *Clostridium perfringens* is the most common bacterium; found in 80–90% of wounds.
- Other clostridial bacteria include *Clostridium novyi*, *Clostridium septicum*, *Clostridium histolyticum*, *Clostridium bifermentans*, and *Clostridium fallax*.
- 2 distinct mechanisms for introduction of clostridial organisms:
 - _ Traumatic and postoperative
 - _ Nontraumatic associated with diabetes mellitus, peripheral vascular disease, alcoholism, IV drug abuse, and malignancies



DIAGNOSIS

SIGNS AND SYMPTOMS

- Sudden severe pain of extremity or involved area
- Low-grade fever
- Tachycardia out of proportion to fever
- Bronzing of the skin over involved area; later can turn purple or red
- Crepitus
- Formation of blebs and bullae
- Thin, serosanguinous exudate and sweet odor
- Rapid local extension
- Obtunded sensorium
- Systemic toxicity

ESSENTIAL WORKUP

- History and physical exam with special attention to clinical evidence of crepitus in soft tissue

- Soft tissue x-rays of involved area to detect gas dissecting along fascial planes:
 - The absence of gas does not exclude significant disease.
- Stat Gram stain of wound exudate for gram-positive bacillus with paucity of leukocytes

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC with differential, electrolytes, BUN, and creatinine
- Coagulation studies
- Evaluate for hemolysis
- Stat Gram stain of wound exudates
- Anaerobic cultures of wound or tissue biopsy

Imaging

- Radiographs may reveal soft tissue gas.
- CT if area involves abdomen or flank.

Diagnostic Procedures/Surgery

All patients with gas gangrene must undergo surgical débridement.

DIFFERENTIAL DIAGNOSIS

- Cellulitis
- Necrotizing fasciitis
- Nonclostridial myositis and myonecrosis
- Other causes of gas in tissues, as from dissection from respiratory or GI tracts



TREATMENT

PRE HOSPITAL

Establish IV and infuse isotonic fluids

INITIAL STABILIZATION/THERAPY

Manage airway and resuscitate as indicated:

- Rapid sequence intubation as needed.
- Supplemental oxygen:
 - Cardiac and oxygen saturation monitors should be placed.
- IV access; consider central venous pressure monitoring; sepsis protocol is appropriate
- Aggressive volume expansion, including crystalloid, plasma, packed RBCs, and albumin if there is septic shock.

ED TREATMENT/PROCEDURES

- Parenteral antibiotic therapy:
 - Initial empiric therapy should cover *Clostridium* species and group A *Streptococcus* as well as mixed aerobes and anaerobes
 - Primary definitive therapy: Penicillin G + clindamycin
 - Alternative: Ceftriaxone or erythromycin
 - If mixed infection: Penicillin + clindamycin, metronidazole, or vancomycin and gram-negative coverage with gentamicin
 - Follow local sepsis protocols
- Surgical consultation:
 - Débridement, amputation, or fasciotomy is required.
- Hyperbaric oxygen (HBO) as adjunctive therapy:
 - Early transfer to hyperbaric facility may be lifesaving.
 - Lack of randomized trials with HBO but nonrandomized studies suggest benefit
- Tetanus prophylaxis
- Observe for major complications including ARDS, renal failure, myocardial irritability, and DIC.
- Polyvalent antitoxin is not made in US and studies have not demonstrated efficacy:
 - Because of the unacceptable hypersensitivity reactions, it is not routinely recommended.

MEDICATION

- Ceftriaxone: 2 g (peds: 100 mg/kg/24h max. 4 g) IV q12h
- Clindamycin: 900 mg (peds: 40 mg/kg/d q6h) IV q8h
- Erythromycin: 1 g (peds: 50 mg/kg/d q6h) q6h IV
- Gentamicin: 2 mg/kg (peds: 2 mg/kg IV q8h) IV q8h
- Metronidazole: 500 mg (peds: Safety not established) IV q8h
- Penicillin G: 24 million IU/24h (peds: 250,000 IU/kg/24h) IV q4–6h
- Tetanus immune globulin: 500 IU IM
- Tetanus toxoid: 0.5 mg IM

First Line

Primary definitive therapy for clostridial species; combination of penicillin G and clindamycin



FOLLOW-UP

DISPOSITION

Admission Criteria

- All patients with gas gangrene and evidence of myonecrosis *must be admitted* for surgical débridement and IV antibiotics.
- Use of HBO therapy is an important adjunct.

Discharge Criteria

No patient with acute gangrene should be discharged.

Issues for Referral

After stabilization with antibiotics and surgical débridement, consider referral for HBO treatment as an adjunct.

PEARLS AND PITFALLS

- Bacteremia occurs in about 15% and can progress quickly to intravascular hemolysis.
- HBO as adjunctive therapy to surgical débridement and early antibiotics if patient is hemodynamically stable

ADDITIONAL READING

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- Bryant AE, Stevens DL. Clostridial myonecrosis: New insights in pathogenesis and management. *Curr Infect Dis Rep.* 2010;12(5):383–391.
- Headley AJ. Necrotizing soft tissue infections: A primary care review. *Am Fam Physician.* 2003;68(2):323–328.
- Pinzon-Guzman C, Bashir D, McSherry G et al. Clostridium septicum gas gangrene in a previously healthy 8-year old female with survival. *J Pediatr Surg.* 2013;48(4):e5–e8.
- Stevens DL, Aldape MJ, Bryant AE. Life-threatening clostridial infections. *Anaerobe.* 2012;18(2):254–259.



CODES

ICD9

- 040.0 Gas gangrene
- 785.4 Gangrene

ICD10

- A48.0 Gas gangrene
- I96 Gangrene, not elsewhere classified

GASTRIC OUTLET OBSTRUCTION

Jenny J. Lu

BASICS

DESCRIPTION

- Any process impeding the passage of gastric contents into the duodenum
- Causes not limited to gastric pathology and may be duodenal or extraluminal in origin
- Benign and malignant causes, including:
 - Neoplasms (most common cause in adults), intrinsic or extrinsic neoplasms (pancreatic, gastric lymphoma, duodenal, gallbladder). Extrinsic masses may cause compression at pylorus or proximal duodenum
 - Peptic ulcer disease (PUD), no longer most common cause in adults, with treatment of *Helicobacter pylori* and use of H₂ blockers
 - Pyloric stenosis (most common pediatric cause): Incidence 2–5/1,000
 - Postoperative complications, especially from gastric surgeries (e.g., edema, scarring, stricture, or hyperplasia of pylorus or duodenum)
 - Mechanical causes: Gastric volvulus, polyps, bezoars, duplication cysts
 - Edema, scarring, strictures/webs, or hyperplasia of pylorus or duodenum from various causes (e.g., caustic injury, chronic pancreatitis)

ETIOLOGY

- Regardless of exact cause, gastric outlet obstruction characteristically leads to nausea and nonbilious vomiting
- Persistent vomiting may lead to dehydration, electrolyte and acid–base derangements
 - Chronic symptoms may lead to weight loss, malnutrition, failure to thrive
 - Hypokalemic, hypochloremic metabolic alkalosis is classic finding

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Symptoms may be intermittent until obstruction becomes complete
- Nausea and vomiting, *usually* nonbilious
- Abdominal pain, variable in character and often vague
- Early satiety and epigastric fullness
- Epigastric discomfort relieved with emesis
- Weight loss, failure to thrive

Physical-Exam

- Vital signs:
 - May be normal
 - Tachycardia, hypotension if volume depletion is significant
- Abdominal exam:
 - Variable amount of epigastric/abdominal distention
 - Tympanitic to auscultation
 - Succession splash > 4 hr after eating
 - Digital rectal exam: Evaluate for occult blood
- Signs of dehydration in eyes, oral pharynx, mucous membranes, skin turgor
- Signs of malnutrition in chronic or late obstruction
- Weight loss when chronic and with malignancy

Geriatric Considerations

- Abdominal pain, nausea/vomiting: GI symptoms may be more vague/subtle in elderly patients
- If appropriate, consider other causes of symptoms (cardiac causes, neurologic causes)

Pediatric Considerations

- Idiopathic hypertrophic pyloric stenosis:
 - Most common cause in pediatric population
 - “Typical” patient is male (Caucasian and US-born Asians more common)
 - Usually 2–8 wk old but may be diagnosed as early as 1st wk and up to 3 mo of age
 - Initially intermittent, nonprojectile, postprandial vomiting, which progresses to projectile, nonbilious vomiting
 - A midepigastric peristaltic wave occurring prior to vomiting may be visible on exam
 - Epigastric “olive” mass may be palpable in 80–90% of patients

ESSENTIAL WORKUP

Careful history and physical exam

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC:
 - Anemia if malignancy or GI blood loss
 - High hematocrit indicating hemoconcentration
- Electrolytes, BUN/creatinine, glucose:
 - Hypokalemia
 - Hypochloremic metabolic alkalosis

- Hypoglycemia
- Prerenal azotemia
- Urinalysis
- Amylase/lipase
- Liver profile, if malignancy suspected
- *H. pylori*, if PUD suspected

Imaging

- Plain abdominal radiographs (obstructive series):
 - Often nondiagnostic
 - Dilated stomach or absence of air in bowel distally may be suggestive
- Abdominal US in pediatric patients:
 - No ionizing radiation
 - Elongated hypertrophic pyloric sphincter
- Abdominal CTs are often very helpful for detecting neoplastic, intraluminal, and extraluminal causes of obstruction.
 - Likely to be most commonly used modality in adults
 - Radiation load is especially undesirable in pediatric population; ultrasound and fluoroscopic UGI series are preferred initial approaches

Diagnostic Procedures/Surgery

- Upper GI series:
 - To demonstrate site and character of obstruction
 - “String sign,” “double track sign,” “beak sign,” “shoulder sign” are characteristic findings in pyloric stenosis
- Upper endoscopy:
 - To visualize gastric interior, gastric outlet, proximal duodenum

DIFFERENTIAL DIAGNOSIS

- Proximal bowel obstruction
- Exacerbation of PUD
- Gastroenteritis
- Cholelithiasis
- Cholecystitis
- Acute pancreatitis
- Diabetic gastroparesis
- Psychogenic vomiting



TREATMENT

PRE HOSPITAL

- Vital signs, airway stabilization, oxygen administration, IV access

- Fluid resuscitation if dehydrated, vomiting

INITIAL STABILIZATION/THERAPY

- 0.9% NS IV fluid resuscitation significant volume losses:
 - _ Adults: 1 L bolus
 - _ Peds: 20 mL/kg bolus
- Correction of electrolyte abnormalities, especially hypokalemia

ED TREATMENT/PROCEDURES

- Nasogastric tube (NGT)
- Foley catheter to monitor urine output
- Surgical consultation/intervention:
 - _ Endoscopic balloon dilatation of benign strictures
 - _ Enteral stent placement (malignant causes)
 - _ Gastrojejunostomy (malignant causes)
 - _ Vagotomy and antrectomy or pyloroplasty or gastrojejunostomy or other variation (benign causes)

MEDICATION

- Famotidine: Adults: 20 mg (peds: 0.6–0.8 mg/kg/24 h div. q6–8h) IV q12h *or*
- Ranitidine: 50 mg (peds: 2–4 mg/kg/24 h div. q6–8h) IV q8h
- Pantoprazole: Adults: 40 mg IV (also *H. pylori* treatment as needed)



FOLLOW-UP

DISPOSITION

Admission Criteria

Most patients with gastric outlet obstruction will be admitted for fluid resuscitation, electrolyte repletion, gastroenterologic, and surgical evaluation.

Discharge Criteria

Rarely, patients may be considered for discharge if:

- Symptoms of abdominal pain, vomiting have resolved
- Evaluated and cleared by surgeon or gastroenterologist during presentation
- Lab parameters, imaging, and patient's volume status are normal

Issues for Referral

Surgical and gastroenterology consultations

FOLLOW-UP RECOMMENDATIONS

Any discharged patient should follow up with surgeon and/or gastroenterologist:

- [Specific instructions to return if symptoms recur](#)

PEARLS AND PITFALLS

- [Misdiagnosing symptoms of gastric outlet obstruction as gastroenteritis](#)
- [Failure to appreciate limitations of plain radiographs in diagnosing this condition](#)
- [Failure to consider gastric outlet obstruction and malignancy in patient with epigastric pain and vomiting](#)
- [Failure to adequately fluid resuscitation of patients, especially elderly or pediatric patients](#)

ADDITIONAL READING

- [Dada SA, Fuhrman GM. Miscellaneous Disorders and their management in gastric surgery: Volvulus, carcinoid, lymphoma, gastric varices, and gastric outlet obstruction. *Surg Clin North Am.* 2011;91:1123–1130.](#)
- [Kim JH, Shin JH, Song HY. Benign strictures of the esophagus and gastric outlet: Interventional management. *Korean J Radiol.* 2010;11\(5\):497–506.](#)
- [Otjen JP, Iyer RS, Phillips GS, et al. Usual and unusual causes of pediatric gastric outlet obstruction. *Pediatr Radiol.* 2012;42:728–737.](#)
- [Shone DN, Nikoomanesh P, Smith-Meek MM, et al. Malignancy is the most common cause of gastric outlet obstruction in the era of H₂ blockers. *Am J Gastroenterol.* 1995;90:1769–1770.](#)



See Also (Topic, Algorithm, Electronic Media Element)

- [Abdominal Pain](#)
- [Bowel Obstruction](#)
- [Pyloric Stenosis](#)
- [Vomiting](#)



CODES

ICD9

537.0 Acquired hypertrophic pyloric stenosis

ICD10

K31.1 Adult hypertrophic pyloric stenosis

GASTRITIS

Yanina Purim-Shem-Tov

BASICS

DESCRIPTION

- Inflammatory response of gastric mucosa to injury—“gastritis”
- 3 lines of defense of gastric mucosa:
 - Mucous layer that forms protective pH gradient
 - Surface epithelial cells that can repair small defects
 - Postepithelial barrier that neutralizes any acid that has traversed 1st 2 layers
- No definite link between histologic gastritis and dyspeptic symptoms
- Epithelial cell damage with no associated inflammation—“gastropathy”

ETIOLOGY

- Common causes of gastritis: Infections, autoimmune, drugs (i.e., cocaine), hypersensitivity, stress
- Common causes of gastropathy: Endogenous or exogenous irritants, such as bile reflux, alcohol, or aspirin and NSAIDs, ischemia, stress, chronic congestion
- Acute gastritis:
 - Stress (sepsis, burns, trauma):
 - Decrease in splanchnic blood flow leading to decreased mucus production, bicarbonate secretion, and prostaglandin synthesis
 - Results in mucosal erosions and hemorrhage
 - Alcohol:
 - Induces production of leukotrienes that cause microvascular stasis, engorgement, and increased vascular permeability
 - Leads to hemorrhage
 - NSAIDs, including aspirin:
 - Interfere with prostaglandin synthesis, leading to similar cascade as induced by alcohol
 - Results in mucosal erosions
 - Steroids
- Chronic gastritis:
 - Produced by *Helicobacter pylori*
 - Mechanism of *H. pylori* unclear:
 - Gram-negative spiral bacteria found in gastric mucous layer
 - Contains enzyme urease that allows it to change pH level (alkaline) of its microenvironment

SIGNS AND SYMPTOMS

- Dyspepsia
- Bloating
- Nausea/vomiting
- Anorexia
- Epigastric tenderness
- Heartburn

History

- Dyspepsia
- Epigastric pain or discomfort (episodic and chronic)
- Bloating, indigestion, eructation, flatulence, and heartburn
- Anorexia, nausea/vomiting
- Hematemesis, melena

Physical-Exam

- Careful physical exam including stool Hemoccult testing and vital signs with orthostatics
- Dehydration, tachycardia (with vomiting)
- Pallor (hemorrhagic gastritis)
- Abdominal exam
- Nonspecific
- Epigastric tenderness

ESSENTIAL WORKUP

- ABCs
- Hematocrit determination
- Evaluation for dehydration/shock

DIAGNOSIS TESTS & INTERPRETATION***Lab***

- Normal lab values in uncomplicated gastritis
- CBC:
 - Anemia with acute hemorrhagic gastritis
 - Leukocytosis: Infection
- Electrolytes, BUN, creatinine, glucose
- Amylase/lipase for pancreatitis in differential
- Urinalysis:
 - Assess dehydration/ketosis (starvation)

- Bilirubin present with hepatitis

Diagnostic Procedures/Surgery

- ECG:
 - For elderly patients
 - Myocardial ischemia in differential
- Endoscopy:
 - Outpatient unless significant hemorrhage
 - Allows for visualization of bleeding sites, histologic confirmation of mucosal inflammation, and detection of *H. pylori*
- Noninvasive *H. pylori* testing:
 - ¹³C and ¹⁴C urea breath tests
 - Stool antigen test
 - Serology to detect antibodies to *H. pylori*
 - Serum pepsinogen isoenzymes
 - The ratio of pepsinogen isozymes I and II in serum correlates with presence of metaplastic atrophic gastritis (principally autoimmune metaplastic atrophic gastritis and pernicious anemia)

DIFFERENTIAL DIAGNOSIS

- Peptic ulcer disease (PUD)
- Nonulcer dyspepsia (symptoms without ulcer on endoscopy)
- Gastroesophageal reflux
- Biliary colic
- Cholecystitis
- Pancreatitis
- Hepatitis
- Abdominal aortic aneurysm
- Aortic dissection
- Myocardial infarction



TREATMENT

PRE HOSPITAL

- ABCs
- IV fluid resuscitation

INITIAL STABILIZATION/THERAPY

- ABCs with acute erosive or hemorrhagic gastritis that presents with hemodynamic instability
- IV fluid resuscitation with lactated Ringer solution or 0.9% normal saline (NS) via 2 large-bore catheters

- NGT for gastric decompression and lavage when history of hematemesis or unstable vital signs
- Foley catheterization to assess volume replacement

ED TREATMENT/PROCEDURES

- Pain control with:
 - Antacids
 - GI cocktail:
 - 30 mL antacids + 10–20 mL viscous lidocaine
 - H₂ antagonists
 - Proton pump inhibitors (PPIs)
 - Sucralfate
 - Avoid narcotics—may mask serious illness
- Acute hemorrhagic gastritis:
 - IV fluid resuscitation
 - Blood transfusion if low hematocrit
 - Reverse causes (alcohol, sepsis, NSAIDs, or trauma)
 - Prevent *acute* or *erosive* gastritis in critically ill:
 - Antacids hourly or IV PPI or H₂ antagonists
 - Goal is to keep pH level at >4
- Chronic gastritis—*H. pylori* therapy:
 - Treatment of *H. pylori* infection:
 - Invasive or noninvasive testing to confirm infection
 - Oral (PO) eradication antibiotic therapy options
 - Most common therapies for *H. pylori* infection:
 - PPI (omeprazole 20 mg or lansoprazole 30 mg), clarithromycin 500 mg BID for 2 wk, amoxicillin 1 g BID for 2 wk.
 - For penicillin-allergic patients: PPI + clarithromycin 500 mg BID + metronidazole 500 mg BID for 14 days
 - 4-drug therapy: H₂ blocker, bismuth subsalicylate (Pepto-Bismol) + either amoxicillin 1,000 mg BID or tetracycline 500 mg QID in combination with either metronidazole 250 mg QID or clarithromycin 500 mg BID for 14 days
 - Drug resistance in US:
 - Metronidazole: 30–48%
 - Clarithromycin: >10%
 - Amoxicillin: Uncommon
 - Bismuth: None
 - Treatment controversial for asymptomatic or nonulcer dyspepsia gastritis
- Vitamin B₁₂ supplementation for *atrophic gastritis*

MEDICATION

- Bismuth subsalicylate: 525 mg tabs 2 PO QID not to exceed 8 doses in 24 hr
- Cimetidine (H₂ blocker): 800 mg PO at bedtime nightly (peds: 20–40 mg/kg/24 h) for 6–8 wk
- Famotidine (H₂ blocker): 40 mg PO at bedtime nightly (peds: 0.5–0.6 mg/kg q12h) for 6–8 wk
- Lansoprazole (PPI): 30 mg PO BID for 2 wk
- Maalox Plus: 2–4 tablets PO QID
- Misoprostol: 100–200 µg PO QID
- Mylanta II: 2–4 tablets PO QID
- Nizatidine (H₂ blocker): 300 mg PO at bedtime nightly for 6–8 wk
- Omeprazole (PPI): 20 mg PO BID (peds: 0.6–0.7 mg/kg q12–24 h) for 2 wk
- Pantoprazole (PPI): 40 mg PO/IV daily for 2 wk
- Ranitidine (H₂ blocker): 300 mg PO at bedtime nightly (peds: 5–10 mg/kg/24 h given q12h) for 6–8 wk
- Sucralfate: 1 g PO QID for 6–8 wk

First Line

- Triple therapy using a PPI with clarithromycin and amoxicillin or metronidazole given twice daily remains the recommended 1st choice treatment.
- Sequential 10-day therapy in high prevalence areas:
 - Double therapy for 5 days:
 - PPI
 - Amoxicillin
 - Followed by triple therapy for 5 days:
 - PPI
 - Clarithromycin
 - Metronidazole

Second Line

- Bismuth-based quadruple therapies remain the best 2nd choice treatment.
- The rescue treatment should be based on antimicrobial susceptibility testing.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Acute hemorrhagic or erosive gastritis that presents with upper GI tract bleeding, tachycardia, and hypotension
- Uncontrolled pain or vomiting
- Coagulopathy from medication or liver disease

Discharge Criteria

- Unremarkable physical exam with normal CBC and heme-negative stools
- If heme-positive stools, discharge if stable vital signs, normal hematocrit, and negative NGT aspiration for upper GI tract hemorrhage:
 - Outpatient evaluation for endoscopy

Issues for Referral

- Outpatient referral for endoscopy and *H. pylori* testing
- Biopsy for gastric dysplasia and malignancy

FOLLOW-UP RECOMMENDATIONS

Close follow-up with gastroenterologist for endoscopy with biopsy for diagnostic reasons.

PEARLS AND PITFALLS

- Gastritis/gastropathy is a common presentation to ED.
- Symptoms typically are dyspepsia, nausea, and vomiting.
- ED management depends on patient's clinical symptoms, but should include diagnostic and therapeutic components.
- Therapeutic management usually involves treatment of *H. pylori*.
- Drug resistance of *H. pylori* to antibiotics is increasing.
- Close follow-up with gastroenterologist recommended for biopsy and to detect gastric cancers.

ADDITIONAL READING

- Czinn SJ. *Helicobacter pylori* infection: Detection, investigation, and management. *J Pediatr*. 2005;146:S21–S26.
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See Also (Topic, Algorithm, Electronic Media Element)

- GI Bleeding
- Gastroesophageal Reflux Disease
- Peptic Ulcer Disease



CODES

ICD9

- 535.00 Acute gastritis, without mention of hemorrhage
- 535.30 Alcoholic gastritis, without mention of hemorrhage
- 535.50 Unspecified gastritis and gastroduodenitis, without mention of hemorrhage

ICD10

- K29.00 Acute gastritis without bleeding
- K29.20 Alcoholic gastritis without bleeding
- K29.70 Gastritis, unspecified, without bleeding

GASTROENTERITIS

Isam F. Nasr



BASICS

DESCRIPTION

Inflammation of stomach and intestines associated with diarrhea and vomiting; often the result of infectious or toxin exposure.

ETIOLOGY

Infectious

- **Viruses:**
 - 50–70% of all cases with Norovirus cases on the rise in travelers returning from Mexico and India.
- **Invasive bacteria:**
 - *Campylobacter*: Contaminated food or water, wilderness water, birds, and animals:
 - Most common cause
 - Gross or occult blood is found in 60–90%.
 - *Salmonella*: Contaminated water, eggs, poultry, or dairy products:
 - *Typhoid fever (Salmonella typhi)* characterized by unremitting fever, abdominal pain, rose spots, splenomegaly, and bradycardia
 - Immunocompromised susceptible
 - *Shigella*: Fecal–oral route
 - *Vibrio parahaemolyticus*: Raw and undercooked seafood
 - *Yersinia*: Contaminated food (pork), water, and milk:
 - May present as mesenteric adenitis or mimic appendicitis
 - Specific food-borne disease (food poisoning):
 - *Staphylococcus aureus*:
 - Most common toxin-related disease
 - Symptoms within 1–6 hr after ingesting food
 - *Bacillus cereus*:
 - Classic source is fried rice left on steam tables.
 - Symptoms within 1–36 hr
 - Cholera: Profuse watery stools with mucous (rice-water stools)
 - Ciguatera:
 - Fish intoxication
 - Onset 5 min–30 hr (average 6 hr) after ingestion
 - Paresthesias, hypotension, peripheral muscle weakness
 - Amitriptyline may be therapeutic.

- Scombroid:
 - Caused by blood fish: Tuna, albacore, mackerel, and mahi-mahi
 - Flushing, headache, erythema, dizziness, blurred vision, and generalized burning sensation
 - Symptoms last < 6 hr.
 - Treatment includes antihistamines.
- Protozoa:
 - *Giardia lamblia*:
 - High-risk groups: Travelers, day care children, homosexual men, and campers who drink untreated mountain water

Noninfectious Causes

- Toxins:
 - Zinc, copper, cadmium
 - Organic chemicals: Polyvinyl chlorides
 - Pesticides: Organophosphates
 - Radioactive substances
 - Alkyl mercury
- Altered host response to food substance (tyramine, monosodium glutamate, tryptamine)

Pediatric Considerations

- Focus evaluation on state of hydration
- Most of viral origin and self-limited
- Rotavirus accounts for up to 50%
- *Shigella* infections associated with seizures

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Nausea, vomiting, diarrhea
- Bloody/mucous diarrhea
- Abdominal cramps or pain
- Fever
- Malaise, myalgias, headache, anorexia
- Hypotension, lethargy, and dehydration (severe cases)

Physical-Exam

- Dry mucous membranes
- Tachycardia

- Abdominal tenderness
- Perianal inflammation, fissure, fistula

ESSENTIAL WORKUP

- Digital rectal exam to determine presence of gross or occult blood
- Fecal leukocyte determination:
 - Present with invasive bacteria
 - Absent in protozoal infections, viral, toxin-induced food poisoning

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC indications:
 - Significant blood loss
 - Systemic toxicity
- Electrolytes, glucose, BUN, creatinine—indications:
 - Lethargy, significant dehydration, toxicity, or altered mental status
 - Diuretic use, persistent diarrhea, chronic liver or renal disease
- Stool culture indications:
 - Presence of fecal leukocytes
 - Historical markers (immunocompromised, travel, homosexual)
 - Public health (food handler, day/health care worker)
- Blood culture indications:
 - Suspected bacteremia or systemic infections
 - Ill patients requiring admission
 - Immunocompromised
 - Elderly patients and infants

Imaging

Abdominal radiographs have no value unless obstruction or toxic megacolon suspected.

Pediatric Considerations

- Lab studies not required in most cases
- Rotazyme assay detects rotavirus:
 - Rarely indicated in managing outpatients
 - Helpful to cohort and avoid cross-contamination among inpatients
- Stool culture indication:
 - Fecal leukocytes
 - Toxic
 - Infants
 - Immunocompromised

DIFFERENTIAL DIAGNOSIS

- Gastritis/peptic ulcer disease
- Milk and food allergies
- Appendicitis
- Irritable bowel syndrome
- Ulcerative colitis/Crohn's disease
- Malrotation with midgut volvulus
- Meckel diverticulum
- Drugs and toxins:
 - Mannitol
 - Sorbitol
 - Phenolphthalein
 - Magnesium-containing antacids
 - Quinidine
 - Colchicine
 - Mushrooms
 - Mercury poisoning



TREATMENT

PRE HOSPITAL

- Difficult IV access with severe dehydration.
- Avoid exposure to contaminated clothes or body substances.

INITIAL STABILIZATION/THERAPY

- Management of ABCs
- IV fluid with 0.9% NS resuscitation for severely dehydrated

ED TREATMENT/PROCEDURES

- Oral fluids for mild dehydration (Gatorade/Pedialyte)
- IV fluids for:
 - Hypotension, nausea and vomiting, obtundation, metabolic acidosis, significant hypernatremia, or hyponatremia
 - 0.9% NS bolus (adults: 500 mL–1 L, peds: 20 mL/kg) for resuscitation, then 0.9% NS or D₅ 0.45% peds: NS (peds: D₅ 0.25% NS) to maintain adequate urine output
- Bismuth subsalicylate (Pepto-Bismol):
 - Antisecretory agent
 - Effective clinical relief without adverse effects
- Kaolin–pectin (Kaopectate):
 - Reduces fluidity of stools
 - Does not influence course of disease
- Antimotility drugs (diphenoxylate [Lomotil], loperamide [Imodium], paregoric,

and codeine):

- Appropriate in noninfectious diarrhea
- Initial use of sparse amounts to control symptoms in infectious diarrhea
- Avoid prolonged use in infectious diarrhea—may increase duration of fever, diarrhea, and bacteremia and may precipitate toxic megacolon.
- Antibiotics for infectious pathogens:
 - *Campylobacter*: Quinolones or erythromycin
 - *Salmonella*: Quinolones or trimethoprim–sulfamethoxazole (TMP–SMX)
 - Typhoid fever: Ceftriaxone
 - *Shigella*: Quinolone, TMP–SMX, or ampicillin
 - *V. parahaemolyticus*: Tetracycline or doxycycline
 - *Clostridium difficile*: Metronidazole or vancomycin
 - *Escherichia coli*: Quinolones or TMP–SMX
 - *Giardia lamblia*: Metronidazole
- Antiemetics for nausea/vomiting:
 - Ondansetron
 - Prochlorperazine
 - Promethazine

MEDICATION

- Ampicillin: 500 mg (peds: 20 mg/kg/24 h) PO or IV q6h
- TMP–SMX; Bactrim DS: 1 tab (peds: 8–10 mg TMP/40–50 mg SMX/kg/24 h) PO BID
- Ceftriaxone: 1 g (peds: 50–75 mg/kg/12 h) IM or IV q12h
- Ciprofloxacin (quinolone): 500 mg PO or 400 mg IV BID (> 18 yr)
- Doxycycline: 100 mg PO or 400 mg IV BID
- Metronidazole: 250 mg (peds: 35 mg/kg/24 h) PO TID (> 8 yr)
- Ondansetron 4 mg (peds: 0.1 mg/kg) IV
- Prochlorperazine (Compazine): 5–10 mg IV q3–4h; 10 mg PO q8h; 25 mg per rectum (PR) q12h
- Promethazine (Phenergan): 25 mg IM/IV q4h; 25 mg PO/PR (peds: 0.25–1 mg/kg PO/PR/IM)
- Tetracycline: 500 mg PO or IV QID
- Vancomycin 125–500 mg (peds: 40 mg/kg/24 h) PO q6h



FOLLOW-UP

DISPOSITION

Admission Criteria

- Hypotension unresponsive to IV fluids
- Significant bleeding

- Signs of sepsis/toxicity
- Intractable vomiting or abdominal pain
- Severe electrolyte imbalance
- Metabolic acidosis
- Altered mental status
- Children with > 10–15% dehydration

Discharge Criteria

- Mild cases requiring oral hydration
- Dehydration responsive to IV fluids

Issues for Referral

Cases of prolonged symptoms may be referred to a gastroenterologist for further workup.

FOLLOW-UP RECOMMENDATIONS

Most cases are self-limiting; therefore, follow-up is optional.

PEARLS AND PITFALLS

- Viruses account for over 50% of cases
- Avoid antimotility drugs in cases due to infectious pathogens.
- TMP–SMX (Bactrim DS), ciprofloxacin, doxycycline, and tetracycline are contraindicated in pregnancy. Metronidazole may be used during the 3rd trimester.

ADDITIONAL READING

- Bresee JS, Marcus R, Venezia RA, et al. The etiology of severe acute gastroenteritis among adults visiting emergency departments in the United States. *J Infect Dis.* 2012;205:1374–1381.
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- DuPont HL. Clinical practice. Bacterial diarrhea. *N Engl J Med.* 2009;361(16):1560–1569.
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See Also (Topic, Algorithm, Electronic Media Element)

- Diarrhea, Adult
- Diarrhea, Pediatric



CODES

ICD9

- 008.63 Enteritis due to norwalk virus
- 009.0 Infectious colitis, enteritis, and gastroenteritis
- 558.9 Other and unspecified noninfectious gastroenteritis and colitis

ICD10

- A08.11 Acute gastroenteropathy due to Norwalk agent
- A09 Infectious gastroenteritis and colitis, unspecified
- K52.9 Noninfective gastroenteritis and colitis, unspecified

GASTROESOPHAGEAL REFLUX DISEASE

Yanina Purim-Shem-Tov

BASICS

DESCRIPTION

- Spectrum of pathology in which gastric reflux causes symptoms and damage to esophageal mucosa
- Reflux esophagitis vs. nonerosive reflux disease
- 40% of general population experience symptoms monthly

ETIOLOGY

- Incompetent reflux barrier allowing increase in frequency and duration of gastric contents into esophagus
- Lower esophageal sphincter (LES):
 - Main antireflux barrier
 - Crural diaphragm attachment (diaphragmatic sphincter)
 - Contributes to pressure barrier at gastroesophageal junction
 - Esophageal acid clearance via peristalsis and esophageal mucosal resistance are additional barriers.
 - Most healthy individuals have brief episodes of reflux without symptoms.
- Transient lower esophageal sphincter relaxations (TLESRs):
 - Occur with higher frequency in gastroesophageal reflux disease (GERD) patients
 - Exposed esophageal mucosa becomes acidified and with time necroses
- Decreased LES tone:
 - Smoking
 - Foods: Alcohol, chocolate, onion, coffee, tea
 - Drugs: Calcium channel blockers, morphine, meperidine, barbiturates, theophylline, nitrates
- Delayed gastric emptying, increased body mass, and gastric distention contribute to reflux
- Hiatal hernias associated with GERD:
 - Significance varies in any given individual
 - Most persons with hiatal hernias do not have clinically evident reflux disease
- Acid secretion is same in those with or without GERD
- Associated medical conditions: Pregnancy, chronic hiccups, cerebral palsy, Down syndrome, autoimmune diseases, diabetes mellitus (DM), hypothyroidism

DIAGNOSIS

SIGNS AND SYMPTOMS

- Esophageal manifestations
 - Heartburn (or pyrosis)
 - Regurgitation
 - Dysphagia
- Extraesophageal manifestations
 - Bronchospasm
 - Laryngitis
 - Chronic cough

History

- Typical signs and symptoms:
 - Heartburn (pyrosis):
 - Retrosternal burning pain
 - Radiates from epigastrium through chest to neck and throat
 - Dysphagia:
 - Dysphagia suggests esophageal spasm or stricture.
 - Odynophagia:
 - Odynophagia suggests ulcerative esophagitis.
 - Regurgitation
 - Water brash
 - Belching
 - Esophageal strictures, bleeding
 - Barrett esophagus (esophageal carcinoma)
 - Early satiety, nausea, anorexia, weight loss
 - Symptoms worse with recumbence or bending over
 - Symptoms usually relieved with antacids, although temporarily
- Atypical signs and symptoms:
 - Noncardiac chest pain
 - Asthma
 - Persistent cough, hiccups
 - Hoarseness
 - Pharyngeal/laryngeal ulcers and carcinoma
 - Frequent throat clearing
 - Recurrent pneumonitis
 - Nocturnal choking
 - Upper GI tract bleeding

Physical-Exam

- Nonspecific, may have some epigastric tenderness.
- Symptoms worsen with placing patient flat on the bed or Trendelenburg position

Pediatric Considerations

- Regurgitation is common in infants:
 - Incidence decreases from twice daily in 50% of those age 2 mo to 1% of 1-yr-olds.
- Signs:
 - Frequent vomiting, irritability, cough, crying, and malaise
 - Arching the body (hyperextension) at feeding and refusals of feedings
- Failure to thrive
- Formula intolerance
- Sepsis

ESSENTIAL WORKUP

- Differentiate GERD from more emergent conditions such as ischemic heart pain, esophageal perforation, or aortic pathology.
- Obtain typical history
- Perform thorough physical exam: Vital signs, head, ears, eyes, nose, throat (HEENT), chest and abdominal exams

DIAGNOSIS TESTS & INTERPRETATION

No gold standard

Lab

- CBC:
 - Chronic anemia from esophagitis
- Stool testing for occult bleeding

Imaging

- No routine Imaging
- Chest radiograph:
 - Evidence of esophageal perforation, hiatal hernia, aortic disease

Diagnostic Procedures/Surgery

- Diagnostic trial of antacid:
 - Those with persistent symptoms should be referred for endoscopy
 - 90% of GERD patients respond to proton pump inhibitor (PPI) therapy
- Barium esophagram for prominent dysphagia
- Esophageal pH monitoring:
 - Correlate symptoms to acid reflux
- Esophageal manometry (poor sensitivity):
 - Evaluate LES resting pressure and esophageal peristaltic contractions
- Esophagogastroduodenoscopy (EGD)—detects reflux esophagitis and complications (Barrett esophagus, hiatal hernia, stricture, ulcers, malignancy)

DIFFERENTIAL DIAGNOSIS

- Ischemic heart disease
- Asthma
- Peptic ulcer disease
- Gastritis
- Hepatitis/pancreatitis
- Esophageal perforation
- Esophageal foreign body
- Esophageal infection
- Cholecystitis/cholelithiasis
- Mesenteric ischemia



TREATMENT

PRE HOSPITAL

- Esophageal pain may mimic angina
- Airway may need active control secondary to vomiting

INITIAL STABILIZATION/THERAPY

- ABCs need to be evaluated
- IV fluid resuscitation for blood loss or shock

ED TREATMENT/PROCEDURES

- Symptomatic relief:
 - Antacids
 - Antacids with viscous lidocaine
 - Sublingual nitroglycerine for esophageal spasm
 - Analgesics
- Lifestyle modifications:
 - Avoid late-night or heavy/fatty meals.
 - Minimize time in supine position after eating.
 - Elevation of head of bed
 - Weight loss
 - Eliminate smoking and alcohol intake
 - Avoid direct esophageal irritants such as citric juices and coffee
 - Avoid foods that decrease LES pressures such as fatty foods, chocolate, and coffee
 - Avoid drugs that lower LES tone
- PPIs:
 - More potent long-acting inhibitors of gastric acid secretion than H₂-blockers
 - Faster healing than other drug therapies
 - More efficacious in severe GERD and frank esophagitis

- H₂-blockers:
 - Effective for mild to moderate disease
 - Severe disease requires greater dosage than that used for peptic ulcer disease
- Antacids (Maalox, Mylanta):
 - Treatment of mild and infrequent reflux symptoms
 - Not effective for healing esophagitis
 - Alginic acid slurry floats on surface of gastric contents, providing mechanical barrier
- Sucralfate:
 - Binds to exposed proteins on surface of injured mucosa to form protective barrier
 - May also directly stimulate mucosal repair
- Metoclopramide (prokinetic drug):
 - Improves peristalsis
 - Accelerates gastric emptying
 - Increases LES pressure
- Drugs that modify TLESR
 - Baclofen
 - ADX10059
- Endoscopic therapy:
 - Suturing (plication), thermal injury, chemical injection
- Antireflux surgery (goal: Increase LES pressure):
 - Chronic reflux, younger patients, nonhealing ulceration, severe bleeding
 - Fundoplication can be more effective than medical therapy in selected cases
 - Currently newer incisionless procedure called transoral incisionless fundoplication available

Pregnancy Considerations

- Reflux present in 30–50% of pregnancies
- Increased intra-abdominal pressure, hormonal fluctuations lead to increased TLESRs
- EGD reserved for severe presentations
- H₂-blockers—1st-line therapy (longer safety record)
- PPIs—limited safety history in pregnancy

MEDICATION

- Antacids: 30 mL + viscous lidocaine, 10 mL, PO q6h
- Cimetidine: 400 mg PO BID, 300 mg IM/IV q6–8h
- Esomeprazole: 20–40 mg PO daily
- Famotidine: 20 mg PO/IV BID (peds: 0.5–1 mg/kg/d div. q8–12h, max. 40 mg/d)
- Lansoprazole: 15–30 mg daily

- Metoclopramide: 10–15 mg PO/IV/IM q6h before meals and nightly at bedtime
- Nizatidine: 150 mg PO BID
- Omeprazole: 20–40 mg PO daily
- Pantoprazole: 40 mg PO/IV daily
- Rabeprazole: 20 mg PO daily
- Ranitidine: 150 mg (peds: 5–10 mg/kg q12h) PO BID or 300 mg PO nightly at bedtime
- Sucralfate: 1 g PO 1 hr before meals and nightly at bedtime

First Line

- Life style modifications:
 - Head of bed elevation
 - Dietary modification
 - Refraining from assuming a supine position after meals
 - Avoidance of tight-fitting garments
 - Promotion of salivation by either chewing gum
 - Restriction of alcohol use
 - Reduction of obesity
- Acid-suppressive medications:
 - PPI or H₂ blocker
- Treatment of *H. pylori* infections

Second Line

- Prokinetic drugs (bethanechol, metoclopramide)
- Drugs that inhibit TLESRs (baclofen)



FOLLOW-UP

DISPOSITION

Admission Criteria

- Significant esophageal bleeding
- Uncontrolled reactive asthma
- Dehydration
- Starvation and failure to thrive

Discharge Criteria

Uncomplicated GERD: Refer to patient's primary care physician (PCP) or gastroenterologist for further evaluation.

Issues for Referral

Extraesophageal manifestations such as asthma, laryngitis.

FOLLOW-UP RECOMMENDATIONS

Gastroenterologist for endoscopy in patients who require continuous maintenance medical therapy to rule out Barrett esophagus.

PEARLS AND PITFALLS

- GERD therapy should include lifestyle changes.
- In patients with worse than mild and intermittent GERD symptoms initiate acid-suppressive therapy.
- In patients with GERD and moderate to severe esophagitis, provide acid suppression with a PPI rather than H₂ blockers.
- Endoscopy for patients who fail chronic therapy (at least 8 wk).
- Antireflux surgery for patients on high doses of PPIs, specially in young patients who may require lifelong therapy.
- Complications of GERD
 - Esophagitis
 - Peptic stricture and Barrett metaplasia
 - Extraesophageal manifestations of reflux: Asthma, laryngitis, and cough.

ADDITIONAL READING

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- Nwokediuko SC. Current trends in the management of gastroesophageal reflux disease: A review. *ISRN Gastroenterol.* 2012;2012:391631.



See Also (Topic, Algorithm, Electronic Media Element)

- Gastritis
- Peptic Ulcer Disease



CODES

ICD9

- 530.11 Reflux esophagitis
- 530.81 Esophageal reflux

ICD10

- K21 Gastro-esophageal reflux disease
- K21.0 Gastro-esophageal reflux disease with esophagitis
- K21.9 Gastro-esophageal reflux disease without esophagitis

GASTROINTESTINAL BLEEDING

Czarina E. Sánchez • Leon D. Sánchez



BASICS

DESCRIPTION

- Bleeding from GI tract:
 - Upper GI tract: Proximal to ligament of Treitz
 - Lower GI tract: Distal to ligament of Treitz to anus
- Mortality rate:
 - 10% overall; from <5% in children up to 25% for adults of age >70
 - Upper GI bleed (UGIB) 6–8%; variceal 30–50%
 - Lower GI bleed (LGIB) 2–4%

ETIOLOGY

Upper Gi Bleed (UGIB):

- Ulcerative disease of upper GI tract:
 - Peptic ulcer disease (40%):
 - *Helicobacter pylori* infection
 - Drug-induced (NSAIDs, aspirin, glucocorticoids, K⁺ supplements, Fe supplements)
 - Gastric or esophageal erosions (25%):
 - Reflux esophagitis
 - Infectious esophagitis (*Candida*, HSV, CMV)
 - Pill-induced esophagitis
 - Esophageal foreign body
 - Gastritis and stress ulcerations:
 - Toxic agents (NSAIDs, alcohol, bile)
 - Mucosal hypoxia (trauma, burns, sepsis)
 - Cushing ulcers from severe CNS damage
 - Chemotherapy
- Portal HTN:
 - Esophageal or gastric varices (10%)
 - Portal hypertensive gastropathy
- Arteriovenous malformations:
 - Aortoenteric fistula (s/p aortoiliac surgery)
 - Hereditary hemorrhagic telangiectasia (Osler—Weber—Rendu syndrome)
 - Dieulafoy vascular malformations
 - Gastric antral vascular ectasia (GAVE or watermelon stomach)
 - Idiopathic angiomas

- Mallory–Weiss tear (5%)
- Gastric and esophageal tumors
- Pancreatic hemorrhage
- Hemobilia
- *Strongyloides stercoralis* infection

Lower Gi Bleed (LGIB):

- Diverticulosis (33%)
- Cancer or polyps (19%)
- Colitis (18%):
 - Ischemic, inflammatory, infectious, or radiation
- Vascular:
 - Angiodysplasia (8%)
 - Radiation telangiectasia
 - Aortocolonic fistula
- Inflammatory bowel disease:
 - Crohn's disease and ulcerative colitis
- Postpolypectomy
- Anorectal (4%):
 - Hemorrhoids (internal and external)
 - Anal fissures
 - Anorectal varices
 - Rectal ulcer
 - Foreign body

Pediatric Considerations

Meckel diverticulum and intussusception are the most common causes of LGIB in children.

DIAGNOSIS

SIGNS AND SYMPTOMS

- Both UGIB and LGIB may present with signs/symptoms of hypovolemia
- UGIB classic presentation:
 - Hematemesis or coffee ground emesis
 - Melena: Black tarry stool
- LGIB classic presentation:
 - Hematochezia: Bright red or maroon stool

ALERT
Hematochezia classically signals an LGIB, but can also be seen with brisk UGIB.

History

- Hematemesis and melena most common
- Coffee ground emesis
- Black stools
- Bright red blood per rectum
- Abdominal pain
- Weakness or lightheadedness
- Dyspnea
- Confusion or agitation

Physical-Exam

- Tachycardia
- Hypotension
- Pale conjunctiva
- Dry mucous membranes
- Bloody, melanotic, or heme-positive stools
- Shock

ESSENTIAL WORKUP

- CBC, coagulation studies, electrolytes
- Perform ENT exam. Distinguish between hemoptysis and hematemesis:
 - Pulmonary source:
 - Bright red and frothy in appearance
 - Sputum mixed with blood is likely pulmonary
 - pH >7
 - GI source:
 - Dark red/brown blood, ± gastric contents
 - Associated with nausea/vomiting
 - pH <7
- Consider nasogastric lavage:
 - Might help determine if bleeding is ongoing and facilitate endoscopy
 - Controversial studies have failed to demonstrate outcome benefit. False-negatives, if bleeding beyond pylorus.
- Rectal exam:
 - Inspect for hemorrhoids and anal fissures
 - Examine stool color
 - False-positive Hemocult result:
 - Raw red meat
 - Iron supplements
 - Fruits: Cantaloupe, grapefruit, figs
 - Vegetables: Raw broccoli, cauliflower, radish
 - Methylene blue, chlorophyll
 - Iodide, bromide

- False-negative Hemoccult result:
 - Bile
 - Mg-containing antacids
 - Ascorbic acid
- Agents causing black stools, but negative Hemoccult:
 - Iron
 - Charcoal
 - Bismuth (i.e., Pepto-Bismol)
 - Food dyes
 - Beets

Pediatric Considerations

Bloody stool in newborns may be caused by the infant swallowing maternal blood.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC:
 - Anemia (low mean corpuscular volume seen with chronic blood loss)
 - Thrombocytopenia
- Electrolytes, BUN, creatinine, glucose
- Coagulation profile
- Lactate
- LFTs, if upper GI bleeding suspected
- Type and screen/cross for active bleeding or unstable vital signs
- BUN/Cr ratio > 36 has a high sensitivity but low specificity for UGIB

ALERT

Hematocrit can remain normal for a period after acute blood loss; a drop may not be immediately seen.

Imaging

- Upright CXR if concern for aspiration or perforation
- Angiography/arterial embolization:
 - Effective for identifying large, active bleeding
- Radionucleotide (tagged red blood cell) scan:
 - Effective for identifying slow, active bleeding

Diagnostic Procedures/Surgery

- Anoscopy:
 - For suspected internal hemorrhoids or fissures
- Esophagogastroduodenoscopy (EGD):
 - Diagnostic and possibly therapeutic

- Colonoscopy:
 - Diagnostic only
 - Best after adequate bowel prep
- Bowel resection:
 - Reserved for refractory bleeding

DIFFERENTIAL DIAGNOSIS

- Epistaxis
- Oropharyngeal bleeding
- Hemoptysis
- Hematuria
- Vaginal bleeding
- Visceral trauma



TREATMENT

PRE HOSPITAL

- Stabilize airway
 - Intubate for massive UGIB, if patient unable to protect airway
- Establish access
 - Insert large-bore IV (16–18g) and administer crystalloid to keep SBP > 90 mm Hg
 - Attempt 2nd IV line en route to hospital

INITIAL STABILIZATION/THERAPY

- Assess airway, breathing, and circulation
- Control airway in unstable patients, with massive bleeding, or unable to protect airway
- Initiate 2 large-bore (16 g) IVs and place on cardiac monitor
- Provide volume:
 - Administer 1 L NS bolus (peds: 20 mL/kg) and repeat once, if necessary
 - Transfuse RBCs if significant anemia or unstable after crystalloid boluses
 - Cross-matched or type-specific blood, if available
 - Otherwise, O negative for premenopausal women, O positive for others
 - Provide fresh frozen plasma (FFP) along with RBC transfusion in ratio of 1:2–4. For patients requiring massive transfusion, consider adding FFP and platelets in 1:1:1 ratio with RBCs
 - For coagulopathy, administer FFP and vitamin K (if INR > 1.5) and platelets (if platelets < 50,000/uL)

ED TREATMENT/PROCEDURES

- Consult gastroenterology for any significant GI bleeding

- Consider surgical consult and/or interventional radiology for massive active bleeding, unstable patient, or evidence of perforation
- Place Foley catheter to monitor urine output
- Consider nasogastric tube (NGT), as above
- Blood transfusion indications:
 - _ Significant anemia:
 - Hemoglobin <7 g/dL
 - Hemoglobin <10 g/dL when at increased risk of ischemia (e.g., CAD and CVA)
 - Evidence of end-organ ischemia
 - Ongoing chest pain/ischemic EKG changes
 - _ Unstable vital signs despite crystalloid bolus

ALERT

Avoid overtransfusion in variceal bleeding; it can precipitate further bleeding

- UGIB treatment
 - _ IV proton pump inhibitor (PPI) (e.g., pantoprazole)
 - _ Octreotide for suspected variceal bleeding
 - _ Consider vasopressin for active variceal bleeding:
 - Bleeding cessation benefits may be counterbalanced by increased mortality due to ischemia
 - Administer with IV nitroglycerin to reduce tissue ischemia
 - _ High risk for active bleeding with 2 out of 3 risk factors:
 - Bright blood from NGT
 - Hemoglobin <8 g/dL
 - WBC >12,000/uL
 - _ Emergent endoscopy
 - _ Therapeutic options:
 - Cauterization of bleeding ulcers/vessels
 - Endoscopic sclerotherapy
 - _ Balloon tamponade with Blakemore tube is a last resort for varices
 - _ In cirrhotics with UGIB prophylactic antibiotic use reduce bacterial infections and all cause mortality
- LGIB treatment
 - _ Consider angiography for massive, active bleeding with directed vasopressin infusion
 - _ Consider bowel resection for massive bleeding refractory to medical management

MEDICATION

- Pantoprazole: 80 mg (peds: Dosing not approved) IV bolus followed by an infusion of 8 mg/h for 72 hr
- Octreotide: 50 µg (peds: 1–2 µg/kg) bolus, then 50 µg/h (peds: 1–2 µg/kg/h) IV

- Somatostatin: 250 µg (peds: Not established) IV bolus and 250–500 µg/h for 2–5 days (not available in US)
- Vasopressin: 0.4–1 IU/min (peds: 0.002–0.005 IU/kg/min) IV
- Nitroglycerin: 10–50 µg/min (peds: Not established) IV
- Vitamin K: 10 mg (peds: 1–5 mg) PO/SC/IV q24h



FOLLOW-UP

DISPOSITION

Admission Criteria

- Active bleeding
- Age > 65 or comorbid conditions
- Coagulopathy
- Decreased hematocrit
- Unstable vital signs at any time

Discharge Criteria

- Resolution of UGIB with negative nasogastric lavage and EGD
- Minor or resolved LGIB
- Stable hematocrit > 30 or hemoglobin > 10 g/dL
- Otherwise healthy patient

Issues for Referral

Consider referral to gastroenterologist for outpatient colonoscopy and/or EGD

FOLLOW-UP RECOMMENDATIONS

- Patients discharged from the ED should have close follow-up within 24–36 hr
- Give strict discharge instructions to return if further bleeding or other concerning symptoms (lightheadedness, dyspnea, chest pain, etc.) occur
- Patients with UGIB should be discharged on a PPI, and advised to avoid caffeine, alcohol, tobacco, NSAIDs, and aspirin

PEARLS AND PITFALLS

- 10–15% of UGIB present with hematochezia
- Consider GIB in patients presenting with signs of hypovolemia or hypovolemic shock
- Common pitfall: Failure to adequately resuscitate with crystalloid and blood products

Geriatric Considerations

PUD is the predominant cause of GIB in elderly and has a higher associated mortality.

ADDITIONAL READING

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- Wolfson AB, Hendey GW, Ling LJ, et al., eds. *Harwood-Nuss' Clinical Practice of Emergency Medicine*. 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2009.



CODES

ICD9

- 533.40 Chronic or unspecified peptic ulcer of unspecified site with hemorrhage, without mention of obstruction
- 535.51 Unspecified gastritis and gastroduodenitis, with hemorrhage
- 578.9 Hemorrhage of gastrointestinal tract, unspecified

ICD10

- K27.4 Chronic or unsp peptic ulcer, site unsp, with hemorrhage
- K29.71 Gastritis, unspecified, with bleeding
- K92.2 Gastrointestinal hemorrhage, unspecified

GERIATRIC TRAUMA

Charles W. O'Connell • Peter Witucki



BASICS

DESCRIPTION

- Geriatric specific considerations and approach to the elderly trauma patient
- Should be used in conjunction with the accepted standard treatment of traumatic injuries (see trauma, multiple)
- Advanced age is a known risk factor for adverse outcomes following trauma
- Generally age >65, age not well defined, difficult to target due to discrepancies between physiologic and chronologic age in individuals

EPIDEMIOLOGY

Incidence and Prevalence Estimates

ETIOLOGY

Most common mechanisms:

- Falls—most common cause of injury in patients of age >65, often occurs on an even, flat surface
- Motor vehicle crashes—2nd leading cause, most common fatal etiology
- Pedestrian—motor vehicle collisions, diminished cognitive skills, poor vision/hearing, impaired gait contribute to increased incidence
- Burns—higher fatality rate than young adults with same extent of burn
- Violence—less common mechanism than in younger ages, have heightened suspicion for elderly abuse, an under recognized issue
- Elderly more susceptible to serious injury from low-energy mechanisms



DIAGNOSIS

- Triage to major trauma center is determined by local protocols
- Injured patients with potential need for surgical, neurosurgical, or orthopedic intervention should be transferred to major trauma center
- Threshold for scene triage or transfer to trauma center should be lower for elderly

SIGNS AND SYMPTOMS

- The same pattern of assessment using primary survey (ABCDE) and secondary survey should be used with geriatric patients as with younger patients (see trauma, multiple)
- Normal vital signs can lead to false sense of security
 - Hypoperfusion often masked by inadequate physiologic response,

underlying medical pathology, and medication effects

Primary survey (ABCDE)

- Airway, cervical spine—establish and maintain a patent airway with C-spine immobilization
 - Anatomic variation in elderly can lead to more difficult airways (dentures, cervical arthritis, TMJ arthritis)
 - Failure to recognize indications for early intubation is a common mistake
- Breathing—maintain adequate and effective breathing and ventilation
 - Weakened respiratory muscles and degenerative changes in chest wall result in diminished effective ventilation
 - Blunted response to hypoxia, hypercarbia, and acidosis delays onset of clinical distress
 - Lower threshold to intubate elderly patients
- Circulation—ensure adequate perfusion
 - Vigilant hemodynamic monitoring, heart rate, and BP do not always correlate well with cardiac output
 - Geriatric patients often have impaired chronotropic response to hypovolemia
 - Cardiovascular response may be blunted by rate controlling meds (β -blockers, Calcium channel blockers)
 - Baseline hypertension, common in elderly, may obscure relative hypotension
 - Bleeding made worse by antiplatelet and anticoagulation medicines
- Disability—rapid neurologic evaluation to assess for intracranial and spinal cord injury
 - Brain atrophy may delay onset of clinical symptoms from compressive effects
 - Grave error to assume alterations in mental status due solely to underlying dementia or senility
- Exposure—patient should be undressed completely
- Secondary survey
 - After the primary survey has been completed
 - Stabilization at each level
 - Complete physical exam from head to toe

History

- The geriatric trauma patient should be viewed as both a trauma and a medical patient
- Elderly patients can have significant comorbidities, past medical history, medications, and allergies are essential
- Comorbid medical conditions may have precipitated the traumatic event
- Consider hypoglycemia, syncope, cardiac dysrhythmia, CVA, UTI, etc.
- Details of the mechanism, initial presentation, and treatment rendered should be

elicited from EMS personnel

- Concurrent medical conditions impede compensation, confound interpretation of severity and response, and complicate resuscitation.

Physical-Exam

Should follow primary and secondary surveys

DIAGNOSIS TESTS & INTERPRETATION

- Primary and secondary survey
- Cervical spine and chest imaging are mandatory for victims of major traumas
- Pelvic radiographs should be performed with clinical suspicion of pelvic trauma or with hemodynamic instability
- CBC, ABG, blood type
- Electrolytes, renal function, serum glucose
- Urine dip for blood, UA if dip shows positive result
- Coagulation profile
- Base deficit, lactate
- Ethanol screen

Imaging

- Liberal use of head CT is recommended for elderly with closed head trauma
- Nexus criteria has been validated in ages >65; however, cervical spine imaging needed in majority of geriatric traumas. CT scan emerging as study of choice for high suspicion, high-risk mechanism or age related changes likely to limit plain films
- Significant blunt and penetrating chest trauma requires objective evaluations of the heart and great vessels with echocardiography, CT scan, angiography, or direct visualization.
- Blunt abdominal trauma requires objective evaluation, modality depends on patient's condition
- Hemodynamically stable patients should have an abdominal CT with IV contrast
- Ensure adequate hydration and assess baseline renal function prior to contrast load when clinical status permits.
- Unstable patients should have FAST exam or diagnostic peritoneal lavage
- CT with contrast is a valuable diagnostic tool for abdominal trauma, but predispose to risk of contrast related renal impairment
- Extremity injury:
 - Radiographs
 - Suspected vascular damage requires angiography or duplex ultrasound



TREATMENT

PRE HOSPITAL

- Emphasis should be placed on airway maintenance, control of external bleeding and shock, immobilization, and immediate transfer to appropriate facility

INITIAL STABILIZATION/THERAPY

- Airway—take into account anatomical variations when establishing an airway
- Breathing
 - Continuous pulse oximetry and capnometry helpful
 - Administer supplemental oxygen to maintain oxygen saturation > 95%
 - Serial ABGs may provide early insight to respiratory function and reserve
 - Timely intubation in patients with ventilatory compromise and more severe injuries
 - Intubation indications: Respiratory rate > 40 breaths/min, PaO₂ is < 60 mm Hg or PaCO₂ > 50 mm Hg
 - Adequate analgesia of chest wall pain is essential for optimizing ventilation
- Circulation—severity of hemodynamic instability often underappreciated by clinicians
 - Serial crystalloid fluid boluses of 250–500 mL
 - Early invasive monitoring has been advocated, better assess need for volume loading and inotropic support
 - Geriatric patients can decompensate from overly aggressive volume replacement
 - Strong consideration for early and liberal use of red blood cell transfusion
 - Target hemoglobin level is controversial, but many authors recommend 10 g/dL
 - Recognize the harmful effects and complications of red blood cell transfusions
 - Blood viscosity, infection, and impairment of immune response
 - Serial base deficit and lactate levels provide good initial measures of shock and can guide resuscitation decisions
 - Creatinine clearance reduced in elderly
 - Kidneys more susceptible to injury from hypovolemia, medications, and nephrotoxins
- Disability:
 - Head Injury: Age is an independent risk factor for morbidity and mortality
 - Age-related atrophy and mental decline may confound the evaluation of mental status
 - Anticoagulated patients with blunt head injury at increased risk for intracranial bleeds and delayed bleeding.
 - Strongly consider repeat imaging to detect delayed bleeds in anticoagulated patients
 - When indicated, initiate treatment for intracranial hypertension, maintain

- spinal immobilization, and obtain definitive airway
- Exposure: Completely undress patient, but prevent hypothermia
 - Age-related changes and medications make elderly more susceptible to hypothermia
 - Hypothermia not attributable to shock or exposure should raise concern for sepsis, endocrinopathy, or drug ingestion
- Common injury patterns:
- Head injury
 - Less prone to epidural hematomas
 - Higher incidence of subdural hematomas
- Cervical spine injuries
 - Propensity to sustain cervical spine injuries from seemingly minor trauma (fall from standing or seated height)
 - C1–C2 and odontoid fractures are particularly more common among elderly
 - Underlying cervical spine pathology, such as arthritis may predispose to spinal cord injuries
 - With hyperextension injuries, increased risk of developing a central cord syndrome
- Vertebral injuries
 - More susceptible to fractures, especially anterior wedge compression fractures
- Chest trauma
 - Rib fracture is most common; in geriatric patients there is an increased risk of pneumonia and mortality with each additional rib fracture
 - Hemopneumothorax, pulmonary contusion, flail chest, and cardiac contusion can quickly lead to decompensation
- Abdominal trauma
 - Similar pattern of injury as younger adults
 - Paramount to recognize signs of hemodynamic stability early
 - Nonoperative treatment of hemodynamically stable blunt hepatic and splenic injuries has emerged as the trend
 - Should have high index of suspicion for internal injuries with associated pelvic and lower rib cage injuries
- Orthopedic injuries—more predisposed due to osteopenia and osteoporotic changes
 - Uniquely susceptible to pelvic and hip fractures
 - Goal of orthopedic injuries is to undertake the least invasive, most definitive procedure that will permit early return to function
- Anticoagulation—consider fresh frozen plasma, cryoprecipitate, and concentrated factor for significant bleeds depending on indications
 - Beware of fluid overload and thrombotic complications

ED TREATMENT/PROCEDURES

- Early monitoring of pulmonary and cardiovascular systems must be instituted

- Prompt stabilization, early recognition of the need for operative intervention, and appropriate and expedient surgical consultation are paramount
- Definitive treatment is often surgical
- Elderly patients benefit from preferential transfer to trauma centers and aggressive, yet thoughtful care
- No reliable age-based criteria upon which to base decisions to triage away from care
- Good outcomes can be achieved with appropriately aggressive trauma care
- Equally important to limit intensive treatment to injuries which are survivable and allow potentially acceptable quality of life
- Seek existence of advance directives, living will, or similar legal document



FOLLOW-UP

DISPOSITION

Admission Criteria

- Most major trauma patients should be admitted for observation, monitoring, and further evaluation
- Lower threshold for admitting geriatric patients to ward, monitored settings, or ICU
- Elderly patients with polytrauma, significant chest wall injuries, abnormal vital signs, evidence of hypoperfusion should be admitted to the ICU

Discharge Criteria

Patients with minor trauma and negative workup/imaging may be observed in the ED for several hours and then discharged

Issues for Referral

Follow-up should be determined by the types of injuries sustained and specialty care required.

FOLLOW-UP RECOMMENDATIONS

Follow-up and referral should be determined by the types of injuries sustained and specialty care required

PEARLS AND PITFALLS

- Minor mechanisms of injury can produce serious injury and complication because of the effect of limited physiologic reserve, medication effects, and unrecognized hypoperfusion
- Frequent use of medications, especially β -blockers and anticoagulants complicate

assessment and management

- Mistaken impression that “normal” BP and heart rate imply normovolemia.
- Geriatric trauma patients must be treated as both trauma and medical patients.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Specific anatomic injuries
- Shock
- Airway management
- Multiple trauma



CODES

ICD9

- V15.88 History of fall
- 797 Senility without mention of psychosis
- 995.81 Adult physical abuse

ICD10

- R54 Age-related physical debility
- T74.11XA Adult physical abuse, confirmed, initial encounter
- Z91.81 History of falling

GHB POISONING

Amy V. Kontrick • Mark B. Mycyk



BASICS

DESCRIPTION

- Naturally occurring analog of γ -aminobutyric acid (GABA)
- Used medically for narcolepsy
- Nonmedical uses:
 - Bodybuilding agent
 - Euphoric agent
 - Date-rape/predatory agent
- γ -Hydroxybutyrate (GHB) precursors (γ -butyrolactone [GBL], 1,4 butanediol [1,4-BD], GHV [γ -hydroxyvalerate], and GVL) have same effects as GHB.
- Onset of activity: 15–30 min after ingestion
- Duration of effect: 2–6 hr

ETIOLOGY

Deliberate or accidental ingestion of GHB



DIAGNOSIS

SIGNS AND SYMPTOMS

- CNS:
 - CNS depression
 - Ataxia/dizziness
 - Impaired judgment
 - Aggressive behavior
 - Clonic movements of the extremities
 - Coma
 - Seizures
- Pulmonary:
 - Respiratory depression
 - Apnea
 - Laryngospasm (rare)
- GI:
 - Nausea
 - Vomiting
- Cardiovascular:
 - Bradycardia
 - Atrioventricular block

- Hypotension
- Other:
 - Nystagmus
 - Hypothermia
- Withdrawal symptoms:
 - HTN
 - Tachycardia
 - Hyperthermia
 - Agitation
 - Diaphoresis
 - Tremors
 - Nausea, vomiting, and abdominal cramping
 - Hallucinations, delusions, and psychosis

ESSENTIAL WORKUP

- Diagnosis based on clinical presentation and an accurate history
- Exclude coingestants if signs and symptoms inconsistent with GHB intoxication

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Confirmatory GHB screen is typically a send-out lab and does not change ED management.
- Urine toxicology screen to exclude coingestants
- Serum alcohol level
- Urinalysis and creatine kinase (CK) if suspected rhabdomyolysis from prolonged immobilization or agitation

Imaging

- ECG:
 - Sinus bradycardia
 - Atrioventricular block
- CXR:
 - Aspiration pneumonia
- Head CT if suspected occult head trauma

DIFFERENTIAL DIAGNOSIS

- Alcohol intoxication
- Barbiturate overdose
- Benzodiazepine overdose
- Neuroleptic overdose
- Opiate overdose
- Withdrawal:

- Alcohol withdrawal
- Sedative-hypnotic withdrawal



TREATMENT

PRE HOSPITAL

Transport all pills/bottles and drug paraphernalia involved in overdose for identification in ED.

INITIAL STABILIZATION/THERAPY

- ABCs:
 - Airway control essential
 - Administer supplemental oxygen
 - Intubate if indicated
- Administer thiamine, dextrose (or Accu-Chek), and naloxone for depressed mental status.

ED TREATMENT/PROCEDURES

- Supportive care
- Bradycardia:
 - Atropine
 - Temporary pacing
- Hypotension:
 - 0.9% NS IV fluid bolus
 - Trendelenburg
 - Dopamine titrated to pressure
- Seizures:
 - Treat initially with benzodiazepine.
 - Treat refractory seizures with phenobarbital.
- Withdrawal:
 - Treat aggressively with benzodiazepine.
 - Treat with phenobarbital or propofol if large doses of benzodiazepines unsuccessful.

MEDICATION

- Dextrose: 50–100 mL D₅₀ (peds: 2 mL/kg of D₂₅ over 1 min) IV; repeat if necessary
- Diazepam: 5–10 mg (peds: 0.2–0.5 mg/kg) IV q10–15min
- Dopamine: 2–20 µg/kg/min with titration to effect
- Lorazepam: 2–4 mg (peds: 0.03–0.05 mg/kg) IV q10–15min
- Naloxone: 0.4–2 mg (peds: 0.1 mg/kg; neonatal: 10–30 µg/kg) IV or IM
- Phenobarbital: 10–20 mg/kg IV (loading dose) monitor for respiratory depression with IV administration

- Propofol: 0.5–1 mg/kg IV (loading dose), then 5–50 µg/kg/min (maintenance dose)
- Thiamine (vitamin B₁): 100 mg (peds: 50 mg) IV or IM



FOLLOW-UP

DISPOSITION

Admission Criteria

- Intubated patient
- Patient with hypothermia or other hemodynamic instability
- Coingestion prolonging duration of intoxication

Discharge Criteria

- Asymptomatic after 6 hr of observation
- No clinical evidence of withdrawal syndrome

ALERT

Withdrawal from GHB is life-threatening and appears similar to alcohol withdrawal. Prolonged inpatient treatment may be indicated.

FOLLOW-UP RECOMMENDATIONS

- Substance abuse referral for patients with recreational drug abuse.
- Patients with unintentional (accidental) poisoning require poison prevention counseling.
- Patients with intentional (e.g., suicide) poisoning require psychiatric evaluation.

PEARLS AND PITFALLS

- Consider nontoxicologic causes for persistent altered mental status
- Routine hospital drug testing will not confirm GHB or other common recreational drugs of abuse

ADDITIONAL READING

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CODES

ICD9

968.4 Poisoning by other and unspecified general anesthetics

ICD10

- T41.291A Poisoning by oth general anesthetics, accidental, init
- T41.293A Poisoning by other general anesthetics, assault, init enctr
- T41.294A Poisoning by oth general anesthetics, undetermined, init

GIANT CELL ARTERITIS (GCA) (TEMPORAL ARTERITIS)

Donald J. Lefkowitz

BASICS

DESCRIPTION

- Chronic vasculitis of large- and medium-sized vessels that occurs among individuals over 50 yr of age
- Often referred to as temporal arteritis (TA)
- Median age of onset is 72
- Most commonly causes inflammation of arteries originating from the arch of the aorta
- Although usually clinically silent, involvement of the thoracic aorta occurs in a significant minority of patients, and aortic aneurysm or dissection may result
- Thoracic aortic aneurysm is a late manifestation with an incidence 17 times those without TA
- Abdominal aortic aneurysm is about twice as common in those with giant cell arteritis (GCA)
- Pathologic specimens feature patchy mononuclear granulomatous inflammation resulting in a markedly thickened intima and occlusion of the vessel lumen
- Occlusive arteritis may involve thrombosis of the ophthalmic artery resulting in anterior ischemic optic neuropathy (AION) and acute visual loss:
 - Visual symptoms are an ophthalmic emergency
- Inflammation of arteries supplying the muscles of mastication results in jaw claudication and tongue discomfort
- Age is the greatest risk factor:
 - Rare in patients <50 yr old
 - >90% are >60 yr old
 - Prevalence in individuals >50 yr is estimated at 1:500
- Increased prevalence in Northern latitude
- 2 to 4 times more common in women
- Rare in African American patients, common in Whites
- There is a strong association with polymyalgia rheumatica (PMR) ~50%

Genetics

Genetic predisposition is linked to HLA-DR4—60% prevalence

ETIOLOGY

- Unknown
- Genetic, environmental and autoimmune factors have been identified



DIAGNOSIS

- Presence of any 3 or more of the following in patients with vasculitis:
 - ESR > 50
 - Age greater than 50 yr
 - New onset of localized headache
 - Tenderness or decreased pulsation of temporal artery
 - New visual symptoms
 - Biopsy revealing necrotizing arteritis

SIGNS AND SYMPTOMS

- May present with acute, subacute, or chronic symptoms:
 - Headache is the single most frequent symptom (70%)
 - Often localized, boring, or lancinating in quality
 - Often described as unilateral over a temple
- Tongue or jaw claudication upon mastication is the common symptom (50%)
- Constitutional symptoms:
 - Fatigue
 - Malaise
 - Anorexia
 - Weight loss
 - Weakness
 - Arthralgias
 - Low-grade fever
- Visual findings:
 - Findings are usually in 1 eye
 - May develop weeks to months after the onset of other symptoms
 - May fluctuate, but visual impairment does not usually improve over time, even with treatment
 - Amaurosis Fugax
 - Blindness
 - Diplopia
 - Ptosis
 - Extraocular muscle weakness
 - Scotomata
 - Blurred vision
- Scalp tenderness, especially over the temporal artery
- Pulsations over temporal artery:
 - Increased pulsations early in disease
 - Decreased pulsations late in the disease
- Erythema, warmth, swelling, or nodules over scalp arteries
- Bruits or decreased pulses over large arteries
- Sore throat, cough, dysphagia

- Rare findings:
 - Respiratory symptoms
 - Ischemic chest pain
 - Congestive heart failure
- Neurologic problems:
 - Occur in up to one-third of patients:
 - Neuropathies
 - Transient ischemic attacks
 - Cerebral vascular accidents
- Occult manifestations include:
 - Glossitis
 - Lingual infarction
 - Tongue infarction
 - Raynaud phenomenon
- Up to 30% may not present with the classic features of headache, scalp tenderness, visual changes, or jaw claudication
- Frequently associated with PMR (up to 50%):
 - Stiffness
 - Aching pain in the proximal muscles
 - Worse in the morning and decreasing with exercise
- Often associated with synovitis, especially in the knees

ESSENTIAL WORKUP

- Focused physical exam with emphasis on:
 - Temporal artery and scalp abnormalities
 - Complete neurologic exam
 - Ophthalmic exam including visual acuity and visual field testing
- Fundoscopy:
 - Often normal initially
 - Iritis and fine vitreous opacities may be early findings
 - Optic nerve edema
 - Swollen, pale disc with blurred margins
 - Pallor
 - Hemorrhage
 - Scattered cotton-wool spots
 - Vessel engorgement and exudates are seen later
- Any pulse differences in the extremities or bruits over large arteries should be noted

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Elevated ESR, often > 100 mm/hr

- ESR < 40 is rare
- C-reactive protein above 2.45 mg/dL
- Complete blood count (CBC):
 - A mild normochromic anemia is typical
 - Thrombocytosis (often mild)
 - White cell count can be normal or slightly elevated and differential is usually normal
- Liver function tests and prothrombin time may be elevated; creatine phosphokinase, tests of renal function, and urinalysis are generally normal
- Elevation in interleukin-6 (IL-6) is seen during flares

Imaging

- Doppler ultrasound:
 - Decreased blood flow in temporal, facial, and ophthalmic arteries
 - Presence of a halo is highly suggestive of active TA.
- MRI:
 - Indicated for exam of large arteries
- Angiogram:
 - Smooth, tapered occlusions or stenosis

Diagnostic Procedures/Surgery

- Temporal artery biopsy:
 - Multiple sections should be done in as soon as feasible after initiation of steroid therapy
 - Gold standard for diagnosis
 - Contralateral biopsy is recommended if 1st is negative and index of suspicion high

DIFFERENTIAL DIAGNOSIS

- Vasculitides:
 - Polyarteritis nodosa
 - Hypersensitivity vasculitis
 - Systemic lupus erythematosus
 - Takayasu arteritis
 - Wegener granulomatosis
- Thrombosis of retinal, ophthalmic, or temporal arteries
- Lyme disease
- Nonarteritic anterior ischemia optic neuropathy (NAAION)



TREATMENT

PRE HOSPITAL

- Acute symptoms may be confused with stroke
- Initiate appropriate monitoring and oxygen
- Patients may be hypotensive from 1 of the rare sequelae (aortic dissection, abdominal aortic aneurysm, or myocardial infarction)

INITIAL STABILIZATION/THERAPY

Although rare, patients may present with vascular catastrophe such as aortic dissection or myocardial infarction and need appropriate aggressive early management

ED TREATMENT/PROCEDURES

- Steroids:
 - Strong clinical indications should be present if started before temporal artery biopsy
 - Scheduling of TA biopsy should not delay steroid therapy when clinical suspicion is high as pathologic vessel changes resolve slowly and will remain for weeks
 - Early, aggressive treatment can significantly reduce the incidence of blindness
 - Steroids effectively control systemic and local symptoms within days to weeks
 - Treatment with prednisone may continue for years—usual disease length is 3–4 yr
- Symptomatic pain management with NSAIDs, salicylates, and/or narcotics as indicated

MEDICATION

- Prednisone: 60–100 mg PO per day for at least 2 wk before considering tapering
- For acute onset visual symptoms, consider 1,000 mg methylprednisolone IV pulse therapy for the 1st 1–3 days
- Low-dose aspirin therapy to reduce thrombotic risks
- Pain management with NSAIDs or narcotics



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients with impending vascular complications or acute focal neurologic findings
- Patients with associated acute visual loss

Discharge Criteria

- Less symptomatic patients without evidence of end-organ involvement

- Follow-up arranged within 1--2 days

Issues for Referral

- Rheumatology
- Ophthalmology if associated with visual symptoms
- Neurology with acute focal neurologic findings

FOLLOW-UP RECOMMENDATIONS

- Rheumatology for steroid management and search for associated connective tissue disorders
- Ophthalmology and neurology for visual disturbance and/or focal neurologic findings.

PEARLS AND PITFALLS

- Permanent visual loss is the most feared complication of GCA
- Do not delay initiation of steroid therapy to await biopsy if strong clinical suspicion for GCA exists or if visual changes are reported
- Jaw claudication and amaurosis fugax, while often dramatic, are symptoms patients often neglect to report. Query directly and specifically about them if TA/GCA is being considered
- 25–50% of patients who present with acute loss of vision in 1 eye who go untreated will develop bilateral blindness

ADDITIONAL READING

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- Waldman CW, Waldman SD, Waldman RA. Giant cell arteritis. *Med Clin North Am*. 2013;97:329–335.



See Also (Topic, Algorithm, Electronic Media Element)

- Aortic Dissection, Thoracic
- Central Retinal Artery Occlusion
- Central Retinal Vein Occlusion
- Glaucoma
- Systemic Lupus erythematosus
- Vasculitis

ICD9

446.5 Giant cell arteritis

ICD10

- M31.5 Giant cell arteritis with polymyalgia rheumatica
- M31.6 Other giant cell arteritis

GIARDIASIS

Benjamin Mattingly • Joseph B. Schneider



BASICS

DESCRIPTION

- Noninvasive diarrhea
- Found worldwide:
 - 2–15% prevalence in developed nations
 - 20–40% prevalence in developing nations
- 5% of all travelers' diarrhea
- Most common intestinal parasite in US:
 - Highest incidence in early summer months through fall
 - Highest incidence in children aged 1–9 yr and adults aged 30–39 yr
 - In 2010, 19,888 cases reported in US (mostly from Northern States)
- Fecal–oral transmission:
 - Humans are major reservoir
 - Zoonotic reservoir in both domestic and wild mammals
 - Reservoir in contaminated surface water
- Populations at risk:
 - Travelers to endemic areas (developing countries, wilderness areas of US)
 - Children in day care centers and their close contacts
 - Institutionalized persons
 - Practitioners of anal sexual activity

ETIOLOGY

- *Giardia lamblia*:
 - A protozoan flagellate
- Also called *Giardia intestinalis* or *Giardia duodenalis*
- Ingested *Giardia* attach to intestinal villi
- Alters the intestinal brush-border enzymes, impairing digestion of lactose, and other saccharides
- No toxin produced



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Onset 1–2 wk postexposure
- Infection may be asymptomatic (most common).

- Diarrhea of acute onset (90% of symptomatic patients):
 - Foul-smelling stools
 - Steatorrhea
 - Nonbloody
 - Self-limiting within 2–4 wk
 - More severe in immunocompromised patients and patients with underlying bowel disease
- Flatulence and bloating (70–75%)
- Abdominal cramping (70%)
- Nausea (70%)
- Vomiting (30%)
- Malaise (86%)
- Anorexia (66%)
- Weight loss (60–70%)
- Fever is rare (15%)
- 30–50% of acute cases progress to chronic giardiasis (> 4 wk):
 - Fat malabsorption
 - Severe macrocytic anemia secondary to folate deficiency
 - Secondary lactase deficiency (in 20–40% of patients)
- Infection is more severe and harder to eradicate in immunosuppressed patients.

Pediatric Considerations

- Acute infection:
 - Severe dehydration
- Chronic infection:
 - Failure to thrive
 - Growth retardation and cognitive impairment owing to nutrient malabsorption

Physical-Exam

- Abdominal exam is benign.
- Extraintestinal manifestations (10% of patients):
 - Polyarthritits
 - Urticaria
 - Aphthous ulcers
 - Maculopapular rash
 - Biliary tract disease

ESSENTIAL WORKUP

- History:
 - Possible sources of exposure
 - Membership in high-risk group
- Physical exam:

- If gross or occult blood on digital rectal exam, unlikely to be *Giardia*

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Stool sample for microscopy (ova and parasites):
 - 50–70% sensitive if 1 sample
 - 85–90% sensitive if 3 samples taken at 2-day intervals (ideal)
 - 100% specific
 - Ability to detect other parasites as well
- Stool ELISA or immunofluorescent antibody (IFA) assay for *Giardia* antigen:
 - 95% sensitive, 95–100% specific
 - Unlike microscopy, cannot rule out other parasites
- Stool polymerase chain reaction (PCR):
 - 100% sensitive and 100% specific
- Fecal leukocytes and stool culture unnecessary unless enteroinvasive organisms suspected (fever, bloody stool)
- Serology for anti-*Giardia* antibodies not helpful in the ED setting
- Electrolytes, BUN/creatinine, glucose:
 - If prolonged diarrhea or evidence of dehydration
- CBC:
 - Macrocytic anemia in chronic giardiasis
 - Nondiagnostic in acute giardiasis

Imaging

Abdominal CT or ultrasound may show bowel wall thickening and flattened duodenal folds (nonspecific findings)

Diagnostic Procedures/Surgery

- Duodenal sampling:
 - Entero-Test (patient swallows a weighted string, which is later retrieved and examined for *Giardia* using microscopy)
- Endoscopy:
 - Duodenal aspiration
 - Endoscopic duodenal biopsy

DIFFERENTIAL DIAGNOSIS

- Viral gastroenteritis:
 - Norwalk virus
 - Rotavirus
 - Hepatitis A
- Bacterial infections:
 - *Staphylococcus*

- *Escherichia coli*
- *Shigella*
- *Salmonella*
- *Yersinia*
- *Campylobacter*
- *Clostridium difficile*
- *Vibrio cholerae*
- Other protozoa:
 - *Cryptosporidium*
 - Microsporidia
 - *Cyclospora*
 - *Isospora*
 - *Entamoeba*
- Inflammatory bowel disease
- Irritable bowel syndrome
- Lactase deficiency
- Tropical sprue
- Drugs and toxins:
 - Antibiotics
 - Calcium channel blockers
 - Magnesium antacids
 - Caffeine
 - Alcohol
 - Sorbitol
 - Laxative abuse
 - Quinidine
 - Colchicine
 - Mercury poisoning
- Endocrine:
 - Addison disease
 - Thyroid disorders
- Malignancy:
 - Colorectal carcinoma
 - Medullary carcinoma of the thyroid



TREATMENT

INITIAL STABILIZATION/THERAPY

- ABCs: Airway, breathing, circulation
- IV 0.9% NS if signs of significant dehydration

Pediatric Considerations

- For severe dehydration (>10%):
 - IV bolus with 0.9% NS at 20 mL/kg
 - Cardiac monitor
 - Blood glucose determination

ED TREATMENT/PROCEDURES

- Oral fluids for mild dehydration
- Correct any serum electrolyte imbalances.
- Stool sample for microscopy
- If stool sample is positive for *Giardia*: Treat as listed below under medication
- If stool sample negative for *Giardia*:
 - Refer to gastroenterologist for further specialized testing.
 - Consider empiric course of metronidazole if high suspicion for *Giardia*.

MEDICATION

First Line

- Metronidazole or tinidazole are the treatment of choice:
 - 90% cure rate for each
- Metronidazole: 250–500 mg (peds: 15 mg/kg/24h) PO q8h for 5–10 days
- Tinidazole: 2 g (peds [> 3 yr]: 50 mg/kg) PO once

Second Line

Albendazole (78–90% efficacy), quinacrine (90% efficacy), or nitazoxanide (75% efficacy) if 1st-line therapy fails

- Albendazole: 400 mg (peds: 10–15 mg/kg/24h) PO daily for 5–7 days
- Furazolidone: 100 mg (peds: 6–8 mg/kg/24h) PO q6h for 7–10 days (not available in US)
- Nitazoxanide: 500 mg (peds: 100 mg for ages 2–3 yr, 200 mg for ages 4–11 yr) PO BID for 3 days
- Paromomycin: 500 mg (peds: 25–30 mg/kg/24h) PO q8h for 5–10 days
- Quinacrine: 100 mg (peds: 6 mg/kg/24h) PO q8h for 5–7 days (limited availability)

Pediatric Considerations

- Metronidazole is 1st-line therapy (80–95% efficacy)
- Alternatives:
 - Furazolidone (80–85% efficacy)
 - Nitazoxanide (60–80% efficacy)
 - Paromomycin (55–90% efficacy)

Pregnancy Considerations

- Metronidazole contraindicated in 1st trimester

- Albendazole, quinacrine, and tinidazole are contraindicated throughout pregnancy
- Use nitazoxanide instead
- If mild symptoms only, consider deferring treatment until late pregnancy or postpartum

Immunocompromised Considerations

- Immunocompromised patients at risk for disease that is refractory to standard drug regimens:
 - Try drug of a different class/mechanism or metronidazole + quinacrine for at least 2 wk

ALERT

- Use furazolidone in older children only:
 - Causes hemolytic anemia in infants
 - Causes hemolytic anemia in persons with G6PD deficiency
- Avoid quinacrine in G6PD deficiency (causes hemolytic anemia)
- Avoid paromomycin in renal failure



FOLLOW-UP

DISPOSITION

Admission Criteria

- Hypotension or tachycardia unresponsive to IV fluids
- Severe electrolyte imbalance
- Children with > 10% dehydration
- Signs of sepsis/toxicity (rare in isolated giardiasis)
- Patients unable to maintain adequate oral hydration:
 - Extremes of age, cognitive impairment, significant comorbid illness

Discharge Criteria

- Able to maintain adequate oral hydration
- Dehydration responsive to IV fluids

FOLLOW-UP RECOMMENDATIONS

- Gastroenterology referral for diagnostic endoscopy if symptoms persist for > 4 wk despite drug therapy
- Acquired lactose intolerance may develop and last for weeks to months
- Association with postinfectious fatigue syndrome

PEARLS AND PITFALLS

Diagnosis is the greatest challenge in this disease:

- Include giardiasis in the differential diagnosis of all patients with diarrhea:
 - *Giardia* occasionally reported in domestic water supply
 - Patients may not present with the classic history and risk factors to have giardiasis
 - 1 stool sample is frequently insufficient for diagnosis

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Amebiasis
- Diarrhea, Adult



CODES

ICD9

007.1 Giardiasis

ICD10

A07.1 Giardiasis [lambliasis]

GLAUCOMA

Yasuharu Okuda • Lisa Jacobson

BASICS

DESCRIPTION

Disease characterized by elevation of intraocular pressure, optic neuropathy, and progressive loss of vision.

ETIOLOGY

- Primary glaucoma:
 - Open-angle glaucoma:
 - Normal anterior chamber angle
 - Insidious onset with persistent rise in intraocular pressure
 - Most common type accounting for 90% of glaucomas in US
 - Leading cause of blindness in African Americans
 - Risk factors include African American, age > 40 yr, family history, myopia, diabetes, and HTN
 - Acute angle-closure glaucoma:
 - Narrowing or closing of anterior chamber angle precluding natural flow of aqueous humor from posterior to anterior chamber of eye and through its filtering portion of trabecular meshwork
 - Usually abrupt onset with sudden increase in intraocular pressure
 - Risk factors include Asians and Eskimos, hyperopia, family history, increased age, and female gender
- Secondary glaucoma occurs from other diseases, including diseases of eye, trauma, and drugs:
 - Can be either open or closed angle
 - Drugs: Steroids, sertraline, bronchodilators, topiramate
 - Diseases: Neurofibromatosis, uveitis, neovascularization, and intraocular tumors
 - Trauma
 - Rapid correction of hyperglycemia

DIAGNOSIS

SIGNS AND SYMPTOMS

Classic descriptions:

- Open angle:
 - Painless and gradual loss of vision
- Closed angle:

- Painful loss of vision with fixed midsized pupil

History

- Primary open-angle glaucoma:
 - Gradual reduction in peripheral vision or night blindness
 - Typically bilateral
 - Painless
- Primary angle-closure glaucoma:
 - Severe deep eye pain and ipsilateral headache often associated with nausea and vomiting
 - Decrease in visual acuity often described as visual clouding with halos surrounding light sources
 - Associated abdominal pain, which may misdirect diagnosis
 - Concurrent exposure to dimly lit environment such as movie theater
 - Use of precipitating medications:
 - Mydriatic agents: Scopolamine, atropine
 - Sympathomimetics: Pseudoephedrine, albuterol
 - Antihistamines: Benadryl, Antivert
 - Antipsychotics: Haldol
 - Phenothiazines: Compazine, Phenergan
 - Tricyclic antidepressants: Elavil
 - Sulfonamides: Topiramate

Physical-Exam

- Primary open-angle glaucoma:
 - Decreased visual acuity
- Primary angle-closure glaucoma:
 - Decreased visual acuity
 - Pupil is mid-dilated and nonreactive.
 - Corneal edema with hazy appearance
 - Conjunctival injection, ciliary flush
 - Firm globe to palpation

ESSENTIAL WORKUP

- Detailed ocular exam
- Visual acuity:
 - Hand movements typically all that is seen
- Tonometry:
 - Normal pressures are 10–21 mm Hg.
 - Primary open-angle glaucoma:
 - Degree of elevation can vary, but 25–30% of patients may have normal intraocular pressures.
 - Primary angle-closure glaucoma:

○ Any elevation is abnormal, but usually seen in ranges >40 mm Hg.

- Slit-lamp exam:
 - Evaluation of anterior chamber angle
 - Used to eliminate other possibilities in differential including corneal abrasion and foreign body

DIAGNOSIS TESTS & INTERPRETATION

Lab

Directed toward workup of differential

Imaging

Directed toward workup of differential

Diagnostic Procedures/Surgery

Gonioscopy:

- This is direct measurement of the angle of closure

DIFFERENTIAL DIAGNOSIS

- Cavernous sinus thrombosis
- Acute iritis and uveitis
- Retinal artery or vein occlusion
- Temporal arteritis
- Retinal detachment
- Conjunctivitis
- Corneal abrasion



TREATMENT

PRE HOSPITAL

- No specific interventions need occur prior to arrival at the hospital in regard to the eye:
 - Pain control may be necessary
 - In traumatic etiologies, stabilize other injuries

INITIAL STABILIZATION/THERAPY

- Initiate steps to lower intraocular pressure in acute closed-angle glaucoma:
 - Address other effects of trauma if this was the etiology
 - Discontinue inciting medication when involved

ED TREATMENT/PROCEDURES

- Primary open-angle glaucoma:

- Recognition and prompt ophthalmologic referral
- Patients maintained on topical β -blockers or prostaglandin analogs to decrease IOP
- Primary angle-closure glaucoma (ophthalmologic emergency):
 - Intraocular pressure reduction:
 - Topical β -blocker, timolol maleate, to decrease aqueous humor production
 - Topical α_2 -agonist, apraclonidine, to decrease aqueous humor production
 - Carbonic anhydrase inhibitor, acetazolamide, for reduction of formation of aqueous humor
 - Hyperosmotic agent, mannitol, to draw aqueous humor from vitreous cavity into blood (indicated for severe attacks).
 - Movement of iris away from trabecular meshwork:
 - Topical parasympathomimetic, pilocarpine hydrochloride, to constrict pupil once intraocular pressure is < 40 mm Hg
 - Reduction of inflammation:
 - Topical corticosteroid, prednisolone acetate
 - Emergent ophthalmology consultation for possible definitive surgical treatment, laser iridectomy, if no improvement with medical management
 - Adequate narcotic analgesia and antiemetics as needed

MEDICATION

- Acetazolamide: 500 mg IV or PO
- Mannitol 20%: 1–2 g/kg IV over 30–60 min
- Pilocarpine hydrochloride 1–2% solution: 1 drop q15–30min until pupillary constriction occurs, then 1 drop q2–3h
- Prednisolone acetate 1% solution: 1 drop q15–30min for total of 4 doses

First Line

- β -Agonists:
 - Timolol maleate 0.25 or 0.5%:
 - 1 drop to affected eye BID
 - Levobunolol 0.25 or 0.5%:
 - 1 drop to affected eye BID
 - Carteolol HCL 1%:
 - 1 drop to affected eye BID
 - Betaxolol 0.25 or 0.5%:
 - 1–2 drop(s) to affected eye BID

Second Line

- Adrenergic agonists:

- Apraclonidine 0.5%, 1%:
 - 1–2 drop(s) to affected eye BID
- Brimonidine:
 - 1 drop to affected eye TID
- Carbonic anhydrase inhibitors:
 - Acetazolamide:
 - 125–250 mg PO QID
 - Methazolamide:
 - 50–100 mg PO BID
 - Dorzolamide HCl 2%:
 - 1 drop in affected eye TID
 - Brinzolamide:
 - 1 drop to affected eye TID
- Prostaglandin analogs:
 - Latanoprost:
 - 1 drop in affected eye QHS
 - Bimatoprost 0.03%:
 - 1 drop in affected eye QHS
 - Travoprost:
 - 1 drop in affected eye QHS
 - Unoprostone:
 - 1 drop to affected eye BID

Considerations in Prescribing

- Prostaglandin analogs have become standard of care for open-angle glaucoma due to an improved side-effect profile
- Due to cost, topical β -blockers are often still used primarily



FOLLOW-UP

DISPOSITION

Admission Criteria

- Severe pain, nausea, or vomiting
- Patients receiving parenteral medications should be observed for side effects.
- Patients without improvement of symptoms or intraocular pressures should be admitted for continued monitoring of intraocular pressure, medical treatment, and possible definitive surgical management:
 - Laser intervention is more likely than operative

Discharge Criteria

Patients with minor symptoms and significant improvement of intraocular pressure may

be safely discharged once seen by ophthalmology and with close, <24-hr follow-up.

Issues for Referral

If no ophthalmologist is available, treatment should be initiated and patient transferred to nearest hospital with ophthalmologic consultation.

FOLLOW-UP RECOMMENDATIONS

- Open-angle glaucoma patients need urgent ophthalmology follow-up to optimize medical management
- Closed-angle glaucoma patients need immediate intervention

PEARLS AND PITFALLS

- Increased IOP can cause vascular insufficiency and with delayed treatment vision loss can be permanent
- Eye pain/headache can be associated with severe abdominal pain—do not ignore the eye and miss the diagnosis
- Patients maintained on topical β -blockers for open-angle glaucoma may present with systemic side effects including orthostatic hypotension, bradycardia, or syncope

ADDITIONAL READING

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- Tse DM, Titchener AG, Sarkies N, et al. Acute angle closure glaucoma following head and orbital trauma. *Emerg Med J*. 2009;26(12):913.



See Also (Topic, Algorithm, Electronic Media Element)

- Red Eye
- Visual Loss



CODES

ICD9

- 365.9 Unspecified glaucoma
- 365.11 Primary open angle glaucoma
- 365.22 Acute angle-closure glaucoma

ICD10

- H40.9 Unspecified glaucoma
- H40.11X0 Primary open-angle glaucoma, stage unspecified
- H40.219 Acute angle-closure glaucoma, unspecified eye

GLOBE RUPTURE

Alexander T. Limkakeng, Jr. • Megan G. Kemnitz



BASICS

DESCRIPTION

- A full-thickness corneal or scleral injury owing to trauma
- Blunt trauma/globe rupture:
 - Causes an abrupt rise in intraocular pressure diffusely
 - Subsequent rupture of the eye either opposite the point of impact or at the weakest points:
 - Extraocular muscle insertion
 - Corneoscleral junction
 - Limbus, where the sclera is thinnest
- Penetrating injury/globe laceration:
 - Occurs with sharp objects or projectiles injuring the sclera or anterior eye directly
 - Most commonly anterior—the bony orbit protects the globe laterally and posteriorly
 - Posterior injury can occur with fracture of the bony orbit or with penetrating injuries of the eyelid or eyebrow.
- Prognosis worse with:
 - Larger lacerations
 - Injury posterior to the rectus insertion
 - Blunt injury
 - Intraocular foreign body, especially if made of organic material
 - Vitreous extrusion
 - Lens damage
 - Hyphema
 - Retinal detachment
 - Poor visual acuity at presentation
 - Afferent pupillary defect
 - Increased time to OR

ETIOLOGY

- Falls, impact injuries
- Sport-related injuries (e.g., elbow, ball impacts, arrows, game controllers, etc.)
- Indirect concussive injuries (explosions)
- Sharp instrument/stabbing injuries, accidental or intentional
- Projectile injuries (industrial, firearms, BB pellets, blast explosion shrapnel—glass, etc.)

SIGNS AND SYMPTOMS

- Pain
- Localized ecchymosis and swelling
- Scleral or corneal laceration
- Extrusion of intraocular contents
- Markedly decreased visual acuity
- Limited extraocular motion
- Hyphema
- Severe subconjunctival hemorrhage and edema, especially if circumferential bloody chemosis
- Abnormally deep or shallow anterior chamber
- Low intraocular pressure:
 - Note: Do not perform tonometry if there is suspected rupture.
- Irregular pupil (points toward lesion)
- Subluxed lens
- Commotio retinae—gray-white discoloration of the retina

History

- Mechanism of injury:
 - Assess for possibility of retained intraocular foreign body
- History of previous eye surgery
- Preinjury visual acuity
- Assess tetanus status
- Ascertain time of last PO intake

Physical-Exam

- Penlight or slit-lamp exam observing for signs of globe rupture
- If the diagnosis of ruptured globe is made, defer further ocular exam until the time of surgical repair:
 - Prevents placing any undue pressure on the eye and risking extrusion of the intraocular contents
- If no evidence of globe rupture on initial survey, proceed with thorough ophthalmologic exam:
 - Visual acuity
 - Slit-lamp exam
 - Cornea
 - Anterior chamber
 - Iris
 - Sclera
 - Fundus

- Retina
- Seidel test: Observe if fluorescein moves away as contents (which appear yellow-green) leak out at site of rupture:
- Measure intraocular pressure
 - Perform only if globe rupture is definitely *not* present.
- Ultrasound (only if rupture not suspected)

ESSENTIAL WORKUP

Perform thorough ocular exam as outlined above:

- Once diagnosis of globe rupture is suspected or made, defer further exam until time of repair.

DIAGNOSIS TESTS & INTERPRETATION

Lab

Preoperative labs:

- CBC
- Electrolytes
- Coagulation studies

Imaging

- Orbital radiograph (anteroposterior/lateral) for metallic intraocular foreign body
- CT scan of the orbits (axial and coronal views)
- MRI scan of the orbits after retained metallic foreign body is ruled out
- B-scan US of the eye

Diagnostic Procedures/Surgery

- Slit-lamp
- Fluorescein

DIFFERENTIAL DIAGNOSIS

- Intraocular foreign body
- Hyphema
- Severe subconjunctival hemorrhage and chemosis
- Partial corneal laceration
- Partial scleral laceration



TREATMENT

PRE HOSPITAL

- Place a shield (not patch) over eye with no pressure on the globe.
- Use a Styrofoam cup if no shield available.

INITIAL STABILIZATION/THERAPY

- Keep manipulation of the eye to a minimum if ruptured globe is suspected.
- Try to prevent any activity that will cause an increase in intraocular pressure such as straining, coughing, or vomiting.

ED TREATMENT/PROCEDURES

- Prepare for definitive surgical management:
 - _ Emergent ophthalmologic consultation
 - _ Thorough physical exam to identify concurrent injuries
 - _ Preoperative labs and ECG as indicated
 - _ No food or drink (NPO)
- Minimize intraocular pressure to reduce further injury
 - _ Administer antiemetic for nausea/vomiting
 - _ Elevate the head of the bed
 - _ Protective eye shield (NO pressure on the globe itself)
- Update tetanus status.
- Administer prophylactic antibiotics IV:
 - _ Skin organisms (staph, strep) most common
 - _ Consider injury-specific contaminants in cases of animal bites, organic foreign body, etc.
 - _ Vancomycin + ceftazidime OR vancomycin + ciprofloxacin if allergic to penicillin
- Succinylcholine is relatively contraindicated:
 - _ However, with a defasciculating dose of a nondepolarizing agent and sufficient anesthesia, it may be used.

Pediatric Considerations

- Consider nonaccidental trauma
- Because of risk of extrusion of intraocular contents, straining/crying should be avoided. Try to keep them happy!

MEDICATION

- Ceftazidime: 1–2 g (peds: 30–50 mg/kg) IV q8h
- Ciprofloxacin: 400 mg (peds: 10 mg/kg) IV q12h
- Clindamycin: 450 mg (peds: 8–12 mg/kg) IV q8h
- Ondansetron (Zofran): 4 mg IV
- Prochlorperazine (Compazine): 5–10 mg IV/IM
- Tobramycin: 2 mg/kg (peds: 2 mg/kg) IV q8h
- Vancomycin: 15 mg/kg IV q8–12h (peds: 10 mg/kg IV q6h)



FOLLOW-UP

DISPOSITION

Admission Criteria

- All patients with globe rupture/penetrating eye injuries
- Early enucleation for severe injury

Discharge Criteria

Globe penetration excluded

Issues for Referral

- Emergent ophthalmologic consultation in the ED may be needed to definitively rule out globe rupture owing to difficulty with exam and the desire to minimize manipulation of the eye.
- Speed is of the essence since the risk of infection increases with prolonged time to operative repair.
- If appropriate, patient should be counseled on use of protective eyewear to prevent recurrence.

FOLLOW-UP RECOMMENDATIONS

Postoperative ophthalmology follow-up

PEARLS AND PITFALLS

- Do not manipulate the eye if you suspect or confirm a ruptured globe:
 - Place eye shield over affected eye.
- Administer antiemetic for patients with nausea and vomiting to prevent elevation of intraocular pressure and extrusion of globe contents.
- Update tetanus
- Empiric antibiotics tailored to clinical scenario

ADDITIONAL READING

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- Skarbek-Borowska SE, Campbell KT. Globe rupture and nonaccidental trauma: Two case reports. *Pediatr Emerg Care.* 2011;27(6):544–546.



See Also (Topic, Algorithm, Electronic Media Element)

- Blowout Fracture
- Corneal Abrasion
- Corneal Foreign Body
- Hyphema
- Retinal Detachment
- Visual Loss



CODES

ICD9

- 871.0 Ocular laceration without prolapse of intraocular tissue
- 871.2 Rupture of eye with partial loss of intraocular tissue

ICD10

- S05.20XA Oclr lac/rupt w prolaps/loss of intraoc tiss, unsp eye, init
- S05.30XA Oclr lac w/o prolaps/loss of intraoc tissue, unsp eye, init

GLOMERULONEPHRITIS

Melissa H. White • Carolyn Maher Overman

BASICS

DESCRIPTION

- Syndrome characterized by:
 - Hematuria
 - Proteinuria
 - Red blood cell casts
 - Hypertension
 - Renal insufficiency
- Common pathway of multiple diseases resulting in intraglomerular inflammation and cellular proliferation
- Contributing factors:
 - Genetics
 - Infectious
 - Rheumatologic
 - Leading to antibody deposition:
 - Antibody attaches to glomerular antigen (native or implanted).
 - Circulating antigen–antibody complex deposited
 - Causing an influx and activation of inflammatory mediators:
 - Leukocytes, complement, cytokines
 - Cell-mediated immune mechanisms
- Results in glomerular dysfunction
- Persistent inflammation that can lead to scarring and permanent damage.

ETIOLOGY

- Postinfectious:
 - Poststreptococcal glomerulonephritis (PSGN):
 - Occurs 7–21 days after *Streptococcal pharyngitis* or skin infection
 - Highest prevalence in ages 2–14 and the elderly
 - Male predominance
 - Ranges from asymptomatic hematuria to oliguric renal failure
 - Can follow other bacterial, fungal, viral, or parasitic infections
- IgA nephropathy (Berger disease):
 - Most common in men in the 3rd and 4th decades of life
 - Possibly related to increased production of IgA after infection usually a URI
 - Henoch–Schönlein purpura (HSP) has IgA nephropathy but affects a younger age and has systemic symptoms
- Rapidly progressive glomerulonephritis (RPGN):

- Can destroy renal function in days
- Crescentic deposits in glomeruli destroy function.
- Pauci-immune (small vessel vasculitides):
 - Often antineutrophil cytoplasmic antibody (ANCA)-positive
 - Can involve other areas (i.e., lungs, skin)
 - Wegener granulomatosis
 - Microscopic polyangiitis
 - Churg–Strauss syndrome
- Immune complex deposits:
 - Postinfectious
 - Endocarditis associated
 - Systemic disease (i.e., systemic lupus erythematosus [SLE], HSP)
- Anti-glomerular basement membrane (GBM) deposits:
 - Older patients of age > 60
 - Goodpasture disease with pulmonary involvement
- Membranoproliferative glomerulonephritis (MPGN):
 - Complement deposits in basement membrane
 - Hepatitis C
 - Non-Hodgkin lymphoma
 - Occult infection



DIAGNOSIS

SIGNS AND SYMPTOMS

- Cardinal signs:
 - Hematuria
 - Proteinuria
- Edema:
 - Owing to renal salt and water retention
 - Periorbital
 - Ascites
 - Pleural effusion
- HTN
- Oliguria
- Azotemia
- CHF
- Renal failure
- Nonspecific manifestations:
 - Fatigue
 - Weight loss
 - Abdominal pain
 - Nausea/vomiting

- Autoimmune disorders:
 - Arthralgias
 - Arthritis
 - Rash
 - Fever
- Goodpasture syndrome:
 - Hemoptysis
- Wegner granulomatosis:
 - Purulent rhinorrhea/sinus pain
 - Arthritis/arthralgias
 - Hemoptysis
- HSP:
 - Abdominal pain
 - Purpura
 - Arthritis

Geriatric Considerations

- Pauci-immune RPGN is common in this population (hematuria, proteinuria, and elevated CR).
- Urgent diagnosis and biopsy are indicated as this may progress to ESRD.
- Consult nephrology to discuss steroids, cyclophosphamide, and plasma exchange.

ESSENTIAL WORKUP

Urinalysis for:

- Hematuria, proteinuria, and RBC casts

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Electrolytes, BUN, Creatinine, GFR:
 - Renal function
 - Hyperkalemia
- Albumin, total protein:
 - Varying degrees of hypoalbuminemia depending on clinical process
- CBC:
 - Anemia secondary to chronic renal disease, neoplasm, Goodpasture, and Wegener
 - With or without elevated WBC in infections
- PT, PTT:
 - Coagulation factors consumed in certain types of GN
- Labs to be considered for consultants:
 - Cultures—throat, skin, blood
 - 24-hr urine collection—protein, urine electrolytes

- Streptozyme or antistreptolysin O titer
- Complement levels (C1, C3, C4, CH₅₀)—reduced in PSGN, MPGN, SLE
- ANA, rheumatoid factor—connective tissue diseases
- ESR and CRP inflammatory markers
- Anti-GBM—Goodpasture
- c-ANCA—Wegener; p-ANCA-pauci-immune
- Anti-DNA antibodies (SLE)
- Hepatitis B and C serologies
- HIV

Imaging

- Renal ultrasound (if GFR is decreased):
 - Kidney size predictor of potential reversibility of disease, alternative diagnosis (i.e., neoplasm, stone)
- Chest radiograph (CXR): Heart size, pulmonary edema, or hemorrhage

Diagnostic Procedures/Surgery

- Renal biopsy: Discern primary glomerulopathies vs. other causes
- Cystoscopy: If concern for bladder neoplasm

DIFFERENTIAL DIAGNOSIS

- Hematologic:
 - Sickle cell disease
 - Coagulopathy
- Renal:
 - Infectious
 - Malformation
 - Neoplasm
 - Ischemic
 - Trauma
 - Vasculitis
- Postrenal:
 - Mechanical (i.e., stones, reflux, obstruction, catheterization)
 - Inflammatory (i.e., cystitis, prostatitis, epididymitis, endometriosis, periurethritis)
 - Neoplasm
- Factitious:
 - Food
 - Drugs
 - Pigmenturia (i.e., myoglobin, porphyria, hemoglobin)
 - Vaginal bleeding



TREATMENT

PRE HOSPITAL

- Supportive
- ABCs and fluid restriction in stable patients with significant edema.

INITIAL STABILIZATION/THERAPY

ABCs: Airway, breathing, circulation

ED TREATMENT/PROCEDURES

- Treatment mainly supportive care:
 - _ BP control: < 125/75 mm Hg
 - Loop diuretics
 - ACE inhibitor for maintenance
 - Treat hypertensive emergencies
 - _ Dialysis:
 - Fluid overload
 - Hyperkalemia
 - Uremia
- PSGN:
 - _ Supportive care
 - _ Usually resolves spontaneously
 - _ No benefit to antibiotics
- IgA nephropathy:
 - _ Supportive care
 - _ Immunosuppressives if inflammation on biopsy
 - _ Variable course, most recover but may relapse
- RPGN:
 - _ Can irreversibly destroy renal function in days
 - _ Emergently consult nephrologist to discuss starting potentially toxic therapies.
 - _ Immunosuppressives and high-dose steroids:
 - Methylprednisolone and prednisone
 - Cyclophosphamide
 - Rituximab
 - Plasmapheresis for anti-GBM antibody
- MPGN:
 - _ Treat underlying disease if known.
 - _ Emergently consult nephrologist to discuss starting potentially toxic therapies.
 - _ May include plasma exchange, cyclophosphamide, and/or steroids

MEDICATION

- Benazepril: 5–40 mg PO daily (or any other ACE inhibitor)
- Cyclophosphamide: Dose in conjunction with nephrology
- Diazoxide: 1–3 mg/kg IV, max. 150 mg, repeat q15min
- Furosemide: 20–80 mg IV; max. 2 mg/kg/d
- Methylprednisolone: 30 mg/kg IV on alternative days for 3 doses, followed by oral prednisone (dose in conjunction with nephrology)
- Nitroprusside: 0.3–10 µg/kg/min IV
- Prednisone: 0.5–2 mg/kg/d



FOLLOW-UP

DISPOSITION

Admission Criteria

- Unstable vital signs
- Oliguria, anuria
- Uremia
- Acute renal failure
- Electrolyte abnormality
- Hypertensive emergency
- CHF
- Infectious cause of GN

Discharge Criteria

Healthy patients with no comorbid illness who present with mild hematuria and proteinuria with:

- Stable vital signs
- No signs of infection
- Otherwise normal lab work
- Close follow-up recommended

FOLLOW-UP RECOMMENDATIONS

All patients with glomerulonephritis should follow-up with nephrology

PEARLS AND PITFALLS

- Discussion with nephrology if management with immunosuppressives is sought.
- The finding of proteinuria or hematuria should always prompt follow-up to ensure that the patient is not progressing to GN.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Nephritic Syndrome
- Nephrotic Syndrome
- Renal Failure



CODES

ICD9

- 580.0 Acute glomerulonephritis with lesion of proliferative glomerulonephritis
- 583.4 Nephritis and nephropathy, not specified as acute or chronic, with lesion of rapidly progressive glomerulonephritis
- 583.9 Nephritis and nephropathy, not specified as acute or chronic, with unspecified pathological lesion in kidney

ICD10

- N00.9 Acute nephritic syndrome with unsp morphologic changes
- N01.9 Rapidly progr nephritic syndrome w unsp morphologic changes
- N05.9 Unsp nephritic syndrome with unspecified morphologic changes

GONOCOCCAL DISEASE

Sunil D. Shroff



BASICS

DESCRIPTION

- 2nd most frequently reported STD in US:
 - _ Estimated 820,000 new cases per year
 - _ <50% reported
 - _ Highest rates in 15–24-yr-old males and females, African Americans
 - _ Increasing incidence in men who have sex with men (MSM):
 - Higher in HIV-positive individuals
 - _ Humans only known host
- Concurrent infection with *Chlamydia trachomatis* is common
- Affects the urethra, rectum, cervical canal, pharynx, upper female genital tract, and conjunctiva
- Urethritis most common presentation in men
- Often asymptomatic in women

ETIOLOGY

Neisseria gonorrhoeae:

- Gram-negative aerobic diplococci



DIAGNOSIS

SIGNS AND SYMPTOMS

- Cervicitis:
 - _ Defined as:
 - Mucopurulent endocervical discharge; OR
 - Easily induced endocervical bleeding
 - _ Most common site of infection
 - _ Up to 80% asymptomatic
 - _ Most symptoms nonspecific:
 - Vaginal discharge
 - Menorrhagia
 - Pelvic pain
 - Dyspareunia
 - Frequency and dysuria
- Pelvic inflammatory disease (PID):
 - _ Up to 20% of untreated cases
 - _ Lower abdominal pain—most common presenting symptom

- Other common signs and symptoms:
 - Dyspareunia, abnormal bleeding, abnormal cervical or vaginal discharge
- Symptoms often occur at onset of menses.
- Fever (50%)
- 2/3 have mild, vague symptoms; may go unrecognized
- Fitz-Hugh–Curtis syndrome: (perihepatitis):
 - 10% occurrence rate
 - Right upper quadrant pain/tenderness
- Bartholin abscess
- Urethritis:
 - Incubation period 2–5 days
 - Symptoms:
 - Penile discharge
 - Dysuria
- Prostatitis—can occur in untreated urethritis
- Epididymitis:
 - Acute, unilateral testicular pain and swelling
- Proctitis:
 - Often asymptomatic
 - Only site of infection in 40% of MSM
 - Rectal infection occurs in 35–50% of women with endocervical infection
 - 3-fold increase in HIV infection risk
 - Symptoms:
 - Perianal pruritus, mucopurulent discharge, mild rectal bleeding, severe rectal pain, tenesmus, and constipation
- Pharyngitis:
 - Sore throat, exudative tonsillitis
- Disseminated gonococcal infections (DGI):
 - Gonococcal bacteremia
 - Arthritis: Dermatitis syndrome:
 - 0.5–3% of untreated mucosal infections
 - Triad of tenosynovitis, dermatitis, and polyarthralgia
 - Fever, chills, malaise
 - Dermatitis:
 - Tender necrotic pustules on an erythematous base, few lesions, begin distally
 - Acute monoarticular or oligoarticular arthritis:
 - Knee most common
 - Warm, erythematous joint with effusion and pain with range of motion
 - Female > male, 3:1:
 - Risk factors: Recent menstruation or recent pregnancy

- Rare manifestations:
 - Hepatitis
 - Myocarditis
 - Endocarditis
 - Meningitis

Physical-Exam

- Cervicitis:
 - Cervical edema, congestion, friability
- PID:
 - Uterine tenderness, adnexal or cervical motion tenderness
- Urethritis:
 - Yellow-white thick discharge, urethral meatal erythema

ESSENTIAL WORKUP

- Clinical diagnosis in male gonorrhea:
 - Gram stain 95% sensitive
- Cervical culture in female gonorrhea
- Also test for chlamydia and syphilis

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Cultures (gold standard):
 - Thayer-Martin medium
 - Mainstay for blood and synovial fluid
- Gram stain:
 - Intracellular gram-negative diplococci:
 - Approaches 100% sensitive in symptomatic men
- Nucleic acid amplification tests (NAATs):
 - DNA or RNA sequences using polymerase chain reaction (PCR)
 - Many also test for chlamydia
 - Useful in urethral, cervical, and urine specimens
- Pharyngeal/rectal cultures for local symptoms in high-risk individuals
- DGI:
 - Synovial fluid analysis:
 - Neutrophilic leukocytosis, $> 50,000$ cells/mm³
 - Positive cultures when $> 80,000$ cells/mm³
 - 2 or more sets of blood cultures
 - Synovial, skin, urethral or cervical, and rectal cultures:
 - Thayer-Martin media
- PID/lower abdominal pain in female:

- CBC
- Urinalysis
- Pregnancy test
- Consider pelvic ultrasound for tubo-ovarian abscess
- Rapid plasma reagin (RPR): For associated syphilis

DIFFERENTIAL DIAGNOSIS

- Urethritis:
 - Chlamydia
 - Trichomonas
 - UTI
 - Syphilis
- DGI:
 - Bacterial arthritis:
 - Meningococcus (rash)
 - Hepatitis B
 - Connective tissue disease:
 - Reiter syndrome
 - Rheumatoid arthritis
 - Psoriatic arthritis
 - Acute rheumatic fever:
 - Poststreptococcal arthritis
 - Infective endocarditis
 - Others:
 - HIV
 - Secondary syphilis
 - Viral infection
 - Lyme disease (rash)
 - Gout (arthritis)



TREATMENT

ED TREATMENT/PROCEDURES

- Hydration (0.9% NS) for nausea/vomiting
- Treat sexual partner. For expedited partner therapy jurisdiction – www.cdc.gov/std/ept
- Patient with gonorrhea should often be presumptively treated for chlamydial infection.
- Cervical, urethral, and anorectal infection:
 - Ceftriaxone: 250 mg IM once OR
 - Also treat for chlamydia:
 - Azithromycin: 1 g PO once OR

- Doxycycline: 100 mg PO BID for 7 days
- PID:
 - Outpatient:
 - Ceftriaxone: 250 mg IM once or cefoxitin 2 g IM and probenecid 1 g PO once or another 3rd-generation cephalosporin (ceftizoxime or cefotaxime) + doxycycline 100 mg BID for 14 days with or without metronidazole 500 mg PO BID for 14 days
 - Inpatient:
 - Cefoxitin 2 g IV q6h or cefotetan 2 g IV q12h + doxycycline 100 mg PO or IV q12h
 - Clindamycin 900 mg IV q8h + gentamicin loading dose (2 mg/kg) followed by (1.5 mg/kg) q8h or 3–5 mg/kg q24h
- Pharyngitis:
 - Ceftriaxone 250 mg IM single dose + treatment for chlamydia
- Epididymitis:
 - Ceftriaxone 250 mg IM once + doxycycline 100 mg BID for 10 days
- Treat sexual partner
- DGI:
 - Ceftriaxone: 1 g IV/IM daily (recommended)
 - Cefotaxime: 1 g IV q8h OR
 - Ceftizoxime: 1 g IV q8h OR
 - 24–48 hr after improvement, additional 7 days with:
 - Cefixime: 400 mg PO BID OR
 - Cefpodoxime: 400 mg PO BID
 - Neonates, incl. gonococcal scalp abscess
 - Ceftriaxone 25–50 mg/kg/d IV/IM for 7 days OR
 - If hyperbilirubinemia-Cefotaxime 25 mg/kg IV/IM q12h for 7 days
- Conjunctivitis:
 - Adults:
 - Ceftriaxone 1 g IM once
 - Ophthalmia neonatorum:
 - Ceftriaxone 25–50 mg/kg IM/IV once
 - Saline irrigation, hospitalize
- Meningitis/endocarditis:
 - Ceftriaxone 1–2 g IV q12h:
 - 10–14 days for meningitis
 - At least 4 wk for endocarditis
- Severe cephalosporin allergy:
 - Consult infectious disease
 - Cephalosporin use postdesensitization best alternative
 - Azithromycin 2 g PO for uncomplicated gonococcal infection:
 - Limit use to prevent resistance

Pediatric Considerations

- Gonococcal ophthalmia neonatorum:
 - Mother with genital tract infection
 - Bilateral conjunctivitis 2–5 days postpartum:
 - If untreated, leads to globe perforation

Pregnancy Considerations

- Gonorrhea: Ceftriaxone/spectinomycin
- Chlamydia: Erythromycin



FOLLOW-UP

DISPOSITION

Admission Criteria

PID—CDC recommendations

- Severely ill (e.g., nausea, vomiting, and high fever)
- Pregnant
- Does not respond to or cannot take oral medication
- Tubo-ovarian abscess
- Other emergency surgical condition possible (e.g., appendicitis).

Discharge Criteria

Uncomplicated genital, pharyngeal, or conjunctival infection

Issues for Referral

- Infertility
- Recurrent infection despite multiple therapy

PEARLS AND PITFALLS

- Epididymitis—rule out torsion
- DGI—strongly consider in young sexually active patient with acute nontraumatic oligoarthritis or tenosynovitis

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Chlamydia
- Urethritis



CODES

ICD9

- 098.0 Gonococcal infection (acute) of lower genitourinary tract
- 098.7 Gonococcal infection of anus and rectum
- 098.15 Gonococcal cervicitis (acute)

ICD10

- A54.00 Gonococcal infection of lower genitourinary tract, unsp
- A54.03 Gonococcal cervicitis, unspecified
- A54.6 Gonococcal infection of anus and rectum

GOUT/PSEUDOGOUT

Delaram Ghadishah



BASICS

DESCRIPTION

- Uric acid deposition into tissues, affecting mainly middle-aged men and postmenopausal women:
 - Most common crystalline diseases
 - 4 phases:
 - Asymptomatic hyperuricemia (serum urate >7 mg/dL)
 - Acute gout
 - Intercritical gout: Quiet intervening periods
 - Tophaceous gout (up to 45% of cases)
 - Risk factors:
 - Age >40
 - Male/female ratio 2:1–6:1 <65 yr old; 1:1 ≥ 65 yr old
 - Hypertension
 - Use of loop or thiazide diuretics
 - High intake of alcohol, meat, seafood, and fructose-sweetened beverages
 - Obesity
 - Urologic deposition of uric acid calculi may cause renal dysfunction.
 - Associated with avascular necrosis and deforming arthritis
 - Most frequent in previously damaged joints, tissues:
 - Synovium
 - Subchondral bone
 - Bursae (olecranon, infrapatellar, prepatellar)
 - Achilles tendon
 - Extensor surface of the forearms, toes, fingers, ear
 - Rarely CNS or cardiac (valves)
- Pseudogout: A disorder caused by calcium pyrophosphate crystal deposition:
 - Most common cause of acute monoarthritis >60 yr of age
 - Risk factors:
 - Hypercalcemia (e.g., hyperparathyroidism, familial)
 - Hemochromatosis; hemosiderosis
 - Hypothyroidism and hyperthyroidism
 - Hypophosphatemia, hypomagnesemia
 - Amyloidosis
 - Gout

ETIOLOGY

- Deposition of monosodium urate crystals in tissues from supersaturated extracellular fluid owing to:
 - Underexcretion (most commonly) or excessive production of uric acid
 - Any rapid change in uric acid levels
 - Initiation or cessation of diuretics
 - Alcohol, salicylates, niacin
 - Cyclosporine
 - Lead acetate poisoning
 - Uricosurics or allopurinol
- Pseudogout occurs secondary to excess synovial accumulation of calcium pyrophosphate crystals
- Precipitants for both gout and pseudogout include minor trauma and acute illnesses:
 - Surgery, ischemic heart disease

DIAGNOSIS

SIGNS AND SYMPTOMS

- Gout and pseudogout both present as acute monoarticular or polyarticular arthritis:
 - Increased warmth, erythema, and joint swelling are present.
 - Early attacks subside spontaneously within 3–21 days, even without treatment.
 - Later attacks may last longer, cluster, be more severe, and be polyarticular.
- Gout:
 - Symptoms present maximally within 12–24 hr.
 - Tophi and joint desquamation may be present.
 - Women predominantly present after menopause and have polyarticular predominance (up to 70%).
 - Less dramatic presentations in immunosuppressed and elderly
 - Most common: 1st metatarsophalangeal joint (75%) > ankle; tarsal area; knee > hand; wrist
- Pseudogout:
 - Typically involves larger joints than with gout
 - Most common: Knee > wrist > metacarpals; shoulder; elbow; ankle > hip; tarsal joints
 - Monoarticular (25%)
 - Asymptomatic (25%)
 - Pseudo-osteoarthritis (45%): Progressive degeneration, often symmetric
 - Pseudorheumatoid arthritis (in elderly)
- Polyarticular variant with fever and confusion

ESSENTIAL WORKUP

- Arthrocentesis and aspiration of tophi:
 - Examine aspirant for crystals, Gram stain, cultures, leukocyte count, and differential
 - Fluid is typically thick pasty white:
 - Gout: 20,000–100,000 WBC/mm³; poor string and mucin clot; no bacteria
 - Pseudogout: Up to 50,000 WBC/mm³; no bacteria
- Microscopic exam of crystals under polarized light:
 - Gout:
 - Needle shaped
 - Strong birefringence
 - Negative elongation
 - Pseudogout:
 - Rhomboid
 - Weak birefringence
 - Positive elongation

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC often shows leukocytosis.
- Chemistry panel to assess for renal impairment
- Magnesium and calcium, thyroid-stimulating hormone (TSH), and serum iron
- Uric acid level has limited value.
- If infectious arthritis is suspected:
 - Blood and urine cultures
 - Urethral, cervical, rectal, or pharyngeal gonococcal cultures

Imaging

- Plain radiographs to assess the presence of:
 - Effusion
 - Joint space narrowing
 - Baseline status of joint
 - Contiguous osteomyelitis
 - Fractures or foreign body
- Acute gout: Soft tissue swelling; normal mineralization; joint space preservation
- Chronic gout: Calcified tophi; asymmetric bony erosions; overhanging edges; bony shaft tapering
- Pseudogout: Chondrocalcinosis; subchondral sclerosis or cysts (wrist); radiopaque calcification of cartilage, tendons, and ligaments; radiopaque osteophytes
- Dual energy CT to assess for kidney stones or soft tissue urate crystals

Diagnostic Procedures/Surgery

- Arthrocentesis
- Aspiration of tophi

DIFFERENTIAL DIAGNOSIS

- Infectious arthritis
- Trauma
- Osteoarthritis
- Reactive arthritis
- Miscellaneous crystalline arthritis
- Aseptic necrosis
- Rheumatoid arthritis
- Systemic lupus erythematosus
- Sickle cell
- Osteomyelitis
- Psoriatic arthritis



TREATMENT

INITIAL STABILIZATION/THERAPY

- Relieve pain.
- Rule out infectious cause.

ED TREATMENT/PROCEDURES

- NSAIDs are 1st-line treatment.
- If NSAIDS ineffective or contraindicated:
 - Steroids (oral, intravascular, IM, intra-articular)
 - Colchicine (limited by toxicity)
- Joint aspiration
- Avoid aspirin
- Reduction of hyperuricemia and long-term management of gout and pseudogout are not within the usual scope of ED care:
 - Careful withdrawal of gout-producing agent
 - Uricosurics (e.g., probenecid, sulfinpyrazone)
 - Allopurinol to reduce uric acid synthesis
 - Increased fluid intake and urine alkalization to prevent renal stones
 - Long-term colchicine or NSAIDs prophylactically

MEDICATION

- Anakinra: 100 mg SQ QD:
 - Off label use for chronic, treatment refractory gout or pseudogout and with renal failure
- Allopurinol: 100 mg PO QD, increased weekly to max. 800 mg QD:

- Start 1–2 wk after attack has resolved
- Adjust for kidney disease
- Discontinue with rash or fever
- Treatment of choice with uric acid kidney stones
- Doses >400 mg should be taken in divided doses
- Colchicine: 1.2 mg PO upon gout flare followed by 0.6 mg 1 hr later:
 - Can cause bone marrow suppression at high doses
 - Not dialyzable
 - Long-term use may cause myopathy.
 - Adjust dose for liver or kidney disease.
 - Does not prevent monosodium urate deposition or joint damage of chronic gout
- Corticosteroids:
 - Corticotropin: 40 units IM, q8h, up to 2 doses
 - Methylprednisolone: 40 mg (peds: 1–2 mg/kg) IM or IV QD for 3–4 days
 - Prednisone: 40 mg (peds: 1–2 mg/kg) PO QD for 3–4 days; taper over 7–14 days
 - Triamcinolone: 10–40 mg + dexamethasone 2–10 mg intra-articularly
- Febuxostat: 40–80 mg QD:
 - Give with NSAID or colchicine when 1st started
 - Inhibits urate production
 - Safe for mild or moderate kidney disease
- NSAIDs in maximal doses initially for 3 days, then taper over 4 days:
 - Ibuprofen: 800 mg (peds: 10 mg/kg) PO QID
 - Indomethacin: 25–50 mg PO TOD–QID (peds: 2 mg/kg/d TID–QID; *not* for children <14 yr old)
 - Ketorolac: 15–30 mg IM/IV in ED, may repeat for 1 dose (peds: 1 mg/kg to max. 30 mg IM or 0.5 mg/kg to max. 15 mg IV) IM:
 - Naproxen: 500 mg PO TID (peds: 5 mg/kg PO BID)
 - Sulindac: 200 mg PO TID
- Pegloticase: 8 mg IV over 2+ hr q2wk
 - For gout refractory to conventional treatment
 - Premedicate with antihistamine and steroids
 - Associated with anaphylaxis
 - Stop if uric acid increases to >6 mg/dL
 - Contraindicated with G6PD deficiency
- Probenecid: 250–500 mg PO q12h, max. 3 g QD:
 - Promotes excretion of uric acid
 - Not effective or less effective with renal disease or aspirin or diuretic use
 - Relatively contraindicated with presence of uric acid kidney stones
- Riloncept: 2 × 160 mg (2 × 2 mL) SC injected into 2 different sites on the same day, then 160 mg (1 × 2 mL) SC every week –Off label use for acute gout or prophylaxis

– Given during initiation of urate-lowering therapy

- Sulfipyrazone: 200–400 mg PO in divided doses BID with food, maintenance dose 400 mg in divided doses BID, max. 800 mg QD

Geriatric Considerations

NSAIDs may worsen renal function, fluid retention, gastropathy, hepatotoxicity, and cognitive function, particularly in the elderly.

Pediatric Considerations

Gout not usually seen in children, although possible during chemotherapy treatment for cancer.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Suspected infectious arthritis
- Acute renal failure
- Intractable pain

Discharge Criteria

- No evidence of infection
- Adequate pain relief

Issues for Referral

- Septic arthritis
- Renal failure

FOLLOW-UP RECOMMENDATIONS

- Rheumatology follow-up in severe or difficult to control cases
- Renal follow-up if renal insufficiency is present
- Urology follow-up if uric acid stones are present
- Orthopedic follow-up in cases of septic arthritis or significant joint damage
- Advise patient to follow a low-purine diet.

PEARLS AND PITFALLS

- Septic arthritis can occur simultaneously with an acute gout attack.
- NSAIDs are 1st-line treatment if tolerated.
- Attacks generally tend to be self-limited.
- Gout and pseudogout can lead to bony and cartilaginous damage.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

www.Epocrates.com



CODES

ICD9

- 274.00 Gouty arthropathy, unspecified
- 274.9 Gout, unspecified
- 712.30 Chondrocalcinosis, unspecified, site unspecified

ICD10

- M10.00 Idiopathic gout, unspecified site
- M10.9 Gout, unspecified
- M11.20 Other chondrocalcinosis, unspecified site

GRANULOCYTOPENIA

Richard E. Wolfe • William D. Timbers • Elicia Sinor Kennedy



BASICS

DESCRIPTION

- A significant decrease in the number of granulocytes in the peripheral blood.
- 3 classes of granulocytes:
 - Neutrophils or polymorphonuclear (PMN) cells and bands
 - Eosinophils
 - Basophils
- As PMN cells predominate, the term neutropenia is often used interchangeably with granulocytopenia, as almost all granulocytopenic patients are neutropenic.
- Granulocytes are a key component of the innate immune system.
- The clinical risks resulting from granulocytopenia are best defined by the level of the absolute neutrophil count (ANC):
 - $ANC = WBC \times \text{percentage (PMN + bands)}$
 - Modern automated instruments often calculate and report ANC.
- Neutropenia: $ANC < 1,500 \text{ cells/mm}^3$:
 - Mild: Between 1,000 and 1,500
 - Moderate: Between 500 and 1,000
 - Severe: < 500
 - Agranulocytosis: < 100
 - Patients with a count $< 1,000$ that has recently or rapidly fallen are at greater risk for infection than those with a count < 500 but rising.
 - Patients with myelodysplastic syndromes should be considered granulocytopenic with higher counts because of defective neutrophils.
- 4 basic mechanisms cause granulocytopenia:
 - Decreased production
 - Ineffective granulopoiesis
 - Shift of circulating PMN cells to vascular endothelium
 - Enhanced peripheral destruction.
- Mortality of fever and neutropenia is as high as 50% if untreated:
 - Mortality correlates with the duration and severity of the neutropenia and the time elapsed until the 1st dose of antibiotics.
- 21% of patients with cancer and neutropenic fever develop serious complications.

Pediatric Considerations

- Newborn infants have a physiologically elevated ANC in the 1st few days of life and may be granulocytopenic with levels $> 1,500/\mu\text{L}$.
- Children $> 3 \text{ mo}$ without underlying immunodeficiency or a central venous

catheter unexpectedly found to have isolated moderate neutropenia are not at high risk of serious bacterial infection.

ETIOLOGY

- Most common in patients undergoing myelosuppressive drug therapy or radiation treatment for neoplasms. Most common 5–10 days after chemo.
- Adverse reaction to drugs is the 2nd most common cause:
 - Excludes cytotoxic drugs and requires at least 4 wk of administration prior to the onset of granulocytopenia
 - Discontinuation usually results with correction within 30 days.
 - Drugs with the highest risk:
 - Antipsychotic: Clozapine
 - Antibiotic: Sulfasalazine
 - Antithyroid: Thioamides
 - Antiplatelet agents
 - Antiepileptic drugs
 - NSAIDs
- Drugs that suppress the bone marrow:
 - Methotrexate
 - Cyclophosphamide
 - Colchicine
 - Azathioprine
 - Ganciclovir
- Chemicals
- Bacterial infections:
 - Typhoid
 - Shigella enteritis
 - Brucellosis
 - Tularemia
 - Tuberculosis
- Parasitic infections:
 - Kala azar
 - Malaria
- Rickettsial infections:
 - Rickettsialpox
 - Ehrlichiosis
 - Rocky Mountain spotted fever
- Viral infections
- Postinfectious neutropenia:
 - Most severe and protracted following HIV, hepatitis B, and Epstein–Barr viral infections
- Immune-related:
 - Primary immune neutropenia:

- Due to antineutrophil antibodies
 - Crohn's disease
 - Systemic lupus erythematosus
 - Rheumatoid arthritis
 - Goodpasture disease
 - Wegener granulomatosis
 - Thymoma
 - Complement activation
- Bone marrow infiltration
- Transfusion reaction
- Alcoholism
- Vitamin deficiency (B₁₂/folate/copper)
- Chronic idiopathic neutropenia
- Pure white cell aplasia

Pediatric Considerations

- Congenital neutropenia:
 - Neutropenia with abnormal immunoglobulins
 - Reticular dysgenesis
 - Severe congenital neutropenia or Kostmann syndrome
 - Cyclic neutropenia
- Chronic benign neutropenia
- Neonatal isoimmune neutropenia
- Shwachman–Diamond syndrome
- Cartilage–hair hypoplasia
- Dyskeratosis congenita
- Barth syndrome
- Chédiak–Higashi syndrome
- Myelokathexis
- Lazy leukocyte syndrome
- Cohen syndrome
- Hermansky–Pudlak syndrome type 2



DIAGNOSIS

SIGNS AND SYMPTOMS

- Signs of bacterial or fungal infection:
 - Fever
 - Localized erythema or fluctuance
- Signs of pancytopenia:
 - Fatigue
 - Pallor

- Petechiae
- Epistaxis and other spontaneous bleeding

History

- Medical list should be reviewed for causative drugs.
- Family history of granulocytopenia in neonates and children
- Records of past ANC levels to assess for chronicity
- Question the patient carefully about fever, chills, dizziness, and vomiting as indicators of an underlying serious infection.
- Ask about localizing signs of infection such as cough; shortness of breath; chest pain; dysuria; urinary retention, urgency, or frequency; abdominal pain; and rectal pain.

Physical-Exam

Focus on finding signs of infection:

- Oral exam: Thrush, ulcers, periodontal disease, mucositis
- Lungs: Rales, rhonchi
- Abdominal: Splenomegaly
- Skin: Rashes, ulcers, abscesses
- Perirectal: Although the rectal exam is relatively contraindicated until antibiotics are started, check for abscesses and mucosal lesions.
- Evaluate indwelling catheter sites

ESSENTIAL WORKUP

Complete physical exam:

- Detailed exam of oral mucosa and perianal area
- Palpation of skin
- Location of fluctuance or tenderness
- Careful lung exam and abdominal
- Rectal exam after antibiotics if symptoms suggest perirectal abscess

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC with differential:
 - ANC
- LFTs
- Blood culture before antibiotics from 2 different sites, with 1 from IV catheter site if present
- Urinalysis and urine culture:
 - May not show WBCs or leukocytes esterase OR may be normal
- Sputum Cx if applicable
- Stool Cx if applicable

- Culture indwelling catheters
- Cerebrospinal fluid analysis for altered mental status/signs of meningitis

Imaging

CXR even in absence of lung findings

DIFFERENTIAL DIAGNOSIS

- Lab error
- Neoplasm and chemotherapy
- Medication reaction
- Chemical exposure
- Infections (viral/bacterial/rickettsial)
- Autoimmune syndrome
- Genetic etiology
- Transfusion reaction
- Nutritional deficiency
- Tumor lysis syndrome
- Hypersplenism
- African Americans may have a lower but normal ANC value of 1,000 cells/mm³



TREATMENT

INITIAL STABILIZATION/THERAPY

For patients presenting in shock:

- Administer 1 L 0.9% NS IV fluid bolus (peds: 20 cc/kg).
- Initiate pressors as needed to stabilize BP if no response to IV fluids.
- Consider starting goal-directed therapy.

ED TREATMENT/PROCEDURES

- Strict isolation in a negative airflow room if possible
- Administer broad-spectrum combination antibiotics after cultures for suspected or documented infection:
 - Imipenem/cilastatin or fluoroquinolone
 - Ceftazidime alone or with aminoglycoside (amikacin, tobramycin, gentamicin)
- Cefepime alone
- Aminoglycoside + antipseudomonal β -lactam (mezlocillin, piperacillin, or ticarcillin)
- Vancomycin if patient is at risk to be carrier of *MRSA*

MEDICATION

- Amikacin: 15 mg/kg/24 h (peds: 15–30 mg/kg/24 h) div. q8–12h IV

- Cefepime: 0.5–2 g q12h
- Ceftazidime: 1–2 g (peds: 30–50 mg/kg q8h) q8–12h IV
- Gentamicin: 1 mg/kg (peds: 2–2.5 mg/kg) q8h or 5 mg/kg q24h
- Imipenem/cilastatin: 250–1,000 mg q6–8h
- Levofloxacin: 500 mg IV QID
- Mezlocillin: 3 g q4h over 30 min
- Tobramycin: 1 mg/kg q8h IV (peds: 2–2.5 mg/kg q8h IV)
- Vancomycin: 15 mg/kg q8–12h IV



FOLLOW-UP

DISPOSITION

Risk Stratification

MASCC Score: Identifies febrile neutropenic patients who are at a lower risk of complications.

Admission Criteria

- Signs of infection
- Unreliable patient
- Close follow-up unavailable

Discharge Criteria

- Previously diagnosed granulocytopenia
- Completely asymptomatic
- Close follow-up ensured
- Reliable patient

Issues for Referral

All patients with granulocytopenia should be referred to their physician or a hematologist.

FOLLOW-UP RECOMMENDATIONS

- Patient should return immediately to the ED with fever.
- Follow-up within 48 hr with the patient's physician

PEARLS AND PITFALLS

- Usual signs of infection may be masked because of the impaired immune response in patients with granulocytopenia.
- Rectal exams and rectal temperatures are relatively contraindicated in neutropenic patients but should be performed once antibiotics are started to avoid missing a

perirectal abscess.

- Patients with fever and an ANC < 500 requires immediate and aggressive therapy with broad-spectrum antibiotics and IV fluids.
- Hepatosplenic candidiasis: Complication of resolving neutropenia. Abscess formation as ANC rises. Treat with amphotericin B.

ADDITIONAL READING

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- Perrone J, Hollander JE, Datner EM. Emergency Department evaluation of patients with fever and chemotherapy-induced neutropenia. *J Emerg Med*. 2004;27(2):115–119.
- Segel GB, Halterman JS. Neutropenia in pediatric practice. *Pediatr Rev*. 2008;29:12–23.



CODES

ICD9

- 288.00 Neutropenia, unspecified
- 288.03 Drug induced neutropenia
- 288.09 Other neutropenia

ICD10

- D70.1 Agranulocytosis secondary to cancer chemotherapy
- D70.8 Other neutropenia
- D70.9 Neutropenia, unspecified

GUILLAIN–BARRÉ SYNDROME

Erin F. Drasler • Jeffrey Druck



BASICS

DESCRIPTION

- Group of peripheral nerve disorders characterized by autoimmune demyelination and axonal degeneration of peripheral nerves leading to acute ascending paralysis
- Humoral and cellular immune mediated
- Leading cause of acute flaccid paralysis worldwide (since polio vaccination)
- Triggered by antecedent bacterial/viral infection
- Increasing incidence with advancing age, male gender
 - Average 1.1 per 100,000 per yr
- Weakness reaches nadir at 2–4 wk
- Spontaneous resolution occurs over weeks to months
 - 80% full recovery at 1 yr
 - 20% unable to walk at 6 mo
 - 5% die of complications (PE, infection, cardiac)
- Acute inflammatory demyelinating polyradiculoneuropathy (AIDP):
 - Most common form of Guillain–Barré syndrome (90% of all GBS cases)
 - Demyelination sometimes accompanied by axonal loss
- Other forms of GBS:
 - Acute motor axonal neuropathy (AMAN):
 - Pure motor axonal involvement
 - 67% seropositive for *Campylobacter jejuni*
 - Recovery often rapid
 - Often pediatric patients
 - Acute motor sensory axonal neuropathy (AMSAN):
 - Degeneration of myelinated motor and sensory nerves without significant inflammation or demyelination
 - Similar to AMAN, but also involves sensory nerves
 - Acute panautonomic neuropathy:
 - Very rare
 - Involves sympathetic and parasympathetic nerves
 - Postural hypotension, dysrhythmias, tachycardia, hypertension
 - Blurry vision, dry eyes, anhidrosis
 - Recovery gradual, often incomplete
 - Miller Fisher syndrome:
 - Rare
 - Rapidly evolving ataxia, areflexia, and ophthalmoplegia without weakness

- Demyelination and inflammation of cranial nerves II and VI, spinal ganglia, and peripheral nerves
- Resolves in 1–3 mo

ETIOLOGY

- Postinfectious:
 - 2/3 with antecedent illness, usually respiratory or GI
 - Different autoantibodies associated with different subtypes
 - 1–3 wk between prodromal illness and neurologic symptoms
 - *C. jejuni* most common antecedent bacterial infection
 - *Cytomegalovirus* most common antecedent viral infection
 - Epstein–Barr virus, VZV, HIV, mycoplasma also associated
- Relationship to vaccines is questionable
 - Slight increased risk ascribed to 1976 swine flu vaccine, no other vaccines have same risk

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Rapidly evolving, symmetric, ascending paralysis
- Absent or mild sensory symptoms (e.g., paresthesias of fingertips or toes)
- Pain, commonly of pelvis, shoulder girdles, posterior thighs
- Cranial nerve involvement may affect swallowing, facial muscles, eye movements
- Preceding bacterial or viral infection
- Progression of symptoms over 8 wk not consistent with GBS, but chronic inflammatory demyelinating polyradiculoneuropathy (CIDP)

Physical-Exam

- Ascending symmetric weakness, legs more affected than arms
- Loss of deep tendon reflexes
- Look for cranial nerve involvement
- Respiratory insufficiency (25% patients)
- Normal sensory exam
- Other subtypes:
 - Autonomic dysfunction:
 - Hypertension
 - Orthostatic hypotension
 - Ileus
 - Dysrhythmias
 - Urinary retention

- Miller Fisher variant:
 - Ataxia
 - Areflexia
 - Ophthalmoplegia
 - Mild limb weakness
- Features that suggest alternative diagnosis:
 - Fever
 - Normal reflexes
 - Upper motor neuron signs
 - Asymmetric neurologic deficits
 - Sharply demarcated sensory level

DIAGNOSIS TESTS & INTERPRETATION

- Diagnosis is generally made on clinical grounds
- Lab and imaging tests can assist with diagnosis and rule out other causes of symptoms

Lab

- Electrolytes (some patients have SIADH)
- Lumbar puncture:
 - Albuminocytologic dissociation-increased protein with few or no WBCs
 - Protein typically 55–350, may be present only after 7–10 days as disease and blood–brain barrier dysfunction progress
 - WBCs > 10–50 suggests other etiology
 - Normal opening pressure

Imaging

CT or MRI to rule out cord compression

Diagnostic Procedures/Surgery

Electrophysiologic studies will be abnormal (nerve conduction confirmatory)

DIFFERENTIAL DIAGNOSIS

Polyneuropathies:

- Acute intermittent porphyria
- Chronic heavy metal poisoning
- Diphtheria
- Lyme disease
- Paraneoplastic disease
- Poliomyelitis
- Sarcoidosis
- Tick paralysis
- Vasculitis

Spinal cord disorders:

- Cord compression
- Transverse myelitis

Neuromuscular junction disorders:

- Botulism
- Eaton–Lambert syndrome
- Myasthenia gravis

Muscle disorders:

- Acute polymyositis
- Critical illness myopathy

Other:

- Hypokalemia
- Psychogenic, malingering



TREATMENT

PRE HOSPITAL

Attention to airway management

INITIAL STABILIZATION/THERAPY

Airway assessment and management:

- Progression to respiratory failure can be rapid

ED TREATMENT/PROCEDURES

- Airway management:
 - _ ~ 30% will need ventilatory support.
 - _ May need intubation within 24–28 hr of onset
 - _ Frequent monitoring of respiratory parameters:
 - Forced vital capacity (FVC) or negative inspiratory flow (NIF) helpful
 - ICU admission if FVC < 20 mL/kg or NIF < 30 cm H₂O
 - Intubation recommended if FVC < 15 mL/kg
- Watch for autonomic dysfunction
- Supportive therapy
- Early neurology consult

MEDICATION

- Plasmapheresis or IV immunoglobulin (IVIG), in conjunction with neurologic consultation:
 - _ Unclear benefit for Miller Fisher or mild GBS
- IVIG: 400 mg/kg/d for 5 days
- Pain control:
 - _ Acetaminophen: 500 mg PO q6h; not to exceed 4 g/24h

- Ibuprofen: 400–800 mg PO q8h
- Gabapentin: Start 300 mg PO per day
- Steroids not beneficial for pain or deficits:
 - Oral steroids delay recovery
 - IV steroids with no benefit



FOLLOW-UP

DISPOSITION

Admission Criteria

- All patients with GBS or suspected GBS warrant admission for close observation
- ICU admission for those with signs of respiratory compromise, autonomic dysfunction or need for frequent monitoring for progression of illness

Discharge Criteria

Patients should be considered for discharge only after consultation with neurologist

FOLLOW-UP RECOMMENDATIONS

- Follow-up determined by neurologist
- Poor outcome associated with:
 - Older age
 - Longer time to nadir
 - Necessity for ventilator support
 - Preceding diarrheal illness, *C. jejuni*

PEARLS AND PITFALLS

- Pearls:
 - Check FVC and/or NIF frequently to anticipate airway compromise
 - Consider other etiologies if CSF WBC count > 10–50
- Pitfalls:
 - Failure to obtain appropriate imaging of the brain and spinal cord to rule out other potential causes
 - Failure to consult neurology and admit or observe all patients with suspected GBS

ADDITIONAL READING

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- Sejvar JJ, Baughman AL, Wise M, et al. Population incidence of Guillain-Barré syndrome: A systematic review and meta-analysis. *Neuroepidemiology*. 2011;36:123–133.
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See Also (Topic, Algorithm, Electronic Media Element)

- Myasthenia Gravis
- Spinal Cord Syndromes
- Spine Injury
- Tick Bites



CODES

ICD9

357.0 Acute infective polyneuritis

ICD10

G61.0 Guillain-Barre syndrome

HALLUCINATIONS

Michael Ganetsky

BASICS

DESCRIPTION

Hallucinations are a symptom or feature and not a diagnosis. They may be auditory, visual, tactile, gustatory, or olfactory. Hallucinations and similar phenomena are often defined as follows:

- **Hallucination:**
 - Sensory perception that has the compelling sense of reality of a true perception without external stimulation of the relevant sensory organ and is experienced as a sensation through that organ
 - Patients may or may not have insight that they are having the hallucination
- **Illusion:**
 - Misperception or misinterpretation of a real external stimulus
- **Flashback:**
 - Recurrence of a memory, feeling, or perceptual experience from the past that may have the compelling sense of reality
- **Pseudohallucination:**
 - Hallucination that is not experienced by a sensory organ (i.e., voices inside head or “inner voice” as opposed to hearing voices)

EPIDEMIOLOGY

Incidence and Prevalence Estimates

- Lifetime incidence of auditory hallucinations is 4–8% in general population (although some estimates are higher due to vague definitions or inclusion of pseudohallucinations)
- More than 50% of elderly patients with dementia have paranoia or hallucinations

ETIOLOGY

There are numerous causes of hallucinations. The following are common: (An exhaustive list is beyond the scope of this chapter)

- **Psychiatric**
 - Schizophrenia
 - Bipolar disorder, mania
 - Major depression
- **Acute intoxications**
 - Ethanol
 - Cannabis

- Marijuana alternatives (i.e., *K2*, *Spice*)
- Sympathomimetics
 - Amphetamine
 - Methamphetamine
 - Cocaine
 - Synthetic cathinones (i.e., MDPV, ``bath salts'')
- NMDA antagonists
 - Ketamine
 - PCP
 - Dextromethorphan
- Serotonergic
 - MDMA (Ecstasy)
 - LSD
 - Peyote cactus (mescaline)
 - Mushrooms (psilocybin)
 - 2C series (i.e., 2CB, 2CT-7)
 - 5-MeO series (i.e., 5-MeO-DMT)
- Kappa opioid receptor agonist
 - *Salvia divinorum* (cause synesthesias – i.e., hearing colors or smelling sounds)
- Opiates
- Inhalants
 - Toluene
 - Nitrous oxide
- Medications
 - Anticholinergic agents
 - Steroids
 - Methylphenidate
- Withdrawal
 - Ethanol
 - Benzodiazepines
 - Barbiturates
 - GHB
- Substance-induced disorders
 - Methamphetamine-associated psychosis
 - Prolonged duration of psychosis, auditory hallucinations and recurrence without relapse of using drug
 - Cannabis-induced psychosis
- Infectious
 - Meningitis
 - Encephalitis
 - In patients with dementia, any infection (i.e., UTI, pneumonia) can trigger hallucinations

- Metabolic
 - Hypoglycemia
 - Electrolyte imbalances
 - Thyroid disease
 - Adrenal disease
 - Wilson’s disease
 - Thiamine deficiency
- Neurologic
 - Seizures
 - Partial simple or complex seizures can result in visual, auditory, olfactory, and gustatory hallucinations
 - Migraines
 - CNS hemorrhage or tumor
 - CVA
 - Tourette syndrome
 - Neurodegenerative disorders
 - Parkinsons
 - Dementia (Lewy body, Alzheimer)
 - HIV
- Ocular
 - Glaucoma
 - Macular degeneration
 - Charles Bonnet syndrome
- Others
 - Food, sensory, sleep deprivation
 - Fatigue, extreme stress
 - Heat-related illness
 - Religious and ritual activities
 - Falling asleep and awakening from sleep

Pediatric Considerations

Hallucinations are relatively common in children and adolescents and are often developmentally normal. Most children with hallucinations do not have psychosis. Hallucinations can occur as part of a delirium, such as from fever. As with the adult patient, carefully conduct a search for a medical or neurologic etiology.

Geriatric Considerations

In the elderly patient, hallucinations are most often from an organic cause. They can commonly accompany dementia, depression, medication reactions and substance abuse, and are often associated with agitation. Atypical antipsychotic agents are effective treatment for hallucinations with agitation in the elderly.

SIGNS AND SYMPTOMS

History

- Obtaining accurate and thorough history often difficult
 - Collateral history should be obtained from bystanders, EMS, police, family, physicians
- Assess for changes in behavior from baseline
- Explore for delusions or persecutory beliefs
- Previous episodes of hallucinations
- Change in medications
- Substance abuse history
- Alteration in cognition that rapidly develops and waxes and wanes throughout course of the day suggests delirium
- Headache may suggest CNS lesion or migraine

Physical-Exam

- Acute psychosis
 - Disorganized thought
 - Responding to internal stimuli
- Mania
 - Excessive talking or pressured speech
- Delirium
 - Altered level of consciousness
 - Not oriented
 - Abnormal vital signs
- CNS lesion
 - Cranial nerve deficit
 - Aphasia
 - Any focal neurologic finding
 - Gait abnormality
 - External signs of trauma
- Systemic or infectious illness
 - Asterixis
 - Fever
 - Nuchal rigidity
 - Myoclonus
 - Jaundice
 - Ascites
- Signs of intoxication or withdrawal
 - Sympathomimetic intoxication, ethanol/benzodiazepine withdrawal

- Agitation, excited delirium
- Mydriasis
- Tachycardia, hypertension
- Hyperthermia
- Diaphoresis
- Opiate
 - Miosis
 - Bradypnea
 - Needle marks
- Serotonergic
 - Tachycardia, hypertension
 - Hyperreflexia
 - Clonus
 - Tremor
- NMDA antagonism
 - Nystagmus

DIAGNOSIS TESTS & INTERPRETATION

- Common tests:
 - CBC, serum chemistries
 - Ethanol, acetaminophen, salicylate serum concentrations
 - Urinalysis
- More focused studies depending on comorbid conditions or clinical concerns:
 - Urine drug of abuse screen
 - Interpretation can be difficult as this is a test of use and not intoxication. In addition, it is not designed to detect newer drugs of abuse, although some may cross-react with this assay.
 - EKG
 - Thyroid function
 - Liver function tests
 - RPR, folate, B₁₂, thiamine
 - Specific drug concentrations

Imaging

- Brain imaging (CT, MRI)
- Chest x-ray

Diagnostic Procedures/Surgery

- If suspicion exists for medical cause, should consider procedures such as:
 - Lumbar puncture
 - EEG
- If hallucinations are from acute psychiatric illness or decompensation of chronic

psychiatric illness

- Obtain emergent psychiatric consultation

ESSENTIAL WORKUP

Patients with a clear psychiatric history with characteristic symptoms need minimal testing (CBC, chemistries). However, patients with undifferentiated hallucinations, especially those in high-risk groups, require extensive testing.

DIFFERENTIAL DIAGNOSIS

The primary goal of ED evaluation is to differentiate psychiatric from nonpsychiatric cause of hallucination. (See Psychosis, Medical vs. Psychiatric)

- More likely to be from psychiatric illness:
 - Auditory and command hallucination
 - Hallucinations and illusions incorporated into delusional system
 - Age of onset 13–40 yr old
 - Flat affect
 - Normal orientation
 - Disorganized attention
- The following groups are considered to be at higher risk for nonpsychiatric illness:
 - Elderly
 - History of substance abuse
 - No pre-existing psychiatric history
 - Presence of pre-existing medical disorders
 - Lower socioeconomic level
- Visual hallucinations more common:
 - Delirium
 - Dementia
 - Migraines
 - Dopamine agonist therapy (i.e., carbidopa)
 - Posterior cerebral infarcts
 - Narcolepsy



TREATMENT

PRE HOSPITAL

Observe details of patient's environment not available to hospital care team

- Disorganized living environment
- Drug paraphernalia

INITIAL STABILIZATION/THERAPY

- Address ABC's and any abnormal vital signs (i.e., supplemental oxygen for hypoxia)
- Check FSBG

- Consider thiamine 100 mg IV/PO
- Treat acute agitation (see Agitation)
 - De-escalation techniques
 - Physical restraints
 - Chemical sedation

ED TREATMENT/PROCEDURES

- If underlying medical cause identified
 - Treat medical etiology
 - These patients typically do not require antipsychotic medications
- In patients with acute psychosis or decompensation of chronic psychotic illness
 - Use antipsychotics and benzodiazepines (see Psychosis, Acute)
- In patients with hallucinations due to intoxication with excited delirium
 - General supportive care
 - Benzodiazepines
- If dementia with hallucinations
 - Treat underlying medical etiology, if any
 - Atypical antipsychotics have benefits and harm (CVA, extrapyramidal symptoms)

ALERT

When treating hallucinations with an excited delirium due to acute intoxication (except for ethanol), use benzodiazepines.



FOLLOW-UP

DISPOSITION

Admission Criteria

Disposition determined by medical condition or psychiatric evaluation. Hallucinations from some intoxications such as methamphetamine or cannabis may persist even after drug is metabolized.

Admission Criteria

- Medical condition requiring admission
- Acute psychiatric illness or decompensation of chronic psychiatric illness requiring psychiatric hospitalization

Discharge Criteria

- Symptoms have resolved and reversible medical cause (i.e., intoxication, UTI)
- Decompensation of chronic psychiatric condition has been addressed, home environment appropriate and mental health follow-up available.

Issues for Referral

Alcohol/drug treatment as appropriate

FOLLOW-UP RECOMMENDATIONS

As appropriate for medical or chronic psychiatric condition(s)

PEARLS AND PITFALLS

- Do not assume that auditory hallucinations are always from psychiatric illness whereas visual, tactile, olfactory, and gustatory hallucinations are nonpsychiatric – always perform thorough evaluation.
- Even though 10% of cases of schizophrenia occur in patients older than 45, do not assume hallucinations are from psychiatric cause in older age group without extensive workup.
- Do not treat hallucinations with excited delirium from an acute intoxication (except due to ethanol) with antipsychotic agents.

ADDITIONAL READING

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- Piechniczek-Buczek J. Psychiatric emergencies in the elderly population. *Emerg Med Clin North Am*. 2009;24(2):467–490.
- Sood TR, Mcstay CM. Evaluation of the psychiatric patient. *Emerg Med Clin North Am*. 2009;27(4):669–683.
- Sosland MD, Edelson GA. Hallucinations in children and adolescents. *Curr Psychiatry Rep*. 2005;7(3):180–188.



CODES

ICD9

- 291.3 Alcohol-induced psychotic disorder with hallucinations
- 368.16 Psychophysical visual disturbances
- 780.1 Hallucinations

ICD10

- F10.951 Alcohol use, unsp w alcoh-induce psych disorder w hallucin
- R44.1 Visual hallucinations
- R44.3 Hallucinations, unspecified

HALLUCINOGEN POISONING

Joanne C. Witsil

BASICS

DESCRIPTION

- Predominantly alters perception, cognition, and mood
- All hallucinogens potentiate neurotransmitter release or bind directly to receptors:
 - Serotonin (5-hydroxytryptamine; 5-HT): Many hallucinogens are agonists or antagonists at 5-HT receptor subtypes.
 - Norepinephrine, N-methyl-D-aspartate (NMDA), dopamine

ETIOLOGY

- Most exposures are intentional
- Common hallucinogens:
 - Indoleamine:
 - Lysergic acid diethylamide (LSD) (duration 6–12 hr)
 - Morning glory (*Ipomoea* spp.)
 - Tryptamines:
 - Psilocybin (*Psilocybe* mushrooms); frequently adulterated with LSD
 - N,N-dimethyltryptamine (DMT); 5-MeO-DMT (“foxy-methoxy”), and other tryptamine congeners
 - Phenylethylamines (hallucinogenic amphetamines):
 - Methylenedioxyamphetamine (MDA)
 - Methylenedioxymethamphetamine (MDEA)
 - Methylenedioxymethamphetamine (MDMA; “ecstasy”; duration 8–12 hr)
 - Paramethoxyamphetamine
 - Dimethoxyamphetamine
 - Mescaline (peyote cactus); frequently adulterated with LSD (duration 6–12 hr)
 - Arylcycloalkylamines:
 - Phencyclidine (PCP), (duration is variable 11–96 hr in 1 report)
 - Ketamine, (duration depends on route of administration 30–120 min)
 - Anticholinergic:
 - Deadly nightshade (*Atropa belladonna*)
 - Jimsonweed (*Datura stramonium*)
 - Other:
 - Piperazines: Benzyl piperazine (BZP) and trifluoromethyl phenylpiperazine (TFMPP)
 - Dextromethorphan (DXM), (duration 3–6 hr)

- Marijuana



DIAGNOSIS

SIGNS AND SYMPTOMS

- Considerable individual variation; effects may last 4–12 hr and ≤ 96 hr depending on agent/dose
- Symptoms characterized by sympathetic arousal
- Usually oriented and able to give history of exposure even while having delusions
- Initial symptoms:
 - Nausea, flushing, chills, tremor
- Neurologic symptoms:
 - Restlessness and dizziness early after ingestion
 - Desire to laugh (especially with *Psilocybe* mushrooms)
 - Anxiety, despair, helplessness, dread
 - Intensified perceptions, visual distortions/intensification
 - Tactile distortions (especially with mescaline)
 - Synesthesia (blending of sensory modalities, e.g., seeing sounds)
 - Religious or mystical experiences
 - Sleep disruption
- Neurologic signs:
 - Unusual/bizarre behavior
 - Speech disruption
 - Mydriasis
 - Piloerection
 - Hyperreflexia
 - Coma with massive exposure
 - Convulsions:
 - Hallucinogenic amphetamines
 - Children who become hyperpyrexia after *Psilocybe* mushroom ingestion
- Pulmonary:
 - Mild tachypnea
 - Respiratory arrest (with massive exposure)
- Cardiovascular:
 - Tachycardia
 - HTN (with hallucinogenic amphetamines)
 - Dysrhythmia (with hallucinogenic amphetamines)
 - Intracerebral hemorrhage (with hallucinogenic amphetamines)
- GI:
 - Nausea/vomiting (especially with mescaline)
- Metabolic:

- Hyperpyrexia:
 - Especially with MDMA use at “rave” clubs
 - Hepatic failure, renal failure, and disseminated intravascular coagulopathy may follow.
 - May be lethal
- Hyponatremia: Has been reported with MDMA use
- Hematopoietic:
 - Coagulopathies and hemorrhage at high doses owing to disruption of platelet serotonin function

History

Identity of agent or agents used including:

- Method of usage
- Quantity
- Time of exposure
- Knowledge of place of exposure

Physical-Exam

- Obtain accurate vital signs including temperature.
- Perform detailed neurologic and psychiatric exam

ESSENTIAL WORKUP

- Core temperature measurement and other vital signs
- ECG monitoring
- Determination of risk of rhabdomyolysis:
 - Creatine kinase level
 - Urine dip or myoglobin level

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Electrolytes, BUN, creatinine, glucose levels, coagulation screen, arterial, or venous blood gas
- Urine toxicology screen:
 - Rarely indicated
 - Distinguishing between hallucinogens is of little value
 - Most hallucinogenic substances are not tested for on routine drug screens
 - Amphetamine screen is frequently negative for hallucinogenic amphetamines (e.g., MDMA)

Imaging

- Consider chest x-ray if looking for aortic dissection, pulmonary aspiration, or trauma-related injury

- Consider CT of head if looking for intracranial bleeds or lesions
- Consider abdominal x-ray if there is suspicion of ingested packets

DIFFERENTIAL DIAGNOSIS

- Hypoglycemia
- Meningitis, encephalitis
- Sepsis
- Intracranial bleeds or lesions
- Withdrawal (ethanol, sedative–hypnotic, baclofen)
- Serotonin syndrome (especially with concomitant serotonergic agents involved)
- Psychiatric illnesses:
 - LSD associated with prolonged psychoses resembling schizoaffective disorders
 - Chronic amphetamine/cocaine abuse
 - Steroids
- Infectious/febrile seizures in hyperpyretic child

Pediatric Considerations

Assess parent–child relationships for possibility of neglect or abuse.



TREATMENT

PRE HOSPITAL

- Controversies:
 - Sedation with benzodiazepines vs. haloperidol vs. physical restraints:
 - Benzodiazepines are generally preferred
 - Sedation masks symptoms and may limit history
- Cautions:
 - Sedate or restrain patient to ensure safe transport
 - For hyperthermic patient:
 - Use sedation rather than physical restraint
 - Begin cooling measures

INITIAL STABILIZATION/THERAPY

- Management of ABCs
- Aggressive cooling if hyperthermic
- IV access/rehydration with isotonic fluids for significant fluid loss or evidence of rhabdomyolysis
- Naloxone, Accu-Chek, dextrose, and thiamine if patient has altered mental status

ED TREATMENT/PROCEDURES

- Cooling measures:

- Cool mist and fans
- Benzodiazepines if agitated
- Paralytics with intubation if needed (generally not succinylcholine)
- Sedate for agitation or autonomic signs:
 - Benzodiazepines
 - Rarely neuroleptics:
 - May intensify hallucinogenic experience
 - Possibly lower seizure threshold
- Activated charcoal (AC) is not expected to be helpful for most agents owing to rapid absorption and delayed patient presentation:
 - Consider AC for oral ingestions within 2–3 hr in patients with intact protective airway reflexes; especially for anticholinergics or seeds owing to delayed GI motility/absorption
- Place in a quiet, calm environment
- Maintain urine output of 2–3 mL/kg/hr and consider urine alkalinization for treatment of rhabdomyolysis

MEDICATION

- Benzodiazepines (diazepam): 5–10 mg IV (peds: 0.2–0.5 mg/kg) IV or lorazepam: 1–4 mg IV/IM (peds: 0.02–0.05 mg/kg) IV/IM, may repeat as needed
- Dextrose (D₅₀W) for hypoglycemia: 1 ampule: 25 g/50 mL (peds: D₂₅W, 0.5–1 g/kg or 2–4 mL/kg) IV, may repeat as needed
- Haloperidol (Haldol): 2.5–5 mg IV or IM may repeat every 30–60 min until calm, usual max. dose is 10–20 mg; not recommended for children
- Naloxone (Narcan): Initial dose: 2 mg (peds: 0.01–0.1 mg/kg) IV or IM, may repeat as needed
- Sodium bicarbonate infusion for rhabdomyolysis: 3 ampules in 1 L of D₅W; infuse at 1.5–2 times maintenance rate (keep urine pH > 7.5)
- Thiamine (vitamin B₁): 100 mg (peds: 25 mg) IV or IM × 1 dose



FOLLOW-UP

DISPOSITION

Admission Criteria

- Severely intoxicated
- Atypical presentation
- Prolonged symptoms (> 12 hr after exposure)
- Prolonged periods of agitation and hyperthermia:
 - Risk of rhabdomyolysis or organ damage

Discharge Criteria

After receiving supportive therapy and observation, most patients can be discharged once they are asymptomatic.

Pediatric Considerations

Suspected cases of child abuse or neglect require referral to child protective services.

FOLLOW-UP RECOMMENDATIONS

Upon discharge, patients should receive follow-up care from their PCP, psychiatrist, or drug counseling facility.

PEARLS AND PITFALLS

- Do not delay in the diagnosis and treatment of hyperthermia. Once hyperthermia is identified, aggressively lower body temperature.
- Use appropriate physical and chemical restraints to control violent and agitated patients to protect the patient and staff from physical injury.
- Conduct serial exams and vital signs (especially temperature). Do not assume once a violently agitated patient is calm that the patient is recovering. The patient may be progressing to serious illness.

ADDITIONAL READING

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- Hall AP, Henry JA. Acute toxic effects of “ecstasy” (MDMA) and related compounds: Overview of pathophysiology and clinical management. *Br J Anaesth*. 2006;96:678–685.
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- Richardson WH 3rd, Slone CM, Michels JE. Herbal drugs of abuse: An emerging problem. *Emerg Med Clinic North Am*. 2007;25:435–457.



CODES

ICD9

- 968.3 Poisoning by intravenous anesthetics
- 969.6 Poisoning by psychodysleptics (hallucinogens)
- 969.72 Poisoning by amphetamines

ICD10

- T40.8X1A Poisoning by lysergide, accidental (unintentional), init
- T40.901A Poisoning by unsp psychodyslept, accidental, init
- T43.621A Poisoning by amphetamines, accidental (unintentional), init

HAND INFECTION

Chester D. Shermer

BASICS

DESCRIPTION

- Hand infections are commonly seen in the ED.
- The range of pathology is broad and may include acute *and* chronic conditions.

ALERT

- Serious hand infections are potential liability issues and must be handled with extreme caution.
- Referral to hand surgeon is almost always indicated.

ETIOLOGY

- Bacterial infection of the hand is associated with skin pathogens:
 - Staphylococcus or Streptococcus spp
 - History of a puncture wound
- Anaerobes are identified in 75% of paronychia in children owing to thumb sucking and nail biting.
- Chronic paronychia may be caused by *Candida albicans*.
- Herpetic whitlow is caused by type 1–2 herpes simplex virus.
- Clenched fist injuries involve a variety of pathogens, including anaerobic Streptococcus and Eikenella spp.

DIAGNOSIS

SIGNS AND SYMPTOMS

- Paronychia:
 - Localized edema, erythema, and pain in proximal portion of lateral nail fold
 - Fluctuance may be present and may extend beneath the nail margin.
 - Systemic signs and symptoms are usually not present.
- Felon:
 - Erythema and tense swelling of the distal pulp space that does *not* extend proximal to the proximal interphalangeal (PIP) joint
 - Aching pain early, severe throbbing pain late
 - Systemic signs are usually not present.
- Herpetic whitlow:
 - Distal pulp space is swollen, but remains soft.
 - Lateral nail folds may be affected.

- Throbbing pain of the distal pulp space
- Vesicles containing nonpurulent fluid are present and may form bullae.
- Systemic symptoms may be present:
 - Fever
 - Lymphadenopathy
 - Constitutional symptoms
- Flexor tenosynovitis:
 - Kanavel signs:
 - Severe pain and symmetric edema of the digit
 - Tenderness over the course of tendon sheath
 - Flexed position of the finger at rest
 - Pain on passive extension of the finger—may be the only finding in early infection
- Clenched fist injury:
 - Laceration over the metacarpophalangeal (MCP) joint from striking an object with a clenched fist
 - Any laceration over the MCP must be assumed to be a *human bite wound* until proven otherwise.
- Web space abscess:
 - Pain and edema of the affected web space and adjacent palm
 - Fingers are held abducted.
- Palmar space infections:
 - Thenar space infection:
 - Pain, tenderness, tense edema of thenar eminence
 - Dorsal edema without tenderness
 - Thumb is held abducted and flexed, and passive adduction is painful.
 - Midpalmar space infection:
 - Pain, edema, and tenderness of the midpalmar space
 - Dorsal edema without tenderness
 - Motion of middle and ring fingers is painful
 - Hypothenar space infection:
 - Pain and fullness over hypothenar eminence
 - No limitation of finger movement

History

See Signs and Symptoms.

Physical-Exam

See Signs and Symptoms.

ESSENTIAL WORKUP

Most hand infections are diagnosed by history and physical exam with special attention to neurovascular status.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Although usually not necessary, herpetic whitlow may be confirmed by Tzanck test.
- Gram stain and culture may guide antibiotic choice in felons.
- Blood cultures, CBC are not routinely indicated.

Imaging

- Radiographs are usually not helpful unless there has been trauma or a suspected foreign body.
- With felon, flexor tenosynovitis, and palmar space infection, radiograph may identify osteomyelitis or foreign body.
- Radiographs in clenched fist injury may reveal a fracture.

DIFFERENTIAL DIAGNOSIS

- Paronychia should be differentiated from herpetic whitlow and felon.
- The differential for palmar space infection includes flexor tenosynovitis, cellulitis, and web space infection.



TREATMENT

PRE HOSPITAL

Hand immobilization as appropriate

ED TREATMENT/PROCEDURES

- Paronychia:
 - Early paronychia/simple cellulitis without purulence present may be managed with oral antibiotics and rest:
 - Cephalexin, dicloxacillin
 - Clindamycin or erythromycin, if associated with nail biting or oral contact
 - Superficial infections are drained by inserting a No. 11 blade between nail and eponychium, and lifting the eponychium from the nail.
 - If necessary, the lateral nail fold may be incised tangential to the curvature of the nail.
 - When pus is present under the adjacent nail, 1/4 of the nail should be removed.
 - When pus is present under the dorsal roof of the proximal nail, remove 1/3 of the proximal nail.
- Felon:
 - A lateral incision avoiding the neurovascular bundle is preferred.

- More extensive felons are drained through a unilateral longitudinal incision that does not cross the distal interphalangeal (DIP) flexor crease.
- Disruption of fibrous septa is no longer recommended:
 - Results in an unstable fingertip
 - Loculations may need to be broken up.
- Give oral antibiotics to cover skin pathogens, place a drain, and recheck in 48 hr:
 - Cephalexin, dicloxacillin
- Herpetic whitlow:
 - Usually self-limited; do not incise and drain.
 - Oral acyclovir may be given to patients with systemic involvement.
- Flexor tenosynovitis, web space abscess, palmar space infection:
 - Elevation, IV antibiotics, and pain control:
 - Ampicillin/sulbactam, cefoxitin, ticarcillin/clavulanate
 - All of these infections require immediate consultation with a hand surgeon.
- Clenched fist injury:
 - Elevation, IV antibiotics, tetanus prophylaxis, and pain control in the ED:
 - Ampicillin/sulbactam, cefoxitin, ticarcillin/clavulanate
 - All bite wounds with evidence of infection or joint involvement require emergent consultation with a hand surgeon.
 - If there are no signs of infection and no joint penetration, patients may be considered for outpatient treatment with oral antibiotics after appropriate irrigation and wound care:
 - Ampicillin/clavulanate or penicillin V + cephalexin or dicloxacillin
 - Do *not* primarily close lacerations associated with a human bite; delayed primary closure or healing by secondary intention is appropriate.

MEDICATION

- Acyclovir: 400 mg PO TID for 10 days (peds: Not recommended for herpetic whitlow)
- Amoxicillin/clavulanate: 875/125 mg PO BID (peds: 40 mg/kg/d PO div. q6h)
- Ampicillin/sulbactam: 1.5–3 g IV q6h (peds: Safety not established)
- Cefoxitin: 2 g IV q8h (peds: 80–160 mg/kg/d IV or IM div. q6h)
- Cephalexin: 500 g PO QID for 7 days (peds: 40 mg/kg/d PO div. q6h)
- Clindamycin: 300–450 mg PO QID for 7 days. Can use IV in severe cases: 600–900 mg IV q8h (peds: 20–40 mg/kg/d div. q8h PO IV or IM)
- Dicloxacillin: 500 mg PO QID for 7 days (peds: 12.5–50 mg/kg/d PO div. q6h)
- Erythromycin: 500 mg PO QID for 7 days (peds: 40 mg/kg/d div. q6h PO)
- Penicillin V: 250 mg PO QID (peds: 40 mg/kg/d PO div. q6h)
- Ticarcillin/clavulanate: 3.1 g IV q4–q6h (peds: 150–300 mg/kg/d IV div. q6–8h)

Tailor to etiology

Second Line

Tailor to etiology



FOLLOW-UP

DISPOSITION

Admission Criteria

- Flexor tenosynovitis, web space abscess, palmar space infections:
 - All these infections require admission for IV antibiotics and drainage.
- Clenched fist injury with signs of infection:
 - Requires admission for surgical débridement and IV antimicrobials

Discharge Criteria

- Paronychia and felons:
 - Patients with uncomplicated paronychia or felon may be discharged from the ED with a recheck and drain removal in 48 hr.
- Herpetic whitlow:
 - Patients with herpetic whitlow may be discharged from the ED with appropriate follow-up.
- Clenched fist injury without infection:
 - May be discharged on oral antibiotics with follow-up in 24 hr

Issues for Referral

Immediate consultation in emergency department is indicated

FOLLOW-UP RECOMMENDATIONS

Usually arranged by admitting physician after operative therapy

PEARLS AND PITFALLS

- Missed or delay in diagnosis
- Failure to obtain history of clenched fist injury
- Failure to consult surgeon promptly

ADDITIONAL READING

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Staphylococcus aureus hand infections in an urban setting. *J Hand Surg Am.* 2007;32(3):380–383.

- Ong YS, Levin LS. Hand infections. *Plast Reconstr Surg.* 2009;124(4):225e–233e.



See Also (Topic, Algorithm, Electronic Media Element)

Hand Infections <http://emedicine.medscape.com/article/783011-overview>



CODES

ICD9

- 112.3 Candidiasis of skin and nails
- 681.02 Onychia and paronychia of finger
- 914.9 Other and unspecified superficial injury of hand(s) except finger(s) alone, infected

ICD10

- B37.2 Candidiasis of skin and nail
- L03.019 Cellulitis of unspecified finger
- S61.439A Puncture wound w/o foreign body of unsp hand, init encntr

BASICS

DESCRIPTION

- Hazmat refers to exposure to hazardous materials causing local or systemic toxicity.
- Pathophysiology:
 - Acids cause coagulation necrosis with eschar, usually limiting penetration to deeper tissue.
 - Alkalis cause liquefaction necrosis and soluble complexes that penetrate into deep tissues.
 - Damage also occurs through oxidation, protein denaturation, cellular dehydration, local ischemia, and by metabolic competition/inhibition.

ETIOLOGY

- Hazardous materials are encountered in household, industry, agriculture, transportation accidents, and in criminal/terrorist activities.
- The toxicity of the materials relates to the particular substances and their effects.

DIAGNOSIS

SIGNS AND SYMPTOMS

- Skin:
 - Chemical burns; may appear deceptively mild initially
 - Visible liquid or powder on skin
 - Absorption through skin may cause systemic toxicity.
- Mucous membranes (eyes, nasopharynx; see Corneal Burn):
 - Ranges from subjective irritation to serious mucosal burns
 - Potential airway compromise
- Pulmonary:
 - Cough
 - Pleuritic chest pain
 - Bronchospasm
 - Dyspnea
 - Pulmonary edema (immediate or delayed)
- Systemic (after skin or pulmonary absorption):
 - Altered mental status
 - Seizures
 - Tachy/brady dysrhythmias

- Hypotension/HTN
- GI symptoms
- Electrolyte disturbances
- Carboxyhemoglobinemias and methemoglobinemias
- Cyanide toxicity
- Cholinergic syndrome (see Chemical Weapons Poisoning, Nerve Agents)

History

Elicit type, circumstances, and duration of exposure

ESSENTIAL WORKUP

- Attempt to identify substance using pre-hospital providers, Material Safety Data Sheet (MSDS), and *Chemical Transportation Emergency Center (Chemtrec)*.
- MSDS:
 - Identifies chemicals
 - Differentiates vapor vs. skin hazard
 - Determines need for decontamination
 - Limited treatment data
- Determine route and duration of exposure.
- Inhalation injury more likely in an enclosed space
- Determine toxicity using poison control; computerized databases, such as POISINDEX or TOXNET; or standard toxicology test.
- Observe as needed for systemic toxicity.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Depends on substance
- Electrolytes, BUN, creatinine, and glucose levels
- LFTs
- Calcium level
- Magnesium level
- Phosphorus level
- Arterial blood gases:
 - Metabolic acidosis
 - Carboxyhemoglobinemias and methemoglobinemias
 - Respiratory failure

Imaging

Chest radiograph for pulmonary edema

DIFFERENTIAL DIAGNOSIS

- Skin:

- Hypersensitivity reaction
- Thermal burns
- Pulmonary:
 - Pneumonia
 - Pulmonary embolism
 - Anaphylaxis
- Systemic:
 - Status epilepticus
 - Overdose
 - Psychiatric illness
 - Myocardial infarction



TREATMENT

PRE HOSPITAL

- Recognize a HAZMAT situation:
 - Accident at industrial/agricultural site
 - Accident involving transport of hazardous materials
 - Suspected terrorist mass casualty incident
 - Cholinergic syndrome
 - Irritant mucous membrane symptoms
 - Chemical burns
- Protect yourself:
 - Approach from upwind.
 - Do not enter scene until safety of material is determined.
 - Use Level A protective gear if safety not established
 - Anyone able to walk and talk is minimally contaminated.
- Personal chemical protective equipment:
 - Level A: Positive-pressure self-contained breathing apparatus (SCBA), fully encapsulated chemical-resistant suit, double chemical-resistant gloves, chemical-resistant boots, and airtight seals between suit, gloves, boots
 - Level B: SCBA, nonencapsulated chemical suit, double gloves, boots
 - Level C: Air-purification device, suit, gloves, boots
 - Level D: Common work clothes
 - Identify substance:
 - Department of Transportation (DOT) placard, MSDS, shipping papers, hazard labels
 - If unsuccessful, call Chemtrec (1[800] 424-9300) to determine substance and toxicity.
 - Hazmat teams can do chemical testing.
- Determine toxicity and need for decontamination:
 - Poison control (1[800] 222-1222)

- Chemtrec
- Decontaminate:
 - Hazmat team
- Treat:
 - Provide basic life support and advanced life support care as indicated.
 - Generally basic life support only in a “hot zone”
 - Irrigate skin and ocular burns immediately and continue until arrival at hospital.

INITIAL STABILIZATION/THERAPY

- Protect ED personnel:
 - Secondary contamination can occur from dermal contact or through inhalation of volatile gases/particles.
- Keep patients outside in designated hot zones until decontaminated.
- When in doubt, decontaminate.
- Expect contaminated patients to arrive via emergency medical services or private vehicle.
- If treatment is required before/during decontamination:
 - Use minimum necessary staff in appropriate personal protection gear.
 - Focus on life- and limb-saving care only.
- Decontamination:
 - Security to enforce hot zone
 - Remove, label, and double-bag clothing (including contact lens).
 - Copious irrigation with soap and water for 10–15 min with special attention to obviously contaminated areas, wounds, and exposed eyes
 - Recapture water to prevent contamination of the sewer and downstream areas:
 - In an emergency or mass casualty situation, it is acceptable to let water drain into sewer.
 - Hydrotherapy:
 - Mainstay of therapy for chemical burns
 - Contraindicated only for elemental metals (sodium and potassium)
 - Allow patient to decontaminate himself or herself or use trained decontamination team.
 - Decontaminate children, dependent elderly, mentally/physically challenged and their appliances (e.g., wheelchairs) with caregivers
 - Gloves, masks, goggles, and disposable gowns provide some protection
 - Remove/replace bandages, tourniquets, airway adjuncts, IV sets
 - Re-triage after decontamination.

ED TREATMENT/PROCEDURES

- Provide supportive care as needed.
- Determine if antidotal treatment would be effective and available.

- Hazmat incidents provoke extreme fear:
 - Expect casualties suffering from collective hysteria.
 - Knowledge of toxicologic profile can exclude contamination in these patients.
- ED staff may become symptomatic even if chemical concentrations in the air are below toxic levels and may need to be escorted to fresh air.
- Chemical burns:
 - Irrigation should be started as soon as possible and, if owing to a strong alkali, may need to be continued for hours.
 - Aggressive fluid resuscitation with 2–4 mL/kg lactated Ringer solution per total burn surface area (TBSA) percent over 24 hr with 1/2 given over the 1st 8 hr
 - Pain control
- Pulmonary symptoms:
 - Bronchodilators, oxygen, intubation, and mechanical ventilation
- Selected special treatments:
 - Hydrofluoric acid burns:
 - Calcium gluconate via topical cutaneous gel, SC, or intra-arterial
 - For systemic toxicity: IV calcium gluconate and magnesium
 - Phenol burns:
 - Remove phenol from skin with polyethylene glycol 300 or 400 or with isopropyl alcohol.
 - Nitrates:
 - Ingested or extensive burns may cause methemoglobinemia.
 - Treat levels >30% with high-flow oxygen and IV methylene blue.
 - Elemental metals (sodium/potassium):
 - Water lavage is contraindicated and dangerous.
 - Cover with oil until substance can be débrided from skin.
 - Cyanide toxicity:
 - Hydroxocobalamin administration
 - Organophosphates/carbamate insecticides (see Chemical Weapons Poisoning)

MEDICATION

- Albuterol: 2.5–5.0 mg nebulized
- Calcium gluconate: 10 mL of 10% solution applied topically. Consult poison center for instructions.
- Magnesium: 2 g IV over 20 min
- Methylene blue: 1–2 mg/kg slow IV (peds: *Not recommended for <6 yr old; >6 yr old: 1 mg/kg IV/IM over 5 min*)
- Hydroxocobalamin: 5 mg IV over 5 min, repeat once



FOLLOW-UP

DISPOSITION

Admission Criteria

- Airway compromise, respiratory difficulty (hypoxia)
- Significant systemic symptoms
- Admit patients with chemical burns to burn center.

Discharge Criteria

- Patients who are well after a period of observation and consultation with poison control
- Superficial chemical burns owing to a toxin without potential for systemic toxicity (weak acid/alkali)

FOLLOW-UP RECOMMENDATIONS

Psychiatric or social work referral for victims of chemical terrorist attacks.

PEARLS AND PITFALLS

- Decontaminate stable victims on site when possible.
- Protect medical providers (pre-hospital and ED) with appropriate personal protective equipment.
- Provide specific antidotes for exposures when indicated.
- Victims who can walk and talk are minimally contaminated.

ADDITIONAL READING

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- Freyberg CW, Arquilla B, Fertel BS, et al. Disaster preparedness: Hospital decontamination and the pediatric patient—Guidelines for hospitals and emergency planners. *Prehosp Disaster Med.* 2008;23(2):166–172.
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See Also (Topic, Algorithm, Electronic Media Element)

- Chemical Weapons Poisoning

- Cyanide Poisoning
- Radiation Injury



CODES

ICD9

- V87.09 Contact with and (suspected) exposure to other hazardous metals
- V87.2 Contact with and (suspected) exposure to other potentially hazardous chemicals
- V87.39 Contact with and (suspected) exposure to other potentially hazardous substances

ICD10

- Z77.018 Contact with and (suspected) exposure to other hazardous metals
- Z77.098 Contact w and expsr to oth hazard, chiefly nonmed, chemicals
- Z77.128 Contact with and (suspected) exposure to other hazards in the physical environment

HEAD TRAUMA, BLUNT

Gary M. Vilke

BASICS

DESCRIPTION

Blunt trauma to head resulting in a variety of injuries ranging from closed head injury to death

ETIOLOGY

Blunt trauma to head may cause several types of closed head injuries:

- **Concussion:** Transient (LOC) or amnesia with normal head CT
- **Subdural hematoma:** Tearing of subdural bridging veins and bleeding into the subdural space
- **Epidural hematoma:** Dural arterial injury, especially the middle meningeal artery often associated with a skull fracture:
 - Classically, transient LOC followed by a lucid interval, then rapid demise
- **Subarachnoid hemorrhage:** Bleeding into the subarachnoid space following trauma
- **Cerebral contusion:** Focal injuries to the brain characterized as coup (beneath area of impact) or contrecoup (area remote from impact)
- **Intracerebral hemorrhage:** Mass intracranial lesion with bleeding into the brain parenchyma
- **Diffuse axonal injury:** Microscopic injuries scattered throughout the brain in a patient in deep coma

DIAGNOSIS

SIGNS AND SYMPTOMS

- Evidence of trauma to head includes:
 - Scalp laceration, cephalohematoma, or ecchymosis
 - Raccoon eyes: Bilateral ecchymosis of orbits associated with basilar skull fractures
 - Battle sign: Ecchymosis behind the ear at mastoid process associated with basilar skull fracture
 - Hemotympanum
 - Cerebral spinal fluid rhinorrhea or otorrhea
- Evidence of increasing intracranial pressure includes:
 - Decreasing level of consciousness, falling score on Glasgow Coma Scale
 - Cushing response, bradycardia, HTN, and diminished respiratory rate
 - Dilated pupils associated with decorticate or decerebrate posturing

History

- Mechanism
- LOC or amnesia for event
- Use of anticoagulants
- Headache, visual changes, or hearing loss
- Focal neurologic complaints
- Associated neck pain

Physical-Exam

- Evaluation of head for hematoma, Battle sign, raccoon eyes
- Complete neurologic exam
- Exam of neck/cervical spine

ESSENTIAL WORKUP

- Imaging indicated for patients with any of the following:
 - LOC or amnesia of events
 - Progressive headache
 - Alcohol or drug intoxication
 - Unreliable history or dangerous mechanism
 - Post-traumatic seizure
 - Repeated vomiting
 - Signs of basilar skull fracture
 - Possible skull penetration or depressed skull fracture
 - Glasgow Coma Scale score < 15
 - Focal neurologic findings
- Patients on Coumadin, heparin, or other anticoagulants and those with a history of bleeding dyscrasias must undergo imaging.
 - If initial head CT is negative and < 4 hr post injury, patient must be monitored and repeat head CT 4–6 hr post injury, or earlier if clinical deterioration.
- Alcoholics have an increased risk for bleeding, low threshold for imaging

Geriatric Considerations

- Older patients (> 60–65 yr of age) are at higher risk of intracranial hemorrhage.
- Many are on anticoagulation, take a careful hx.
- Have a low threshold for obtaining CT scan.

DIAGNOSIS TESTS & NTERPRETATION

Lab

- Rapid check of blood glucose level
- CBC, platelet count, and coagulation parameters
- Type and cross-match for surgical candidates.

- Baseline electrolytes, BUN, and creatinine levels
- Blood alcohol level if indicated

Imaging

- CT or MRI of head as indicated
- Cervical spine radiographs or helical CT when indicated

Diagnostic Procedures/Surgery

Lumbar puncture if question of subarachnoid blood on head CT

DIFFERENTIAL DIAGNOSIS

- Penetrating head trauma
- Any condition that alters mental status that may have produced a fall and caused external evidence of head trauma (e.g., hypoglycemic episode, seizure)



TREATMENT

PRE HOSPITAL

- Blunt head trauma patients with risk for intracranial lesion must go to a trauma center:
 - High-risk patients include those with depressed consciousness, focal neurologic signs, multiple trauma, or palpable depressed skull fractures.
- Moderate-risk patients should go to a hospital with availability of prompt neurosurgical consultation:
 - Moderate-risk patients include those with progressive headache, alcohol or drug intoxication, unreliable history, post-traumatic seizure, repeated vomiting, post-traumatic amnesia, signs of basilar skull fracture.
- Protect and manage the airway, including intubation:
 - Routine hyperventilation without signs of cerebral herniation should be avoided.
- If evidence of cerebral herniation (see Signs and Symptoms) or progressive neurologic deterioration in a normotensive patient, initiate measures to decrease intracranial pressure:
 - Mild hyperventilation to keep ETCO₂ about 30–35 mm Hg:
 - 20 breaths/min in adults
 - 25 breaths/min in children
 - 30 breaths/min in infants < 1 yr
 - Elevating head of bed 20–30°
- Cervical spine precautions must be maintained in all patients.
- Cautions:
 - Avoid hypotension (systolic BP < 90 mm Hg); use IV crystalloid solutions to maintain BP.

- Avoid hypoxia (oxygen saturation < 90%); administer 100% oxygen.
- Check blood glucose level.

INITIAL STABILIZATION/THERAPY

Management of ABCs:

- Control airway as needed:
 - Rapid sequence intubation if Glasgow Coma Scale score < 8, unable to protect airway, or evidence of hypoxia
 - Normalize Pco₂, avoid hyperventilation and hypoventilation.
- Treatment with etomidate or fentanyl as induction agent, succinylcholine (pretreat with minidose paralytic), rocuronium, or vecuronium; morphine for ongoing sedation
- Caution with fentanyl in hemodynamically labile patients
- IV catheter placement with crystalloid solution as needed to avoid hypotension (keep systolic BP > 90 mm Hg)
- Cervical spine precautions

ED TREATMENT/PROCEDURES

- Early neurosurgical consultation
- If patient has evidence of cerebral herniation (see Signs and Symptoms), initiate measures to decrease intracranial pressure:
 - *Mild* hyperventilation: 20 breaths/min in adults, 25 breaths/min in children, and 30 breaths/min in infants < 1 yr to keep ETCO₂ about 30–35.
 - Elevate head of bed 20–30°
 - Mannitol boluses IV: Do not administer mannitol unless systolic BP > 100 mm Hg and patient is adequately fluid resuscitated
- Phenytoin to prevent *early* post-traumatic seizures
- Reverse hypocoagulable states
- The use of glucocorticoids is *not* recommended to lower intracranial pressure in head trauma patients.
- Barbiturates are *not* recommended in the initial ED treatment of head-injured patients.
- If definitive neurosurgical care is not immediately available, a single burr hole may preserve life until neurosurgical intervention can be obtained:
 - Perform only in comatose patients with decerebrate or decorticate posturing on the side of a known mass lesion who have not responded to hyperventilation and mannitol.
- Transfuse as needed to keep hematocrit > 30%.
- Avoid hypothermia, which will increase risks of coagulopathy during surgery.
- Maintain NPO status.
- Surgery:
 - Surgical procedure based on findings of CT scan and neurosurgical

MEDICATION

For RSI intubation, increased ICP, seizures, anticoagulation reversal, and pain control

First Line

- Etomidate: 0.2–0.3 mg/kg IV
- Fentanyl: 3–5 µg/kg IV if systolic BP > 100 mm Hg
- Mannitol: 0.25–1 g/kg IV bolus
- Morphine sulfate: 2–20 mg IV (peds: 0.1 mg/kg IV up to adult doses)
- Phenytoin: 15–20 mg/kg IV up to 1,000 mg
- Rocuronium: 0.6 mg/kg IV
- Succinylcholine: 1–2 mg/kg IV
- Vecuronium bromide: 0.1 mg/kg IV; minidose pretreatment: 0.01 mg/kg IV
- Vitamin K:
 - To be used in patients on Coumadin with intracranial hemorrhage
 - 10 mg in 50 mL NS infused over 30 min
- Protamine sulfate:
 - To be considered if taking low molecular weight heparin (LMWH) with intracranial hemorrhage
 - If LMWH used < 8 hr prior, use 1 mg protamine for each mg of LMWH slow IV push over 1–3 min
 - If LMWH used > 8 hr prior, use 0.5 mg protamine for each mg of LMWH slow IV push over 1–3 min



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients with mass lesion associated with head trauma must be admitted to the ICU or undergo surgery.
- Patients with subarachnoid hemorrhage and diffuse axonal injury should be initially admitted to the ICU.
- Patients with ongoing symptoms including repetitive questioning, anterograde amnesia, or disorientation should be admitted to a monitored unit for neurologic evaluation.

Discharge Criteria

- Patients with resolved symptoms, negative findings on head CT, and no other comorbid factors (e.g., intoxication, additional trauma needing treatment) may be discharged.

- Patients on anticoagulation need to be observed and have negative findings on a head CT at 4–6 hr after the injury prior to discharge.
- Patients with minor head trauma, no LOC or amnesia, and normal neurologic exam findings may be discharged home with a friend or family member and head injury instructions.

Pediatric Considerations

Cases of suspected nonaccidental trauma must be reported to the appropriate legal agency.

Issues for Referral

If there are symptoms of concussion, patient will need follow-up with PMD, sports medicine physician, or neurologist to determine whether return to sports will be safe.

FOLLOW-UP RECOMMENDATIONS

Return if worsening headache, visual changes, confusion, focal neurologic changes, or other changes in clinical status.

PEARLS AND PITFALLS

- Failure to query about anticoagulant use and image appropriately
- Failure to aggressively reverse hypocoagulable states
- Failure to counsel patient with a concussion for no contact sports until cleared by PMD, sports medicine physician, or neurologist.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Head Trauma, Penetrating
- Spine Injury: Cervical, Adult



CODES

ICD9

- 850.9 Concussion, unspecified
- 852.20 Subdural hemorrhage following injury without mention of open intracranial wound, unspecified state of consciousness
- 959.01 Head injury, unspecified

ICD10

- S06.0X0A Concussion without loss of consciousness, initial encounter
- S06.5X0A Traum subdr hem w/o loss of consciousness, init
- S09.90XA Unspecified injury of head, initial encounter

HEAD TRAUMA, PENETRATING

Gary M. Vilke

BASICS

DESCRIPTION

Penetrating injury to intracranial contents:

- **High-velocity penetration:** Usually bullets, which cause trauma directly to brain tissue and also have a “shock wave” injury to local surrounding brain
- **Low-velocity penetration:** Usually knives, picks, or other sharp objects, with direct local trauma to brain tissue

ETIOLOGY

- **Direct penetration of the skull into the intracranial cavity by foreign object:**
 - Direct or local damage to brain tissue
 - Intracranial hemorrhage, including subdural, epidural, and intraparenchymal bleeds
- A bullet that hits the skull, ricochets off, and does not fracture the skull can still cause significant trauma to the underlying brain tissue.

DIAGNOSIS

SIGNS AND SYMPTOMS

- **Alteration in level of consciousness and neurologic exam varies based on object and location.**
- **Evidence of increasing intracranial pressure:**
 - Decreasing level of consciousness
 - Falling Glasgow Coma Scale score
 - Cushing response: Bradycardia, hypertension, and diminished respiratory rate
 - Blown pupil associated with decorticate or decerebrate posturing
- **Evidence of penetrating injury to head or basilar skull fracture, or object still remaining in head:**
 - **Raccoon eyes:** Bilateral ecchymosis of orbits associated with basilar skull fractures
 - **Battle sign:** Ecchymosis behind the ear at mastoid process associated with basilar skull fracture
 - Hemotympanum
 - CSF rhinorrhea or otorrhea

History

- Determine the weapon type or caliber of weapon at scene.
- Loss of consciousness (LOC) or amnesia for event
- Use of anticoagulants
- Headache, visual changes, or hearing loss
- Focal neurologic complaints

Physical-Exam

- Evaluation of head for evidence of penetrating injury and if a projectile, for multiple sites
- Complete neurologic exam
- Alteration in level of consciousness and neurologic exam varies based on object and location.
- Evidence of penetrating injury to head

ESSENTIAL WORKUP

- Thorough history and exam to assess extent of injuries
- Imaging study

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC
- Platelet count
- Coagulation parameters
- Type and cross-match
- Electrolytes, BUN, and creatinine baseline levels

Imaging

- CT of head depicts location of lesion and extent of damage.
- Skull radiographs may reveal depth of impalement, location of bone fragments, and presence of fragments within the cranium.
- Cervical spine evaluation (when indicated):
 - Helical CT scanning or anteroposterior, lateral, and odontoid views plain radiographs

DIFFERENTIAL DIAGNOSIS

- Blunt head trauma
- Basilar skull fracture
- Any condition that alters mental status that may have induced a fall and caused secondary penetrating trauma



TREATMENT

PRE HOSPITAL

- Stabilize but do not remove foreign object (e.g., knife).
- Determine the weapon type or caliber of weapon at scene.
- Protect and manage the airway to avoid hypoxemia.
- Avoid hyperventilation.
- Maintain cervical spine precautions.
- Transport to trauma center.
- Avoid hypoxia (oxygen saturation < 90%):
 - 100% oxygen
- Avoid hypotension (systolic BP < 90 mm Hg):
 - Administer IV crystalloid solutions

INITIAL STABILIZATION/THERAPY

- Management of ABCs
- Rapid sequence intubation:
 - For Glasgow Coma Scale score < 8, inability to protect airway, hypoxia, or cerebral herniation
 - Medications include etomidate or fentanyl as induction agent, succinylcholine (pretreat with minidose paralytic), rocuronium, or vecuronium; and morphine sulfate for ongoing sedation
 - Caution with fentanyl in the hemodynamically labile patient
 - Normalize Pco₂. Avoid hyperventilation or hypoventilation.
- IV catheter placement
- Crystalloid solution to maintain systolic BP > 90 mm Hg
- Address other sources of associated trauma.
- Cervical spine precautions should be maintained.

ED TREATMENT/PROCEDURES

- Early neurosurgical consultation
- If patient has evidence of cerebral herniation (see Signs and Symptoms), initiate measures to decrease intracranial pressure:
 - *Mild* hyperventilation: 20 breaths/min in adults, 25 breaths/min in children, and 30 breaths/min in infants < 1 yr to keep ETCO₂ about 30–35 mm Hg.
 - Elevate head of bed 20–30°.
 - Mannitol boluses IV: Do not administer mannitol unless systolic BP > 100 mm Hg and patient is adequately fluid resuscitated.
- Phenytoin intravenously to prevent *early* post-traumatic seizures
- Reverse hypocoagulable states
- Glucocorticoids are *not* recommended to lower intracranial pressure in head trauma patients.
- Barbiturates are *not* recommended in the initial ED treatment.
- Transfuse as needed to keep hematocrit > 30%.

- If definitive neurosurgical care is not immediately available, a single burr hole may preserve life until neurosurgical intervention can be attained:
 - Perform only in comatose patients with decerebrate or decorticate posturing who have not responded to initial treatment on the side of a known mass lesion/hematoma.
- Avoid hypothermia, which will increase risks of coagulopathy during surgery.
- Maintain NPO status.
- Surgery:
 - Based on clinical and radiologic findings and neurosurgical consultation

MEDICATION

For RSI intubation, increased ICP, seizures, and pain control

First Line

- Etomidate: 0.2–0.3 mg/kg IV
- Fentanyl: 3–5 µg/kg IV:
 - If systolic BP > 100 mm Hg
- Mannitol: 0.25–1 g/kg IV bolus
- Morphine sulfate: 2–20 mg IV (peds: 0.1 mg/kg up to adult doses)
- Phenytoin: 15–20 mg/kg IV up to 1,000 mg
- Rocuronium: 0.6 mg/kg IV
- Succinylcholine: 1–2 mg/kg IV
- Vecuronium bromide: 0.1 mg/kg IV:
 - Pretreatment minidose: 0.01 mg/kg IV
- Vitamin K:
 - To be used in patients on Coumadin with intracranial hemorrhage
 - 10 mg in 50 mL NS infused over 30 min
- Protamine sulfate:
 - To be considered if taking low molecular weight heparin (LMWH) with intracranial hemorrhage
 - If LMWH used < 8 hr prior, use 1 mg protamine for each mg of LMWH slow IV push over 1--3 min
 - If LMWH used > 8 hr prior, use 0.5 mg protamine for each mg of LMWH slow IV push over 1--3 min



FOLLOW-UP

DISPOSITION

Admission Criteria

Admit all patients to ICU or transport directly to surgery.

Discharge Criteria

Do not discharge.

FOLLOW-UP RECOMMENDATIONS

All patients with penetrating skull injuries should have been admitted.

PEARLS AND PITFALLS

- Failure to query about anticoagulant use and image appropriately
- Failure to aggressively reverse hypocoagulable states

ADDITIONAL READING

- Badjatia N, Carney N, Crocco TJ, et al. Guidelines for prehospital management of traumatic brain injury 2nd edition. *Prehosp Emerg Care*. 2008;12(suppl 1):S1–S52.
- Brain Trauma Foundation, American Association of Neurological Surgeons, Congress of Neurological Surgeons, et al. Guidelines for the management of severe traumatic brain injury. XV. Steroids. *J Neurotrauma*. 2007;24(suppl 1):S91–S95.
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- Espinosa-Aguilar A, Reyes-Morales H, Huerta-Posada CE, et al. Design and validation of a critical pathway for hospital management of patients with severe traumatic brain injury. *J Trauma*. 2008;64(5):1327–1341.
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- Warner KJ, Cuschieri J, Copass MK, et al. The impact of prehospital ventilation on outcome after severe traumatic brain injury. *J Trauma*. 2007;62(6):1330–1336.



See Also (Topic, Algorithm, Electronic Media Element)

- Head Trauma, Blunt
- Spine Injury: Cervical, Adult



CODES

ICD9

- 803.60 Other open skull fracture with cerebral laceration and contusion, unspecified state of consciousness
- 803.90 Other open skull fracture with intracranial injury of other and unspecified

nature, unspecified state of consciousness

- 854.10 Intracranial injury of other and unspecified nature with open intracranial wound, unspecified state of consciousness

ICD10

- S02.91XB Unspecified fracture of skull, init encntr for open fracture
- S06.2X0A Diffuse TBI w/o loss of consciousness, init
- S06.330A Contusion and laceration of cerebrum, unspecified, without loss of consciousness, initial encounter

HEADACHE

Josh W. Joseph

BASICS

DESCRIPTION

- Pain in the cranium, orbits, or upper neck
- Pain within the skull is projected to the surface:
 - Intracranial:
 - Arteries, veins, dura, meninges
 - Extracranial:
 - Skin, scalp, fascia, muscles
 - Mucosal linings of the sinuses
 - Arteries
 - Temporomandibular joints, teeth
- Pain is transmitted via the V cranial nerve.
- May be caused by a number of mechanisms:
 - Nerve irritation
 - Traction on pain-sensitive vessels
 - Vasodilatation of pain-sensitive vessels
 - Hypoxia, hypercapnia, fever, histamine injection, nitroglycerin ingestion
- Complaint in 2–4% of all ED visits:
 - 95% have a benign etiology (lower in patients older than 50 yr)
 - Life-threatening etiologies are rare and can be difficult to diagnose.

ETIOLOGY

- Migraine:
 - Intra/extracranial vasodilatation and constriction of pain-sensitive blood vessels
 - May also involve cortical depression
 - Throbbing headache
- Tension:
 - Requires ≥ 10 attacks of a similar nature
 - Unknown etiology (possibly serotonin imbalance, decreased endorphins, spasm)
 - Most common type of recurring headache
 - Triggered by poor posture, stress, anxiety, depression, cervical osteoarthritis
 - Bilateral, nonpulsatile, band like
 - Mild to moderate intensity
 - 4–13 hr duration

- Cluster headaches:
 - Triggered by alcohol, certain foods, altered sleep habits, strong emotions
 - May involve vasospasm near cranial nerves
- Intracranial (traction, pressure):
 - Mass lesions
 - Idiopathic intracranial hypertension
- Extracranial (compression):
 - Pathology causing pain in a peripheral nerve of the head and neck
- Inflammation:
 - Temporal arteritis
 - Cerebral vasculitis
- Thrombosis:
 - Cerebral venous sinus thrombosis (CVST)
- Impaired vascular autoregulation/endothelial dysfunction:
 - Posterior reversible leukoencephalopathy syndrome (PRES)
 - Reversible cerebral vasoconstriction syndrome (RCVS)

Pediatric Considerations

Serious causes of headache in children are rare but those who come to the ED for this complaint should all have follow-up with a pediatrician.

Geriatric Considerations

Older patients with new headache have a higher likelihood of a serious etiology and should have more thorough evaluation with a low threshold for imaging.

Pregnancy Considerations

In addition to all other causes of headache, pregnant women (and recently postpartum women) are at increased risk for CVST, eclampsia, PRES, and RCVS.

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Attributes of the pain—PQRST:
 - Provocative and palliative features:
 - Position of the head, coughing or straining (increase suggests elevated ICP), and movement
 - Quality:
 - Throbbing or continuous
 - Deep or superficial
 - Change compared to prior headaches

- Region
- Severity
- Worst headache of life?
- Timing
- Sudden or gradual?
- Associated findings:
 - Visual symptoms, dizziness, nausea, vomiting
- Historical factors indicating additional testing:
 - New onset:
 - Age > 50
 - HIV, transplant, or cancer patient?
 - Trauma or falls (even without headstrike)
 - Persistent vomiting
 - Any new focal neurologic or visual symptoms
- Risk factors for cerebral sinus thrombosis:
 - Malignancy
 - Pregnancy (or postpartum)
 - Protein S or protein C deficiency
 - Oral contraceptive
 - Ulcerative colitis
 - Behçet syndrome

Physical-Exam

- Complete neuro exam including cranial nerves, motor, sensation, deep tendon reflexes, gait
- Examine for papilledema.
- Evaluate skin for rashes:
 - Zoster
 - Purpura
- Palpate temporal arteries

ESSENTIAL WORKUP

- Detailed history, CNS, HEENT, and neck exam
- Factors indicating testing beyond the history and physical exam:
 - Severely elevated diastolic BP
 - Fever
 - Altered level of consciousness
 - Papilledema
 - Abnormal neurologic exam or meningismus

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CSF:
 - Essential in suspected meningitis, subarachnoid hemorrhage (SAH)
- ESR:
 - If temporal arteritis or other inflammatory disorders suspected:
 - See Giant Cell Arteritis

Imaging

- Head CT scan:
 - Indications:
 - Uncertain diagnosis based on history and physical exam (leaving open the possibility of serious causes)
 - Signs of increased ICP
 - “First or worst” headache
 - Abrupt onset
 - New focal neurologic abnormalities
 - Papilledema
 - Recurrent morning headache
 - Persistent vomiting
 - Associated with fever, rash, and nausea
 - Trauma with loss of consciousness, focal deficits, or lethargy
 - Altered mental status, meningismus
 - Definitive test for SAH if performed within 6 hr of onset and read by an attending radiologist
 - Within 24 hr, > 95% sensitive (sensitivity falls rapidly with time and is 50% at 7 days out)
- Sinus imaging may show acute sinusitis; chronic sinusitis rarely causes acute headache.
- MRI:
 - Indicated to assess for etiologies that are missed by CT scan and LP:
 - Posterior fossa lesion
 - Pituitary apoplexy
 - CVST
- MRA:
 - Indicated if SAH suspected, CT is negative, and unable to perform lumbar puncture
 - Suspicion of carotid or vertebral dissection (e.g., recent neck manipulation or trauma)
 - Nonmigrainous vascular cause suspected (e.g., RCVS)

Diagnostic Procedures/Surgery

Lumbar puncture:

- Perform CT 1st if:

- New focal neurologic finding
- Papilledema
- Abnormal mental status
- HIV positive or immunosuppressed
- Detect intracranial and meningeal infections
- Detect blood not evident on CT scan:
 - There is no specific threshold number of red cells below which SAH is excluded – the RBC count is a function of time from onset.
- Opening pressure:
 - Essential to diagnose pseudotumor cerebri and CVST
 - Can distinguish traumatic tap vs. true hemorrhage.
- Xanthochromia:
 - Should be visible by 12 hr after onset of a SAH
 - Visual inspection is the most commonly used method – spectrometry (is more sensitive but has a high false-positive rate).

DIFFERENTIAL DIAGNOSIS

- Note: There can be significant overlap in these groupings.
- Acute single headache:
 - SAH
 - Meningitis
 - Vascular:
 - Acute intracerebral hemorrhage
 - Hypertensive encephalopathy
 - Cranial artery dissection
 - CVST
 - Cerebellar stroke
 - Ocular:
 - Acute narrow-angle glaucoma
 - Pituitary apoplexy
 - Temporal neuritis
 - Traumatic
 - Acute sinusitis
 - Toxic/metabolic:
 - Fever
 - Hypoglycemia
 - High-altitude disease
 - Carbon monoxide poisoning
 - Narcotic, alcohol, or benzodiazepine withdrawal
 - Post lumbar puncture
 - Cold stimulus headache
- Acute recurrent headache:
 - Presenting within days to weeks of onset

- CVST
- Pseudotumor cerebri
- Temporal arteritis
- SAH (rebleed)
- Migraine, cluster, tension
- Hypoxic
- Trigeminal neuralgia
- Postherpetic neuralgia
- Coital and exertional headache
- Subacute headache:
 - Within weeks to months of onset
 - Chronic subdural hematoma
 - Brain tumor
 - Brain abscess
 - Chronic sinusitis
 - Temporomandibular joint syndrome
 - Chronic post-traumatic headache
 - Pseudotumor cerebri (idiopathic intracranial HTN)
 - Temporal arteritis
- Chronic headache:
 - Months to years since onset
 - Chronic tension headache
 - Analgesic abuse/rebound
 - Depression
 - Extracranial:
 - Trigeminal neuralgia: Transient, shock like facial pain
 - Temporal arteritis: Elderly, severe, scalp artery tenderness/swelling
 - Metabolic: Severe anemia
 - Acute glaucoma: Nausea, eye pain, conjunctival injection, increased IOP
 - Cervical: Spondylosis, trauma, arthritis



TREATMENT

INITIAL STABILIZATION/THERAPY

- ABCs if altered mental status
- Empiric antibiotics if bacterial meningitis is suspected, acyclovir if immunocompromised

ED TREATMENT/PROCEDURES

- Migraine (See Headache, Migraine)
- Tension:

- Aspirin
- Acetaminophen
- NSAID
- Nonpharmacologic (meditation, massage, biofeedback)
- Cluster (See Headache, Cluster)
- Temporal arteritis (See Giant Cell Arteritis)
- Intracranial infection (See Meningitis)
- Intracranial hemorrhage (See Subarachnoid Hemorrhage)



FOLLOW-UP

DISPOSITION

Admission Criteria

- Headache secondary to suspected organic disease
- Intractable vomiting and dehydration
- Pain refractory to outpatient management
- Consider ICU admission:
 - Suspected symptomatic aneurysm
 - Acute subdural hematoma
 - SAH
 - Stroke
 - Increased ICP
 - Intracranial infection

Discharge Criteria

- Most migraine, cluster, and tension headaches after pain relief
- Local or minor systemic infections (e.g., URI)

Issues for Referral

Patients with recurrent headaches should have follow-up with a neurologist or PCP.

MEDICATION

- Chlorpromazine: 25–50 mg IM/IV (peds: 0.5–1 mg/kg/dose IM/IV/PO) q4–6h
- Dexamethasone: 10–24 mg IV once
- Dihydroergotamine: 1 mg IM/IV, repeat q1h; max. dose 3 mg
- Ergotamine: 2 mg PO/SL at onset, then 1 mg PO q30min; max. dose 10 mg/wk
- Ketorolac: 30–60 mg IM; 15–30 mg IV once, then 15–30 mg q6h (peds: 1 mg/kg IV q6h)
- Lidocaine 4%: 1 mL intranasal on same side as symptoms
- Metoclopramide: 5–10 mg IM/IV/PO q6–8h
- Morphine: 2.5–20 mg (peds: 0.1–0.2 mg/kg/dose) IM/IV/SQ q2–6h

- Prochlorperazine: 5–10 mg IM/IM/PO TID–QID; max. 40 mg/d
- Sumatriptan: 6 mg SQ, repeat in 1 hr, up to 12 mg/24h

ALERT

DO NOT use the response to any medication to indicate a benign cause of a headache.

PEARLS AND PITFALLS

- The sensitivity for detecting SAH on CT scan falls rapidly after 24 hr. LP remains essential for all patients with suspected SAH presenting after 6 hr of symptom onset.
- Neurology consultation should not delay urgent imaging in patients with high-risk features.
- Use dopamine antagonists with caution in patients with QT prolongation or electrolyte abnormalities. Use ergotamines and triptans carefully in patients with a documented history of CAD.
- Patients with chronic headaches and multiple visits benefit from consistent protocols for pain management; however, be alert to significant changes in their symptoms
- Do not wait for LP results to empirically treat cases of suspected meningitis.

ADDITIONAL READING

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- Perry JJ, Stiell IG, Sivilotti ML, et al. Sensitivity of computed tomography performed within six hours of onset of headache for diagnosis of subarachnoid haemorrhage: Prospective cohort study. *BMJ.* 2011;343:d4277.
- Pope JV, Edlow JA. Avoiding misdiagnosis in patients with neurological emergencies. *Emerg Med Int.* 2012;2012:949275.
- Swadron, SP. Pitfalls in the management of headache in the emergency department. *Emerg Med Clin North Am.* 2010;28(1):127–147.



CODES

ICD9

- 339.00 Cluster headache syndrome, unspecified
- 346.90 Migraine, unspecified, without mention of intractable migraine without mention of status migrainosus
- 784.0 Headache

ICD10

- G43.909 Migraine, unsp, not intractable, without status migrainosus
- G44.009 Cluster headache syndrome, unspecified, not intractable
- R51 Headache

HEADACHE, CLUSTER

Andrew K. Chang

BASICS

DESCRIPTION

- Excruciatingly painful primary headache disorder
 - _ Infrequent cause of ED visits and affects only 0.1% of the population
- Often has abated by time of presentation
 - _ Attacks last between 15 and 180 min (75% last <60 min)
- More common in men (~3:1)
- Onset usually between 30 and 50 yr of age
- Headaches occur in clusters lasting weeks to months followed by remission >1 mo
- Commonly occur 1–3 times per day during cluster period that lasts 2–3 mo
- Often occur during the same time of day
- Often occur during the same time of the year
 - _ Highest incidence in spring and fall
- Chronic cluster headache:
 - _ Remission <1 mo
 - _ Do not experience remission
 - _ 10% of patients
- May have many clinical and pathophysiologic similarities with migraine and variants
- Often follows a trigeminal nerve dermatome

ETIOLOGY

A well-described physiologic reflex arc, the trigeminovascular reflex, potentiates the trigeminal pain and cranial autonomic features of cluster headache by positive feedback mechanisms.

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Unilateral pain (usually does not change sides between headaches)
- Sharp, stabbing, boring
- Acute onset and builds in intensity quickly with climax at 5–15 min
- Pain stops abruptly
- Often exhausted after episode
 - _ Location:

- Eye
- Temple
- Radiation to:
 - Ear
 - Cheek
 - Jaw
 - Teeth (often have had extensive dental workup for pain in the past)
 - Nose
 - Ipsilateral neck
- Episodes are often nocturnal
- Attacks are more likely after ingestion of alcohol, nitroglycerine, or histamine-containing compounds
- More likely in times of stress, prolonged strain, overwork, and upsetting emotional experiences
- No prodrome or aura

Physical-Exam

- Agitated, restless
- Prefer to stand and move around as opposed to migraine patients who prefer to lie quietly in a dark room
- Accompanying autonomic symptoms:
 - Ipsilateral to headache
 - Nasal congestion or rhinorrhea (or both)
 - Conjunctival injection or lacrimation (or both)
 - Facial flushing
 - Eyelid edema
 - Ptosis, miosis, or both (partial Horner's syndrome)
 - Sweating of face/forehead

ESSENTIAL WORKUP

- An accurate history and physical exam should confirm the diagnosis
- Life-threatening alternatives should be ruled out

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Lumbar puncture (if meningitis or subarachnoid hemorrhage is suspected)
- Erythrocyte sedimentation rate (ESR); if temporal arteritis is suspected

Imaging

CT scan/MRI if suspect hemorrhage, tumor, etc.

DIFFERENTIAL DIAGNOSIS

- Migraine headache
- Trigeminal neuralgia
- Meningitis
- Temporal arteritis
- Intracerebral mass lesion
- Herpes zoster
- Intracerebral bleed
- Dental causes
- Orbital/ocular disease (acute glaucoma)
- Temporal mandibular joint syndrome



TREATMENT

PRE HOSPITAL

- Recognize more severe life-threatening causes of headache
- Administration of oxygen by face mask may alleviate symptoms

INITIAL STABILIZATION/THERAPY

- Rule out life-threatening causes of headache
- Administration of supplemental oxygen

MEDICATION

- Ergots: DHE 0.5–1 mg IV; repeat in 1 hr if necessary
- Fentanyl: 2–3 µg/kg IV
- Morphine: 2–4 mg IV or IM, may repeat q10min
- NSAIDs: Ketorolac 15–30 mg IM or IV
- Oxygen: 100% via face mask
- Prochlorperazine: 10 mg IM or IV
- Somatostatin: 100 µg SQ
- Sumatriptan: 6 mg SC, may repeat in 1 hr (max. of 2 doses in 24 hr)
- Verapamil immediate release: Preventive drug of choice. Start at 80mg TID

First Line

- Oxygen: 12 L/min via nonrebreather mask for 15 min:
 - May increase to 15 L/min if refractory headache
- Sumatriptan
- DHE

Second Line

- Narcotics
- Corticosteroids

FOLLOW-UP

DISPOSITION

Admission Criteria

- Persistent headache unresponsive to usual measures
- Unclear headache diagnosis

Discharge Criteria

- Patients with moderate to complete pain relief, a normal neurologic exam, and with a confident diagnosis of cluster headache
- Consider prescribing oxygen and/or SC sumatriptan for management at home

Issues for Referral

Follow-up with a neurologist should be arranged

PEARLS AND PITFALLS

- History is essential to diagnose cluster headache as pain may be improved upon presentation
- 100% oxygen should be the 1st treatment initiated
- Cluster headaches may be so severe that they lead to suicide
 - _ Follow-up is essential to manage clusters which may last months

ADDITIONAL READING

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- Friedman BW, Grosberg BM. Diagnosis and management of the primary headache disorders in the emergency department setting. *Emerg Med Clin North Am*. 2009;27:71–87.
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- Nesbitt AD, Goadsby PJ. Cluster headache. *BJM*. 2012;344:e2407.

See Also (Topic, Algorithm, Electronic Media Element)

- Headache
- Headache, migraine

CODES

ICD9

- 339.00 Cluster headache syndrome, unspecified

- 339.01 Episodic cluster headache
- 339.02 Chronic cluster headache

ICD10

- G44.009 Cluster headache syndrome, unspecified, not intractable
- G44.019 Episodic cluster headache, not intractable
- G44.029 Chronic cluster headache, not intractable

HEADACHE, MIGRAINE

Benjamin W. Friedman



BASICS

DESCRIPTION

- Chronic episodic headache disorder
- Neurovascular pathophysiology:
 - Aberrant trigeminal nerve activation
 - Activation of nociceptive pathways within brainstem
 - Vascular dilation reactive rather than causative
 - No longer considered primarily a vascular headache
 - Disordered sensory processing and autonomic dysfunction
 - Cortical spreading depression underlies aura
- 1 million ED visits per year
- Causes majority of ED headache visits
- 3 × as common in women
- Prevalence peaks in 4th decade of life
- Established criteria for migraine without aura:
 - A. 5 attacks fulfilling criteria B, C, D, E
 - B. Attack lasts 4–72 hr
 - C. Headache has 2 of the following:
 - 1. Unilateral location
 - 2. Pulsating
 - 3. Moderate to severe pain (impairs activities)
 - 4. Aggravation by or avoidance of physical activity
 - D. During headache, nausea, or vomiting and/or photophobia + phonophobia
 - E. Not attributable to other cause
- Migraine with aura:
 - Less common
 - Classically, reversible neurologic symptoms that precede headache
 - Some patients report aura at the same time or after the headache
 - Rarer subtypes of migraine include:
 - Basilar type migraine
 - Dysarthria, vertigo, ataxia, diplopia, or decreased level of consciousness
 - Hemiplegic migraine
 - Full reversible motor weakness
 - Retinal migraine
 - Repeated attacks of monocular visual disturbance

Pediatric Considerations

- More commonly bilateral pain and shorter duration of headache
- Associated symptoms may be difficult to elicit and can be inferred from behavior
- Cyclical vomiting syndrome associated with migraine
- High placebo response

ETIOLOGY

Genetic disorder with variable penetrance, influenced by the environmental factors

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- May be precipitated by chocolate, cheese, nuts, alcohol, sulfites, monosodium glutamate (MSG), stress, or menstruation
- Prodrome precedes migraine by several days
 - May consist of cognitive or emotional alterations, yawning, drowsiness
- Aura precedes migraine by 1 hr
 - Most commonly consists of visual or sensory disturbances
 - Scintillating scotoma
 - Fortification spectra
 - Numbness or tingling
- Headache typically unilateral, throbbing
- Sufficiently intense to impair activity
- Can be bilateral
- Usually associated with osmophobia, photophobia, phonophobia, nausea, or vomiting
- Usually gradual onset
- History often reflects similar headache previously

Physical-Exam

- Allodynia (sensitivity to normally non-noxious stimuli) may be present and signifies more refractory migraine
- Physical exam should otherwise be normal
- Physical exam should include exam of fundi and assessment of visual fields
- Elevated blood pressure does not exclude migraine as diagnosis
- Sinus tenderness does not exclude migraine as diagnosis

ESSENTIAL WORKUP

- An accurate history and physical exam confirm the diagnosis
- Patients with new headache syndrome may require workup including imaging and spinal fluid analysis

DIAGNOSIS TESTS & NTERPRETATION

Lab

Clinical diagnosis: None required

Imaging

Clinical diagnosis: None required

Diagnostic Procedures/Surgery

Clinical diagnosis: None required

DIFFERENTIAL DIAGNOSIS

- Cluster headache
- Medication overuse headache
- Tension-type headache
- Allergic or viral rhinosinusitis
- Idiopathic intracranial hypertension (pseudotumor cerebri)
- Reversible cerebral vasoconstriction syndrome



TREATMENT

PRE HOSPITAL

- Allow patients with migraine headache to be in a calm, dark environment
- Oxygen may be beneficial

INITIAL STABILIZATION/THERAPY

- Exclude secondary causes of headache
- Rapid and effective analgesia

ED TREATMENT/PROCEDURES

- Detailed history will exclude secondary cause of headache in most patients
- Provide analgesia without relying upon opioid analgesics
- IV saline hydration is often helpful
- Provide patient with diagnosis – “You have a migraine”, education about trigger avoidance

Pregnancy Considerations

Metoclopramide, prochlorperazine best treatment options in pregnancy

MEDICATION

- Abortive therapy in ED:
 - Dopamine antagonists:

- Prochlorperazine 10 mg IV coadministered with diphenhydramine 25 mg IV to prevent akathisia
- Droperidol 2.5 mg IV coadministered with diphenhydramine 25 mg IV to prevent akathisia (check EKG prior)
- Metoclopramide 10 mg IV
- Trimethobenzamide 200 mg IM
- Triptans:
 - Sumatriptan: 6 mg SC (avoid if cardiac risk factors)
 - Eletriptan 40 mg PO
- Ergot alkaloids:
 - Dihydroergotamine: 1 mg IV, coadministered with an antiemetic (avoid if cardiac risk factors; avoid if on macrolide or antiretrovirals)
- Nonsteroidals
 - Ketorolac 30 g IV
- Corticosteroids
 - Dexamethasone 10 mg IV or IM
 - Prednisone taper

Treatment strategy

- Abortive therapy with antiemetics, triptans, DHE, or nonsteroidals
- Opioids only if no response to several of the above
- Corticosteroids to avoid post-ED headache recurrence



FOLLOW-UP

DISPOSITION

Admission Criteria

- Persistent severe headache or focal neurologic deficits
- Intractable vomiting, electrolyte imbalance, or inability to take oral food or fluid
- Coexisting medication overuse headache

Discharge Criteria

- Headache relief
- Pathologic cause of headache excluded

Issues for Referral

Chronic migraine or frequent episodic migraine should be referred to a clinician with relevant expertise

FOLLOW-UP RECOMMENDATIONS

- Maintain headache diary to identify and avoid triggers
- Persistent primary care follow-up to identify an effective oral migraine therapeutic

PEARLS AND PITFALLS

- Opioids should not be used as first-line therapy in the ED
- Migraine likely to recur after ED discharge—patients should go home with prescription
- Distinguish between migraine, a chronic, recurrent disorder, and new onset progressive headaches

ADDITIONAL READING

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- Schellenberg ES, Dryden DM, Pasichnyk D, et al. Acute Migraine Treatment in Emergency Settings. *Comparative Effectiveness Review* No. 84. AHRQ Publication No. 12 (13)-EHC142-EF. Rockville, MD: Agency for Healthcare Research and Quality. November 2012. www.Effectivehealthcare.gov/reports/final.cfm.



CODES

ICD9

- 346.00 Migraine with aura, without mention of intractable migraine without mention of status migrainosus
- 346.10 Migraine without aura, without mention of intractable migraine without mention of status migrainosus
- 346.90 Migraine, unspecified, without mention of intractable migraine without mention of status migrainosus

ICD10

- G43.009 Migraine w/o aura, not intractable, w/o status migrainosus
- G43.109 Migraine with aura, not intractable, w/o status migrainosus
- G43.909 Migraine, unsp, not intractable, without status migrainosus

HEART MURMUR

Elizabeth M. Foley • Leon D. Sánchez



BASICS

DESCRIPTION

- Sounds created by physiologic processes or functional and structural anomalies of the heart.
- Stenotic lesions:
 - Pressure overload in the chamber preceding the valve, leading to hypertrophy of the chamber in an attempt to overcome the increased resistance
- Regurgitant lesions:
 - Volume overload of the chamber preceding the valve, leading to chamber dilatation in an attempt to accommodate the regurgitant blood volume
- Genetic abnormalities:
 - Congenital defects associated with abnormal cardiac blood flow

ETIOLOGY

- Aortic stenosis:
 - Rheumatic heart disease
 - Congenital bicuspid valve
 - Calcification
 - Prosthetic valve
- Aortic regurgitation:
 - Rheumatic heart disease
 - Endocarditis
 - Aortic dissection
 - Prosthetic valve
- Mitral stenosis:
 - Rheumatic heart disease
 - Rheumatologic disorders (systemic lupus erythematosus)
 - Calcification
 - Cardiac tumors (atrial myxoma)
 - Congenital
 - Prosthetic valve
- Mitral regurgitation, acute:
 - Endocarditis
 - Papillary muscle rupture or dysfunction
 - Rupture of chordae tendineae
 - Prosthetic valve

- Mitral regurgitation, chronic:
 - Rheumatic heart disease
 - Mitral valve prolapse
 - Connective tissue disease (Marfan syndrome)
- Mitral valve prolapse:
 - Congenital
 - Connective tissue disease
- Tricuspid stenosis:
 - Rheumatic heart disease
- Tricuspid regurgitation:
 - Rheumatic heart disease
 - Endocarditis
 - Pulmonary HTN
- Pericardial friction rub:
 - Pericarditis
 - Pericardial effusion
- Ventricular septal defect:
 - Congenital
 - Traumatic
 - Postinfarction
- Ventricular assist device
 - Implantable pump supplements or replaces ventricular function

Pediatric Considerations

- Pulmonic stenosis:
 - Congenital
 - Maternal–fetal rubella exposure
 - Rheumatic heart disease
- Pulmonic regurgitation:
 - Congenital
 - Rheumatic heart disease
 - Pulmonary HTN
- Atrial septal defect:
 - Congenital
- Patent ductus arteriosus:
 - Congenital
 - Prematurity
 - Maternal–fetal rubella exposure
- Coarctation of the aorta:
 - Congenital
 - Turner syndrome
- Hypertrophic cardiomyopathy/idiopathic hypertrophic subaortic stenosis:
 - Congenital

- Genetic predisposition



DIAGNOSIS

SIGNS AND SYMPTOMS

- Aortic stenosis:
 - Systolic crescendo–decrescendo murmur radiating to carotids
 - Carotid pulse described as parvus et tardus: Diminished intensity and late upstroke
 - Angina
 - Dyspnea on exertion
 - Exertional syncope
- Aortic regurgitation:
 - Diastolic blowing murmur at left sternal border
 - Pulmonary edema
 - Dyspnea
 - Tachycardia
 - Chest pain
 - Widened pulse pressure
 - Austin Flint murmur: Diastolic rumble from exposure of mitral valve to regurgitant flow
 - Corrigan pulse or water hammer pulse: Rapid upstroke and downstroke of the carotid pulse
 - Quincke pulse: Pulsations seen at nail beds
 - de Musset sign: Head bobbing with carotid pulse
- Mitral stenosis:
 - Diastolic, rumbling murmur at apex
 - Loud S₁ with opening snap
 - Dyspnea
 - Orthopnea
 - Hemoptysis
 - Pulmonary edema
 - Emboli to systemic circulation
 - Atrial fibrillation
- Mitral regurgitation, acute:
 - Systolic, harsh, crescendo–decrescendo murmur at apex
 - Pulmonary edema
- Mitral regurgitation, chronic:
 - Holosystolic murmur at apex radiating to axilla
 - Dyspnea on exertion
 - Fatigue
 - Atrial fibrillation

- Mitral valve prolapse:
 - Early to mid-systolic click often followed by systolic murmur
 - Palpitations
 - Chest pain
- Tricuspid stenosis:
 - Diastolic, high-pitched murmur
 - Peripheral edema
 - Hepatosplenomegaly
 - Ascites
 - Fatigue
 - Atrial fibrillation
 - Large A wave in the jugular venous pulse
- Tricuspid regurgitation:
 - Holosystolic, blowing murmur along left sternal border
 - Peripheral edema
 - Hepatosplenomegaly
 - Ascites
 - Atrial fibrillation
 - Large V wave in the jugular venous pulse
- Patent ductus arteriosus:
 - Continuous machinery murmur
 - CHF
- Pericardial friction rub:
 - Intermittent murmur
 - Systolic and/or diastolic component
- Ventricular septal defect:
 - Harsh, holosystolic murmur loudest along lower left sternal border
- Ventricular assist device:
 - Mechanical hum at apex
 - Continuous or pulsatile
 - May have adequate perfusion without palpable pulse or measurable BP

Pediatric Considerations

- Pulmonic stenosis:
 - Systolic crescendo–decrescendo ejection murmur at left upper sternal border
 - Severe lesions may have a thrill
 - Widely split S₂
 - Dyspnea with exertion in serious cases
 - May have signs of right heart failure
- Pulmonic regurgitation:
 - High-pitched, early, decrescendo diastolic murmur
 - Widely split S₂

- Associated with Graham Steell murmur of pulmonary HTN (high-pitched early diastolic murmur)
- May have signs of right heart failure
- Atrial septal defect:
 - Systolic ejection murmur in secundum defect
 - Secundum associated with pulmonary HTN
 - Wide fixed S₂
 - No murmur in PFO
- Patent ductus arteriosus:
 - Continuous machinery murmur at upper left sternal border with systolic thrill
 - Cyanosis
 - Bounding peripheral pulses
 - Tachypnea
- Coarctation of the aorta:
 - Continuous or late systolic murmur
 - Possible aortic click related to bicuspid valve
 - Difference between upper and lower extremity pulses
- Hypertrophic cardiomyopathy/idiopathic hypertrophic subaortic stenosis:
 - Systolic, harsh, crescendo–decrescendo murmur at left sternal border
 - Increased intensity with Valsalva
 - Dyspnea
 - Chest pain
 - Exertional syncope
 - Sudden death

Physical-Exam

- Auscultation of heart and lung sounds
- Evaluation of pulses, peripheral perfusion, and edema

ESSENTIAL WORKUP

For more details, see Valvular Heart Disease, Mitral Valve Prolapse, Congenital Heart Disease, Patent Ductus Arteriosus, Pericarditis, and Pericardial Effusion/Tamponade.

DIAGNOSIS TESTS & INTERPRETATION

Imaging

- EKG
- CXR
- Echo:
 - Evaluate valves, chambers, flow
- CT:

- Rule out aortic dissection

Diagnostic Procedures/Surgery

Acute regurgitant lesions: Cardiac catheterization

DIFFERENTIAL DIAGNOSIS

See Etiology.



TREATMENT

PRE HOSPITAL

- IV fluids:
 - Patients with critical aortic stenosis are very sensitive to fluid shifts.
- Oxygen as appropriate

INITIAL STABILIZATION/THERAPY

- Oxygen
- IV access
- Cardiac monitor
- Treat symptoms (CHF, dysrhythmias)
- Exercise care with fluids and medications in aortic stenosis

ED TREATMENT/PROCEDURES

For more details, see Endocarditis, Valvular Heart Disease, Mitral Valve Prolapse, Congenital Heart Disease, Patent Ductus Arteriosus, Pericarditis, and Pericardial Effusion/Tamponade.

MEDICATION

- Digoxin: 0.5 mg IV, then 0.25 mg IV 6 and 12 hr later
- Diltiazem (Cardizem):
 - 0.25 mg/kg (17.5 mg for 70 kg person) IV over 2 min
 - May reboles after 15 min with 0.35 mg/kg IV
 - Start drip at 5–15 mg/hr
- Furosemide (Lasix):
 - 20–80 mg IV; may increase dose if necessary
 - Max. of 600 mg per 24 hr
- Heparin:
 - 80 U/kg bolus IV, then drip at 18 U/kg/hr
 - Monitor partial thromboplastin time
- Metoprolol (Lopressor): 5 mg IV q5–15 min for 3 doses, as tolerated
- Nitroglycerin:
 - 10–20 µg/min IV
 - Titrate to effect

- Max. 300 µg/min
- Nitroprusside:
 - 0.3 µg/kg/min IV
 - Titrate to effect
 - Max. 10 µg/kg/min
 - Protect bag from light
 - Thiocyanate toxicity from prolonged use
- Propranolol (Inderal):
 - 1--3 mg IV q2--5min up to max 5 mg
 - No additional doses for 4 hrs after reaching max dose or desired response



FOLLOW-UP

DISPOSITION

Admission Criteria

- Signs of cardiac ischemia
- Syncope or near syncope
- Pulmonary edema
- Hemodynamic instability
- Endocarditis
- Arrhythmia

Discharge Criteria

- Asymptomatic
- Hemodynamically stable

Issues for Referral

Patients with new murmurs should be referred to their caregiver or a cardiologist.

FOLLOW-UP RECOMMENDATIONS

- Patients should always inform their medical and dental caregivers that they have a heart murmur.
- To avoid an infection of the lining of the heart, antibiotics may be needed before procedures such as teeth cleaning.

PEARLS AND PITFALLS

Patients with new heart murmurs and fever need to be assessed for endocarditis.

ADDITIONAL READING

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management of patients with valvular heart disease. *Circulation*. 2006;114:e84–e231.

- Frank JE, Jacobe KM. Evaluation and management of heart murmurs in children. *Am Fam Physician*. 2011;84:793–800.
- Nishimura RA, Carabello BA, Faxon DP, et al. ACC/AHA 2008 Guideline update on valvular heart disease: Focused update on infective endocarditis. *Circulation*. 2008;118:887–896.
- www.blaufuss.org/tutorial/
- www.easyauscultation.com/heart-sounds.aspx



CODES

ICD9

- 394.0 Mitral stenosis
- 424.1 Aortic valve disorders
- 785.2 Undiagnosed cardiac murmurs

ICD10

- I05.0 Rheumatic mitral stenosis
- I35.0 Nonrheumatic aortic (valve) stenosis
- R01.1 Cardiac murmur, unspecified

HELLP SYNDROME

Michael J. Bono



BASICS

DESCRIPTION

- HELLP syndrome: *Hemolysis Elevated Liver enzymes, Low Platelets*
- Continuum with severe preeclampsia as most patients will be hypertensive
- Liver involvement is hallmark:
 - Other organs may be involved (e.g., brain, kidneys, lungs)
- HELLP syndrome divided into 3 groups, representing severity of the disease; severity is directly related to the platelet count:
 - Class 1: Most severe form; platelet nadir $< 50,000$ platelets/ μL
 - Class 2: Less severe; platelet nadir between 50,000 and 100,000 platelets/ μL
 - Class 3: Least severe; platelet nadir between 100,000 and 150,000 platelets/ μL
- Most maternal deaths occur with class 1
- Increased mortality rate is associated with hepatic hemorrhage or CNS or vascular insult to the cardiopulmonary or renal systems
- Incidence: 0.2% of all pregnancies
- 12–18% have normal BP
- Occurs in 20% of pregnancies with severe preeclampsia or eclampsia
- At diagnosis:
 - 52% preterm
 - 18% term
 - 32% postpartum

RISK FACTORS

Frequently white, multiparous, older

Pediatric Considerations

Infant mortality is greater in women with HELLP

ETIOLOGY

- Unclear, but vasospasm is the basis:
 - Fetal-placental debris is released into maternal circulation, causing systemic inflammatory response
 - Vascular constriction causes resistance to blood flow and HTN
 - Vasospasm probably damages vessels directly
 - Angiotensin II causes endothelial cells to contract
 - Endothelial cell is damaged and interendothelial cell leaks are the result

- Small-vessel leaks:
 - Platelets and fibrinogen get deposited in the subendothelium
 - Fibrin deposition develops in severe cases
- Vascular changes and local tissue hypoxia lead to hemorrhage, necrosis, and end-organ damage

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- History and physical exam with attention to symptoms of abdominal pain, nausea, vomiting, and headache
- Obstetric history:
 - Parity
 - Deliveries
 - History of hypertensive disorder during pregnancy
 - Estimated gestational age
 - Prenatal care
- May present with flulike symptoms, such as fatigue or malaise
- Nausea, usually with vomiting
- Right upper quadrant or epigastric pain:
 - Pain increases with severity of disease
- Headache, often with visual changes
- Symptoms which carry higher morbidity:
 - Dyspnea and/or fluid overload to suggest cardiogenic/noncardiogenic pulmonary edema
 - Dyspnea associated with pulmonary embolus
 - Chest pain suggestive of myocardial ischemia
 - Altered mental status, seizures or focal neurologic deficit:
 - Hypertensive encephalopathy
 - Cerebral edema
 - Hemorrhagic cerebrovascular accident
 - Peripheral edema
 - Ascites
 - Hematuria
 - Low urine output

ALERT

Determination of gestational age and fetal viability is critical in HELLP.

Physical-Exam

- Vital signs with attention to BP
- May not have systolic or diastolic HTN
- Many patients will have right upper quadrant pain, concern for liver subcapsular hematoma
- Evidence of fluid overload
- Careful neurologic exam
- Fetal heart tones

ESSENTIAL WORKUP

- Immediate CBC with platelet count and smear, BUN, creatinine, LFTs, coagulation profile, and magnesium level
- Urinalysis for protein; screen for UTI
- Weigh patient to determine recent weight gain

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC:
 - Anemia
 - Thrombocytopenia
 - Peripheral smear demonstrates microangiopathic hemolytic anemia (burr cells or schistocytes)
 - Other hemolysis markers are elevated lactate dehydrogenase (LDH) levels, increased reticulocyte count, and elevated bilirubin levels
- Platelet count and smear:
 - $< 100,000$ platelets/ μL
- Disseminated intravascular coagulation screen
- Coagulation profile:
 - PT
 - PTT
- BUN, creatinine, and magnesium levels
- LFTs to assess hemolysis markers and hepatic dysfunction:
 - Elevated aspartate aminotransferase level: > 40 IU/L
 - Elevated alanine aminotransferase level: > 40 IU/L
 - Elevated LDH: > 600 IU/L
 - Elevated serum bilirubin: > 1.2 mg/dL

Imaging

- CXR:
 - Suspected pulmonary edema
- CT of head:
 - Mental status changes or focal neurologic deficit
- US of the pelvis (transabdominal or transvaginal):

- Image fetus and placenta

DIFFERENTIAL DIAGNOSIS

- GI:
 - Cholecystitis
 - Cholelithiasis
 - Biliary colic
 - Pancreatitis
 - Hepatitis
 - Ulcer disease
 - Acute fatty liver of pregnancy
 - Acute gastritis
 - Hiatal hernia
 - Severe gastroesophageal reflux
- Hematologic:
 - Preeclampsia-associated thrombocytopenia
 - Gestational thrombocytopenia
 - Idiopathic thrombocytopenic purpura
 - Thrombotic thrombocytopenic purpura
 - Hemolytic uremic syndrome
- Neurologic:
 - Epilepsy
 - Encephalitis
 - Meningitis
 - Encephalopathy
 - Brain tumor
 - Intracranial hemorrhage
- Other:
 - Drug abuse
 - Pyelonephritis
 - Sepsis



TREATMENT

PRE HOSPITAL

Cautions:

- Transport patient in left lateral decubitus position to prevent inferior vena cava syndrome
- Venous access for anticipated seizure activity
- Routine seizure management (preferably with magnesium sulfate) if the patient seizes

ALERT

Transport to a facility capable of providing high-risk obstetric care.

INITIAL STABILIZATION/THERAPY

- ABC management
- Left lateral decubitus position to prevent inferior vena cava syndrome
- High-flow oxygen via face mask
- Maternal monitoring:
 - Cardiac
 - Pulse oximetry
 - Tocography
- Fetal monitoring

ED TREATMENT/PROCEDURES

- Control HTN with antihypertensives (see Medication):
 - Avoid ACE inhibitors because of fetal side effects
- Heparin should be avoided because of bleeding complications
- Treat preeclampsia or eclampsia with IV magnesium sulfate:
 - Magnesium sulfate is not given to treat HTN
- Order type and screen for possible transfusion
- Call for emergent obstetric consult, consider neonatology consult:
 - Consider emergent delivery
 - Early plasma exchange therapy has shown promise in postpartum patients with severe disease
- Discuss administration of glucocorticoid with consultant:
 - Helps fetal lung maturity
 - IV dexamethasone more effective than IM betamethasone
 - Depends on gestational age of fetus
 - Does not reduce disease severity or duration, but improves platelet counts
- Limit IV fluid administration unless clinical evidence of dehydration:
 - Excess fluids promote further capillary leak
 - Lactated Ringers or NS at 60 mL/hr (no more than 125 mL/hr)
 - Monitor urine output with Foley catheter
- Correct thrombocytopenia by platelet transfusion in women with platelet counts $< 20,000$ platelets/ μL , even without active bleeding, as risk of postpartum bleeding is significantly increased
- Platelet counts $> 40,000$ platelets/ μL are safe for vaginal delivery
- Correct thrombocytopenia to platelet counts $> 50,000$ platelets/ μL if cesarean delivery planned
- If coagulation dysfunction is present, transfusion with fresh frozen plasma and packed RBCs in consultation with obstetrics
- Transfusion with packed RBCs for hemoglobin < 10 g/dL

MEDICATION

First Line

- Hydralazine: 2.5 mg IV, then 5–10 mg q15–20min:
 - Up to 40 mg total dose, to keep diastolic BP < 110 mm Hg
 - IV drip 5–10 mg/hr titrated
- Labetalol: 10 mg IV, then 20–80 mg IV q10min:
 - Up to 300 mg total dose
 - IV drip 1–2 mg/min titrated

Second Line

- Nitroprusside: 0.25 µg/kg/min as a drip:
 - Increase 0.25 µg/kg/min q5min
 - Use only if no response to hydralazine or labetalol
- Magnesium sulfate: 4–6 g in 100 mL IV over 15–20 min as loading dose:
 - Maintenance drip starting at 2 g/hr
 - Titrate to clinical effect
 - Watch for toxicity (antidote is calcium gluconate 10%, 10 mL IV over 3 min).
 - Measure magnesium sulfate level at 4–6 hr; adjust drip to achieve levels between 4 and 7 mEq/L.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Admit all patients to obstetric service for continuous monitoring of mother and fetus
- ICU admission:
 - Pulmonary edema
 - Respiratory failure
 - Cerebral edema
 - GI bleeding with hemodynamic instability

Discharge Criteria

Patients with HELLP syndrome should always be admitted. Discharge should be a decision of the OB Consultant

Issues for Referral

After stabilization in the ED, transfer to facility capable of managing high-risk obstetric conditions unless delivery is imminent.

FOLLOW-UP RECOMMENDATIONS

Patients should be followed closely by OB:

- May develop HELLP after delivery, usually within 48 hr

PEARLS AND PITFALLS

- Hypertensive pregnant women with abdominal pain, elevated LFTs, and decreased platelets need emergent treatment and OB consultation
- Patients with HELLP syndrome may have a normal BP
- Transport to a facility capable of caring for these patients after stabilization is essential

ADDITIONAL READING

- Ciantar E, Walker JJ. Pre-eclampsia, severe pre-eclampsia and hemolysis, elevated liver enzymes and low platelets syndrome: What is new? *Women's Health*. 2011;7(5):555–569.
- Deak TM, Moskovitz JB. Hypertension and pregnancy. *Emerg Med Clin North Am*. 2012;30:903–917.
- Giannubilo SR, Bezzeccheri V, Cecchi S, et al. Nifedipine versus labetalol in the treatment of hypertensive disorders of pregnancy. *Arch Gynecol Obstet*. 2012;286:637–642.
- Woudstra DM, Chandra S, Hofmeyr GJ, et al. Corticosteroids for HELLP (hemolysis, elevated liver enzymes, low platelets) syndrome in pregnancy (Review). *Cochrane Database Syst Rev*. 2010; (9):CD008148.
- Yoder SR, Thornburg LL, Bisognano JD. Hypertension in pregnancy and women of childbearing age. *Am J Med*. 2009;122:890–895.



See Also (Topic, Algorithm, Electronic Media Element)

Preeclampsia



CODES

ICD9

- 642.50 Severe pre-eclampsia, unspecified as to episode of care
- 642.51 Severe pre-eclampsia, delivered, with or without mention of antepartum condition
- 642.52 Severe pre-eclampsia, delivered, with mention of postpartum complication

ICD10

- O14.20 HELLP syndrome (HELLP), unspecified trimester
- O14.22 HELLP syndrome (HELLP), second trimester

- O14.23 HELLP syndrome (HELLP), third trimester

HEMATURIA/PROTEINURIA

Andrew Rinne • Edward Ullman



BASICS

DESCRIPTION

- Microscopic hematuria: ≥ 3 red blood cells per high-power field in 2 of the 3 properly collected urine specimens
- Gross hematuria: Visible blood in properly collected urine specimen
- Proteinuria: Urinary protein excretion of > 150 mg/day
- Risk factors for disease in asymptomatic proteinuria:
 - Diabetes
 - HTN
 - NSAID abuse
 - Heroin use
- Risk factors for disease in asymptomatic hematuria:
 - Tobacco use
 - Occupational exposure to benzenes, aromatic amines, and dyes
 - History of gross hematuria
 - Age > 40 yr old
 - History of urologic disorder or disease
 - History of painful voiding
 - History of UTI
 - Analgesic abuse
 - History of pelvic irradiation

ETIOLOGY

- Proteinuria:
 - Glomerular:
 - Nephritic (postinfectious, IgA, lupus, vasculitis)
 - Nephrotic (minimal change, diabetes, preeclampsia)
 - Tubular
 - Overflow (hemolysis, rhabdomyolysis, multiple myeloma)
- Hematuria:
 - UTI
 - Stones (renal, bladder)
 - BPH
 - Cancer (bladder, renal, prostate)
 - Transient unexplained
 - Acute glomerulonephritis

SIGNS AND SYMPTOMS

- Dysuria
- Blood in urine
- Fever
- Flank pain
- Flank ecchymosis
- Initial hematuria (anterior urethral lesion)
- Terminal hematuria (posterior urethra, bladder, neck, trigone)
- Cyclic hematuria (endometriosis or urinary tract)
- Previous upper respiratory tract infection (10–21 days prior)
- Previous skin infection (10–21 days prior)
- Deafness (Alport syndrome)
- Peripheral edema
- CHF
- Hemoptysis (Goodpasture disease)
- Concurrent menstruation
- Testicular, epididymal, and prostatic tenderness or trauma
- Terminal urethral lesion
- Enlarged prostate
- Penile/scrotal hematoma
- Atrial fibrillation:
 - Renal artery embolus or thrombus
- Organomegaly, flank mass
- Pregnancy consideration
- Headache
- HTN (> 140/90 mm Hg)
- Right upper quadrant pain

History

- Characteristics of complaint (onset, duration)
- Associated symptoms (recent illness)
- Past medical history (DM, HTN, pregnancy)
- Medications (nephrotoxic, anticoagulation, antibiotics)

Physical-Exam

- Complete physical exam, special attention to:
 - Edema, including periorbital
 - Thorough GU exam, including prostate
 - Rashes
 - Flank (ecchymosis, tenderness)

ESSENTIAL WORKUP

- Urine dipstick
- Urinalysis with microscopic analysis
- Consider urine culture.
- BUN level
- Serum creatinine level
- CBC
- Pregnancy consideration
- Liver function test
- Platelet count
- Consider coagulation panel.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Urine:
 - Culture
 - Cytology
 - 24 hr urine protein and creatinine levels
 - Spot ratio of urine protein to creatinine
 - Spot ratio of urine protein to osmolality
 - Protein electrophoresis
- Serum:
 - Coagulation studies
 - Protein electrophoresis

Imaging

- Helical CT scan
- Renal US

Diagnostic Procedures/Surgery

- Cystourethroscopy
- Urethrogram
- Cystogram
- Retrograde pyelogram
- IV pyelogram

DIFFERENTIAL DIAGNOSIS

- Glomerular hematuria:
 - IgA nephropathy (Berger disease)
 - Postinfectious glomerulonephritis
 - Membranoproliferative glomerulonephritis
 - Focal glomerular sclerosis

- Lupus nephritis
- Wegener granulomatosis
- Polyarteritis nodosa
- Henoch–Schönlein syndrome
- Thrombotic thrombocytopenic purpura:
 - Hemolytic uremic syndrome
 - Alport syndrome
 - Goodpasture disease
- Nonglomerular hematuria:
 - Infection (pyelonephritis, tuberculosis, schistosomiasis)
 - Inflammation (drug induced, radiation induced)
 - Urothelial malignancy
 - Renal and extrarenal tumor
 - Interstitial nephritis
 - Papillary necrosis
 - Polycystic kidney disease
 - Medullary sponge disease
 - Renal artery embolism/thrombosis
 - Renal vein thrombosis
 - Sickle cell disease
 - Malignant HTN
 - Hypercalciuria
 - Hyperuricosuria
 - Urolithiasis
 - Strictures
 - Endometriosis
 - Foreign bodies
 - Benign prostatic hypertrophy
 - Coagulopathy/bleeding disorders
 - Trauma (renal pedicle injuries, urethral disruptions, bladder rupture)
 - Recent instrumentation
 - Frequent or interrupted coitus
 - Factitious
- Glomerular proteinuria (> 2 g/day):
 - Minimal-change disease
 - Membranous glomerulonephritis
 - Focal segmental glomerulonephritis
 - Membranoproliferative glomerulonephritis
 - DM
 - Collagen vascular diseases
 - Amyloidosis
 - Preeclampsia
 - Infection (HIV, hepatitis B, hepatitis C, poststreptococcal infection, syphilis)

- Lymphoma
- Chronic renal transplant rejection
- Heroin use
- Penicillamine
- Tubular proteinuria:
 - Hypertensive nephrosclerosis
 - Uric acid nephropathy
 - Acute hypersensitivity interstitial nephritis
 - Fanconi syndrome
 - Sickle cell disease
- Overflow proteinuria:
 - Monoclonal gammopathy
 - Leukemia
- Proteinuria, other:
 - Dehydration
 - Stress
 - Fever
 - Heat injury
 - Inflammatory process
 - Orthostatic proteinuria



TREATMENT

PRE HOSPITAL

- Airway, breathing, and circulation management
- Control other trauma, if present.

INITIAL STABILIZATION/THERAPY

- Airway, breathing, and circulation management
- Treat hemodynamically unstable injuries 1st, if present.
- Obtain initial labs (urinalysis with microscopic analysis, BUN, serum creatinine, electrolytes).

Pregnancy Considerations

If considering preeclampsia:

- Aggressive BP control
- Magnesium if indicated
- Prompt OB/GYN consultation

ED TREATMENT/PROCEDURES

- Uncomplicated UTIs:
 - Antibiotics (see Urinary Tract Infection, Adult or Urinary Tract Infection,

Pediatric)

- Pyelonephritis:
 - Antibiotics (see Urinary Tract Infection, Adult or Urinary Tract Infection, Pediatric)
 - Analgesics
 - Antipyretics
- Rapidly progressing glomerulonephropathy:
 - Steroid therapy
 - Nephrology consultation
- Acute renal failure:
 - Hemodialysis
 - Renal US
 - Urine electrolytes
 - Nephrology consultation
- Renal colic:
 - IV fluids
 - Analgesics
 - If initial presentation, noncontrast helical CT scan
- Gross hematuria:
 - Insertion of 3-way Foley catheter with bladder irrigation to clear blood clots that may cause urinary retention from bladder obstruction

MEDICATION

- Antibiotic recommendations for UTI are discussed in the relevant chapters.
- Steroids for rapidly progressing glomerulonephropathy should be discussed with a nephrologist.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Acute renal failure:
- Azotemia/uremia/hyperkalemia
- Hemodynamic instability
- Hematuria with traumatic injuries
- Obstructing ureteral stones with evidence of systemic infection or renal failure
- Hypertensive emergency
- Oliguria/anuria
- Pregnant with preeclampsia, pyelonephritis, obstructing nephrolithiasis
- Intractable pain
- Intolerance of PO fluids and medications

Discharge Criteria

- Hemodynamically stable without life-threatening issues
- Infected ureteral stones without renal failure or obstruction
- Mild hematuria or proteinuria without renal failure
- Hematuria:
 - Gross hematuria, except in young women with proven UTIs, needs urology follow-up.
 - Microscopic hematuria needs repeated U/As and PCP follow-up, and may need urology/nephrology in the future.
 - Pregnant patients with possible infections should have close follow-up to ensure treatment success.
- Proteinuria:
 - Mild cases should be referred to their primary care physician for further workup in an outpatient setting.
 - Nephrotic-range proteinuria, as well as proteinuria with renal failure and no alternative explanation, should be referred to a nephrologist promptly.

PEARLS AND PITFALLS

- Missing acute glomerulonephritis in children, either by misdiagnosing as a UTI based on the U/A, or by failing to consider it in the differential is a pitfall.
- Spot urine protein/creatinine ratio correlates well with 24 hr urine protein (i.e., ratio of 3.5 is roughly equivalent to 24 hr protein excretion of 3.5 g)
- Periorbital edema can be a sign of nephritic syndrome, not just allergic reaction.
- Failing to ensure follow-up for asymptomatic hematuria, especially in patients > 40 yr is a pitfall.

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See Also (Topic, Algorithm, Electronic Media Element)

- Urinary Tract Infection, Adult
- Urinary Tract Infection, Pediatric
- Preeclampsia/Eclampsia
- Renal Failure

- Renal Calculus



CODES

ICD9

- 599.70 Hematuria, unspecified
- 791.0 Proteinuria
- 599.71 Gross hematuria

ICD10

- R31.9 Hematuria, unspecified
- R80.9 Proteinuria, unspecified
- R31.0 Gross hematuria

HEMOPHILIA

Steven H. Bowman • Christina H. Georgopoulos

BASICS

DESCRIPTION

- Caused by deficiency of functional factor VIII or factor IX
- Lack of functional factor causes partial inactivation of coagulation cascade and impaired hemostasis.
- 2 types:
 - Hemophilia A: Factor VIII deficiency
 - Hemophilia B (Christmas disease): Factor IX deficiency
- Symptoms dependent on factor activity:
 - 5–30% factor activity (mild hemophilia):
 - Bleeding with major trauma or surgery
 - 1–5% factor activity (moderate hemophilia):
 - Bleeding secondary to trauma/surgery
 - Occasional spontaneous hemarthroses (< 1 time per month)
 - < 1% factor activity (severe):
 - Spontaneous bleeding from infancy
 - May bleed as often as 1–2 times per week, often requiring factor replacement
- Complications:
 - Death from hemorrhage
 - Recurrent joint bleeding leads to joint destruction and loss of function
 - Transfusion-transmitted infections (risk reduced with purification of concentrates)
 - Development of inhibitors (IgG antibodies to factors), which prevent hemostasis

ETIOLOGY

Genetics

- X-linked recessive, though 1/3 have a spontaneous mutation
- Rare disease:
 - Hemophilia A: 1 in 5–7,000 males
 - Hemophilia B: 1 in 30,000 males
- Prevalence of inhibitors: 20% of severe hemophilia A and < 5% of severe hemophilia B

DIAGNOSIS

SIGNS AND SYMPTOMS

- **Bleeding:**
 - Hemarthrosis (most common):
 - Knee (most common) > elbow > ankle > shoulder > wrist
 - Muscle hemorrhage
 - Bleeding from soft tissue lacerations
 - Postextraction or oral mucosal bleeding
 - Epistaxis (only in severe disease)
 - Hematuria
 - Intracranial hemorrhage
 - GI bleeding
 - Pseudotumors (blood cysts)

ESSENTIAL WORKUP

Thorough history and physical exam

DIAGNOSIS TESTS & INTERPRETATION

Lab

- PLT count: Normal
- Bleeding time: Normal
- PT: Normal
- PTT: Increased
- Urinalysis: Asymptomatic hematuria (often)
- Specific factor assays:
 - Factor VIII:Ag (measures quantity): Decreased
 - Factor VIII:c (measures activity): Decreased
 - vWF:Ag and vWF: Normal

Imaging

Radiographic studies may be required in certain circumstances:

- Head CT to evaluate for intracranial bleed
- Renal US/cystoscopy to evaluate excessive hematuria or renal trauma
- Abdominal CT to evaluate for retroperitoneal bleeding

DIFFERENTIAL DIAGNOSIS

- Von Willebrand disease
- Anticoagulant drugs
- Antiplatelet agents
- Thrombocytopenia
- Hepatic dysfunction



TREATMENT

INITIAL STABILIZATION/THERAPY

- Control bleeding proximally.
- Establish IV access, draw type and screen.
- Consider PRBCs for transfusion.

ED TREATMENT/PROCEDURES

General

- Patients generally know their doses, type of factor, and whether or not they have inhibitors. Have low threshold for factor replacement when they present with any symptom.
- Coordinate ED care with patient's hematologist.
- Start replacing factors immediately, even before imaging/consults.

Approach to Factor Replacement

- 1 U in 1 mL of normal plasma is considered 100% clotting factor activity.
- People without hemophilia have factor levels between 60% and 150%.
- Step 1: Determine % activity desired based on location/system involved in bleeding:
 - Low to moderate bleeding – want 30–50% activity
 - Soft tissue injury/lacerations
 - Joint or muscle bleeding (except iliopsoas)
 - Moderate to severe bleeding – want 50–100% activity
 - GI or GU bleeding
 - Major muscle bleeds (iliopsoas)
 - Severe to life-threatening bleeding – want 100% activity
 - CNS injury/intracranial bleed
 - Major bleeding from trauma or postsurgery
 - Intra-abdominal/retroperitoneal bleeding
 - Throat/neck bleeds compromising airway
- Step 2: Calculate factor dose for level of activity desired.
 - Factor VIII required (in U) = $\text{wt (kg)} \times 0.5 \times (\% \text{ factor activity desired})$. 1 IU/kg raises activity by 2%. Give every 12 hr for 1–2 days.
 - Factor IX required (in U) = $\text{wt (kg)} \times 1 \times (\% \text{ factor activity desired})$. 1 IU/kg raises activity by 1%. Give every 24 hr for 1–2 days.
 - Factor dose given IV push over 1–2 min

Specific Management Considerations

- Hemarthrosis:
 - Splint, ace, ice

- Arthrocentesis is rarely indicated
- Muscle hemorrhage:
 - Forearm/calf—consider compartment syndrome
 - Psoas hematoma—groin pain, femoral nerve paresthesias
- Post tooth extraction or oral mucosal bleeding:
 - Treat locally with collagen sponge (Avitene) or other hemostatic agent.
 - Replace factor if severe.
 - Aminocaproic acid (Amicar) or tranexamic acid (Cyklokapron) may be useful.
- Hematuria (generally mild):
 - Hydrate
 - Avoid Amicar and cryoprecipitate.
- Intracranial hemorrhage:
 - All head injuries should be considered significant, especially in children.
 - Do not delay therapy for CT head.
- GI bleeding:
 - Secondary to ulcers, polyps, hemorrhoids
 - Replace factor prior to endoscopy.

MEDICATION

To calculate doses for recombinant and plasma-derived factor VIII and factor IX concentrates refer to section Approach to Factor Replacement above.

Patient without Inhibitors

- **First line: Recombinant factor**
 - Recombinant factor VIII options: Recombinate, Kogenate, Advate, Helixate FS, Xyntha
 - Recombinant factor IX options: BeneFIX
- **Second line: Plasma-derived factor**
 - Plasma-derived factor VIII high-purity concentrates: Monoclote-P, Hemofil-M, Koate DVI, Alphanate
 - Plasma-derived factor VIII intermediate-purity concentrates: Humate
 - Only use when previous options are not available.
 - Plasma-derived factor IX high-purity concentrates: AlphaNine, Mononine
- **Other options**
 - Cryoprecipitate (useful only in hemophilia A):
 - Obtained from FFP after thawing at 4°C
 - Contains factor VIII, vWF, fibrinogen
 - Estimated 80–100 U of factor VIII in 1 U of cryoprecipitate
 - Give 10 bags initially, peds dose 1 bag/6 kg
 - Only if factor VIII concentrates are not available
 - FFP (contains factors VIII and IX):
 - 1 U of FFP contains about 200–300 U each of both factor VIII and

- factor IX
 - 1 U of FFP will raise factor level 5–10% in a 60 kg person
 - Readily available in most EDs; useful in life-threatening bleeds when access to specific factor treatment is delayed
- Adjunct to factor therapy: DDAVP (only in hemophilia A)
 - Raises factor VIII level 2–3 times (only use in patients with mild hemophilia, and only with mild bleeds)
 - Side effects: Mild flushing, headache, tachycardia, hypotension, hyponatremia
 - Dose 0.3 µg/kg of DDAVP diluted in 50 mL 0.9% NS given over 15–30 min given IV or SC (intranasal “Stimate” 1 spray each nostril if >50 kg)
 - Should not be used in children <1 yr old
- Adjunct to factor therapy: Antifibrinolytic agents (for mild bleeds)
 - Inhibit plasmin activity, prevents clot lysis
 - Only for mucosa/oral/dental bleeding
 - Do not use in children.
 - Examples: Aminocaproic acid (Amikar) 75 mg/kg every 6 hr up to 4 g or tranexamic acid (Cyklokapron) 25 mg/kg every 8 hr.
- Adjunct to factor therapy: Topical thrombin
 - Useful for localized control of bleeding from lacerated superficial tissues

Patient with Inhibitors

- First line: Recombinant factor VIIa (NovoSeven)
 - 90–120 µg/kg every 2–3 hr
 - Higher doses of up to 300 µg/kg may promote thrombin formation and stabilize fibrin clot in the case of inefficient hemostasis
 - Bypasses the coagulation cascade and works locally, decreasing risk for systemic coagulation
 - Strong safety profile, minimal risk of inhibitor development, expensive
- Second line: Activated prothrombin complex concentrates (APCCs)
 - Examples: FEIBA and Autoplex-T
 - Dose: 75–100 U/kg every 8–12 hr
 - Contains VIIa, IXa, and Xa
 - Bypasses coagulation cascade
 - Thromboembolic events occur with higher doses: DVT, PE, acute MI, DIC
- Other options
 - Use high-dose recombinant or plasma-derived factor concentrates (2–3 times normal range) in those who have low antibody titers (low responders)
 - Porcine factor VIII is now off the market

- Avoid all IM injections of factor concentrates
- Avoid aspirin and aspirin-containing products
- Replace factor before any imaging, consultations, invasive procedures, or hospital transfers



FOLLOW-UP

DISPOSITION

Admission Criteria

- Low threshold for admission
- All joint/muscle bleeds, internal bleeding, severe bleeds
- Bleeding that may require multiple transfusions
- Severe complications or any head trauma

Discharge Criteria

Minor soft tissue bleeding, superficial lacerations with resolution/control of bleeding

Issues for Referral

Hematology for all bleeds

FOLLOW-UP RECOMMENDATIONS

- Hematologist as outpatient
- Return to ER for recurrent bleeding episodes

PEARLS AND PITFALLS

- Do not wait for a head CT before treatment in the setting of head trauma
- In a suspected bleeding emergency in a patient with unknown clotting factor level, assume the level to be 0 and treat as a severe bleed
- Be familiar with different factor replacement therapies for patients with and without inhibitors
- Consult hematologist for appropriate dosing in ER, length of treatment, inhibitor vs. noninhibitor treatment
- Have a low threshold for admission

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CODES

ICD9

- 286.0 Congenital factor VIII disorder
- 286.1 Congenital factor IX disorder

ICD10

- D66 Hereditary factor VIII deficiency
- D67 Hereditary factor IX deficiency

HEMOPTYSIS

Amy Kiraly • Peter S. Pang • Navneet Cheema



BASICS

DESCRIPTION

- Expectoration of blood originating from the tracheobronchial tree
- Source of bleeding:
 - Bronchial arteries (90%), usually causes profuse bleeding
 - Pulmonary arteries (5%), usually causes small amounts of bleeding
 - Nonbronchial arteries (5%) including intercostal arteries, coronary arteries, thoracic, upper, and inferior phrenic arteries
- Threshold of massive hemoptysis defined has been defined from 100 mL to 1 L/24 hr:
 - > 8 mL/kg/day in children
 - Most common definition is > 300–600 mL/24 hr
- Mortality:
 - Massive hemoptysis (> 500 mL/24 hr): 38%
 - Trivial to moderate hemoptysis (< 500 mL/24 hr): 4.5%
 - Malignancy and coagulopathy increase the risk of mortality

ETIOLOGY

- Infectious (most common cause):
 - Acute or chronic bronchitis
 - Pneumonia
 - Necrotizing pneumonia or lung abscess (*Klebsiella pneumoniae*, *Pseudomonas aeruginosa*, *Staphylococcus aureus*, *Streptococcus pneumoniae*)
 - Tuberculosis
 - Viral (influenza, varicella)
 - Fungal (*Aspergillus*, *Coccidioides*, *Histoplasma*, *Blastomyces*)
 - Parasitic (*Ascariasis*, *Amebiasis*, *Paragonimiasis*, *Echinococcus*)
- Neoplastic:
 - Squamous cell, small cell, carcinoid
 - Bronchogenic carcinoma
 - Metastatic disease
- Pulmonary:
 - Bronchiectasis
 - Pulmonary embolism/infarction
 - Cystic fibrosis
 - Bronchopleural fistula
 - Sarcoidosis

- Cardiac:
 - Mitral stenosis
 - Tricuspid endocarditis
 - Heart failure
- Systemic disease:
 - Goodpasture syndrome
 - Systemic lupus erythematosus
 - Vasculitis (Wegener granulomatosis, Henoch–Schönlein purpura, Behçet disease)
- Hematologic:
 - Coagulopathy
 - Thrombocytopenia
 - Platelet dysfunction
 - DIC
- Vascular:
 - Pulmonary HTN
 - Arteriovenous malformation
 - Aortic aneurysm
 - Pulmonary artery aneurysm (Rasmussen aneurysm, mycotic, arteritis)
 - Aortobronchial fistula
- Drugs/toxins:
 - Aspirin/antiplatelet therapy
 - Anticoagulants
 - Penicillamine, amiodarone, propylthiouracil, bevacizumab
 - Cocaine (“crack”) lung
 - Organic solvents
- Trauma:
 - Tracheobronchial rupture
 - Pulmonary contusion
- Iatrogenic:
 - Bronchoscopy/lung biopsy
 - Pulmonary artery or central venous catheterization
 - Transtracheal aspirate
- Miscellaneous:
 - Foreign-body aspiration
 - Catamenial hemoptysis (pulmonary endometriosis)
 - Amyloidosis
 - Idiopathic or cryptogenic (between 5% and 30%, depending on patient population)

SIGNS AND SYMPTOMS

- Chest pain
- Dyspnea
- Fever
- Weakness
- Fatigue
- Night sweats
- Weight loss

History

- Inquire about prior lung, renal, or valvular heart disease
- History of cigarette smoking
- Chemical, asbestos, or infectious exposure
- Travel history (consider parasitic or fungal infectious etiology)
- Aspirin, NSAID, or anticoagulant use
- Consider Goodpasture syndrome if a history of hematuria is present.
- Recurrent or chronic hemoptysis raises suspicion of arteriovenous malformations, bronchiectasis, and cystic fibrosis.

Physical-Exam

- Clubbing of the fingers (chronic inflammatory lung diseases)
- Cutaneous ecchymosis (blood dyscrasia or anticoagulants)
- Aphthous ulcers (Behçet disease)
- Nasal septal perforation (Wegener granulomatosis)
- Hematuria (Goodpasture syndrome)
- Unilateral lower extremity edema may indicate DVT.
- Suggestive of pseudohemoptysis:
 - Sinusitis, epistaxis, rhinorrhea, pharyngitis, upper respiratory infection, aspiration

Pediatric Considerations

- Thorough head, eyes, ears, nose, and throat exam to exclude nonpulmonary source of bleeding
- Pulmonary exam is often normal.
- Wheezing may suggest obstruction (e.g., foreign body).
- Crackles may indicate an underlying pulmonary etiology (e.g., pneumonia, hemothorax, heart failure).
- Telangiectasias or hemangiomas raise suspicion of arteriovenous malformations.

ESSENTIAL WORKUP

- Differentiate between hemoptysis and pseudohemoptysis:
 - Note any precipitating factors, duration of symptoms, quantity and quality

of blood.

- Pulmonary source:
 - Bright red blood
 - Frothy in appearance
 - Sputum mixed with blood is likely pulmonary
 - pH > 7
- GI source:
 - Dark red or brown blood
 - Accompanied by gastric contents
 - Worsens in the setting of nausea/vomiting
 - pH < 7
 - Gastric lavage may be used to rule out GI source of bleeding; however, nasal or other trauma may cause further bleeding.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC with differential
- Basic metabolic panel
- PT/INR, PTT
- Urinalysis
- Febrile patient or suspected infectious etiology:
 - Blood cultures
 - Sputum culture and Gram stain
 - Cytology
 - KOH prep for fungal causes
 - AFB stain for tuberculosis
- Hypotensive patient (criteria for massive hemoptysis):
 - Type and cross
 - Complete metabolic (liver and renal function) panel
 - Coagulation profile:
 - Fibrin and fibrinogen degradation products (FDP) or antithrombin III if disseminated IV coagulation suspected
- Pediatric patient:
 - Consider sweat-chloride test if cystic fibrosis is suspected.

Imaging

- CXR:
 - Characterizes pathology (e.g., tumor, cavity, effusion, infiltrate, pneumothorax)
 - Early pulmonary hemorrhage may present as infiltrate.
 - ~20% will be normal.
- CT:

- High-resolution CT has become gold standard for diagnosing bronchiectasis.
- Ideal study for stable patients with hemoptysis and a normal CXR
- Can detect active TB by the presence of cavitary lesions and acinar nodules
- Higher sensitivity for peripheral tumors that may not be apparent on bronchoscopy
- CTA:
 - Known variability in bronchial arterial supply
 - Characterizes origin of bronchial arteries and presence of variants
 - May identify a pulmonary artery as a source of bleeding, show a pulmonary or bronchial artery aneurysm
 - Characterizes abnormal nonbronchial arterial supply, eliminates nonbronchial arteries as possible sources of bleeding
 - identifies pulmonary embolism
- V/Q:
 - If PE is suspected and patient cannot get CTA
 - Limited utility if x-ray is abnormal

Diagnostic Procedures/Surgery

Bronchoscopy:

- Allows direct visualization of tumors, foreign bodies, granulomas, and infiltration
- Valuable for collecting bronchial secretions for cytology and histology
- Limited diagnostic yield in lesions outside the bronchial wall, distal to bronchial stenosis or occlusion, or peripheral lesions.

DIFFERENTIAL DIAGNOSIS

Pseudo-hemoptysis:

- Epistaxis
- Pharyngeal bleeding
- GI bleeding



TREATMENT

PRE HOSPITAL

- Respiratory and contact precautions
- Airway management:
 - Oxygen
 - Suctioning as needed
 - Endotracheal intubation if airway compromised, severe respiratory distress, or hypoxemia
- Dual large-bore IV access
- Volume resuscitation as needed
- Continuous pulse oximetry, close hemodynamic and cardiac monitoring

INITIAL STABILIZATION/THERAPY

- Airway and breathing:
 - Endotracheal intubation for impending respiratory failure
 - >8Fr endotracheal tube to facilitate suctioning and subsequent bronchoscopy
 - Selective intubation of nonbleeding lung with single- or double-lumen endotracheal tubes may be required.
 - Supplemental oxygen as needed
 - Continuous pulse oximetry and cardiac monitoring
- Massive hemoptysis:
 - Principal risk to life is asphyxiation, not exsanguination
 - Maintain dual large-bore IV access.
 - Volume resuscitation with crystalloid or blood products as needed

ED TREATMENT/PROCEDURES

- Antimicrobial therapy if concern for or diagnosed infectious cause
- Correct hypoxemia and/or coagulopathy
- If massive hemoptysis:
 - Multiple large-bore IVs or central access with volume resuscitation and blood products as needed
 - Patient should be positioned upright or in lateral decubitus with affected lung positioned down
 - Intubation for airway protection, impending respiratory failure, or to facilitate bronchoscopic evaluation
 - Endobronchial tamponade with Foley or Fogarty (<4Fr) catheter, or double-lumen endotracheal tube (temporary measures)
 - Bronchoscopy for local therapy including vasoconstrictive agents, stent or balloon tamponade, electrocautery, procoagulants
 - Bronchial artery embolization (success rates reported as high as 98%); rebleeding presents in ~20% of cases
 - Surgery:
 - Lobectomy or pneumonectomy if unsuccessful embolization or in the presence of thoracic aneurysm, trauma, or arteriovenous malformation
 - Surgical resection is most effective for patients with localized lesions and adequate cardiopulmonary reserve

MEDICATION

Refer to specific etiology



FOLLOW-UP

DISPOSITION

Admission Criteria

- ICU:
 - Intubation
 - Massive hemoptysis
 - Hemodynamic instability
 - Hypovolemic shock
 - Severe or refractory hypoxemia
 - Impending respiratory failure
 - Impending airway compromise
- General ward:
 - Mild hemoptysis
 - TB (isolation)
 - Stable foreign body
 - Lung abscess
 - Cavitory lung disease

Discharge Criteria

- Hemodynamically stable
- Mild hemoptysis
- No coagulopathy
- No supplemental oxygen requirement
- History of chronic stable hemoptysis
- Close follow-up

Issues for Referral

- PCP within 7–10 days
- Specialist if etiology warrants referral

FOLLOW-UP RECOMMENDATIONS

- Counsel patient not to smoke.
- Avoid medications that may increase the risk of bleeding, including herbal supplements such as garlic, ginkgo, or ginseng.
- The patient should seek care immediately for:
 - Shortness of breath
 - Chest pain
 - Severe dizziness on standing
 - Fainting
 - Persistent or worsening hemoptysis

PEARLS AND PITFALLS

- Consider early airway management as clinical picture warrants.
- If severe unilateral hemorrhage with hypoxemia, place patient “bad lung” down
- Bronchial artery embolization can be very effective. Discuss case early with IR.

ADDITIONAL READING

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CODES

ICD9

- 466.0 Acute bronchitis
- 491.9 Unspecified chronic bronchitis
- 786.30 Hemoptysis, unspecified

ICD10

- J20.9 Acute bronchitis, unspecified
- J42 Unspecified chronic bronchitis
- R04.2 Hemoptysis

HEMORRHAGIC FEVERS

Fraser C. Mackay • Ben Osborne

BASICS

DESCRIPTION

Hemorrhagic fever describes a multisystem syndrome of vasocapillary permeability and/or organ dysfunction. Viral hemorrhagic fever (VHF) is caused by a distinct group of viruses, but the initial phase resembles influenza-like illness. Hemorrhagic stages typify the minority of patients and the later phases of disease.

RISK FACTORS

- Travel in endemic region
- Biologic warfare
- Close animal contact, insect bite or ingestion

PATHOPHYSIOLOGY

- VHF causes endothelial damage and increase vascular permeability, hemorrhage, and may proceed to shock
- VHF shock state is both hypovolemic and distributive, and is often very difficult to reverse. Hypotension can progress swiftly, and indicates very high mortality.
- DIC appears to be a regular feature of Marburg and Crimean-Congo hemorrhagic fever but is less frequent with Arenavirus infections.
- Dengue hemorrhagic fever is immune mediated and is usually the result of secondary infection. It is among the most common causes for VHF.

ETIOLOGY

- RNA viruses that have zoonotic life cycles in specific geographic areas
- Short incubation period (< 10–21 days)
- More common VHF vectors:
 - Filoviruses: Fruit bat reservoir, unclear mode of transmission (sub-Saharan Africa)
 - Ebola
 - Marburg
 - Arenaviruses: Rodent reservoir, aerosolized rodent excreta (sub-Saharan Africa).
 - Lassa
 - South American hemorrhagic fevers
 - Flaviviruses: Human reservoir, via mosquito (tropics, increasingly worldwide)
 - Dengue (common cause of VHF)
 - Yellow fever

- Bunyaviridae: Rodent reservoir, via tick or mosquito (Europe, South Asia, Africa)
 - Rift Valley fever
 - Crimean-Congo hemorrhagic fever
- Hantaviridae: Rodent reservoir, aerosolized rodent excreta (Southwest USA)
 - Hemorrhagic fever with renal syndrome
 - Hantavirus pulmonary syndrome

ALERT

- Potential biowarfare threat:
 - Aerosols (with exception of dengue) and body fluids highly infectious
 - High morbidity/mortality in some cases
 - Replicate well in cell culture, permitting weaponization



DIAGNOSIS

SIGNS AND SYMPTOMS

- Most common (>50%) symptoms:
 - Acute febrile illness
 - Malaise
 - Headache
 - Nausea/vomiting
 - Flushing
 - Diarrhea (nonbloody)
 - Abdominal pain
 - Myalgias
- Less common (<30%) symptoms:
 - Gingival hemorrhage
 - Conjunctival injection/hemorrhage
 - Petechia
 - Hematemesis
 - Melena
 - Epistaxis
 - Ecchymoses
- Hemorrhagic presentations > 3 days into disease
 - Skin, IV sites, gums, nose, lungs, GI tract, or uterus
 - Diffuse alveolar hemorrhage or ARDS
 - More common in CCHF, Lassa, Marburg, Ebola, Hantavirus
- Exanthems
 - Marburg and Ebola: Nonpruritic centripetal, papular, erythematous eruption appearing between days 5 and 7, which then coalesce into well-demarcated macules that may be hemorrhagic
 - Yellow fever: Jaundice

- Dengue: Bright maculopapular truncal erythroderma that blanches dramatically under light pressure (often on lower extremities)
- Hemodynamic collapse, shock, seizures, coma, death.
 - Late stage of disease, often irreversible

History

- Travel to endemic regions
- Sick contacts
- Clustering of cases should raise concerns of a bioweapon attack or outbreak

Physical-Exam

- Protection of health care workers:
 - Universal blood and body precautions
- Vital signs: Monitor BP, fever, tachycardia
 - Narrowed pulse pressure (< 20 mm Hg) may signal imminent cardiovascular collapse
- Hemorrhage (see Signs and Symptoms)
- Exanthems (see Signs and Symptoms)
- RUQ tenderness or hepatomegaly
 - Hepatitis
- Adventitious lung sounds

ESSENTIAL WORKUP

- Focus on differentiating from other acute febrile illnesses, especially in the traveler.
- Investigate lung involvement, as it can indicate systemic disease and worse outcome
- Recognize possible biologic attack when unusual number of patients present with similar and/or unusual findings.
- History to identify potential pathogen:
 - Include recent travel, illnesses, or other sources of exposure.
 - Often patients are unaware of animal contacts

DIAGNOSIS TESTS & NTERPRETATION

Lab

- CBC:
 - May see leukocytosis, leukopenia, thrombocytopenia, or pancytopenia
 - Abnormally high hematocrit can signify hemoconcentration; may indicate increased 3rd spacing impending shock/cardiovascular collapse.
- Electrolytes, BUN, creatinine, and glucose levels:
 - Consider renal failure
- Liver function tests:

- Hepatic involvement is common, but jaundice occurs mainly with yellow fever.
- Type and screen, prothrombin time, partial thromboplastin time, and d-dimer tests:
 - Look for coagulopathy and DIC (seen in Crimean-Congo hemorrhagic fever, Ebola, and Marburg)
- Special lab test:
 - In specialized labs (biohazard level 4), definitive diagnosis can be made by viral isolation, real-time reverse transcriptase polymerase chain reaction (RT-PCR), and immunohistochemistry techniques:
 - Coordinated with CDC
 - Thick and thin smears to help differentiate from malaria

Imaging

CXR, head, and abdominal CT scanning:

- Rule out pneumonia or ARDS
- Intracranial and intra-abdominal bleeding

Diagnostic Procedures/Surgery

Serum and saliva can be analyzed by RT-PCR in specialized labs.

DIFFERENTIAL DIAGNOSIS

- Malaria:
 - A concern for traveler with fever
- Dengue fever:
 - Common source of fever in traveler
- Rickettsial:
 - Rocky Mountain spotted fever
 - Typhus
- Bacterial:
 - Meningococemia
 - Sepsis
 - EHEC (Escherichia coli O157:H7)
- Systemic disease:
 - Leukemia
 - TTP, ITTP
- Pit viper envenomation



TREATMENT

PRE HOSPITAL

ALERT

- Increasing globalization has increased frequency of imported cases of rare diseases.
- Early detection of VHF, natural, or biologic attack is key to control an outbreak. Report to CDC.
- Most cases will derive from patients who traveled to or had contact with persons from parts of the world where the viruses are endemic.

INITIAL STABILIZATION/THERAPY

Protection of health care workers:

- Universal blood and body precautions
- Isolation of patient
- Use of protective clothing plus HEPA-filtered respirators to minimize exposure to aerosols for those involved in procedures such as suctioning, catheter placement, and wound dressing

ED TREATMENT/PROCEDURES

- Supportive therapy
- Empiric therapy with antimalarial regimens until definitive diagnosis is obtained
- Aggressive treatment of secondary infections
- Bleeding is usually mild, and life-threatening blood loss is rare:
 - If indicated, hemorrhage can be managed by replacement of blood, platelets, and clotting factors.
- Fluid support
 - Patients are prone to 3rd spacing and can go into flash pulmonary edema; be judicious with crystalloids.
 - Reserve colloids/blood products for impending shock/cardiovascular collapse.
- Ribavirin—a synthetic nucleoside:
 - Useful for Lassa, South American hemorrhagic fever, Crimean-Congo hemorrhagic fever, and hemorrhagic fever with renal syndrome; ineffective against filoviruses
 - Causes a reversible hemolytic anemia
- Transfusion of immune plasma (convalescent plasma therapy) for South American hemorrhagic fever within 1st week of symptoms

MEDICATION

- Ribavirin (WHO 2006 recommendations):
 - IV loading dose of 33 mg/kg followed by 16 mg/kg q6h for 4 days, then 8 mg/kg q8h for 3 days
 - Prophylactic 500 mg by mouth q6h for 7 days
- Vaccines:
 - Yellow fever is widely available.
 - South American hemorrhagic fever, Rift Valley fever, Hantavirus, Dengue,

and Ebola/Marburg are under development.

- Other medications under investigation:
 - Nucleoside analog inhibitors of *S*-adenosylhomocysteine hydrolase inhibit Ebola replication in mice.
 - Zidampidine—a derivative of AZT—increases survival of mice infected with Lassa virus

First Line

- Hemodynamic support
- Ribavirin
- Antimalarials

Second Line

Contact the CDC about experimental vaccines and antivirals for postexposure prophylaxis (770) 488-7100 or (800) 311-3435 for all suspected cases.



FOLLOW-UP

DISPOSITION

Admission Criteria

Suspected cases of VHF, particularly those with suspected multisystem involvement/compromise

- Isolation precautions
- ICU for signs of shock or multiorgan system failure

Discharge Criteria

None—if you suspect VHF, ***the patient needs to be isolated and the CDC notified***

FOLLOW-UP RECOMMENDATIONS

Consider experimental postexposure prophylaxis for staff and patient contacts that may include antivirals and vaccines. Coordinate with the CDC at (770) 488-7100 or (800) 311-3435.

PEARLS AND PITFALLS

- **EXTREME** caution with volume resuscitation
- Hemoconcentration and pulmonary involvement are **RED FLAGS**
- Consider hemorrhagic viruses in your differential diagnosis when caring for a sick patient returning from endemic regions of the world
- Employ universal precautions and isolation to minimize the spread of the disease
- Contact the CDC at (770) 488-7100 or (800) 311-3435 for all suspected cases.

ADDITIONAL READING

- Jeffs B. A clinical guide to viral hemorrhagic fevers: Ebola, Marburg and Lassa. *Trop Doct.* 2006;36:1–4.
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- Staples JE, Monath TP. Yellow fever: 100 years of discovery. *JAMA.* 2008;300(8):960–962.
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See Also (Topic, Algorithm, Electronic Media Element)

- Dengue Fever
- Disseminated Intravascular Coagulation
- Hemorrhagic Shock
- Malaria
- Meningococemia



CODES

ICD9

- 065.4 Mosquito-borne hemorrhagic fever
- 065.9 Arthropod-borne hemorrhagic fever, unspecified
- 078.89 Other specified diseases due to viruses

ICD10

- A91 Dengue hemorrhagic fever
- A94 Unspecified arthropod-borne viral fever
- A98.3 Marburg virus disease

HEMORRHAGIC SHOCK

Theodore C. Chan



BASICS

DESCRIPTION

- Loss of effective circulating blood volume resulting in inadequate perfusion
- Blood loss exceeds ability to compensate and tissue and organ perfusion decrease. At the tissue level, hypoperfusion leads to inadequate oxygenation, anaerobic metabolism, cell death
- Hemorrhagic shock is the most common cause of shock from injury
- Compensated shock:
 - Patient's physiologic reserve prevents significant alteration in vital signs
- Decompensated shock:
 - Loss of circulating volume overcomes patient's physiologic reserve, resulting in significant alteration in vital signs.
- Blood loss estimate:
 - Total blood volume ~7% of ideal body weight (4,900 mL in 70 kg adult) or 70 mL/kg
 - Multiply 70 mL/kg × body weight (kg) × percentage loss as determined by class of hemorrhage.

ETIOLOGY

- Trauma—penetrating and blunt:
 - Abdominal:
 - Splenic injury
 - Liver injury
 - Chest:
 - Hemothorax
 - Aorta or great vessel injury
 - Pelvis:
 - Pelvic fracture with vascular injury
- Vascular malformations:
 - May lead to thoracic, intraperitoneal, or retroperitoneal bleeding
 - Aneurysms:
 - Abdominal aortic aneurysm most common
 - Mycotic aneurysm secondary to endocarditis
 - Aortogastric fistula
 - Arteriovenous malformations
- Abortion: Complete, partial, or inevitable
- Ectopic pregnancy

- Epistaxis
- Fractures (especially pelvis and long bones)
- GI bleeding
- Hemoptysis
- Malignancies
- Mallory–Weiss tear
- Placenta previa
- Postpartum hemorrhage
- Retroperitoneal bleeds
- Splenic rupture
- Vascular injuries



DIAGNOSIS

SIGNS AND SYMPTOMS

- Class I hemorrhage: Loss of up to 15% of blood volume (up to 750 mL in 70 kg adult):
 - HR < 100
 - SBP normal
 - Respiratory rate (RR) 14–20
 - Increased or normal pulse pressure
 - Slight anxiety
- Class II hemorrhage: Loss of 15–30% of blood volume (750–1,500 mL):
 - Tachycardia: HR > 100
 - SBP normal, or minimally decreased
 - Tachypnea: RR 20–30
 - Narrowed pulse pressure
 - Mild anxiety
 - Small decrease in urine output
- Class III hemorrhage: Loss of 30–40% of blood volume (1,500–2,000 mL):
 - Marked tachycardia: HR > 120
 - Hypotension: SBP decreased
 - Marked tachypnea: RR 30–40
 - Marked narrowing of pulse pressure
 - Significant change in mental status: Confusion
 - Delayed capillary refill
 - Marked decrease in urine output
- Class IV hemorrhage: Loss of > 40% of blood volume (> 2,000 mL):
 - HR > 140
 - Marked hypotension: SBP decreased
 - RR > 35
 - Very narrow pulse pressure

- Depressed mental status: Confusion, lethargy, loss of consciousness
- Negligible urine output
- Cold and pale skin

ALERT

- Reliance solely on SBP as indicator of shock state can result in delayed recognition

Pediatric Considerations

- Children often have greater physiologic reserve than adults and can preserve normal vital signs longer
- Systemic responses to blood loss in the pediatric patient include:
 - Volume loss < 25%: Weak, thready pulse and tachycardia; lethargy, irritability, and confusion; cool, clammy skin; decreased urine output/increased urine specific gravity
 - Volume loss 25–40%: Tachycardia; marked change in consciousness; dulled response to pain; cyanotic, cold extremities with decreased capillary refill; minimal urine output
 - Volume loss > 40%: Hypotension, tachycardia, or bradycardia; comatose; pale, cold skin; no urine output

Pregnancy Considerations

Physiologic maternal hypervolemia requires greater blood loss to manifest maternal perfusion abnormalities which may result in decreased fetal perfusion.

Geriatric Considerations

Underlying disease and medications may alter responses to hemorrhage and blood loss.

History

Thorough health and past medical history:

- Underlying disease, risk factors, age
- Medications
- Trauma

Physical-Exam

- Complete physical exam to determine shock class and assess for hemorrhage source
- Vital signs including HR, RR, BP
- Temperature
- Mental status (anxiety, confusion, lethargy, obtundation, coma)
- Pulse character, capillary refill and skin perfusion
- Pulse pressure
- Abdominal exam
- Pelvic/rectal exam for bleeding as indicated

ESSENTIAL WORKUP

- Thorough history and physical exam
- IV access for resuscitation
- Blood type and cross-match

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC
- Blood type and cross-match
- Coagulation studies:
 - PT, PTT
 - International normalized ratio
- Other measures of tissue hypoperfusion:
 - Arterial blood gas
 - Base deficit
 - Serum lactate level
 - Serum electrolytes
- Pregnancy test/ β -HCG

ALERT

Massive blood loss may only result in minimal decrease in Hb or Hct initially

Imaging

- CXR:
 - Hemothorax:
 - Blunt chest injuries
 - Thoracic arteriovenous malformation
- Pelvic radiograph for possible occult fracture
- Focused abdominal sonography for trauma (FAST exam):
 - Abdominal trauma
 - Possible abdominal aortic aneurysm
 - Nontraumatic intraperitoneal hemorrhage
 - Fluid in Morrison pouch implies significant hemorrhage or ascites.
 - Negative findings do not rule out intraperitoneal hemorrhage.
- Endovaginal US:
 - Positive pregnancy test
 - Fluid in the cul-de-sac
 - Ectopic pregnancy
- Abdominal CT scan (once patient stable):
 - Detects both intraperitoneal and retroperitoneal hemorrhage
 - Abdominal aortic aneurysm

Diagnostic Procedures/Surgery

- Insert Foley catheter:
 - Monitor urine output.
- Nasogastric tube:
 - For undifferentiated hypovolemic shock to rule out GI hemorrhage
- Diagnostic peritoneal lavage:
 - For unstable trauma patients when US fails to show intraperitoneal hemorrhage
- Endoscopy:
 - In the setting of upper or lower GI bleeding
- Angiography:
 - Pelvic fracture
 - Retroperitoneal hemorrhage
 - Lower GI bleeding
 - Embolization therapy for bleeding from arterial sources can be performed.

DIFFERENTIAL DIAGNOSIS

- Cardiac tamponade
- Tension pneumothorax
- Cardiogenic shock
- Sepsis
- Adrenal insufficiency
- Neurogenic shock



TREATMENT

- Treatment should be initiated as soon as shock state recognized while simultaneously identifying underlying bleeding source
- The goal is to restore tissue and organ perfusion and to control source of hemorrhage
- “Balanced” or “controlled” resuscitation: Approach is to balance goal of perfusion and risk of rebleeding and may vary with patient:
 - In blunt trauma, BP maintenance may take precedence to reduce risk of traumatic brain injury
 - In penetrating trauma with hemorrhage, delayed aggressive fluid resuscitation until definitive control may reduce bleeding risk

PRE HOSPITAL

- Rapid assessment and transport to appropriate care center
- IV access and fluid resuscitation are standard, though delayed fluid resuscitation may be warranted in cases of penetrating trauma.

INITIAL STABILIZATION/THERAPY

- Airway and breathing:
 - Intubation as indicated by patient's respiratory and mental status
 - 100% oxygen via face mask should be administered.
- Circulation:
 - 2 large-bore peripheral IV lines (16G or larger)
 - Central venous line or venous cutdown (saphenous) may be necessary
 - Intraosseous route may be considered
 - Fluid resuscitation with warmed, isotonic crystalloid fluid – total volume based on patient response to initial fluid bolus
- Early transfusion for class III or IV shock:
 - Type-specific and cross-matched blood preferred when time permits, often 1 hr.
 - Type-specific blood is usually available within 10–15 min.
 - Type O blood can be used in immediate, life-threatening situations (type O Rh-negative blood only for women of child-bearing age).

ED TREATMENT/PROCEDURES

- Place patient on continuous monitor.
- NPO status, strict bed rest
- Control hemorrhage (direct pressure, pelvic fixation/stabilization, etc.).
- Central venous access may be indicated for CVP monitoring, but placement of such lines should not interfere with resuscitation.
- Continually reassess patient for clinical response/deterioration:
 - Vital signs, mental status, and urine output.
 - Follow serial blood gas, lactate level, and hemoglobin/hematocrit measurements.
 - Maintain urine output at 50 mL/hr.
- Response to initial fluid resuscitation is the key to determining subsequent therapy:
 - Rapid response to fluid indicates minimal (< 20%) blood loss.
 - Transient response indicates ongoing hemorrhage or inadequate resuscitation; continue fluid and blood administration and obtain necessary studies and consultations
 - Minimal or no response to volume resuscitation indicates ongoing severe blood loss; immediate angiography or surgical intervention is warranted
- Use fluids warmed (~ 39°C [102.2°F]) by microwave ovens, fluid warmers
- Transfuse whole blood, RBCs, platelets, and other blood products as indicated
- Consider autotransfusion devices with tube thoracostomy treatment of large hemothoraces.
- Monitor closely for coagulopathy particularly with massive transfusions
- Specialty consultation and additional procedures (surgery) as indicated by cause and source of hemorrhagic shock

Pediatric Considerations

- Access may be obtained by intraosseous route after 1 or 2 unsuccessful attempts at peripheral access
- Maintain urine output at 1 mL/kg/hr for children and 2 mL/kg/hr for infants

Pregnancy Considerations

Optimizing perfusion and treatment of the mother is treatment of choice for fetus.

MEDICATION

First Line

- IV Fluids:
 - Crystalloids: NS or lactated Ringer
 - Adults: 1–2 L bolus
 - Pediatric: 20 mL/kg bolus:
 - Reassess for clinical response/deterioration.
- Blood products: Cross-matched, type-specific, O-positive, or O-negative:
 - O-negative should be reserved for women of child-bearing age
 - Adult: Initiate with 4–6 U
 - Pediatric: 10 mL/kg

Second Line

- Other blood products:
 - Platelets
 - Coagulation factors, such as fresh frozen plasma, cryoprecipitate
- Antifibrinolytic agents, hemoglobin-based oxygen carriers, perfluorocarbons:
 - Under study, but not yet of proven benefit



FOLLOW-UP

DISPOSITION

Admission Criteria

All patients with hemorrhage should be admitted to the appropriate service.

Discharge Criteria

N/A

Issues for Referral

N/A

PEARLS AND PITFALLS

- Severity of hemorrhagic shock class and volume loss can be determined by vital signs and careful physical exam
- Fluid resuscitation should balance goal of restoring organ perfusion and potential risk of exacerbating bleeding before definitive control
- Response to fluid resuscitation should guide subsequent therapy

ADDITIONAL READING

- American College of Surgeons, Committee on Trauma. *Advanced Trauma Life Support*. 9th ed. Chicago, IL: American College of Surgeons; 2012.
- Curry N, Davis PW. What's new in resuscitation strategies for the patient with multiple trauma? *Injury*. 2012;43:1021–1028.
- Santry HP, Alama HB: Fluid resuscitation: Past, present and the future. *Shock*. 2010;33:229–241.
- Theusinger OM, Madjdpour C, Spahn DR. Resuscitation and transfusion management in trauma patients: Emerging concepts. *Curr Opin Crit Care*. 2012;18:661–670.



CODES

ICD9

- 459.0 Hemorrhage, unspecified
- 865.04 Injury to spleen without mention of open wound into cavity, massive parenchymal disruption
- 958.4 Traumatic shock

ICD10

- S36.09XA Other injury of spleen, initial encounter
- S36.93XA Laceration of unspecified intra-abdominal organ, initial encounter
- T79.4XXA Traumatic shock, initial encounter

HEMORRHOID

Julia H. Sone



BASICS

DESCRIPTION

- General:
 - Normal venous sinusoids of the distal rectum and proximal anal canal
 - Normal vascular cushions of anal canal that contribute to anal continence
 - Arteriovenous shunt system exists at the level of the internal hemorrhoids that accounts for the bright red blood per rectum
- When the hemorrhoids become symptomatic, hemorrhoid disease develops.
- Do not cause pain unless thrombosed or strangulated
- Discrete masses of thick submucosa contain:
 - Blood vessels
 - Smooth muscle
 - Elastic and connective tissue
- Sliding down of part of anal canal lining
- External hemorrhoids:
 - Vessels situated below dentate line
 - Covered by skin/anoderm
 - Drain to internal iliac veins
- Internal hemorrhoids:
 - Submucosal vessels above dentate lines
 - Drain to portal system
 - Usually at left lateral, right posterolateral, and right anterolateral positions
 - Grade 1: Painless, bleeding
 - Grade 2: Prolapse with bowel movement (BM), spontaneously reduce
 - Grade 3: Prolapse with BM, require manual reduction
 - Grade 4: Chronically prolapsed, not reducible

ETIOLOGY

- Exact cause unknown
- Gravitational forces and abdominal pressure cause distention of the sinusoids
- Associated with straining and irregular bowel habits:
 - Hard, bulky stools or diarrhea cause tenesmus/straining.
 - Push anal cushions out of anal canal
 - Weaken submucosal tissue leading to prolapse
- Higher resting anal pressures:
 - Erect posture
- Heredity:

- Absence of valves in veins
- Increased intra-abdominal pressure:
 - Ascites
 - Pregnancy
- Portal hypertension

DIAGNOSIS

SIGNS AND SYMPTOMS

- Painless, rectal bleeding with defecation
- Blood on stool or toilet paper
- Bright red blood drips into toilet bowl
- Rectal discomfort or pressure
- Severe pain if:
 - Internal hemorrhoids prolapse and strangulate
 - External thrombosed hemorrhoids
- Pruritus ani
- May also have fissure

History

- Length of bleeding
- Associate pain
- New lumps or masses by rectum
- Stool consistency: Hard or liquid
- Previous history of rectal problems
- Stool caliber

Physical-Exam

- Exam of perianal area:
 - Gently spread buttocks.
 - Discrete, dark blue, tender mass covered with skin: Thrombosed external hemorrhoid:
 - Can have internal component
 - Purplish, tender mucosal covered mass: Prolapsed, strangulated internal hemorrhoid:
 - Usually associated with enlarged, thrombosed external hemorrhoid
 - Have patient bear down to check for prolapsing hemorrhoids.
 - Digital rectal exam mandatory to rule out cancer
- Anoscopy to visualize anal canal:
 - Identify bleeding internal hemorrhoids.

ESSENTIAL WORKUP

Detailed history with thorough anorectal exam

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC if history of significant blood loss:
 - Hemoglobin/hematocrit
- Platelet count
- PT/PTT/INR if patient on anticoagulants or severe comorbid condition

DIFFERENTIAL DIAGNOSIS

- Rectal prolapse
- Anal fissure
- Perirectal abscess/fistula
- Condyloma acuminata
- Carcinoma or melanoma



TREATMENT

PRE HOSPITAL

Establish IV access if severe bleeding

INITIAL STABILIZATION/THERAPY

Direct digital pressure to control bleeding

ED TREATMENT/PROCEDURES

- Conservative therapy for all patients:
 - Hot sitz baths for 15 min TID and after each BM
 - High-fiber diet—30 g/day:
 - Eat more fresh fruits and vegetables.
 - Increase bran intake.
 - 10–12 glasses of water per day
 - Stool softeners
 - Bulk-forming laxatives
- NSAIDs: Analgesic and anti-inflammatory effects
- Excise thrombosed external hemorrhoid if severe pain, < 5 days old and clot not resolving:
 - Follow with conservative therapy.
 - Place patient in prone jackknife position or left lateral decubitus and tape buttocks apart
 - Infiltrate surrounding skin and underneath clot using 27G needle with lidocaine-containing epinephrine.
 - Make an elliptical incision to excise clot/skin.

- May need silver nitrate sticks for hemostasis
- Place a small piece of Gelfoam and/or gauze onto the wound and tape.
- Remove dressing at time of 1st sitz bath in about 6 hr
- Give analgesics:
 - NSAIDs
 - Acetaminophen
 - Lidocaine 5% ointment to anus: Topical anesthetic for pain relief
 - 0.2% topical nitroglycerin ointment to anus—decreases pain by inhibiting sphincter spasm
- Manually reduce nonthrombosed, prolapsed internal hemorrhoids:
 - Follow with conservative therapy.
 - May need topical anesthetic or anal sphincter block with local anesthesia
 - Can sclerose bleeding internal hemorrhoids with 2.5% sodium morrhuate or 3% hypertonic saline
 - Can rubber band ligate 1 or 2 internal hemorrhoids:
 - Avoid in immunocompromised patients due to perineal sepsis
- Nonreducible internal hemorrhoids:
 - Nonstrangulated: Conservative management and surgical referral
 - Strangulated: Immediate surgical referral for excision

Pregnancy Considerations

- Usually become symptomatic in the 3rd trimester and can be treated conservatively.
- Do not use Analpram-HC (class C)

MEDICATION

- Acetaminophen: 325–650 mg (peds: 15 mg/kg) with codeine 15–30 mg (peds: 0.5 mg/kg) PO q4h PRN
- Bran/fiber: 20 g PO daily
- Docusate sodium (Colace): 50–200 mg (peds: < 3 yr, 10–40 mg/d; 3–6 yr, 20–60 mg/d; > 6–12 yr, 40–150 mg/d) PO q12h
- ELA-Max 5 (5% lidocaine anorectal cream): Apply to perianal area q4h PRN pain (peds: not for < 12 yr of age). Caution: Use very small amount; this product contains about 5 g lidocaine/100 g cream and is readily absorbed.
- Hydrocortisone/pramoxine topical (Analpram-HC) 1%/1% cream; 2.5%/1% cream/lotion (peds: Same dosing) apply thin amount to area TID–QID
- Hydrocortisone/lidocaine topical (AnaMantle HC) 0.5%/3% cream; 2.5%/3% gel (peds: Not indicated) apply to anal canal BID
- Ibuprofen (Motrin): 400–600 mg (peds: 40 mg/kg/d) PO q6h
- Nitroglycerin 0.2% ointment: Apply to area TID with cotton-tipped applicator
- Psyllium seeds: 1–2 tsp (peds: 0.25–1 tsp/d) PO q24h



FOLLOW-UP

DISPOSITION

Admission Criteria

- Strangulated grade 4 hemorrhoids:
 - Surgical consult for prolapsed, thrombosed internal hemorrhoids
- Severe anemia with bleeding hemorrhoids
- Severe bleeding hemorrhoid in pt on anticoagulation or with portal hypertension

Discharge Criteria

Most patients will go home

Issues for Referral

Surgical referral for:

- Grade 3 or 4 internal hemorrhoids
- Suspected anorectal or colonic tumors, inflammatory bowel disease, coagulopathy, pregnancy, or immunocompromised

FOLLOW-UP RECOMMENDATIONS

- Colorectal follow up for grade 3 or 4 internal hemorrhoids or suspected tumor
- Primary care follow-up for uncomplicated hemorrhoids.

ALERT

All patients with bright red blood per rectum should be referred to GI or colorectal surgery to r/o malignancy

PEARLS AND PITFALLS

Hemorrhoids are not the only cause of anorectal pain and bleeding. Investigate for other etiologies when indicated.

ADDITIONAL READING

- Acheson AG, Scholefield JH. Management of hemorrhoids. *BMJ*. 2008;336(7640):380–383.
- Kaider-Person O, Person B, Wexner SD. Hemorrhoidal disease. *J Am Coll Surg*. 2007;204(1):102–117.
- Lohsiriwat V. Hemorrhoids: From basic pathophysiology to clinical management. *World J Gastroenterol*. 2012;18(17):1009–1017.
- Wexner SD, Pemberton JH, Beck DE, et al., eds. *The ASCRS Textbook of Colon and Rectal Surgery*. New York, NY: Springer; 2007.



See Also (Topic, Algorithm, Electronic Media Element)

Anal Fissure



CODES

ICD9

- 455.0 Internal hemorrhoids without mention of complication
- 455.3 External hemorrhoids without mention of complication
- 455.6 Unspecified hemorrhoids without mention of complication

ICD10

- K64.0 First degree hemorrhoids
- K64.1 Second degree hemorrhoids
- K64.9 Unspecified hemorrhoids

HEMOTHORAX

Anthony C. Salazar

BASICS

DESCRIPTION

- Accumulation of blood in the intrapleural space after blunt/penetrating chest trauma or other nontraumatic etiology. Bleeding is usually a result of disruption of the tissues/vessels of the chest wall, pleura, or intrathoracic structures:
 - Results in decreased vital capacity, hypoxia, and respiratory compromise.
 - Loss of large intravascular volume results in hemodynamic instability and hemorrhagic shock.
 - Massive hemothorax can cause increased intrathoracic pressure, resulting in compromised venous return and decreased cardiac output.
- Rarely a solitary finding in blunt trauma:
 - Commonly associated with pneumothorax (25% of cases), extrathoracic injuries (73% of cases), and pulmonary contusion.
- Large hemothoraces cause the release of substances that can act as anticoagulants and contribute to continued intrathoracic bleeding.
- If left untreated, can lead to empyema and fibrothorax (lung trapping due to adhesions).

ETIOLOGY

- Traumatic injuries (including iatrogenic) to major blood vessels:
 - Common vessels, including intercostal artery, internal mammary artery, pulmonary artery, pulmonary vein, aorta, vena cava, and heart are associated with hemorrhage into the thoracic cavity.
- Traumatic lung parenchymal injuries:
 - Often stops spontaneously as a result of low pulmonary pressures and high concentrations of thromboplastin in the lung.
 - Often associated with pneumothorax.
- Nontraumatic or spontaneous hemothoraces:
 - Very rare.
 - Consider coagulation disorder, malignancy, primary vascular event (such as aortic dissection, ruptured aneurysm), PE with infarction, infection (TB), bullous emphysema, pulmonary AV malformation, lobar sequestration.
- Torn pleural adhesions as a complication of spontaneous pneumothorax or tube thoracostomy

DIAGNOSIS

SIGNS AND SYMPTOMS

- Small amount of blood in thorax (< 400 mL): Little or no change in patient's appearance, vital signs, or physical findings
- Large amount of blood (> 1,000 mL): Restlessness, anxiety, pallor, pleuritic chest pain, hemoptysis, dyspnea, or air hunger:
 - Signs of shock with loss of blood volume $\geq 30\%$ (1,500–2,000 mL).
 - Tachycardia, tachypnea, hypotension.
- With insidious onset (i.e., malignancy): Dyspnea is the most common presenting sign since blood loss is usually not acute enough to produce a visible hemodynamic response.

History

- Acute blunt or penetrating trauma to chest.
- Recent rib fracture or flail chest.
- Delayed hemothorax can occur hours to days later without initial evidence of intrathoracic pathology on CXR; may be related to rupture of chest wall hematoma or disruption of intercostal vessels by rib fracture edges during movement.
- Malignancy or metastatic disease.
- Recent surgical procedure: Thoracentesis, thoracostomy, etc.

Physical-Exam

- Vitals signs: Depending on severity and time course, hypoxia, tachypnea, tachycardia, and hypotension maybe seen.
- Neck: JVD if increased intrathoracic pressure, tracheal deviation
- Chest inspection: Asymmetric expansion, gross deformity, paradoxical wall movement, abrasion, hematoma, and contusion
- Chest wall palpation: Tenderness or crepitus over ribs, clavicles, scapulae, or the sternum; SC emphysema, dullness to percussion
- Auscultation: Decreased or absent breath sounds over ipsilateral side (best appreciated in the upright patient)

ESSENTIAL WORKUP

CXR is the ideal diagnostic tool:

- In the hemodynamically stable patient, upright posteroanterior (PA) projection at full inspiration is optimal:
 - Fluid collections > 200–300 mL can usually be seen on upright or decubitus CXR.
 - In a normal unscarred pleural space, fluid will be noted as a meniscus/fluid level blunting the costophrenic angle.
- In the supine anteroposterior (AP) radiograph (i.e., portable), up to 1,000 mL of blood may not be readily apparent:
 - Only a slight hazy infiltrate over the involved hemithorax maybe seen.
- Look for associated injuries (pneumothorax, rib fractures, pulmonary contusion,

widened mediastinum, etc.) when reading chest radiography.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Hematocrit may be helpful if it shows a drop or changes on serial evaluations.
- Type and cross-match.
- Pulse oximetry, ABG
- Pleural fluid removed should reveal a hematocrit $> 50\%$ of the blood hematocrit.

Imaging

- US diagnostic imaging is a valuable tool in the evaluation of intrapleural fluid collection:
 - _ An extended FAST scan can diagnose hemothorax with a higher sensitivity than a portable CXR in trained hands.
- CT is useful in detecting small amounts of intrapleural fluid not visible on the chest radiograph.

DIFFERENTIAL DIAGNOSIS

- Hemopneumothorax
- Pneumothorax
- Pulmonary contusion
- Pleural effusion
- Empyema/pneumonia



TREATMENT

PRE HOSPITAL

- Assess vital signs and pulse oximetry; administer oxygen and obtain IV access.
- Fluid resuscitation as needed for hypotension
- Cautions:
 - _ Difficult to differentiate hemothoraces from pneumothoraces clinically:
 - All may present with dyspnea, pleuritic chest pain, decreased breath sounds, and hemodynamic instability.
 - Certain clues aid in making the diagnosis, such as SC emphysema for pneumothorax and dullness to percussion for hemothorax.
 - _ Perform needle thoracostomy for potential tension pneumothorax if the patient is hemodynamically unstable.

INITIAL STABILIZATION/THERAPY

- Manage airway, breathing, circulation:
 - _ Control airway as needed; endotracheal intubation for patients with

- impending respiratory failure
- Supplemental oxygen
- 2 large-bore IV access sites and fluid bolus to restore circulating blood volume
- Needle thoracostomy should be performed in patients with hemodynamic instability unless chest tube kit is immediately available.
- Patient should be positioned to sit upright unless contraindicated.

ED TREATMENT/PROCEDURES

- Obtain upright CXR as quickly as possible, but if patient unstable do not wait to administer definitive therapy.
- Hemothorax is treated by evacuating accumulated blood in the intrapleural space.
- Tube thoracostomy evacuates blood; allows for re-expansion of the lung, as well as constant monitoring of blood loss.
- Tube thoracostomy:
 - Use a large-bore chest tube (36–40Fr).
 - Insert in the 4th–5th intercostal space at the mid-axillary line aiming posteriorly and superiorly.
 - Tube is then connected to underwater-seal drainage and suction (20–30 mL H₂O).
 - Correct placement and adequate drainage is confirmed via CXR.
- Autotransfusion should be used if available to replace blood loss.
- Indications for OR thoracotomy:
 - Initial tube drainage > 20 mL/kg of blood (or 1,000 mL of blood for adults from the pleural cavity).
 - Persistent bleeding at a rate > 7 mL/kg/hr (or 200 mL/hr for 4 hr).
 - Increasing hemothorax seen on chest radiography.
 - Patient remains hypotensive despite adequate blood replacement and other sites of blood loss have been ruled out.
 - Patient decompensates after initial response to resuscitation.
- Indications for ED thoracotomy:
 - Penetrating trauma:
 - Traumatic arrest in the ED or within 10 min of ED arrival.
 - Severe shock with clinical signs of cardiac tamponade
 - Blunt trauma: Traumatic arrest in the ED at a trauma center or with surgeon available within 10 min

MEDICATION

- Local anesthetics for cutaneous anesthesia prior to tube thoracostomy in awake, conscious patients
- Procedural sedation (midazolam) and analgesia (fentanyl) may be used for stable, awake patients prior to tube thoracostomy:
 - Fentanyl: Adult/peds: 2–5 µg/kg per dose

- Midazolam: Adult/peds: 0.02–0.04 mg/kg per dose
- Other sedative agents may be considered.



FOLLOW-UP

DISPOSITION

Admission Criteria

Patients with hemothoraces requiring tube thoracostomies should be admitted for monitoring and thoracostomy tube management to the trauma, cardiothoracic, or general surgery service. The admitting unit should be experienced in managing chest tube equipment.

Discharge Criteria

- Patients with isolated small hemothoraces (detected incidentally on US or CT imaging) may be considered for discharge after 4–6 hr of observation if there is no evidence of continued bleeding, the patient is not hypoxic, and is asymptomatic.
- Patients with asymptomatic blunt chest trauma and normal initial chest radiographs do not require repeat films prior to discharge.

PEARLS AND PITFALLS

- Because the pleural cavity of an average 70 kg man can hold over 4 L of blood, an exsanguinating hemorrhage can occur without any evidence of external blood loss.
- Auscultation and percussion of a supine trauma patient with substantial hemothorax may produce equivocal findings due to distribution of blood along the entire posterior aspect of pleural space.
- Without a clear history of trauma, CXR may be incorrectly read as pneumonia.
- If there is a concurrent diaphragmatic injury, a hemothorax may have an intra-abdominal origin.
- Prepare for autotransfusion early, as most blood loss occurs on initial chest tube insertion.
- In the supine trauma patient, a common error in chest tube insertion is placement too anterior and superior, making complete drainage difficult.
- Be sure all chest tube fenestrations are within the thoracic cavity.
- Prophylactic antibiotics administered with chest tube insertion do not decrease the risk of pneumonia or empyema.

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CODES

ICD9

- 511.89 Other specified forms of effusion, except tuberculous
- 860.2 Traumatic hemothorax without mention of open wound into thorax
- 860.3 Traumatic hemothorax with open wound into thorax

ICD10

- J94.2 Hemothorax
- S27.1XXA Traumatic hemothorax, initial encounter
- S27.2XXA Traumatic hemopneumothorax, initial encounter

HENOCH–SCHÖNLEIN PURPURA

Karem Colindres Duque

BASICS

DESCRIPTION

Vasculitis

ETIOLOGY

Mechanism:

- Increased serum IgA:
 - Circulating IgA complexes
 - Glomerular mesangial deposition of IgA
- Although cause is undefined, there are many associated conditions:
 - Infections
 - Group A streptococcus
 - Mycoplasma
 - Viral: Varicella, Epstein–Barr (EB)
 - Drugs: Penicillin, tetracycline, aspirin, sulfonamides, erythromycin
 - Allergens: Insect bites, chocolate, milk, wheat
- Peak incidence: School-aged children and young adults
- More common in whites
- Males > females
- Occurs more often in winter/spring
- Multisystem involvement can lead to life-threatening or long-term complications:
 - Intussusception
 - Proliferative glomerulonephritis
 - Chronic renal failure:
 - More common in older children and adults (13–14%)
 - Intracranial hemorrhage

DIAGNOSIS

SIGNS AND SYMPTOMS

- General:
 - Well-appearing child, despite nature and extent of rash
 - Recent or current upper respiratory tract infection
 - Malaise
 - Low-grade fever
 - Hypertension, if associated renal failure
 - Children < 3 mo may have only skin manifestations

- Children <2 yr of age may have facial edema alone as presenting symptom
- Skin:
 - Purpuric rash:
 - Presenting sign in 50% of patients
 - 100% of patients develop purpura
 - 1st appears as pink rounded papules that blanch
 - Progresses to 2–3 cm circular palpable purpura within 24 hr; may be discrete or confluent
 - Rash begins in gravity-dependent areas of legs and buttocks, which may extend to upper extremities and trunk
 - Symmetric distribution
 - May involve lower back
 - Rarely involves the face
 - Rash recurs in up to 40% of patients (within 6 wk).
- Abdominal:
 - Abdominal pain:
 - 70–80% of cases
 - Colicky to severe
 - Abdominal findings may precede the rash by 4 wk.
 - GI bleeding:
 - 75% of cases
 - Occult to severe blood loss
 - Intussusception (ileoileal or ileocolic)
- Renal-genitourinary:
 - Asymptomatic hematuria:
 - Occurs in 80% of cases
 - Scrotal pain
 - Testicular swelling
 - Renal failure
- Extremities:
 - Arthritis:
 - 70–80% of cases
 - Migratory periarticular pain
 - Most frequent in knees and ankles
 - Angioedema
- Neurologic:
 - Headache
 - Seizure
 - Altered mental status
 - Focal deficits +/- visual abnormalities and verbal disability

History

- Constitutional symptoms:

- Fever
- Rash:
 - Location, timing, duration, and progression of rash
- Associated symptoms:
 - Abdominal pain, vomiting, and seldom facial edema
 - Timing, duration, and progression of symptoms
- Progression of symptoms:
 - Timing, duration, and progression of symptoms

Physical-Exam

- General appearance:
 - Level of responsiveness, vital signs (high BP)
- Cardiovascular:
 - Quality of heart tones
 - Perfusion (pulses, capillary refill)
- GI:
 - Abdominal distention, tenderness, palpable masses, bloody stools
- Genitourinary:
 - Testicular swelling, tenderness
- Skin:
 - Location
 - Blanching vs. nonblanching
 - Erythematous or purplish raised lesions (papules, purpura) vs. macular lesions (petechia)
 - Hemorrhagic bullous evolution seldom described in children
- Neurologic:
 - Level of consciousness
 - Presence of focal deficits

ESSENTIAL WORKUP

Exclude life-threatening causes of petechia, purpura, severe abdominal pain, hematuria, and CNS findings, if appropriate.

DIAGNOSIS TESTS & NTERPRETATION

Lab

- CBC:
 - Platelet count normal
 - WBC often elevated
- PT, PTT (if bleeding or in shock; or if unsure of diagnosis and concerned about possibility of coagulopathy)
- Electrolytes (if hypertension or urinalysis abnormal)
- BUN, creatinine (if hypertension or urinalysis abnormal):

- May be elevated in cases with serious renal complications
- Urinalysis:
 - Hematuria is common
 - Proteinuria is suggestive of glomerulonephritis
- Cultures to exclude common infections

Imaging

- Abdominal imaging studies:
 - Indicated if abdominal pain or GI bleeding
 - Flat and upright abdominal films of limited use
 - Abdominal US, barium enema, or CT scan may be necessary to rule out intussusception.
- Testicular US:
 - Indicated in patients with testicular pain and swelling
- Head CT:
 - Indicated if CNS findings to exclude bleed

Diagnostic Procedures/Surgery

Lumbar puncture, as clinically indicated

DIFFERENTIAL DIAGNOSIS

- Abdominal pain:
 - Gastroenteritis
 - Appendicitis
 - Inflammatory bowel disease
 - Intussusception
 - Meckel diverticulum
- Arthralgia:
 - Acute rheumatic fever
 - Polyarthriti nodosa
 - Juvenile rheumatoid arthritis
 - Systemic lupus erythematosus
- Rash:
 - Infection:
 - Meningococemia
 - Bacterial sepsis: Streptococcal or staphylococcal
 - Rocky Mountain spotted fever
 - Infectious mononucleosis
 - Bacterial endocarditis
 - Viral exanthem
 - Trauma/child abuse
 - Functional platelet disorders
 - Thrombocytopenia

- Vasculitis
- Erythema nodosum
- Drugs/toxins
- Renal disease:
 - Acute glomerulonephritis
- Testicular swelling:
 - Incarcerated hernia
 - Orchitis
 - Testicular torsion



TREATMENT

PRE HOSPITAL

Stabilize as clinically indicated

INITIAL STABILIZATION/THERAPY

- IV fluids for shock
- Packed RBCs for massive GI hemorrhage

ED TREATMENT/PROCEDURES

- Emergent intervention for life-threatening conditions, if any
- NSAIDs (ibuprofen):
 - Arthralgia
- Prednisone:
 - Severe abdominal pain once life-threatening pathology excluded
 - Painful SC edema or arthritis
 - Renal disease (high-dose pulse therapy required with methylprednisolone)
 - CNS involvement
- Immunosuppressant drugs:
 - Severe, life-threatening disease with HSP nephritis
- Polyclonal immunoglobulin therapy:
 - Severe, life-threatening disease (controversial)

MEDICATION

First Line

Ibuprofen: 600 mg (peds: 5–10 mg/kg per dose) PO q6h

Second Line

Prednisone: 60 mg (peds: 1–2 mg/kg/24 h) PO daily for 5–7 days



FOLLOW-UP

DISPOSITION

Admission Criteria

- Severe abdominal pain
- CNS findings
- GI bleeding
- Intussusception
- Evidence of renal failure

Discharge Criteria

- Normal platelet count
- Normal renal function
- Minimal or no abdominal pain
- If steroids started, follow up within 24 hr.

Issues for Referral

- GI:
 - Severe abdominal pain
- Renal:
 - Evidence of renal failure or insufficiency

FOLLOW-UP RECOMMENDATIONS

Primary care physician:

- Close monitor of BP. Recheck CBC, urinalysis as clinically indicated (recommended for at least 6 mo in children)

PEARLS AND PITFALLS

- Exclude life-threatening causes
- NSAIDs are usually adequate
- Most patients do not require systemic corticosteroids as it has not been shown to affect the prognosis of HSP nephritis

ADDITIONAL READING

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- Zaffanello M, Brugnara M, Franchini M. Therapy for children with Henoch Schonlein purpura nephritis: A systematic review. *ScientificWorldJournal.* 2007;7:20–30.



See Also (Topic, Algorithm, Electronic Media Element)

- [Intussusception](#)
- [Rash, Pediatric](#)



CODES

ICD9

287.0 Allergic purpura

ICD10

[D69.0 Allergic purpura](#)

HEPATIC ENCEPHALOPATHY

Sidney James • Matthew N. Graber

BASICS

DESCRIPTION

Hepatic encephalopathy (HE) is characterized by changes in behavior, consciousness, and motor disturbances, associated with hepatic insufficiency and the accumulation of substances normally metabolized by the liver. HE may result from a combination of:

- Accumulation of ammonia (NH_3) from:
 - Protein degradation by colonic bacteria
 - Deamination of glutamine in small bowel, kidney, and muscle.
 - Accumulated NH_3 crosses the blood–brain barrier. Astrocytes uptake NH_3 and metabolize it into glutamine which causes cellular swelling. Ultimately leads to cerebral edema and cerebral mitochondrial dysfunction.
- Accumulation of other neurotoxins:
 - Short-chain fatty acids
 - Manganese toxicity
 - Neurosteroids
 - Phenols
 - Mercaptans
 - Amino acids such as tryptophan
- Increased levels of inhibitory neurotransmitters:
 - Benzodiazepines
 - γ -aminobutyric acid (GABA)
 - Serotonin
- Decreased levels of excitatory neurotransmitters:
 - Glutamate
 - Dopamine
 - Aspartate
 - Catecholamines
- Other contributing factors to HE:
 - Decreased cerebral blood flow and oxygen
 - Increased glucose consumption and possible hypoglycemia
 - Zinc deficiency
- Genetics:
 - Inherited errors of the urea cycle

ETIOLOGY

- Classification based on the 11th World Congress of Gastroenterology:
 - Type A: HE associated with acute liver injury and fulminant hepatic failure

(FHF).

- Type B: HE associated with portosystemic bypass and no intrinsic liver disease.
- Type C: HE associated with cirrhosis and portal hypertension.
- Precipitating events:
 - GI bleeding (more common in elderly)
 - Hypokalemia and hyponatremia.
 - Alkalosis decreases renal NH_4 excretion.
 - Sepsis (e.g., spontaneous bacterial peritonitis [SBP])
 - Constipation
 - Noncompliance with treatment regimen in chronic liver failure
 - High-protein diet
 - Hypoglycemia
 - Hypovolemia (e.g., post large-volume paracentesis)
 - Azotemia (e.g., diuretic or diarrhea induced)
 - Narcotics or sedatives, including alcohol
 - Zinc deficiency as multiple urea cycle enzymes are zinc dependent
 - Hepatocellular injury
 - Viral- or drug-induced hepatitis
 - Post portosystemic shunt placement
 - Recurrent encephalopathy can occur without precipitating factors



DIAGNOSIS

SIGNS AND SYMPTOMS

- Type A: Rapidly progresses to seizures, decerebrate rigidity, coma, and frequently death from cerebral edema.
- Type B and C are chronic conditions that may manifest as minimal or overt HE. Overt HE may be classified as episodic or persistent:
 - Minimal HE: Characterized by impaired psychomotor speed, visual perception, and attention and concentration; slow mental processing; and memory loss. Only detected by psychometric testing.
 - Overt HE: Slow monotonous speech pattern, loss of fine motor skills, hyperreflexia, clonus, hyperventilation, extrapyramidal type movement disorder, seizures, confusion, coma, decerebrate/decorticate posturing.
 - Overt HE episodic: Characterized by short period of changes in consciousness over hours to days that usually return to a normal mental state with treatment. In persistent HE, patients do not return to a normal mental state.
- Grading (West Haven criteria):
 - Stage 0:
 - No apparent clinical changes

- Abnormal neurophysiologic and neuropsychological tests
- No asterixis
- Stage I:
 - Personality changes, irritability, depression, or euphoria
 - Reversal of normal sleep pattern
 - Impairment of writing, drawing, addition, subtraction
 - Asterixis may be present
- Stage II:
 - Significant behavioral changes, often inappropriate
 - Lethargy
 - Slow responses
 - Asterixis
 - Slurred speech
 - Ataxia
- Stage III:
 - Disorientation to time and place
 - Amnesia
 - Paranoia
 - Nystagmus
 - Hypoactive reflexes
 - Positive Babinski reflex
 - Semistupor to stupor
- Stage IV:
 - Dilated pupils
 - Stupor or coma

ESSENTIAL WORKUP

- Elicit history of liver disease and prior episodes of HE.
- Search for precipitating cause (particularly GI bleeding and infection).
- Check for electrolyte abnormalities:
 - Even minimal abnormalities may manifest as significant clinical changes.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Blood NH₃ level:
 - Level correlates poorly with the degree of HE or the presence of cerebral edema.
 - Helpful in detecting HE in cases of altered mental status (AMS) of unknown cause
 - Normal NH₃ level with suspected HE warrants search for other causes of AMS.

- Must be kept on ice and assayed within 30 min.
- Consider hemocult testing and nasogastric (NG) lavage to rule out GI bleeding
- CBC to search for anemia
- Electrolytes, BUN, creatinine, glucose
- PT/INR with elevations suggesting significant liver failure
- Liver profile/liver enzymes
- Urinalysis for possible infection
- Culture urine and ascitic fluid to search for infectious cause
- Toxicology screen for alternate cause of altered level of consciousness:
 - Acetaminophen and alcohol level as necessary
- Additional labs as clinical scenario dictates:
 - TSH
 - Blood gases
 - Magnesium
 - Viral serology

Imaging

- CXR for pneumonia and signs of CHF
- Head CT scan: For new-onset AMS, focal neurologic deficit, suspected cerebral edema, or trauma.
- MRI of the brain is especially helpful in diagnosing HE in patients with portosystemic shunts but no intrinsic liver disease.

Diagnostic Procedures/Surgery

- ECG for arrhythmia and electrolyte imbalance
- CSF exam:
 - For new-onset or unexplained worsening of HE
 - CSF glutamine level correlates with severity of HE.
- Paracentesis for SBP workup and culture of ascitic fluid
- EEG is usually abnormal, most commonly with generalized slowing and other nonspecific changes.

DIFFERENTIAL DIAGNOSIS

- Alcohol withdrawal syndromes including delirium tremens
- Cerebrovascular accident
- Congestive heart failure
- CO₂ narcosis
- Head trauma with concussion or intracranial hemorrhage
- Hypocalcemia and hypercalcemia
- Hypoglycemia
- Hypokalemia
- Meningitis or encephalitis

- Metabolic encephalopathy
- Neuropsychiatric disorders
- Toxic confusional states secondary to:
 - _ Sedative overdose
 - _ Alcohol intoxication
 - _ Illicit drugs
 - _ Medications
- Uremia

Pediatric Considerations

- Consider Reye syndrome early (most common cause of fulminate hepatic failure in children) even if PT is only mildly prolonged.
- Consider fatty acid β -oxidation disorder:
 - _ Freeze serum and urine sample for subsequent testing



TREATMENT

INITIAL STABILIZATION/THERAPY

- Oxygen
- Airway protection:
 - _ Patients with grade 3 or 4 HE may require intubation for airway protection. Propofol is the preferred agent for sedation.
- Cardiac monitor
- Fluid resuscitation
- Initial AMS treatment:
 - _ Naloxone
 - _ D₅₀W (or bedside glucose)
 - _ Thiamine

ED TREATMENT/PROCEDURES

Identification and removal of precipitating factors is key and may improve clinical picture alone.

ALERT

- Liver failure predisposes patients to both hypoglycemia and HE, and these can be additive to the clinical picture; therefore, frequent glucose checks are of absolute importance.
- Identification of early cerebral edema is important as brain perfusion must be preserved and herniation prevented (associated but not limited to grade 3/4 HE)
- Treatment of complicating conditions:
 - _ Acute GI bleeding

- Sepsis
- Electrolyte and pH abnormalities
- Coagulopathy
- Renal and electrolyte disturbances
- Avoid sedative/narcotics if possible:
 - If necessary, use agents not metabolized by the liver
- Increase NH₃ elimination:
 - Bowel cleansing with nonabsorbable sugars (i.e., lactulose is mainstay of treatment). Retention enema preferred in grade 3/4 HE
- Decrease NH₃-producing intestinal flora (in combination with lactulose):
 - Neomycin (nephrotoxic and ototoxic)
 - Metronidazole (PO)
 - Vancomycin (PO)
 - Rifaximin: Recommended for lactulose-resistant HE. Current data suggests it is as effective as lactulose and neomycin and has a favorable safety and tolerability profile, although more expensive
- Treat associated cerebral edema if present
- Correct zinc deficiency
- Short-term restriction of protein intake in diet
- Flumazenil in patients who have received benzodiazepines may provide moderate, short-term improvement.
 - Avoid flumazenil unless you are sure that the patient is not a chronic alcoholic or benzodiazepine user as resultant seizures may occur.
- Precautions to prevent bodily harm to the confused patient with HE
- Liver transplantation provides cure for severe, spontaneous, or recurrent HE
- Possibly of benefit:
 - L-carnitine
 - Albumin dialysis
 - Broad-spectrum antibiotic coverage
 - N-acetylcysteine
 - *Lactobacillus acidophilus* supplementation
 - Ornithine aspartate
 - Benzoate or Bromocriptine
 - Branched-chain amino acids
 - Chelation of manganese with edetate calcium disodium

MEDICATION

- Dextrose: 1–2 amps (25 g) of 50% dextrose (child: 2 mL/kg D₂₅W) IV
- Lactulose: 30 mL (peds: 0.3 mL/kg) PO/NG tube q6h titrated to produce 2 or 3 soft stools per day and stool pH < 5 or retention enema at 300 mL + 700 mL saline/tap water q4–6h
- Metronidazole: 250 mg PO/NG (peds: 10–30 mg/kg/d) q8h for 2 wk

- Narcan: 2 mg (peds: 0.1 mg/kg) IV/IM initial dose
- Neomycin: 1–3 g (peds: 50–100 mg/kg/d) PO q6h
- Rifaximin: 550 mg PO/NG q12h. (safety not established for children < 12 yr)
- Mannitol: 0.5 –1 g/kg IV
- Thiamine (vitamin B₁): 100 mg (peds: 50 mg) IV/IM
- Zinc acetate or sulfate: 220 mg PO/NG q8h



FOLLOW-UP

DISPOSITION

Admission Criteria

- HE grade 2, 3, or 4 or inadequate social support
- Type A HE (any grade) and type B or C (grade 2 or above) should be admitted to the ICU with urgent GI consult
- Associated complicating condition (GI bleeding and sepsis)
- Uncertainty about cause of AMS

Discharge Criteria

- Known chronic or intermittent HE
- Grade 0 or 1 with remediable cause
- Adequate supervision with close follow-up
- Those appropriate for discharge should go home with:
 - Low-protein diet
 - Lactulose prescription

Issues for Referral

- Refer to primary physician or GI for consideration of medication or diet changes if recurrent early stage HE episodes
- For any grade of type A HE, consider transfer to a liver transplant facility

FOLLOW-UP RECOMMENDATIONS

- Dietary consultation if possible cause of exacerbation
- Alcohol counseling if it is a concern

PEARLS AND PITFALLS

- Consider rifaximin for lactulose-resistant HE
- Hypoglycemia is common in HE patients.
- Avoid sedatives and narcotics if possible in HE patients. If necessary, use medications not metabolized by liver.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Hypoglycemia



CODES

ICD9

- 070.6 Unspecified viral hepatitis with hepatic coma
- 070.9 Unspecified viral hepatitis without mention of hepatic coma
- 572.2 Hepatic encephalopathy

ICD10

- B19.0 Unspecified viral hepatitis with hepatic coma
- K72.90 Hepatic failure, unspecified without coma
- K72.91 Hepatic failure, unspecified with coma

HEPATIC INJURY

Stephen R. Hayden

BASICS

DESCRIPTION

- The size and location of the liver places it at significant risk for injury:
 - The liver is the solid organ most frequently injured in penetrating trauma.
 - The liver is the 2nd most commonly injured in blunt abdominal trauma, 2nd to the spleen.
 - Highly susceptible to blunt injuries, by direct blow or deceleration forces
- Mechanism of injury and description of forces are important factors in evaluating patients for possible hepatic injury:
 - Blunt trauma:
 - Obtain information about the forces and direction (horizontal or vertical) of any deceleration or compressive forces.
 - Penetrating trauma:
 - Type and caliber of the weapon
 - Distance from the weapon
 - Variety and length of knife or impaling object
- Hepatic injuries are graded by severity, ranging from subcapsular hematoma and lacerations to severe hepatic fragmentation.
- Associated conditions include rib fractures and injuries to the spleen, diaphragm, kidney, lung, gallbladder, pancreas, and blood vessels.
- Overall mortality of hepatic injury is reported at 8–10%.
- More often nonoperative management is becoming more common in isolated blunt hepatic trauma.

Pediatric Considerations

Poorly developed musculature and relatively smaller anteroposterior diameter increase the vulnerability of liver to compressive forces in children.

ETIOLOGY

Trauma:

- Blunt mechanism:
 - Deceleration
 - Acceleration
 - Compression
- Penetrating mechanism:
 - Stab wound
 - Gunshot wound

- Impaled object



DIAGNOSIS

SIGNS AND SYMPTOMS

Physical exam and history can be variable.

History

History of trauma usually available from patient or pre-hospital providers

Physical-Exam

- Neither sensitive nor specific for hepatic injury
- Systemic signs related to acute blood loss:
 - May present with dizziness and weakness
 - Signs of shock including tachycardia and hypotension
- Local signs:
 - Right upper quadrant tenderness
 - Guarding
 - Abdominal distention
 - Rigidity
 - Rebound
 - Tenderness
 - Contusions/abrasions
 - Penetrating wounds to the right chest, flank, or abdomen

ESSENTIAL WORKUP

- Physical exam is unreliable.
- Objective evaluation includes imaging of the abdomen or surgical exploration.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- No hematologic lab studies are specific for diagnosis of injury to the liver.
- Obtain baseline hemoglobin level.
- Liver function tests are not helpful in the acute setting.
- Consider type and cross if active hemorrhage is suspected.

Imaging

- Consider imaging based on mechanism and physical exam.
- Plain abdominal radiographs:
 - Little value
- Bedside US:

- Screening tool for both blunt and penetrating abdominal trauma
- Procedure of choice in unstable patient
- May identify intra-abdominal fluid in the hepatorenal (Morison) pouch or parenchymal injuries
- Suggests intra-abdominal hemorrhage in patient with blunt, multiple-organ trauma
- CT scan with IV contrast:
 - Best depicts extent of hepatic injury and injuries to adjacent organs
 - Requires patient to be hemodynamically stable
 - Extravasation of IV contrast during arterial phase of CT injection indicative of vascular or high-grade liver injury requiring surgical intervention

Diagnostic Procedures/Surgery

- Diagnostic peritoneal lavage (DPL):
 - Rarely performed; usually done in conjunction with trauma surgeon
 - Sensitive for presence of hemoperitoneum
 - Nonspecific for source of bleeding
 - Surgery and exploratory laparotomy if positive
 - Operative management may be necessary for unstable patients and those with high-grade lesions.
- Lower-grade injuries have been increasingly managed successfully without surgery.

DIFFERENTIAL DIAGNOSIS

- Other causes of intraperitoneal injury
- Retroperitoneal injury
- Thoracic injury
- Diaphragmatic injury
- Splenic injury
- Vascular injury



TREATMENT

PRE HOSPITAL

- Obtain details of mechanism of injury.
- Initiate large-bore IV access:
 - Hemorrhage may be rapid and life-threatening.
- Moist saline dressings over penetrating wounds or evisceration
- Direct pressure to control active bleeding
- Full spinal immobilization except in isolated penetrating trauma

INITIAL STABILIZATION/THERAPY

ABCs:

- Control airway as needed; may have associated injuries including head injury.
- Supplemental oxygen, cardiac monitor, pulse oximetry
- Adequate IV access, including central line, intraosseous line, and cut down as dictated by the patient's status
- Fluid resuscitation, initially with 2 L of crystalloid (normal saline or Ringer lactate), followed by blood products such as packed red blood cells. Consider fresh frozen plasma (FFP) as needed.

ED TREATMENT/PROCEDURES

- Immediate laparotomy may be appropriate in the acutely injured patient with the following conditions:
 - Hemodynamic instability
 - Gunshot wounds to the anterior abdomen
 - Frank signs of intraperitoneal hemorrhage
 - Indications based on diagnostic procedures, such as DPL
 - Failure of nonoperative management
- Stab wounds can be managed by local wound exploration followed by US or DPL when intraperitoneal penetration is not demonstrated or equivocal.
- Consider nonoperative management for the following patients:
 - Hemodynamically stable with normal mental status
 - No evidence of other intra-abdominal injury
 - Isolated low-grade (1–3) hepatic injury confirmed by imaging study
 - Transfusion requirements ≤ 2 U of packed red blood cells
- High-grade liver injuries (4 and 5) have less successful nonoperative rates.
- Diet: Nothing per mouth (NPO)
- Activity: Strict bedrest
- Special therapy:
 - Angiography with embolization: Selective use in patients with persistent bleeding may decrease the need for operative management and blood transfusions
 - Factor VIIa and prothrombin complex have been used as an adjunct in nonoperative management to control significant bleeding.

MEDICATION

- Crystalloid IV fluids: NS or lactated Ringer
- Packed red blood cells
- FFP
- Recombinant factor VIIa: 15–30 $\mu\text{g}/\text{kg}$ IV bolus every 4–6 hr until hemostasis is achieved
- Prothrombin complex concentrate (PCC): 50 U/kg IV



FOLLOW-UP

DISPOSITION

Admission Criteria

- All patients with hepatic injury require hospitalization for definitive laparotomy or close hemodynamic observation with serial exams or CT scans, as well as hematocrit measurements.
- ICU admission is often indicated in the 1st 48 hr after injury.

Discharge Criteria

Patients with proven or suspected hepatic injuries should not be discharged.

Issues for Referral

Report all gunshot and stab wounds to appropriate local authorities.

FOLLOW-UP RECOMMENDATIONS

Follow-up US, physical exam, and hematocrits are crucial in noting changes in initially benign presentations.

PEARLS AND PITFALLS

- Obtain early surgical consultation in unstable patients.
- Failing to obtain appropriate and adequate imaging studies is a pitfall.
- Do not rely on negative US to rule out hepatic injury.
- Failing to adequately resuscitate with IV fluids and blood products is a pitfall.
- If hepatic injury is confirmed, ensure no trauma to surrounding organ systems.
- Check for pregnancy in women.

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See Also (Topic, Algorithm, Electronic Media Element)

- Abdominal Trauma, Blunt
- Abdominal Trauma, Penetrating

- Abdominal Trauma, Imaging
- Colon Trauma
- Diaphragmatic Trauma
- Pancreatic Trauma
- Renal Injury
- Small Bowel Injury
- Trauma, Multiple



CODES

ICD9

- 864.00 Injury to liver without mention of open wound into cavity, unspecified injury
- 864.10 Injury to liver with open wound into cavity, unspecified injury
- 864.11 Injury to liver with open wound into cavity, hematoma and contusion

ICD10

- S36.112A Contusion of liver, initial encounter
- S36.118A Other injury of liver, initial encounter
- S36.119A Unspecified injury of liver, initial encounter

HEPATITIS

Jason Koob • Christopher T. Richards

BASICS

DESCRIPTION

- Inflammation of the liver owing to infectious, toxic, and autoimmune disorders
 - Progression from hepatocellular injury (hepatitis) to scarring (cirrhosis)
- Infectious causes are the most common

ETIOLOGY

- Unknown etiology in 5–10% of acute and chronic hepatitis cases and up to 50% in fulminant hepatic failure (FHF)
- Hepatitis A (HAV):
 - Transmission: Fecal–oral
 - Incubation period: 2–7 wk
 - FHF in 0.1%
 - No chronic phase
 - 10% will have a relapsing course over months
- Hepatitis B (HBV):
 - Transmission: Mucous membrane, percutaneous exposure to bodily fluids, perinatal
 - Incubation period: 8–22 wk
 - Subclinical in 70%
 - FHF in 1%
 - Risk of chronic hepatitis increased with age at infection and comorbidities:
 - Neonatal: > 90%
 - Infant: 50%
 - Child: 20%
 - Immunocompetent adult: 1–5%
 - Immunocompromised adult: 50%
 - Risk of cirrhosis, hepatocellular carcinoma
- Hepatitis C (HCV):
 - Transmission: Blood >> sexual and perinatal
 - Incubation period: 6–10 wk
 - FHF rare
 - 80% progress to chronic disease
 - Risk of cirrhosis, hepatocellular carcinoma
- Hepatitis D (HDV):
 - HDV can be transmitted only in the presence of HBV infection
 - 2 patterns of infection: Simultaneous infection with HBV and HDV or

HDV superinfection in an individual with chronic HBV

- Same transmission as HBV
- Incubation period: 3–7 wk
- FHF in 3%
- 5% progress to chronic disease
- Risk of cirrhosis 3 times higher in HDV-infected individual compared with HBV infection alone
- Hepatitis E (HEV):
 - Most common cause of acute hepatitis and jaundice worldwide
 - Rarely found outside developing countries
 - Typically seen as outbreaks
 - Transmission: Fecal–oral, waterborne, foodborne
 - Incubation period: 3–8 wk
 - FHF in 10%
 - Chronic infection almost exclusively in immunocompromised persons
- Alcoholic hepatitis:
 - Associated with > 14 drinks/wk in women and > 21 drinks/wk in men
 - Sequelae of chronic use:
 - Hepatic steatosis in 90–100%
 - Hepatitis in 10–35%
 - Cirrhosis in 5–15%
 - Increased association with chronic viral hepatitis
 - Maddrey discriminant function (MDF) ≥ 32 associated with only 50–65% survival
 - $MDF = 4.6 \times [\text{prolongation of PT above control}](s) + \text{serum bilirubin}(mg/dL)$
- Abscess-induced hepatitis:
 - Entamoeba histolytica, pyogenic
- Secondary hepatitis viruses:
 - CMV, EBV, HSV, HIV
- Medication and toxin induced
- Autoimmune hepatitis:
 - Display concurrent stigmata of autoimmune disease

Pediatric Considerations

- Vast majority of cases are caused by HAV
- Perinatal HBV infection develops into chronic disease 90% of the time

Pregnancy Considerations

- 20% case fatality for HEV during pregnancy.
- Acute fatty liver of pregnancy (AFLP):
 - May progress to DIC

- Hemolysis, Elevated Liver enzymes, and Low Platelets (HELLP) syndrome
- Immunoprophylaxis is safe during pregnancy



DIAGNOSIS

SIGNS AND SYMPTOMS

- Often asymptomatic
- Preicteric phase:
 - Often misdiagnosed as a nonspecific viral syndrome or gastroenteritis
- Icteric phase:
 - Present in 70% of HAV, 30% of HBV, and 20% of HCV
- FHF:
 - Coagulopathy
 - Encephalopathy
 - Cerebral edema

History

- Preicteric phase:
 - Fever, chills
 - Malaise
 - Nausea, vomiting, anorexia
 - Arthralgia
 - Aversion to smoking
- Icteric phase:
 - Jaundice
 - Dark urine
 - Light stools
 - Pruritus
 - Rash
 - Right upper quadrant pain
- FHF:
 - Bleeding
 - Altered mental status

Physical-Exam

- Preicteric phase:
 - Fever
 - Arthritis
 - Dehydration
- Icteric phase:
 - Fever
 - Icterus of skin, sclerae, mucous membranes, and tympanic membranes

- Nonspecific maculopapular or urticarial rash
- Dehydration
- Tender hepatomegaly
- FHF:
 - Bruising
 - Disorientation
 - Asterixis

ESSENTIAL WORKUP

- Detailed history of risk factors for hepatitis, including toxic exposure and drug use
- Viral serologies are the mainstay of diagnosis of viral causes

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC with differential
- Basic metabolic panel:
 - Azotemia with hepatorenal syndrome in FHF
 - Hypoglycemia with severe liver damage
 - Hyponatremia
- LFTs:
 - Elevation in transaminases reflects hepatocellular injury
 - Degree of elevation does not always correlate with severity
 - If alkaline phosphatase more than 4 times normal, consider primary cholestatic process rather than viral hepatitis.
 - Mild to moderate elevation of conjugated bilirubin due to decreased excretion
- Amylase, lipase may indicate pancreatic or biliary etiology
- PT/PTT/INR, albumin
 - Measure of synthetic function of liver
 - Prolonged INR reflects more severe injury
- Ammonia level:
 - For patients with altered mental status
- Viral serologies:
 - HAV:
 - Anti-HAV IgM: Acute infection
 - Anti-HAV IgG: Previous exposure, immunity
 - HBV:
 - HBsAg: Acute infection (appears before symptoms), chronic infection
 - Anti-HBs: Past infection, carrier state, postimmunization
 - Anti-HBc IgM: Acute infection
 - Anti-HBc IgG: Past infection, chronic infection, carrier state
 - HBeAg: Acute infection, some chronic states

- Anti-HBe: Past infection, chronic infection, carrier state
- Postimmunization: Anti-HBs only
- HCV:
 - Anti-HCV: Acute infection, chronic infections, first-line test
 - HCV RNA: Acute infection, chronic infections; confirmatory
- HDV: Anti-HDV or viral RNA, not routine
- HEV:
 - Anti-HEV IgM: Acute infection, detectable for only 3–12 mo
 - Anti-HEV IgG: Persists for years, if not for life
- α -fetoprotein:
 - For chronic HBV or HCV to evaluate for hepatocellular carcinoma
- Monospot: For EBV
- Urinalysis for bilirubin

Imaging

- Head CT to evaluate hepatic encephalopathy
- RUQ US to evaluate for biliary obstruction

DIFFERENTIAL DIAGNOSIS

- Nonalcoholic fatty liver disease
- Cholecystitis and cholangitis
- Reye syndrome
- Liver abscess
- Wilson disease
- Heat stroke
- Fitz-Hugh—Curtis syndrome
- Ischemic hepatitis (“shock liver”)
- Congestive heart failure
- Hemochromatosis
- Budd–Chiari syndrome



TREATMENT

INITIAL STABILIZATION/THERAPY

ABCs and IV fluid resuscitation for FHF and severe hepatic encephalopathy.

ED TREATMENT/PROCEDURES

- Treat hypovolemia judiciously with isotonic fluids
- Correct electrolyte imbalance
- Treat vomiting with ondansetron and metoclopramide
- Avoid hepatotoxic agents: Acetaminophen, alcohol, phenothiazines
- Avoid medications metabolized by liver

- Propofol for sedation preferred
- Fentanyl for pain preferred
- Correct coagulopathy if active bleeding.
- N-acetylcystine (NAC) for acetaminophen-induced hepatitis and consider for FHF
- Consider steroids for severe acute alcoholic hepatitis
- Ursodeoxycholic acid or cholestyramine for cholestasis-induced itching
- Paracentesis for tense ascites leading to respiratory compromise
- Antidotes and activated charcoal for select ingestions
- Postexposure prophylaxis (PEP):
 - HAV:
 - HAV IG 0.02 mL/kg IM within 2 wk of exposure
 - HAV vaccine 1 mL (peds: 0.5 mL) IM
 - HBV:
 - HBV IG 0.06 mL/kg IM within 7 days of exposure
 - HBV vaccine 1 mL (peds: 0.5 mL) IM
 - No effective immunoprophylaxis for HCV or HDV
 - HEV vaccine not available in US

MEDICATION

- Cholestyramine: 4 g PO 2–4 times per day for pruritus
- Metoclopramide: 10 mg IV/IM q6–8h, 10–30 mg PO QID
- NAC 140 mg/kg IV loading dose
- Ondansetron 4 mg IV
- Prednisone 40 mg/d PO
- Thiamine: 100 mg (peds: 50 mg) IV/IM/PO:
 - Prior to glucose if malnourished
- Ursodeoxycholic acid: 3 mg/kg TID
- Vitamin K 10mg IV/PO



FOLLOW-UP

DISPOSITION

Admission Criteria

- Intractable vomiting, dehydration, or electrolyte imbalance not responding to ED treatment
- ICU and consider transfer to transplant center for FHF and acute hepatitis with evidence of significant liver dysfunction:
 - PT > 50% of normal or INR > 1.5
 - Bilirubin > 20 mg/dL
 - Hypoglycemia
 - Albumin < 2.5 g/dL

- Hepatic encephalopathy
- Pregnancy
- Immunocompromised host
- Possibility of toxic hepatitis
- Age > 50

Discharge Criteria

- Normalized electrolytes
- PO tolerance
- Mild hepatic impairment

Issues for Referral

- Hepatology, gastroenterology, and/or infectious disease follow-up for further serologic diagnosis and definitive treatment
- Alcoholics anonymous referral and social work referral for alcohol-related disease

FOLLOW-UP RECOMMENDATIONS

- Strict personal hygiene instructions
- Avoid acetaminophen and alcohol
- Avoid prolonged physical exertion

PEARLS AND PITFALLS

- Acute hepatitis is often misdiagnosed as a nonspecific viral syndrome—screen with urinalysis or serum LFTs
- ED treatment is primarily supportive
- Ask detailed social and travel history
- Early transfer to transplant center for FHF
- Counsel patient on prevention – vaccinations and personal hygiene precautions
- Maintain high index of suspicion for AFLP and HELLP in pregnant patients with compatible symptoms

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- [Acetaminophen Poisoning](#)
- [Ascites](#)
- [Cirrhosis](#)
- [Hepatic Encephalopathy](#)
- [Hepatorenal Syndrome](#)
- [Jaundice](#)
- [Mushroom Poisoning](#)

We wish to acknowledge the previous authors of this chapter for their contributions on this topic: Michael Schmidt, Amer Aldeen, and LucasRoseire.



CODES

ICD9

- [070.1 Viral hepatitis A without mention of hepatic coma](#)
- [070.30 Viral hepatitis B without mention of hepatic coma, acute or unspecified, without mention of hepatitis delta](#)
- [573.3 Hepatitis, unspecified](#)

ICD10

- [B15.9 Hepatitis A without hepatic coma](#)
- [B19.10 Unspecified viral hepatitis B without hepatic coma](#)
- [K75.9 Inflammatory liver disease, unspecified](#)

HEPATORENAL SYNDROME

Matthew T. Keadey • Richard D. McCormick



BASICS

DESCRIPTION

- Renal failure (RF) in patients with acute or chronic liver disease with no other identifiable cause of renal pathology.
- Hepatorenal syndrome (HRS) represents significant decline in renal perfusion due to severe liver disease:
 - Type I HRS:
 - Acute form with spontaneous RF in patients with liver disease
 - Rapidly progressive
 - Decrease in creatinine clearance (CrCl) by 50% or doubling of Cr in 2 wk (>2.5)
 - 90% mortality within 3 mo
 - Seen with acute liver failure or alcoholic hepatitis
 - Oliguric or anuric
 - Type II HRS:
 - Slow course of RF
 - Seen in patients with diuretic resistant ascites
 - Lower mortality rate than type I HRS
- Hallmarks of HRS:
 - Prerenal disease
 - Reversible renal vasoconstriction and mild systemic hypotension
 - Kidneys have normal histology and structure.
 - Lack of improvement in renal function after volume expansion
- Liver disease causes systemic vasodilation with decrease in arterial blood volume:
 - Reflex activation of sympathetic nervous system
 - Activation of rennin–angiotensin–aldosterone system (RAAS)
 - Stimulation of numerous vasoactive substances:
 - Nitric oxide
 - Prostacyclin
 - Atrial natriuretic peptide (ANP)
 - Arachidonic acid metabolites
 - Platelet-activating factor
 - Endothelins
 - Catecholamines
 - Angiotensin II
 - Thromboxane
- Action of vasoconstrictors prevails over vasodilator effects:

- Renal hypoperfusion ensues due to renal cortical vasoconstriction.
- Decrease in renal blood flow and glomerular filtration rates (GFRs)
- Decreased urine sodium excretion (U Na < 10 mEq/day)
- Incidence of HRS:
 - 18% at 1st year, 39% at 5 yr
- Hyponatremia and high plasma renin levels are risk factors.

ETIOLOGY

- Chronic liver disease, especially alcohol related (cirrhosis, severe alcoholic hepatitis)
- Fulminate hepatic failure
- Precipitating factors:
 - Decreased effective blood volume:
 - GI bleeding
 - Vigorous diuresis
 - Large-volume paracentesis
 - Use of nephrotoxic agent:
 - NSAIDs
 - Aminoglycoside
 - Sepsis:
 - Spontaneous bacterial peritonitis (SBP) leads to a 33% chance of developing RF during that year
 - Prophylaxis of SBP reduces the chance of developing acute RF

DIAGNOSIS

SIGNS AND SYMPTOMS

Signs of acute or chronic liver disease:

- Signs of portal hypertension
- Ascites, often tense
- Progressive oliguria
- Jaundice or hepatic encephalopathy
- Coagulopathy
- Tachycardia
- Hypotension
- Dyspnea, tachypnea due to tense ascites

History

Acute or chronic hepatic disease with advanced hepatic failure and portal hypertension:

- Worsening liver function often predates acute renal dysfunction

Physical-Exam

- Consistent with severe hepatic disease
- Vital signs may show:
 - Fever in signs of sepsis
 - Hypotension in sepsis, intestinal bleeding, or even a low baseline intrinsic to liver disease

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC:
 - Anemia due to GI bleed
- Electrolytes:
 - Hyperkalemia
 - Acidosis
- Glucose
- Elevated BUN, creatinine (Cr):
 - Normal Cr found with low GFR) in association with muscle wasting, poor nutrition, and ascites.
 - Cr increased by some medications (cimetidine, trimethoprim, and spironolactone) due to inhibition of tubular secretion of Cr.
 - Hyperbilirubinemia can create a falsely lower serum Cr.
- PT, PTT
- Urinalysis:
 - Absence of casts distinguishes HRS from acute tubular necrosis (ATN).
 - Check for UTI.
- Spot urine sodium and Cr, and serum and urine osmolality:
 - Spot urine $\text{Na}^+ < 10 \text{ mEq/L}$
 - Fractional excretion of $\text{Na}^+ < 1\%$
 - Urine/plasma Cr $> 30:1$
 - Hyperosmolar urine $>$ plasma osmolality
- 24 hr urine output (low in the absence of diuretics)
- 24 hr urine CrCl:
 - Accurately assess GFR
- Blood, ascitic fluid, and urine culture as indicated
- Urinary excretion of β_2 -microglobulin—useful marker of acute tubular damage

Imaging

- CXR for signs of CHF or fluid overload
- Renal US: Rule out obstructive uropathy:
 - Duplex Doppler US can be used to assess degree of renal vasoconstriction.

Diagnostic Procedures/Surgery

- ECG for dysrhythmia or signs of hyperkalemia
- Foley catheter placement to assess for urine output and exclude urinary retention as cause of RF
- Central venous pressure (CVP) measurements may help assess volume status:
 - Differentiates prerenal (low) from HRS (elevated)

DIFFERENTIAL DIAGNOSIS

- HRS is diagnosis of exclusion
- Glomerulopathy:
 - Hepatitis B can lead to glomerulonephritis
 - Hepatitis C can cause intrinsic renal damage due to cryoglobulinemia
- ATN:
 - Urine sodium > 30 mEq/L
 - Urine osmolality equals plasma osmolality
 - Urine casts and cellular debris
- Prerenal azotemia:
 - Over diuresis
 - GI bleeding
 - Urine output improves following correction of hypovolemia
- Obstructive uropathy
- Infections or sepsis
- Medications—NSAIDs
- Interstitial nephritis
- Post liver transplant renal dysfunction due to:
 - HRS due to failure of transplanted liver
 - Medications (e.g., cyclosporine)
 - Pre-existing renal disease
 - Perioperative hypovolemia



TREATMENT

PRE HOSPITAL

Attention to ABCs:

- Airway control may be a concern in severe encephalopathy.
- Respiratory failure seen with tense ascites as well as volume overload
- Correction of hypotension and ensure adequate IV access

INITIAL STABILIZATION/THERAPY

- ABCs
- Aggressive correction of hypovolemia with:
 - 0.9% NS IV fluid
 - Colloid volume expanders: 100 g albumin in 500 mL of NS

- Closely monitor clinical status including use of CVP
- Urine output should improve with correction of prerenal azotemia
- Manage life-threatening emergencies of RF:
 - Hyperkalemia
 - Severe acidosis
 - Hypoxemia
 - Uremic pericarditis

ED TREATMENT/PROCEDURES

- Exclude reversible or treatable causes of HRS.
- Supportive care until hepatic function recovers
- *Do no harm*—discontinue potentially nephrotoxic agents:
 - NSAIDs
 - Aminoglycosides
 - Demeclocycline
- Treat primary disease
- Search for and treat coexisting renal disease
- Correct electrolyte imbalances
- Treat any associated cardiopulmonary disorder and hypoxia
- Initiate broad-spectrum antibiotics if sepsis suspected
- Correct liver-associated complications:
 - Obstructive jaundice
 - Hepatic encephalopathy
 - Hypoglycemia
 - Peritonitis
- Consider large-volume paracentesis with IV albumin replacement (to relieve tense ascites):
 - Increases renal blood flow
 - May briefly improve HRS
- Transhepatic intrahepatic portosystemic shunt (TIPS):
 - Promising results, but small studies
 - Those who survived the procedure had 40% survival at 12 mo compared to 90% at 3 mo.
- Dialysis:
 - Useful in correcting fluid, electrolytes, acid–base imbalances, pulmonary edema
 - Indicated for patients who have likelihood of hepatic regeneration, hepatic recovery, or liver transplantation
- Liver transplant:
 - Is currently the only definitive therapy

MEDICATION

- No medications are first line and should only be considered after other causes of

renal dysfunction excluded

- Dopamine (renal dose): 2–5 µg/kg/min:
 - May improve renal function
 - Not curative
- Midodrine (7.5–12.5 mg PO TID) and octreotide (100–200 µg SC TID):
 - Octreotide is the analog of somatostatin
 - Midodrine is a sympathomimetic drug
- Misoprostol: 0.4 mg PO QID:
 - Synthetic analog of prostaglandin E₁
- Ornipressin:
 - Vasopressin analog
 - Increases renal perfusion pressure and function
 - Not available in US
- Terlipressin: 2 mg/d for 2 days:
 - Synthetic analog of vasopressin
 - Intrinsic vasoconstrictor activity
 - Not available in US



FOLLOW-UP

DISPOSITION

Admission Criteria

- All suspected HRS with GI and nephrology consults
- ICU admission for associated cardiopulmonary disease, hepatic encephalopathy, marked electrolyte imbalances

Discharge Criteria

None

PEARLS AND PITFALLS

Any degree of renal dysfunction needs to be evaluated very seriously in patients with liver disease.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Hepatic Encephalopathy
- Hepatitis



CODES

ICD9

- 572.4 Hepatorenal syndrome
- 997.49 Other digestive system complications

ICD10

- K76.7 Hepatorenal syndrome
- K91.83 Postprocedural hepatorenal syndrome

HERNIAS

Jenny J. Lu



BASICS

DESCRIPTION

- Protrusion of bodily structure or organ through a defect in tissues normally containing it.
- Classified as external (hernia protrudes visibly to outside), internal (herniated contents occur within body cavity), or interparietal (hernial sac contained within abdominal wall)
- Abdominal wall hernia due to weakness or disruption of fibromuscular layer of abdominal wall
- “Groin” hernias include femoral, direct, and indirect inguinal hernias
- “Ventral” hernias include epigastric, umbilical, and spigelian hernias
- External:
 - Indirect inguinal hernia:
 - Results from persistent process vaginalis
 - Peritoneal contents herniate through internal ring
 - Right side more common than left
 - 27% lifetime risk of repair for men; 3% for women
 - Direct inguinal hernia:
 - Due to weakness or defect in transversalis area in Hesselbach triangle:
 - Inguinal ligament inferiorly
 - Inferior epigastric vessels laterally
 - Lateral border of rectus abdominus medially
 - Incisional hernia:
 - Resultant breakdown of previous surgical fascial closure
 - Femoral hernia:
 - Peritoneum herniates into femoral canal beneath inguinal ligament.
 - Incarceration frequent due to protrusion through small orifice
 - Internal: Diaphragmatic, hernias from mesenteric/omental tears, foramen of Winslow
- Other hernias:
 - Obturator (pelvic) hernia:
 - Passes through obturator membrane and exits beneath pectineal muscle
 - Epigastric hernia:
 - Midline between xiphoid and umbilicus
 - Spigelian hernia:

- Protrusion through oblique fascia lateral to rectus abdominus muscle
- Lumbar hernia:
 - Occur in superior and inferior lumbar triangle of posterior abdominal wall (incarcerate in 25% cases)
 - Usually middle-aged men, chronic low back pain with palpable mass
- Umbilical hernia:
 - Congenital failure of umbilical ring to close
 - Protrusion through fibromuscular umbilical ring/umbilicus
 - Often incarcerate in adults, although rarely in infants (often spontaneously close)
 - 20–45% recurrence rate

EPIDEMIOLOGY

- Hernia repair (herniorrhaphy) extremely common general surgical procedure (>750,000 performed in US annually)
- Prevalence: 5% of population
- Groin and femoral hernias account for 85% of hernias:
 - Umbilical and incisional hernias account for additional 10%

ETIOLOGY

- Reducible hernia:
 - Protruding structures can be returned to abdominal cavity
- Incarcerated hernia:
 - Contents of hernia cannot be manipulated back into abdominal cavity
- Strangulated hernia:
 - Vascular compromise of entrapped bowel contained within hernia leading to ischemia and gangrene (skin color changes may be apparent)
 - Higher risk in hernias with small openings and large sacs
 - Signs and symptoms of bowel obstruction or ischemia may occur (nausea/vomiting, fever, leukocytosis)

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Pain and swelling:
 - Localized to region of hernia
- Persistent pain, vomiting, fever may indicate:
 - Incarceration
 - Strangulation
 - Bowel obstruction

Physical-Exam

- Vital signs:
 - Frequently normal
 - Tachycardia with pain, dehydration, infection
 - Hypotension with dehydration, strangulation, infection/sepsis
 - Fever with infection/sepsis
 - Skin color changes with strangulation
- Inguinal hernia:
 - Pain:
 - Localized to inguinal region
 - Exacerbated by straining/positional changes
 - Relieved by rest
 - Swelling:
 - Males: Bulge in scrotum
 - Females: Bulge immediately inferior to inguinal ligament or in labia
 - Swelling of spermatic cord, scrotum, or testes
 - Valsalva maneuver performed with finger directed toward internal ring—may allow hernia sac to descend against finger
- Femoral hernia:
 - Pain/swelling:
 - Localized to femoral orifice inferior to inguinal ligament
- Incisional hernia:
 - Pain/swelling:
 - Localized to previous incision/scar
- Obturator hernia:
 - Nonspecific abdominal pain
 - Intermittent intestinal obstruction
 - Weight loss
 - Pain:
 - Owing to pressure on obturator nerve from hernia (Howship–Romberg sign)
 - Along medial thigh
 - Radiating to hip
 - Relieved with thigh flexion
 - Exacerbated by hip extension, adduction, or external rotation
- Spigelian hernia:
 - Abdominal pain/mass along anterior abdominal wall
 - Increased pain with maneuvers increasing intra-abdominal pressure
 - Intermittent bowel obstruction
 - Palpable mass along spigelian line:
 - Convex line extending from costal arch to pubic tubercle along lateral edge of rectus muscle

Pediatric Considerations

- Diagnosis often difficult:
 - Parents describe bulge in inguinal area often no longer present at time of exam.
 - Incarcerated hernias may present with irritability, abdominal pain, or intermittent vomiting.
- Incidence of incarceration/strangulation is 10–20%:
 - >50% in patients younger than 6 mo of age
 - Incidence of incarceration higher in girls than boys
- Umbilical hernias:
 - Strangulation and incarceration rare
 - Most close spontaneously
 - Most surgeons will delay closure until 4 yr of age, although timing is controversial
- Inguinal hernias (consider hydrocele):
 - If hydrocele, neck narrows at external inguinal canal without extension into inguinal canal

Pregnancy Considerations

- Hernias uncommon during pregnancy, manifesting before or during
- Inguinal hernia: 1:1,000–3,000 incidence, 75% occurring in multiparas
- Recognition of emergent situations (incarceration, strangulation) may be a diagnostic and management challenge
- No consensus exists regarding treatment of unreducible hernia during pregnancy; complications during pregnancy may outweigh elective hernioplasty and emergent surgical consultation recommended

Geriatric Considerations

- Higher risk of bowel resection if older than 65 years of age with incarcerated hernias
- Higher postoperative pulmonary and cardiovascular complications

ESSENTIAL WORKUP

Careful history and physical exam:

- Palpate inguinal/femoral area for tenderness/masses.
- Attempt exam with the patient standing or straining (Valsalva maneuver) if hernia not obvious.
- Pelvic exam in women to evaluate gynecologic etiologies of groin pain

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC:

- Leukocytosis with strangulation
- Electrolytes, BUN/creatinine, glucose:
 - If vomiting/dehydration
- Urinalysis:
 - Genitourinary causes of groin pain

Imaging

- Plain abdominal radiographs:
 - Obstructive bowel pattern with incarceration or strangulation
- US:
 - For identifying masses in groin or abdominal wall
 - May be difficult in obese patients
- CT:
 - To diagnose obturator or spigelian hernia
 - Consider in symptomatic patients in whom body habitus precludes adequate physical exam or US study

DIFFERENTIAL DIAGNOSIS

- Hydrocele
- Varicocele
- Lymphadenitis
- Testicular torsion
- Testicular tumor
- Undescended testis
- Renal calculi
- UTI
- Ovarian torsion
- Lymphogranuloma venereum



TREATMENT

INITIAL STABILIZATION/THERAPY

- 0.9% NS IV fluid resuscitation for dehydration, bowel strangulation, obstruction, or sepsis:
 - Adults: 1 L bolus
 - Peds: 20 mL/kg bolus

ED TREATMENT/PROCEDURES

- Incarcerated or strangulated hernias:
 - IVFs
 - Nasogastric tube (NGT)
 - Surgical consultation

- Preoperative broad-spectrum antibiotics for strangulated hernia (controversial)
- Hernia reduction procedure:
 - IV sedation (benzodiazepines) and analgesia (opiates) if necessary
 - Place patient in Trendelenburg position.
 - For spontaneous reduction, allow 20–30 min
 - For manual reduction:
 - Place constant, gentle pressure on hernia.
 - For inguinal hernias, achieve reduction by putting fingers of 1 hand on internal ring while gently pulling then pressing on hernia distal to external ring.
 - Obtain surgical consultation if reduction is unsuccessful after 1 or 2 attempts.
 - Contraindications to reduction include:
 - Fever
 - Leukocytosis
 - Signs of strangulation
 - Complications:
 - Introduction of strangulated bowel into abdomen
 - Further ischemia/necrosis occurs with no clinical improvement.
 - Reduction in girls may be more difficult if ovary encased within hernia.

MEDICATION

- Analgesics:
 - Morphine sulfate: 2–10 mg per dose (peds: 0.1–0.2 mg/kg IV/IM/SC q2–4h) IV/IM/SC
 - Fentanyl: 1–4 µg/kg (peds: 1–4 µg/kg IV) IV
- Sedatives:
 - Lorazepam: 1–2 mg IV
 - Midazolam: 2.5–5 mg (peds: 0.07 mg/kg) IV



FOLLOW-UP

DISPOSITION

Admission Criteria

- Strangulated hernias require immediate surgical intervention.
- Incarcerated hernias require admission for urgent surgical intervention.
- Intestinal obstruction
- Peritonitis
- Vomiting/dehydration
- Severe pain

Discharge Criteria

After successful reduction has been achieved and patient asymptomatic

Issues for Referral

Referral to surgery with instructions to return if recurrent persistent pain, fever, vomiting

FOLLOW-UP RECOMMENDATIONS

General surgery referral

PEARLS AND PITFALLS

- Failure to recognize signs and symptoms of an incarcerated or strangulated hernia
- Forcing reduction of incarcerated hernia
- Reintroducing strangulated bowel back into abdominal cavity

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Abdominal Pain



CODES

ICD9

- 553.00 Femoral hernia without mention of obstruction or gangrene, unilateral or unspecified(not specified as recurrent)
- 553.9 Hernia of unspecified site without mention of obstruction or gangrene
- 550.90 Inguinal hernia, without mention of obstruction or gangrene, unilateral or unspecified (not specified as recurrent)

ICD10

- K40.90 Unil inguinal hernia, w/o obst or gangr, not spcf as recur
- K41.90 Unil femoral hernia, w/o obst or gangrene, not spcf as recur
- K46.9 Unspecified abdominal hernia without obstruction or gangrene

HERPES SIMPLEX

Benjamin Mattingly • Benjamin Wilks

BASICS

DESCRIPTION

- Viral disease characterized by recurrent painful vesicular lesions of mucocutaneous areas
- Lips, genitalia, rectum, hands, and eyes most commonly involved
- Infection is characterized by 2 phases:
 - *Primary*, in which virus becomes established in a nerve ganglion;
 - *Secondary*, involves recurrence of disease at the same site
- Incubation period is ~4 days from exposure
- Viral shedding occurs from 7–10 days (up to 23 days) in primary infection and 3–4 days in recurrent infections
- Neonatal infections can occur in utero, intrapartum (most common), or postnatal
 - Occur in 1/3,500 births per year in the US
- Human-to-human transmission
- 60–90% of population is infected with herpes simplex type 1 (HSV-1) or type 2 (HSV-2)
- More common in blacks than whites in ages <40 yr
- Females affected more than males

ETIOLOGY

- HSV-1 or HSV-2 are DNA viruses of the Herpesviridae family
- Viral transmission may occur via respiratory droplets, contact with mucosa or abraded skin with infected secretions:
 - Recurrent mucosal shedding of HSV may transmit the virus
 - Rate of recurrence varies with virus type and anatomic site
- Both viruses infect oral or genital mucosa:
 - Most common for HSV-1 to cause oral infections and HSV-2 to cause genital infections

DIAGNOSIS

SIGNS AND SYMPTOMS

- Many primary infections go unrecognized and can only be detected by an elevated IgG Ab titer
- Clinically, infection presents with grouped 1–2 mm vesicles on an erythematous base
- Vesicles may be filled with clear or cloudy fluid or may appear as frank pustules

Orofacial infection:

- Primary infection:
 - Gingivostomatitis or pharyngitis:
 - Ulcerative exanthem involving gingival and mucous membranes
 - Fever, malaise, irritability, headache, myalgias, cervical adenopathy
 - Primary infection symptoms typically last 2--4 weeks unless secondarily infected and heal without scarring
 - Inability to eat owing to pain is a risk for dehydration
- Recurrent infection (recrudescence):
 - Usually involves lips, specifically the vermillion border
 - Commonly incited by sunlight, heat, stress, trauma (chapping, abrasions), or immunosuppression
 - Prodrome of itching, tingling, throbbing, or burning followed by erythema, papule/vesicle, ulcer, crust, and healing
 - Transmission can occur in the absence of recognizable lesions
 - Fewer constitutional symptoms
 - Many individuals have a rise in Ab titer and never experience recurrence
 - HSV-1 oral infections recur more often than genital HSV-1 infections. HSV-2 genital infections recur 6 times more frequently than HSV-1 genital infections

Skin infection:

- History of exposure to HSV-1 or HSV-2
- Abrupt onset of fever, edema, erythema, and localized tenderness
- Herpetic whitlow:
 - HSV-2 more common than HSV-1
 - Infection of pulp and lateral aspect of finger with single or multiple vesicles
 - May occur from autoinoculation with primary oral or genital infection or from direct inoculation from occupational exposure
 - Can last 3–4 wk
 - Recurrence possible
 - In young children, it is associated with HSV-1 inoculation through thumb sucking during gingivostomatitis
- Traumatic herpes:
 - Can occur following cosmetic procedures of face, surgical and dental interventions, sun exposure, or burns
- Herpes gladiatorum:
 - Mucocutaneous infection of athletes involving chest, face, and hands transmitted through traumatized skin (often wrestlers)
- Eczema herpeticum:
 - Association between atopic dermatitis and HSV infection
 - HSV-1 more common than HSV-2
 - Occurs in children and young adults with atopic dermatitis
 - Secondary staphylococcal infection commonly occurs

- Higher risk if on steroids or infected with HIV
- Varicelliform eruption with spread to surrounding skin
- Fever, headache, and fatigue
- HSV-associated erythema multiforme:
 - Usually presents on palms and soles
 - Lasts 2–3 wk

Eye:

- Most common cause of corneal blindness
- Caused by extension of facial lesions or direct inoculation
- Acute onset of pain and photophobia
- Periauricular adenopathy, blurry vision, chemosis, and conjunctivitis
- May be unilateral or bilateral
- Dendritic lesions of cornea noted on fluorescein exam
- Different from herpes varicella zoster as dermatome not involved
- Hutchinson sign:
 - Vesicles on tip of nose may indicate ocular disease
 - Involvement of nasociliary nerve

CNS/encephalitis:

- Most common cause of severe sporadic encephalitis in the western world
- Usually from HSV-1 reactivation disease

History

May or may not have known history of exposure to HSV-1 or HSV-2

Physical-Exam

Vesiculoulcerative lesions in orofacial or genital area

Pediatric Considerations

- Up to 60–80% of babies who develop neonatal HSV are born to mothers without history of genital herpes
- Vesicular skin lesions may or may not be present on initial exam
- Primary genital disease of the mother increases risk of transmitting virus to fetus
- Most primary infections occur during childhood; symptomatic in only 5–10% of children
- Orofacial disease is most likely to present as gingivostomatitis in children younger than 5 yr of age
- Whitlow may be caused by thumb-sucking children with oral herpes

ESSENTIAL WORKUP

- Herpes encephalitis:
 - Lumbar puncture if herpes encephalitis is considered
- Herpes ophthalmicus:

- Fluorescein exam if ocular herpes is a concern

DIAGNOSIS TESTS & INTERPRETATION

- Orofacial:
 - Presumptive diagnosis made by history and exam
 - If definitive diagnosis is necessary (e.g., systemic disease, child abuse):
 - Viral culture or polymerase chain reaction (PCR) testing of swabs from vesicles
 - PCR is the most accurate and reliable method for detecting the virus
 - Fluorescent antibody detection of antigen; serum antibody studies
 - Scrapings for Tzanck smear or Papanicolaou stain
 - Skin biopsy if hyperkeratotic or lichenoid lesions
- Eye:
 - Dendritic corneal lesions by fluorescein exam
 - Swab of affected area for viral culture or fluorescent antibody detection
- CNS/encephalitis:
 - Lumbar puncture with CSF pleocytosis and negative bacterial antigens
 - CSF PCR
 - MRI/CT (abnormalities in temporal lobe may be visualized)
 - EEG diagnostic if spike and waves in temporal region

Lab

- Lesion scrapings can be sent for culture or PCR testing
- Tzanck smear demonstrating multinucleated giant cells, atypical keratinocytes, and large nuclei
- Serum testing has limited ED use
- ELISA testing may demonstrate HSV antibodies, determining past exposure only
- Requires 2 wk to > 3 mo to detect seroconversion

DIFFERENTIAL DIAGNOSIS

- Orofacial and skin:
 - Bacterial pharyngitis
 - Mycoplasma pneumoniae pharyngitis
 - Stevens–Johnson syndrome
 - Herpes zoster
 - Varicella
 - Pemphigus
 - Contact or chemical dermatitis
 - Impetigo
 - Syphilis
- Eye:
 - Conjunctivitis: Viral, bacterial, or allergic
 - Herpes zoster ophthalmicus

- Scleritis/episcleritis
- Angle-closure glaucoma
- Corneal abrasion



TREATMENT

PRE HOSPITAL

- Maintain universal precautions.
- Pain control

INITIAL STABILIZATION/THERAPY

Protect airway in comatose or obtunded patients with suspected CNS disease

ED TREATMENT/PROCEDURES

- Orofacial/gingivostomatitis:
 - Primary disease in healthy children is generally not treated
 - Primary disease in normal host with mild disease requires only supportive treatment with hydration and analgesia
 - Severe disease or immunocompromised patients: IV or oral acyclovir, valacyclovir, or famciclovir
 - Oral acyclovir is first-line medication
 - If recurrent disease, oral antivirals are most helpful if started with prodrome or at 1st sign of lesion:
 - Reduces lesions and symptoms by 1–2 days
 - Consider prophylaxis in patients with more than 6 episodes per year; history of herpes-associated erythema multiforme or herpes gladiatorum; upcoming intense sun exposure or stress; perioral/intraoral surgery; cosmetic facial procedures:
 - Prophylaxis reduces frequency and severity of herpes labialis and may help decrease asymptomatic shedding, leading to decreased transmission
 - Does not cure or terminate the disease
 - When prophylaxis is stopped, most patients have recurrences
- Skin (other than orofacial or genital):
 - May be treated with oral acyclovir
 - Antibiotics if secondary bacterial infection
 - *Do not incise and drain*: May lead to spread of infection
- Eye:
 - Oral acyclovir and topical antiviral therapy with trifluridine or vidarabine
 - Vidarabine ointment for children
 - *Do not treat with steroids*: May cause increased viral replication
 - Ophthalmology consult

Pregnancy Considerations

Acyclovir has been used to suppress genital herpes near end of pregnancy and appears safe, but is not FDA approved

MEDICATION

- **Acyclovir:**
 - Orofacial and skin: 400 mg PO TID for 7–10 days or 5–10 mg/kg IV (5–10 mg/kg) q8h for 7–14 days
 - Pediatric mucocutaneous primary infection: 40–80 mg/kg PO in 3–4 div. doses for 5–10 days; max. dose 1 g/d
 - Eyes for suppression therapy: 400 mg PO BID
 - Encephalitis: 60 mg/kg/24h IV div. q8h for 14–21 days
- **Famciclovir:**
 - Primary orofacial: 250 mg PO TID for 7–10 days (immunocompetent), 500 mg PO BID for 7–10 days (immunocompromised)
- **Trifluridine:**
 - Adults and peds older than 6 yr: 1 drop of 1% ophthalmic ointment to eye q2h while awake (max. 9 drops per day) for at least 10 days and then taper under ophthalmology consultation
- **Valacyclovir:**
 - Adults primary mucocutaneous: 1,000 mg PO BID for 7 days
 - Adult recurrent mucocutaneous (nongenital): 500 mg PO BID for 3 days
- **Vidarabine:**
 - Adults or peds older than 2 yr: Topical 0.5 in ribbon of 3% ophthalmic ointment to eye 5 times per day
- **Recurrent mucocutaneous herpes:**
 - Acyclovir: 400 mg PO TID for 5 days
 - Famciclovir: 1,000 mg PO BID for 1 day
 - Valacyclovir: 500 mg PO BID for 3 days
- **Long-term prophylaxis:**
 - Acyclovir: 400 mg PO BID
 - Valacyclovir: 500 mg PO daily
 - Famciclovir: 250 mg PO BID

ALERT

- Antiviral dosing may need adjustment for renal failure
- Topical antivirals are available but have not been shown to reduce the length of symptoms or decrease recurrence



FOLLOW-UP

DISPOSITION

Admission Criteria

- Encephalitis, disseminated disease, dehydration
- Severe local or disseminated disease in immunocompromised host
- Neonatal HSV
- ICU vs. ward based on toxicity and need for airway support
- Ophthalmology consult vs. admission for ocular involvement

Discharge Criteria

Uncomplicated local disease

Issues for Referral

- Suppressive treatment options
- Herpes infection during pregnancy

FOLLOW-UP RECOMMENDATIONS

Skin/genital infection:

- Follow-up with the patient's primary doctor to discuss risks and benefits of suppressive therapy

PEARLS AND PITFALLS

- Failure to consider herpes simplex encephalitis in patients whom you have a concern for meningitis/encephalitis
- Failure to consider ocular herpes in patients presenting with eye pain, decreased vision, and/or lesions on nose
- Failure to warn patients about the risk of transmission to others especially during outbreaks and for 1–2 wk thereafter
- Failure to warn patients to avoid touching the lesions during outbreaks to prevent spread of the lesions to other body areas

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- [Genital Herpes](#)
- [Varicella](#)
- [Zoster](#)



CODES

ICD9

- [054.9 Herpes simplex without mention of complication](#)
- [054.41 Herpes simplex dermatitis of eyelid](#)
- [054.79 Herpes simplex with other specified complications](#)

ICD10

- [B00.1 Herpesviral vesicular dermatitis](#)
- [B00.9 Herpesviral infection, unspecified](#)
- [B00.59 Other herpesviral disease of eye](#)

HERPES ZOSTER

Aaron Hexdall • Stephen M. Kelly



BASICS

DESCRIPTION

- Commonly known as shingles
- Characterized by unilateral eruption of painful vesicles along a single dermatome
- Disseminates rarely in normal hosts and frequently in immunocompromised hosts
- Most common in patients with decreased cell-mediated immunity:
 - Older than 50 yr of age
 - Neoplastic diseases
 - Immunosuppressive drugs

ETIOLOGY

- Caused by varicella zoster virus (VZV), a DNA virus in the Herpesviridae family
- Reactivation of dormant virus in dorsal root ganglia
- Mostly in individuals who previously had chickenpox and very rarely in vaccinated individuals

Pregnancy Considerations

Zoster in pregnancy is not associated with increased risk of congenital varicella syndrome

Pediatric Considerations

May occur in childhood, most commonly when primary varicella occurred in utero or in the first 6 mo of life



DIAGNOSIS

SIGNS AND SYMPTOMS

- Dermatomal zoster
 - Prodrome of pain and paresthesias in 75% of patients
 - Pain may be sharp, dull, tingling, burning, or intense pruritus
 - Classical rash is grouped vesicles on erythematous base
 - Progress to scab and crust formation over 7–10 days; crusts fall off in 2–3 wk
 - Most common nerve distributions are thoracic and lumbar, followed by trigeminal and cervical
- Zoster sine herpete
 - Syndrome occurs without the rash

- Herpes zoster ophthalmicus (HZO)
 - Involvement of ophthalmic division of trigeminal nerve
 - Hutchinson sign – lesion on tip of nose
 - May cause punctate keratitis or corneal pseudodendrites (elevated mucous plaques, less ulcerative and less fluorescein uptake than HSV dendrites)
- Ramsay Hunt syndrome
 - From VII and VIII cranial nerve involvement
 - Lesions in the external auditory canal, peripheral facial palsy, vertigo and anesthesia of anterior 2/3 of hemitongue
 - Progress to scab and crust formation over 7–10 days; crusts fall off in 2–3 wk
 - Most common nerve distributions are thoracic and lumbar, followed by trigeminal and cervical
- Disseminated disease may cause:
 - Myelitis
 - Meningoencephalitis
 - Peripheral neuropathy
 - Hepatitis
 - Pneumonitis
- Postherpetic neuralgia (PHN) is a complication of zoster:
 - Described as pain that persists at site of zoster lesions for > 3 mo after cutaneous disease has healed
 - 10–70% of patients will have pain after resolution of lesions
 - Incidence increases with age older than 50 yr, severe rash, and severe pain

ESSENTIAL WORKUP

- Clinical presentation is sufficient for diagnosis in most patients
- Labs may aid diagnosis in patients with atypical rash or disseminated disease

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Tzanck Smear
 - Cheap and easy
 - Scrape base of unroofed vesicle
 - Multinucleated giant cells on Giemsa stain
 - Cannot distinguish VZV from HSV
 - Low sensitivity
- PCR is the preferred method
 - From vesicle scraping, blood, CSF or BAL
 - Can distinguish between HSV and VZV
 - More sensitive and specific
- Serology (IgM/IgG)

- More difficult to interpret
- Less sensitive/specific
- Viral culture
 - Slow, insensitive

DIFFERENTIAL DIAGNOSIS

- Primary varicella
- Herpes simplex virus (HSV)
- Cellulitis
- Poison Ivy
- Insect bites
- Bullous impetigo
- Molluscum contagiosum
- Trigeminal neuralgia
- Angina
- Biliary/renal colic
- Radiculopathy
- Bell palsy
- Peripheral vertigo
- Conjunctivitis (nonherpetic)
- HSV keratitis



TREATMENT

PRE HOSPITAL

- Zoster is contagious and may cause varicella in nonimmune health care workers:
 - Lesions should be covered
 - Maintain universal precautions

ED TREATMENT/PROCEDURES

- Generally a self-limited disease
- Goals of treatment are to decrease pain and duration of illness, and to prevent PHN
- Immunocompetent patient
 - Antiviral therapy
 - PO valacyclovir has easiest dosing and seems to be the most effective
 - May also use PO acyclovir (cheapest option) or famciclovir
 - Should be started within 72 hr of rash, but some experts recommend starting later if new vesicles are still appearing
 - Speeds acute healing and resolution of acute pain
 - Unclear if decreases the rate of PHN
 - Analgesics

- Over-the-counter agents (mild disease)
 - Long-acting opioids
- Corticosteroids (controversial)
 - Several studies showed modest improvement in cutaneous healing and acute neuritis
 - Does not help prevent PHN
 - If not otherwise contraindicated, consider in patients with severe disease or CNS involvement
- Immunocompromised patient
 - Antiviral therapy
 - IV acyclovir
 - Analgesics
 - As above
 - Corticosteroids
 - As above
- Herpes zoster ophthalmicus
 - Necessitates ophthalmologic consultation
 - Valacyclovir PO as above
 - IV acyclovir if immunocompromised or cranial nerve involvement
 - Erythromycin ointment for secondary bacterial infection
 - Topical cycloplegic if associated iritis
 - Ophthalmologist may recommend topical steroids
- PHN
 - Antivirals not indicated
 - Long-acting opioids
 - Tricyclic antidepressants are effective
 - Gabapentin and pregabalin may also be helpful
 - Topical lidocaine provides short-term relief
- Postexposure prophylaxis
 - VariZIG is recommended within 72 hr of exposure for the following patients:
 - Immunocompromised
 - Pregnant
 - Exposed premature neonates born < 28 wks gestation
 - Exposed premature neonates born > 28 wks gestation to seronegative mom
 - Neonates born to mother with symptomatic varicella between 5 days predelivery and 2 days postdelivery
 - Must wait 5 mo before giving subsequent vaccine
- Vaccine
 - Zostavax (Merck) recommended for all patients over the age of 60 (unless immunocompromised)
 - Does not reduce risk of recurrence or PHN in patients with zoster

Pregnancy Considerations

- Same treatment as immunocompetent patients
- Vaccine contraindicated in pregnancy

Pediatric Considerations

Neonatal zoster requires treatment with IV acyclovir

MEDICATION

First Line

- **Antivirals:**
 - Valacyclovir 1 g PO q8h × 7 days, Acyclovir 800 mg PO q4h × 7–10 days, Acyclovir 10 mg/kg IV q8h × 7 days
- **Analgesics:**
 - Acetaminophen 500 mg PO q6h; not to exceed 4g/d
 - Ibuprofen 600 mg PO q6h
 - Oxycodone CR 10 mg PO q12h
 - Amitriptyline 25 mg PO qhs, increase as tolerated to 100 mg daily
 - Cyclopentolate ophthalmic 1% apply 1 gtt q8h to affected eye
- **PEP:**
 - Varicella zoster immunoglobulin (VariZIG): 125 U IM/IV per 10 kg body weight, up to max. of 625 U
- **Vaccine:**
 - Zostavax (Merck) one-time SC injection

Second Line

- **Antivirals:**
 - Famciclovir 500 mg PO q8h × 7 days
 - Foscarnet: 90 mg/kg IV as 2 hr infusion every 12 hr (acyclovir-resistant immunocompromised patient)
- **Antibiotics:**
 - Erythromycin ophthalmic ointment USP 0.5% apply 1 in q4h to affected eye
- **Analgesics**
 - Gabapentin: 100–300 mg daily increasing 100–300 mg every 3 days until adequate response or max. 3,600 mg/d
 - Pregabalin: Start 50 mg PO q8h or 75 mg PO q12h, increase to 100 mg q8h within 1 wk
 - Lidocaine patch 5%: Apply up to 3 patches for max. 12 hr within a 24 hr period for severe pain
- **Corticosteroids:**
 - Prednisone: Taper over 7 days (do not extend beyond duration of antiviral therapy)



FOLLOW-UP

DISPOSITION

Admission Criteria

- Immunocompromise
- Disseminated disease
- HZO with cranial nerve involvement
- Intractable pain
- Isolation:
 - Airborne precautions for all patients with primary varicella or disseminated zoster, or immunocompromised patients with dermatomal zoster
 - Patients are infectious from 48 hr before appearance of rash until crusting of all lesions

Discharge Criteria

- Most are managed as outpatients
- Patients should be instructed that lesions may heal with scarring or leave depigmented areas
- Recommend isolation from pregnant or immunocompromised persons until all lesions are crusted
- PHN may require long-term follow-up and/or referral to pain specialist

Pregnancy Considerations

Usually treated as outpatients

Pediatric Considerations

Admit all neonates with zoster

PEARLS AND PITFALLS

- Look for ocular involvement if rash involves the tip of the nose (Hutchinson sign)
- Expose the skin of every patient with chest pain
- Failure to consider the diagnosis in the absence of rash
- Failure to warn patients of the risk of PHN

ADDITIONAL READING

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CODES

ICD9

- 053.9 Herpes zoster without mention of complication
- 053.29 Herpes zoster with other ophthalmic complications
- 053.71 Otitis externa due to herpes zoster

ICD10

- B02.9 Zoster without complications
- B02.21 Postherpetic geniculate ganglionitis
- B02.30 Zoster ocular disease, unspecified

HERPES, GENITAL

Kathleen A. Kerrigan • Jason L. Grimsman



BASICS

DESCRIPTION

- Genital herpes is a lifelong recurrent infection
- ~1 in 4 Americans older than age 30 are seropositive for herpes simplex virus type 2 (HSV-2):
 - Most are asymptomatic
- 1st episode/primary HSV infection:
 - 2–12 day incubation
 - Symptoms peak 8–10 days after onset
 - Lesions heal in 3 wk
 - Primary infection may have more prominent clinical syndrome and complications (e.g., encephalitis, meningitis, hepatitis)
 - Primary infection may also go unnoticed:
 - >50% of 1st recognized signs and symptoms are not primary infection
- Recurrent HSV infection:
 - Average patient has 4 recurrences per year, by herpes simplex virus type 1 (HSV-1) recurs less than HSV-2
 - Virus reactivated from dorsal root ganglia
 - Triggered by local trauma, emotional stress, fever, sunlight, cold or heat, menstruation, or infection
 - Milder clinical syndrome and fewer lesions that usually heal within 10 days
- Asymptomatic HSV infection:
 - Virus is shed intermittently and often transmitted by persons who are without lesions or symptoms

ETIOLOGY

- 70–90% caused by a DNA virus HSV-2:
 - Remainder caused by HSV-1
- Increasing prevalence of genital HSV-1 infection:
 - Higher rates of oral sex
 - Falling incidence of childhood (nonsexual) transmission owing to improved social conditions resulting in a larger pool of susceptible adolescents and adults
- Primary genital infection by HSV-1 is similar to HSV-2 in symptoms and duration, but recurs much less frequently
- Acquisition of HSV-2 in patients with pre-existing HSV-1 infection is less commonly

associated with systemic symptoms:

- Acquisition of HSV-1 in persons with pre-existing HSV-2 infection is rare
- HSV vaccines unsuccessful to date, research is ongoing
- High association with HIV and other STDs

ALERT

- Contact isolation and universal precautions should be maintained
- Patients with HIV coinfection have higher HIV viral levels in the blood and skin lesions during HSV recurrence



DIAGNOSIS

SIGNS AND SYMPTOMS

- Local pain and itching
- Herpetic cervicitis, vaginitis, or urethritis may present with dysuria, urinary hesitance or retention, vaginal discharge, or pelvic pain
- Herpetic pharyngitis or gingivostomatitis may occur with oral acquisition
- Systemic symptoms like fever, headache, malaise, photophobia, anorexia, myalgias, and lymphadenopathy are more common with primary infection

History

- 1–2 day prodrome of local tingling, burning, itching, or pain prior to eruption (can mimic sciatica)
- Classically, lesions are noted on day 2 as macules and papules, then progress to vesicles, pustules, and then ulcerate by day 5
- Skin lesions crust over; mucosal membrane lesions heal without crusting

Physical-Exam

- Lesions on vulva, vagina, cervix, perineum, buttocks; penile shaft or glans
- Grouped vesicles on an erythematous base
- On moist mucosal surfaces, ulcers may predominate
- Atypical features may include localized edema, erythema, crusts, or fissures

Pediatric Considerations

- Neonatal infections are often disseminated or involve the CNS with high morbidity and mortality
- Congenital HSV in the neonate without vesicles may mimic rubella, cytomegalovirus (CMV), or toxoplasmosis
- Consider sexual abuse in children with genital HSV; culture lesions and test for other STDs in suspected cases

ESSENTIAL WORKUP

Diagnosis based on history and physical exam

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Viral load in lesions of primary infection are greater than those seen in recurrence
- Tzanck preparation and staining of fluid from lesions is insensitive and nonspecific
- Viral culture of vesicle fluid or ulcer base positive in 80–95% of cases, decreasing sensitivity as lesions crust and heal:
 - 3–10 days for result
- PCR 1.5–4 times more sensitive than viral culture; test of choice for CSF analysis in suspected CNS infection
- Serologic tests not helpful in acute disease:
 - Highly sensitive and specific; detect anti-gG1 and anti-gG2 antibodies
 - Require 2 wk to > 3 mo to detect seroconversion
 - Cannot distinguish acute from chronic disease
 - HerpeSelect HSV-1/HSV-2 ELISA:
 - Takes hour to days in lab
 - POCKit HSV2, bedside results in 10 min

Imaging

No imaging generally indicated

DIFFERENTIAL DIAGNOSIS

- Syphilis (*Treponema pallidum*)
- Chancroid (*Haemophilus ducreyi*)
- Lymphogranuloma venereum (LGV)
- Granuloma inguinale (*Klebsiella granulomatis*)
- Candidiasis
- Behçet syndrome



TREATMENT

PRE HOSPITAL

Universal precautions should be maintained

INITIAL STABILIZATION/THERAPY

Rarely required unless associated with systemic symptoms requiring hospitalization:

- Disseminated infection
- Hepatitis
- Pneumonitis

- Meningoencephalitis

ED TREATMENT/PROCEDURES

- Treatment partially controls symptoms and lesions; does not eradicate latent virus nor affect recurrences after drug is discontinued
- Episodic treatment of recurrences may shorten duration of lesions or ameliorate recurrences
- Daily suppressive therapy in patients with frequent recurrences (6 or more per year) reduces frequency of recurrences by 75%
- Famciclovir and valacyclovir are equally effective medications with less frequent dosing regimens, all interfere with viral DNA polymerase
- Resistance to acyclovir in immunocompromised individuals is 5–10%:
 - Foscarnet 40 mg/kg IV q8h may be effective
- Consider testing for concomitant STDs, those with an HSV outbreak are more likely to contract HIV
- Consider bladder catheterization, either indwelling or intermittent, for women with difficulty urinating due to possible sacral nerve involvement

Pregnancy Considerations

- Women with primary HSV infection during pregnancy should receive antiviral therapy:
 - High rates of neonatal morbidity in both symptomatic and asymptomatic patients
- Suppressive antiviral therapy after 36 wk associated with decreased incidence of lesions at delivery:
 - Decreased cesarean delivery rates

MEDICATION

- Systemic or severe infection requiring hospitalization:
 - Acyclovir: 5–10 mg/kg IV over at least 1 hr q8h for 5–10 days
 - Neonate/peds: 10–20 mg/kg IV over at least 1 hr q8h for 7–10 days
- 1st episode (7–10 day therapy; extend if not healed in 10 days):
 - Acyclovir: 400 mg PO TID or 200 mg PO 5 times per day:
 - Peds: 20 mg/kg PO TID or 5 mg/kg IV q8h.
 - Famciclovir: 250 mg PO TID for 7–10 days
 - Valacyclovir: 1,000 mg PO BID for 7–10 days
- Recurrent infection (5 day therapy):
 - Must start within 1 day of appearance of lesion or during prodrome
 - Acyclovir: 800 mg PO TID for 2 days or 800 mg PO BID for 5 days.
 - Famciclovir: 1,000 mg PO BID for 1 day or 125 mg PO BID for 5 days.
 - Valacyclovir: 500 mg PO BID for 3 days or 1,000 mg PO daily for 5 days.
- Suppressive therapy (daily):
 - Acyclovir: 400 mg PO BID

- Famciclovir: 250 mg PO BID
- Valacyclovir: 500 mg PO daily or if > 10 recurrences yearly, 1,000 mg PO daily
- Treatment of patients with HIV coinfection:
 - Recurrent infection (5–10 days therapy):
 - Acyclovir: 400 mg PO TID
 - Famciclovir: 500 mg PO BID for 5–10 days
 - Valacyclovir: 1,000 mg PO BID
 - Suppressive therapy:
 - Acyclovir: 400–800 mg PO BID–TID
 - Famciclovir: 500 mg PO BID
 - Valacyclovir: 500 mg PO BID



FOLLOW-UP

DISPOSITION

Admission Criteria

- Systemic involvement (encephalitis, meningitis), significant dissemination
- Severe local symptoms (pain, urinary retention)
- Severely immunocompromised patient

Discharge Criteria

- Immunocompetent patient without systemic involvement
- Discharge counseling:
 - Avoid sexual contact during prodrome until healed
 - Practice safe sex techniques even if there are no lesions
 - Expect future recurrences; consider suppressive therapy if frequent
 - Analgesics and antipruritics as needed
 - Dysuria and urinary retention may be relieved with sitz baths or pouring warm water over lesions during urination

Issues for Referral

- Neonatal herpes infection
- Sexual abuse in children
- Herpes infection during pregnancy

PEARLS AND PITFALLS

- Treat primary infections
- Consider sexual abuse in children with genital herpes
- Herpes is a lifelong infection

ADDITIONAL READING

- ACOG Committee on Practice Bulletins. ACOG Practice Bulletin No. 82: Management of herpes in pregnancy. *Obstet Gynecol*. 2007;109:1489–1498.
- Borhart J, Birnbaumer DM. Emergency department management of sexually transmitted infections. *Emerg Med Clin North Am*. 2011;29:587–603.
- Cernik C, Gallina K, Brodell RT. The treatment of herpes simplex infections: An evidence based review. *Arch Intern Med*. 2008;168:1137–1144.
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- Roett MA, Mayor MT, Uduhiri KA. Diagnosis and management of genital ulcers. *Am Fam Physician*. 2012;85:254–262.
- Sexually transmitted diseases treatment guidelines 2010. Centers for Disease Control and Prevention. Available at <http://www.cdc.gov/std/treatment/2010/genital-ulcers.htm>



See Also (Topic, Algorithm, Electronic Media Element)

Herpes Simplex



CODES

ICD9

- 054.10 Genital herpes, unspecified
- 054.11 Herpetic vulvovaginitis
- 054.13 Herpetic infection of penis

ICD10

- A60.01 Herpesviral infection of penis
- A60.04 Herpesviral vulvovaginitis
- A60.9 Anogenital herpesviral infection, unspecified

HICCUPS

Jeffrey A. Horenstein • Carrie Tibbles



BASICS

DESCRIPTION

- Sudden, involuntary contraction of the diaphragm (usually unilateral) and other inspiratory muscles terminated by abrupt closure of the glottis
- Medical terminology: Singultus
- Usually occur with a frequency of 4–60/min
- Occurs as a result of stimulation of the hiccup reflex arc:
 - Irritation of the vagus and phrenic nerves
 - The “hiccup center” is located in the upper spinal cord or brainstem
- Classification:
 - Hiccup bout: < 48 hr
 - Persistent hiccups: 48 hr–1 mo
 - Intractable hiccups: > 1 mo
- Male > female (4:1)

ETIOLOGY

- GI:
 - Gastric distention, overeating, eating too fast
 - Esophageal: Gastroesophageal reflux, achalasia, candida esophagitis, cancer
 - Gastric: Ulcers, cancer
 - Hepatic: Hepatitis, hepatoma
 - Pancreatic: Pancreatitis, pseudocyst, cancer
 - Bowel obstruction
 - Inflammatory bowel disease
 - Cholelithiasis, cholecystitis
 - Appendicitis
 - Abdominal aortic aneurysm
 - Postoperative, abdominal procedure
- Diaphragmatic irritation:
 - Hiatal hernia
 - Intra-abdominal mass
 - Pericarditis
 - Eventration
 - Splenomegaly, hepatomegaly
 - Peritonitis
- CNS:
 - Vascular lesions: Ischemic/hemorrhagic stroke, head trauma, arteriovenous

- malformations
 - Infectious: Encephalitis, meningitis, abscess
 - Structural: Cancer, Parkinson disease, multiple sclerosis, hydrocephalus
 - Ventriculoperitoneal shunt
- Thoracic:
 - Infectious: Pneumonia, TB
 - Cardiac: MI, pericarditis
 - Aortic aneurysm
 - Cancer
 - Mediastinal lymphadenopathy
- Head and neck:
 - Otic foreign body irritating the tympanic membrane
 - Pharyngitis
 - Laryngitis
 - Goiter
 - Retropharyngeal/peritonsillar abscess
 - Neck mass
- Metabolic:
 - Uremia
 - Hyponatremia
 - Hypocalcemia
 - Gout
 - DM
- Toxic/drug induced:
 - Alcohol
 - Tobacco
 - α -methyldopa
 - Benzodiazepines
 - Steroids
 - Barbiturates
 - Narcotics
 - Chemotherapeutic agents
 - Antibiotics
 - General anesthesia
- Psychogenic causes:
 - Stress/excitement
 - Grief
 - Malingering
 - Conversion disorder
- Idiopathic



SIGNS AND SYMPTOMS

- Characteristic sound abruptly ending an inspiratory effort
- Attacks usually occur at brief intervals and last only a few seconds or minutes.
- Attacks lasting >48 hr or persisting during sleep suggest an organic etiology.

History

- Targeted history and review of systems to determine likelihood of potential underlying etiology:
 - Severity and duration of current episode
 - History of previous episodes and treatment attempts

Physical-Exam

- Careful physical exam in search of an underlying cause, with exam focused on:
 - Head and neck
 - Chest
 - Abdomen
 - Neurologic

ESSENTIAL WORKUP

For persistent or intractable hiccups, a thorough history and physical exam dictate further diagnostic testing.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC with differential
- Electrolytes, BUN, creatinine

Imaging

- CXR
- Further imaging may be indicated depending on clinical suspicion of a particular etiology; this can often be performed on an outpatient basis.

DIFFERENTIAL DIAGNOSIS

Eructation (belching)



TREATMENT

ED TREATMENT/PROCEDURES

- Treat specific causes when identified:
 - Remove foreign bodies from the ear.
 - Relieve gastric distention with a nasogastric tube.

- Nonpharmacologic maneuvers:
 - Catheter stimulation of the posterior pharynx
 - Direct stimulation of the uvula with a cotton swab
 - Supraorbital pressure
 - Carotid sinus massage
 - Digital rectal massage
 - Suboccipital release
 - Gentle traction and pressure applied to the posterior neck, stretching the suboccipital muscles and fascia.
- Pharmacologic treatment:
 - First line, only FDA approved medication for hiccups: Chlorpromazine
- Additional medications:
 - Gabapentin
 - Metoclopramide
 - Baclofen
 - Haloperidol
 - Nebulized lidocaine
 - Amitriptyline
 - Phenytoin

MEDICATION

- Amitriptyline: 10 mg PO TID
- Baclofen: 10 mg PO TID
- Chlorpromazine: 25–50 mg IV/IM, 25–50 mg PO TID–BID
- Gabapentin: 100 mg PO TID–QID
- Haloperidol: 2–5 mg IM
- Lidocaine (4%): 3 mL nebulized, repeat if necessary
- Metoclopramide: 10 mg IV/IM, 10–20 mg PO QID
- Phenytoin: 200 mg IV



FOLLOW-UP

DISPOSITION

Admission Criteria

If hiccups interfere with daily activities and could lead to decreased nutritional or fluid intake, aspirations, insomnia, wound dehiscence

Discharge Criteria

- If hiccups last < 48 hr
- Workup inconsistent with underlying organic etiology

Issues for Referral

Referral in cases of intractable hiccups for investigation into underlying cause and more definitive therapeutic measures:

- Phrenic nerve block, crush, or transection
- Hypnosis
- Behavioral modification
- Acupuncture
- Psychiatric interventions

FOLLOW-UP RECOMMENDATIONS

Home remedies in case of recurrence:

- Swallowing a spoonful of sugar
- Sucking on a hard candy or swallowing peanut butter
- Breath holding/Valsalva maneuver
- Biting a lemon
- Tongue traction
- Lifting the uvula with a cold spoon
- Drinking from the far side of a glass
- Fright
- Noxious stimuli
- Rebreathing into a paper bag

PEARLS AND PITFALLS

Protracted hiccups are strongly suggestive of underlying organic disease.

ADDITIONAL READING

- Calsina-Berna A, García-Gómez G, González-Barboto J, et al. Treatment of chronic hiccups in cancer patients: A systematic review. *J Palliat Med.* 2012;15(10):1142–1150.
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CODES

ICD9

- 306.1 Respiratory malfunction arising from mental factors
- 786.8 Hiccough

ICD10

- F45.8 Other somatoform disorders
- R06.6 Hiccough

HIGH-ALTITUDE ILLNESS

Christopher B. Colwell



BASICS

DESCRIPTION

- Incidence dependent on:
 - Rate of ascent
 - Final altitude
 - Sleeping altitude
 - Duration at altitude
- Acute mountain sickness (AMS) incidence:
 - Up to 67% incidence with rapid ascent (1–2 days) to >14,000 ft
 - 22% incidence for skiers visiting resorts and sleeping at 7,000–9,000 ft, 40% at 10,000 ft
- AMS risk factors:
 - Previous history of high-altitude illness
 - Physical exertion
 - Younger persons (< 50 yr)
 - Physical fitness not protective
 - Obesity and existing lung disease increase the risk
- High-altitude pulmonary edema (HAPE) incidence:
 - < 1–2%
 - Varies with rate of ascent
- High-altitude cerebral edema (HACE) incidence < 1%
- HACE and HAPE are unusual at altitudes under 13,000 ft (4000 m)

Pregnancy Considerations

- Relationship between pregnancy and high-altitude illness is not clearly established.
- Pregnancy-induced hypertension, proteinuria, and peripheral edema are more common at high altitude, which may be related to maternal hypoxemia.
- No evidence of increase in spontaneous abortions, placental abruption, or placenta previa at high altitudes
- Travel by pregnant women with normal pregnancies to moderate altitudes appears safe, although caution should be exercised when traveling > 13,000 ft and for women with complicated pregnancies

Geriatric Considerations

Although elderly persons are more likely to have underlying health problems that may be affected by altitude, such as HTN, COPD, and coronary artery disease, the risk of development of AMS is less in those older than 55 than in other age groups.

ETIOLOGY

- Rapid ascent to > 8,000 ft (about 2,500 m) without proper acclimatization is the most common cause of high-altitude–related illness.
- Rapidity of ascent, final altitude reached, sleeping altitude, and individual susceptibility all play a role in development of high-altitude illness as well.

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- AMS:
 - Headache + at least 1 of the following:
 - Nausea/vomiting
 - Fatigue/lassitude
 - Dizziness
 - Difficulty sleeping
 - Onset 4–12 hr after ascent
 - Generally benign and self-limited
 - Symptoms may become debilitating.
- HAPE:
 - Onset 2–4 days after ascent, most commonly on 2nd night
 - Can be life threatening
 - Cough (dry at 1st, then productive)
 - Dyspnea at rest
- HACE:
 - Life threatening
 - Occurs in the presence of HAPE and/or AMS:
 - Seen rarely as isolated entity
 - Onset:
 - May occur 12 hr after onset of AMS
 - Generally requires 2–4 days for development
 - Altered mental status
 - Severe or increasing headache
 - Nausea/vomiting

Pediatric Considerations

- AMS in infants and young children manifested by:
 - Increased fussiness
 - Decreased playfulness
 - Decreased appetite
 - Vomiting

- Sleep disturbances
- Incidence of HAPE is greater in younger individuals (< 20 yr) than in older adults
- No cases of HAPE or HACE have been reported in children < 4 yr old

Physical-Exam

- AMS:
 - Without presence of HAPE or HACE, the physical exam will often be normal:
 - Mild stages of AMS are often misdiagnosed as viral syndromes or hangovers from alcohol ingestions
- HAPE:
 - Tachypnea
 - Rales
 - Cyanosis
 - Fever may be present.
 - Severe respiratory distress and death may occur.
- HACE:
 - Ataxia
 - Papilledema, retinal hemorrhages
 - Altered mental status/global encephalopathy:
 - Focal neurologic deficit less common
 - Seizure (rare)
 - Coma
 - Has been described as “end-stage” AMS

ESSENTIAL WORKUP

- Clinical diagnosis in the setting of recent altitude gain
- AMS:
 - Diagnosis made with history of headache + at least 1 of the following:
 - Nausea/vomiting
 - Lassitude/fatigue
 - Dizziness
 - Insomnia
 - No diagnostic lab or imaging studies
- HAPE:
 - Dyspnea on exertion—universal finding at altitude
 - Dyspnea at rest—symptom of HAPE, worse at night
 - Rales, cyanosis, or cough support diagnosis.
 - Tachycardia, tachypnea often correlate with severity.
- HACE:
 - Cerebellar ataxia with or without other symptoms of AMS
 - Papilledema, retinal hemorrhages are associated findings.

DIAGNOSIS TESTS & INTERPRETATION

Lab

Arterial blood gas (ABG) for HAPE:

- Reveals hypoxemia (pO_2 30–50) and respiratory alkalosis, *not* acidosis

Imaging

- CXR in HAPE:
 - Reveals patchy alveolar infiltrates with areas of clearing between patches
 - Unilateral or bilateral infiltrates (right mid-lung field being most common)
 - Cardiomegaly, “batwing” distribution of infiltrates, and Kerley B lines (typical of cardiogenic pulmonary edema)—are generally absent in HAPE
- CT and MRI scans in HACE:
 - Vasogenic edema of white matter

Diagnostic Procedures/Surgery

ECG in HAPE:

- Tachycardia
- Evidence of right-heart strain

DIFFERENTIAL DIAGNOSIS

- AMS:
 - Alcohol hangover
 - Carbon monoxide poisoning
 - Encephalitis
 - Exhaustion
 - Meningitis
 - Viral syndrome
- HAPE:
 - High-altitude bronchitis and pharyngitis
 - Pneumonia
 - Pulmonary embolism:
 - More rapid onset
 - Pleuritic chest pain
- HACE:
 - Cerebrovascular accidents/transient ischemic attacks:
 - Focal neurologic signs suggest vascular lesion.



TREATMENT

PRE HOSPITAL

- Severe cases require immediate evacuation to lower altitude.

- Do not proceed to higher altitude in the presence of symptoms.
- Oxygen delivery or simulated descent in portable hyperbaric chamber (Gamow bag) can be lifesaving temporary measure making self-rescue possible.

INITIAL STABILIZATION/THERAPY

- HAPE and HACE:
 - _ ABCs:
 - Endotracheal intubation for impending respiratory failure or airway protection
 - _ Establish IV access.
 - _ Supplemental oxygen and monitoring
 - _ CPAP for HAPE

ED TREATMENT/PROCEDURES

- AMS:
 - _ Mild cases usually self-limited:
 - Symptomatic treatment
 - Halt ascent until symptoms resolve
 - _ Acetazolamide for moderate to severe symptoms
 - _ Ibuprofen or acetaminophen for headache
 - _ Promethazine or ondansetron for nausea
 - _ Supplemental oxygen in severe cases
 - _ Descent for severe or persistent symptoms
 - _ Acetazolamide for AMS prophylaxis:
 - In high-risk individual with planned rapid ascent
- HAPE:
 - _ Immediate descent for moderate/severe symptoms
 - _ Mild cases may be managed without descent if:
 - Adequate oxygen supplies available
 - Serial medical exam possible
 - Immediate descent for any deterioration in clinical status
 - _ Bed rest to avoid exercise-induced pulmonary HTN
 - _ Supplemental oxygen:
 - High flow rates (6–8 L/min) until improvement, then continue with lower flow rates
 - _ Nifedipine when other interventions are unavailable
 - _ β -agonist inhalers may be helpful.
 - _ Hyperbaric therapy is available and immediate descent is not possible
- HACE:
 - _ Immediate evacuation to lower altitude
 - _ Oxygen
 - _ Dexamethasone
 - _ Bed rest with elevation of head at 30°, and in severe cases, aggressive

management of elevated intracranial pressure

MEDICATION

- Acetazolamide:
 - AMS treatment: 250–500 mg (peds: 5 mg/kg) PO BID
 - AMS prophylaxis: 250 mg PO BID (peds: 5 mg/kg) PO BID; start 24 hr before ascent
- Dexamethasone: 8 mg IV, then 4 mg PO/IV QID
- Ibuprofen: 800 mg (peds: 5–10 mg/kg) PO TID
- Nifedipine: 10 mg PO, then 30 mg sustained release (SR) PO QD
- Promethazine: 12.5–25 mg (peds: 0.25–1 mg/kg) PO/PR (per rectum)/IM q4–6h

First Line

- Acetazolamide (AMS)
- Nifedipine (HAPE)

Second Line

Dexamethasone

The use of multiple medications in treating high-altitude illness is not supported by the current literature



FOLLOW-UP

DISPOSITION

Descent to a lower altitude mandatory in severe cases

Admission Criteria

- Persistent symptoms after observation in lower-altitude ED require admission.
- HAPE
- HACE

Discharge Criteria

Once clinical improvement seen and oxygen saturation > 95% on room air at sea level or appropriate normal saturation at higher altitudes

Issues for Referral

Offer prophylactic therapy for future ascents in patients with recurrent AMS (acetazolamide) or HAPE (nifedipine).

PEARLS AND PITFALLS

- The symptoms of high-altitude illness can resemble mild viral syndromes.

- Failure to consider high-altitude illness is a common pitfall.
- When managing altitude illness, once symptoms have developed and until they resolve, further ascent is contraindicated.
- Ataxia and dyspnea at rest are potentially early indicators of HACE and HAPE, respectively.
- Hyperbaric oxygen and adjunctive medications should be considered when descent is not possible.

ADDITIONAL READING

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- Eide RP, Asplund CA. Altitude illness: Update on prevention and treatment. *Curr Sports Med Rep*. 2012;11:124–130.
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CODES

ICD9

993.2 Other and unspecified effects of high altitude

ICD10

- T70.20XA Unspecified effects of high altitude, initial encounter
- T70.29XA Other effects of high altitude, initial encounter

HIP INJURY

Siobhan Gray

BASICS

DESCRIPTION

- Hip injury includes hip fractures and dislocations of the proximal femur due to minor or major trauma or overuse.
- Hip fracture: Fracture of proximal femur. Classified as intracapsular or extracapsular.
 - Intracapsular fracture: Femoral head or neck; often associated with disruption of femoral neck vessels; significant morbidity due to AVN:
 - Femoral head fracture: Usually associated with hip dislocation (anterior > posterior).
 - Femoral neck fracture: Usually older adults with minor trauma, or young patient with major trauma; symptoms vary. Patient may or may not be ambulatory. Often site of stress fracture in runners/military recruits.
 - Extracapsular fractures: Below acetabular capsule. Normally do not disrupt blood flow. Morbidity typically due to patient immobilization: DVT, PE:
 - Trochanteric fractures: Greater trochanter usually fractured by avulsed at insertion in gluteus medius. Lesser trochanter usually fractured by avulsed from forceful contraction of iliopsoas; seen in young athletes and children.
 - Intertrochanteric fracture: Defined as occurring in line between greater and lesser trochanters. Common in elderly, osteoporosis patients often due to fall. Marked external rotation and shortening. Can be stable or unstable. Nonambulatory.
 - Subtrochanteric fracture: Usually due to direct, significant trauma in younger patients or lesser trauma in elderly. Common site of pathologic fracture. Can be site of significant blood loss and shock.
- Hip dislocation: Disarticulation of femoral head. Classified as posterior, anterior, and central:
 - Posterior dislocation (most common):
 - Often from motor vehicle accident (MVA) in which knees strike dashboard
 - 10% associated with sciatic nerve injury
 - Anterior dislocation: 10% hip dislocations:
 - Often due to trauma with sudden abduction of thigh
 - Associated femoral head fractures, femoral nerve injury
 - Can be anterior superior, or anterior inferior

- Central dislocation with acetabular fracture:
 - Usually from direct impact to greater trochanter
 - Associated significant blood loss, sciatic nerve injury

Pediatric Considerations

- Hip dislocation: Uncommon; often spontaneously reduced at time of injury. Concern for tissue trapped in joint space:
 - Trivial force required for posterior hip locations in children <10 yr old
- Proximal femoral physeal fracture: Fracture at growth plate; great risk for osseous necrosis
- Slipped capital femoral epiphysis: Minimal trauma, decreased ROM.
- Femoral neck fractures: Relatively common; stress fractures in young athletes
- Intertrochanteric fractures: Rare.
- Must suspect nonaccidental trauma (NAT)
- Consider pathologic fracture with minor trauma.

ETIOLOGY

See individual injuries above.

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Groin, hip, thigh, medial knee pain, pain with ambulation/weight bearing in the setting of trauma
- Minor trauma in the elderly due to osteoporosis; high-impact trauma in young adults
- Rarely overuse injury, stress fracture.

Physical-Exam

- Obvious signs of trauma:
 - Deformity or angulation, swelling, open fracture, or missile entrance wound
 - Lower extremity held in position of comfort
- Hip fracture: Flexion, abduction, external rotation
- Posterior hip dislocation: Flexion, *adduction, internal rotation* of hip, flexion of knee, hip immobile
- Anterior hip dislocation: Flexion, *abduction, external rotation* of hip, thigh shortening, hip immobile

Pediatric Considerations

- Pediatric fracture patterns different due to developing cartilaginous components:

- Assess for dislocation of the femoral capital epiphysis.
- Fracture classification and management are also different.
- Suspect NAT without obvious mechanism of injury.
- Consider hip pain due to a separate process (limb-length discrepancy, neuromuscular disorders, neoplastic invasion of bone).

ESSENTIAL WORKUP

- Assess distal pulses, palpate compartments, evaluate sensation and motor function.
- If pulses are not equal or palpable, bedside Doppler or angiography may be necessary.
- Search for associated injuries:
 - Neurologic deficits
 - Vascular injury
 - Pelvic fractures (include acetabular fractures)
 - Spinal fractures
 - Blunt abdominal trauma
- Radiographs as outlined below:
 - Remove splints and clothing when taking films.
 - Positive exam plus negative standard films indicates hip fracture until proven otherwise; further imaging (CT or MRI scan) is indicated.
 - Hip dislocations are orthopedic emergencies and require prompt reduction (<6 hr) with limited attempts.

Pediatric Considerations

- In suspected child abuse, obtain appropriate radiographs to evaluate for other injuries.
- Assess markers for NAT:
 - Delay in presentation; history of mechanism inconsistent with injury
 - Isolated trauma to the thigh, associated burns, bruises, linear abrasions

DIAGNOSIS TESTS & INTERPRETATION

Lab

CBC, type and cross-match, INR if appropriate

Imaging

- Standard films: AP pelvis and true lateral of hip, oblique view.
- Femoral neck fracture: AP pelvis with hip internally rotated 15–20°
- Pubic rami and acetabular fractures: Pelvic inlet and outlet views
- Acetabular fractures: Judet views (oblique views of hip)
- High suspicion with negative plain films: CT, MRI, or bone scan. MRI most sensitive.

- Must get postreduction x-ray and/or CT scan.

Diagnostic Procedures/Surgery

- Joint aspiration with or without arthrogram under fluoroscope if a septic joint, foreign body, or hemarthrosis, especially in gunshot wounds to hip is suspected
- Operative repair or wash out

DIFFERENTIAL DIAGNOSIS

- Pubic ramus fracture
- Acetabular fracture
- Septic joint
- Thigh, knee, ankle, or foot injury
- Trochanteric bursitis
- Iliotibial band tendinitis
- Hip contusion



TREATMENT

PRE HOSPITAL

- Neurovascular exam is essential.
- Immobilize extremity in position of comfort for patient.

INITIAL STABILIZATION/THERAPY

- Airway, head, chest, or abdominal injuries take precedence in multiple trauma.
- Maintain pelvis and hip stability.
- Monitor BP continuously.
- Cautions:
 - DO NOT apply traction.
 - Monitor closely for development of hemorrhagic shock as thigh can contain 4–6 U of blood.

ED TREATMENT/PROCEDURES

- Maintain pelvis and hip stability.
- Remove splint and clothing.
- Pain control:
 - Isolated hip injuries: Parenteral analgesia
 - Multitrauma or pediatric patients: Femoral nerve block
- Orthopedic consultation:
 - Necessary for all hip fractures and dislocations
 - Emergent if neurovascular compromise
 - Open fractures must go directly to the OR for irrigation and debridement.
 - May need reduction in OR after 1–2 quick ED attempts to reduce.

- Fractures requiring surgery:
 - Cefazolin IV
- Open fractures with lacerations, extensive soft-tissue damage, or contamination:
 - Add gentamicin/tobramycin, tetanus.
- If highly contaminated wound: Add penicillin G to cover clostridial species.
- Gunshot wounds:
 - Culture missile track, iodine dressing
- Hip dislocation:
 - A true orthopedic emergency
 - Incidence of avascular necrosis and degenerative joint disease increases linearly with time to reduction:
 - Perform reduction in ED, ideally < 6 hr from onset.
 - Allis or Stimson maneuvers
 - Also described: With patient in lateral decubitus position, move hip from flexed and adducted position to full external rotation with tibia perpendicular to floor.
 - Procedural sedation with etomidate, ketamine, or methohexital + midazolam, propofol + fentanyl
 - Look for fractures on postreduction imaging (plain film, CT).
 - Patients with prior hip arthroplasty may be reduced in the ED with procedural sedation and appropriate monitoring.

MEDICATION

- Antibiotics
 - Cefazolin: 1 g IM/IV q6–8h (peds: 25–50 mg/kg IM/IV div. q6–8h max. 1 g)
 - Gentamicin/tobramycin: 3–5 mg/kg/d IV/IM div. q8h (peds: 2–2.5 mg/kg q8h)
 - Penicillin G: 2 million U IV q4h (peds: 100,000–400,000 U/kg/d IV div. q4–6h to max. 24 million U in 24 hr)
- Moderate sedation:
 - Etomidate: 0.1–0.3 mg/kg IV once (not recommended for < 12 yr)
 - Fentanyl: 1–4 µg/kg IV over 1–2 min once (peds: > 6 mo 1–2 µg/kg IV once)
 - Ketamine: Not recommended in adults owing to emergence reaction (peds: 1–2 mg/kg IV, 4 mg/kg IM once)
 - Methohexital: 1–1.5 mg/kg IV once (peds: Not recommended)
 - Midazolam: 0.07 mg/kg IM or 1 mg slowly q2–3min up to 2.5 mg max. (peds: 0.25–1 mg/kg PO once to a max. of 15 mg PO; 6 mo–5 yr: 0.05–0.1 mg/kg IV titrate to max. 0.6 mg/kg; 6–12 yr: 0.025–0.05 mg/kg IV titrate to max. 0.4 mg/kg)
 - Propofol: 40 mg IV q10sec until induction; 5–10 µg/kg/min IV continuous infusion
- Pain control:

- Hydromorphone: 0.5–2.0 mg IM/SC/slow IV q4–6h PRN; titrate for pain control (peds: 0.015 mg/kg/min per dose IV q4–6h PRN)
- Morphine: 2–10 mg IV q4h, titrate for pain control (peds: 0.1 mg/kg IV q4h, titrate for pain control to max. 15 mg/dose)
 - Morphine pediatrics use preservative free preparation.

First Line

- Antibiotics: Cefazolin IV
- Pain: Morphine
- Sedation: User dependent. Etomidate, adults; ketamine, children.

Second Line

- Antibiotics: Ceftriaxone + gentamicin
- Pain: Hydromorphone, fentanyl, nerve block
- Sedation: Methohexital, midazolam, propofol



FOLLOW-UP

DISPOSITION

Admission Criteria

- All hip fractures
- Septic joint
- Suspicion of occult fracture
- Suspicion of NAT in children
- All pediatric hip fractures and dislocations
- Most dislocations of hip

Discharge Criteria

- Hip pain attributable to other cause
- Fracture ruled out (negative radiographs *plus* negative clinical exam)
- Patient with successful reduction of dislocated hip arthroplasty may be considered for discharge in consultation with orthopedics and with appropriate follow-up.
- Stress fracture, crutches, follow-up with bone scan or repeat x-rays.

Issues for Referral

- Chronic pain may need primary physician and pain specialist.
- Pediatric patients and elderly may need physical therapy.

FOLLOW-UP RECOMMENDATIONS

- Discharged patients with hip pain not due to fracture/dislocation are referred to appropriate primary doctor.

- Stress fracture, nonweight bearing: Follow-up orthopedics 2–3 days

PEARLS AND PITFALLS

- Location of fracture determines risk factors for morbidity such as AVN and bleeding.
- Hip dislocations are orthopedic emergencies and require prompt reduction and few attempts.
- Be suspicious of occult fractures, as x-ray may miss 10% fractures. Follow-up study needed (CT or MRI) and possible admission.

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See Also (Topic, Algorithm, Electronic Media Element)

- Femur Fracture
- Pelvic Fracture



CODES

ICD9

- 820.8 Closed fracture of unspecified part of neck of femur
- 835.00 Closed dislocation of hip, unspecified site
- 959.6 Hip and thigh injury

ICD10

- S72.009A Fracture of unsp part of neck of unsp femur, init
- S73.006A Unspecified dislocation of unspecified hip, init encntr
- S79.919A Unspecified injury of unspecified hip, initial encounter

HIRSCHSPRUNG DISEASE

Sheila McMorrow



BASICS

DESCRIPTION

- Described in 1886 by Harold Hirschsprung as a cause of constipation in early infancy
- Congenital aganglionosis megacolon
- 1:5,000 live births
- Overall mortality of Hirschsprung enterocolitis is 35–50%

ETIOLOGY

- Absence of enteric ganglia in the distal bowel
- Normally, ganglia are derived from neural crest and migrate along the intestine, arriving at the proximal colon by 8 wk of gestation and in the rectum by 12 wk of gestation.
- Failure of neural crest cells to migrate into parasympathetic Meissner (submucosal) and Auerbach (myenteric) ganglions results in an aganglionic segment and clinical disease
- Affected bowel classically presents at the internal anal sphincter and involves the rectosigmoid colon (75% of cases)
- May extend the entire length of the GI tract (often fatal)
- Aganglionic segment chronically contracts, forming an obstruction to the passage of stool, and proximal bowel distends to hold the stool that has not passed
- Stimulation of the anus allows passage of stool
- Genetic and other causes
 - Mutations of the *ret* proto-oncogene found in both familial and spontaneous forms
 - Male-to-female ratio 4:1
 - 8% with positive family history; 5–12% of siblings are affected
 - Associated chromosomal abnormality in 5–15%, most commonly trisomy 21 (Down syndrome)
 - Other congenital anomalies in 18%—GI, cardiac, craniofacial, cleft palate, congenital deafness



DIAGNOSIS

SIGNS AND SYMPTOMS

Children with Hirschsprung disease are usually diagnosed by the age of 2 yr

- Three presentations, varying with age:

- Neonatal
 - Abdominal distension
 - Failure of passage of meconium within the 1st 48 hr of life
 - Vomiting
 - Enterocolitis (abdominal pain, fever, foul-smelling and/or bloody diarrhea, vomiting, leading to sepsis and potential intestinal perforation)
 - Sepsis
- Infancy
 - Constipation
 - Chronic abdominal distension
 - Vomiting
 - Failure to thrive
 - Enterocolitis (abdominal pain, fever, foul-smelling and/or bloody diarrhea, vomiting, leading to sepsis and potential intestinal perforation)
 - Toxic megacolon
- Later childhood and adulthood
 - Chronic constipation with obstruction and rare history of overflow incontinence and often refractory to normal treatment protocols
 - Enterocolitis (abdominal pain, fever, foul-smelling and/or bloody diarrhea, vomiting, leading to sepsis and potential intestinal perforation)
 - Abdominal distension
 - Bloody, foul-smelling diarrhea
 - Malnutrition

History

- Sepsis
- Bowel movements frequently require rectal stimulation or enemas
- Narrow caliber stools
- Encopresis and diarrhea are uncommon
- Absence of inciting factors associated with functional constipation (i.e., fissures, toilet training, diet)

Physical-Exam

Possible palpable colon on the left

- Abdominal distension and symptoms of intestinal obstruction
- Findings consistent with malnutrition, failure to thrive
- Rectal exam commonly reveals an empty rectal vault

ESSENTIAL WORKUP

Plain abdominal radiographs:

- Distended small bowel and proximal colon (megacolon) with an empty rectum are common findings
- Transition zone into a narrowed rectosigmoid segment
- In neonates, films will commonly show a distal obstructive pattern
- In children with chronic constipation, films may show only large amounts of stool
- In children with enterocolitis, bowel wall edema or pneumatosis intestinalis may be present

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC, electrolytes, glucose, BUN, Cr
- Urinalysis
- Blood culture, if toxic

Imaging

Barium enema:

- Obtain after stabilization; contraindicated if perforation and enterocolitis are suspected
- Dilated colon proximal to the contracted aganglionic colon with uncoordinated peristalsis, and transition zone between the narrowed aganglionic segment and the dilated and normally innervated segment
 - A transition zone may not be apparent in neonates, because of insufficient time to develop colonic dilatation, or in infants who have undergone rectal exams and enemas
 - May demonstrate a nondistensible rectum, which is a classic sign of Hirschsprung disease
- Delayed barium evacuation

Diagnostic Procedures/Surgery

- Rectal manometry may assist in diagnosis but is often abnormal in long-standing constipation; children with Hirschsprung disease fail to demonstrate reflex relaxation of the internal anal sphincter in response to inflation of a rectal balloon
- Full-thickness rectal biopsy confirms diagnosis by the lack of ganglion cells
 - The definite diagnosis of Hirschsprung disease rests on histologic review of rectal tissue

DIFFERENTIAL DIAGNOSIS

- Neonates
 - Meconium ileus or meconium plug syndrome from cystic fibrosis
 - Intestinal or anal atresia or hypoplasia
 - Malrotation or duplication with volvulus
 - Necrotizing enterocolitis

- Sepsis
- Infants and children
 - Functional constipation
 - Toxic—opiates, anticholinergics
- Infectious—botulism
- Acquired aganglionic colon
 - Metabolic or endocrine—hypothyroid/parathyroid, adrenal insufficiency, electrolyte abnormality
 - Spinal cord defects
- Abdominal masses



TREATMENT

INITIAL STABILIZATION/THERAPY

- Airway, breathing, and circulation management with monitoring as appropriate
- Initial bolus of isotonic IV fluids (20 cc/kg) for shock, dehydration, sepsis

ED TREATMENT/PROCEDURES

- Infants should be managed for bowel obstruction; in the presence of vomiting, an NG tube may be necessary
- Consultation with a pediatric surgeon
 - Triple antibiotics are indicated in toxic patients or those with enterocolitis
- Unstable patients may require decompression by emergent loop colostomy
 - Stable children may have their workup done as an outpatient
 - Definitive treatment is resection of the aganglionic section of bowel to achieve normal ganglion containing bowel within 1 cm of anal opening
- Enterocolitis may occur at any time, even after operative intervention

MEDICATION

- Ampicillin 50 mg/kg div. q6h IV
- Gentamicin 2.5 mg/kg div. q12–24h IV
- Metronidazole 7.5 mg/kg div. q12–48h IV



FOLLOW-UP

DISPOSITION

Admission Criteria

- Neonates and infants presenting with bowel obstruction
- Enterocolitis
- Ill-appearing infants should be admitted to a neonatal/pediatric intensive care unit with available pediatric surgeons

Discharge Criteria

- Well hydrated and taking oral fluids
- Older children with the chief complaint of constipation
- Responsible parents
- Close follow up with a primary care provider

Issues for Referral

Care should be supervised by pediatric gastroenterology and/or pediatric surgery

PEARLS AND PITFALLS

- Presentation varies with the age of the child
- Continuum may vary from toxicity and enterocolitis to chronic constipation
- Toxic child need stabilization, antibiotics and emergent imaging and surgical intervention

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CODES

ICD9

751.3 Hirschsprung's disease and other congenital functional disorders of colon

ICD10

Q43.1 Hirschsprung's disease

HIV/AIDS

Anika Backster • Murtaza Akhter



BASICS

DESCRIPTION

- **AIDS:** Defined as lab evidence of HIV with CD4 < 200 or AIDS defining illness— infection (e.g., cryptosporidium), malignancy (e.g., Kaposi, cervical cancer), or other (e.g., HIV wasting disease, HIV encephalopathy)
- **Opportunistic diseases:**
 - _ CD < 500 cells/mm³:
 - Oropharyngeal candidiasis
 - Pneumococcal infection
 - Hairy leukoplakia
 - Immune thrombocytopenic purpura
 - _ CD4 < 200 cells/mm³:
 - Pneumocystis jiroveci pneumonia (PCP)
 - Cryptococcal infection
 - Disseminated tuberculosis
 - Cryptosporidiosis
 - Isosporiasis
 - Toxoplasmosis
 - Histoplasmosis
 - _ CD4 < 50 cells/mm³:
 - CNS lymphoma
 - Mycobacterium avium complex (MAC)
 - TB pericarditis or meningitis
 - Cytomegalovirus (CMV)
 - Cholangiopathy: Most common cause *Cryptosporidium parvum*



DIAGNOSIS

SIGNS AND SYMPTOMS

- **Primary HIV infection:** 2–6 wk after exposure:
 - _ Fever and malaise
 - _ Rash on face and trunk
 - _ Flu-like syndrome with lymphadenopathy and hepatosplenomegaly
 - _ Pharyngitis
 - _ Diarrhea
 - _ Up to 90% asymptomatic

- Advanced HIV disease (CD4 < 200):
 - Fatigue
 - Fevers and night sweats
 - Weight loss/wasting
 - Alopecia
 - Chronic diarrhea
 - Cough
 - Dyspnea
 - Hemoptysis
 - Chronic low-grade headache
 - Altered mental status
 - Seizures
 - Dementia
 - Neuropathy
 - Painless visual loss
 - Skin lesions

History

- Risk factors:
 - Sexual promiscuity, multiple sexual partners
 - IV drug abuse
 - Men who have sex with men
 - Blood transfusions prior to 1985
 - Unprotected sex with at-risk partners
 - Uncircumcised
- Most recent CD4 count and viral load, lowest CD4 count
- History of or current use of antiretroviral medications
- Medication compliance
- Length of diagnosis/illness
- History of opportunistic infections
- Previous hospitalizations or ICU admissions

ESSENTIAL WORKUP

- HIV serologic tests as noted below:
 - There is a window of 24 wk between primary infection and seroconversion, during which tests may be negative.
 - DNA amplification testing can be positive within 1–2wks of infection, although may not be practical to perform from ED and requires close follow-up and counseling.
- Respiratory symptoms:
 - Chest radiograph
 - Arterial blood gas (ABG)
 - Sputum for Gram stain, AFB, and culture

- Serum LDH—elevated in PCP
- Blood cultures
- Cardiac symptoms:
 - Serum cardiac markers, electrolytes
 - CXR
 - ECG in cases of suspected pericarditis, effusion, or tamponade
 - Blood cultures if endocarditis is suspected
 - Drug screen for cocaine and amphetamines
- Neurologic symptoms:
 - Head CT with and without contrast
 - Lumbar puncture with opening pressure
 - CSF for glucose, protein, Gram stain and culture, cell count with differential, AFB smear, India ink stain, herpes simplex and cryptococcus antigen, and VDRL
- GI symptoms:
 - Stool for ova and parasites, Gram stain, culture, and *Clostridium difficile* assay
 - Urine analysis
 - For women: Urine pregnancy test, pelvic exam with wet mount, and gonorrhea/chlamydia testing
 - Liver functions tests, amylase, and lipase
 - Hepatitis serologies
 - Low threshold for CT abdomen/pelvis
 - US if biliary symptoms present
 - Low threshold for surgical consult, as HIV patients may not present with classic acute abdomen
- Fever workup:
 - Include aerobic/anaerobic, fungal, AFB, and MAC blood cultures
- Ocular symptoms:
 - Fluorescein staining with slit lamp exam

DIAGNOSIS TESTS & INTERPRETATION

Lab

- ELISA:
 - Detects IgG antibody against HIV
 - Sensitivity and specificity ~ 99%
 - Can be negative during the window period
- Western blot:
 - Detects IgG antibody against HIV proteins p24, gp 120, gp 41
 - Used to confirm a positive ELISA
 - Able to detect HIV during the 6 mo seroconversion period
- Rapid HIV testing:

- Results available in 5–20 min
- 4 types of tests currently available
- Samples include oral swabs, whole blood, serum, or plasma
- All reactive tests require confirmatory testing with western blot or ELISA
- >99% specific and sensitive
- Absolute lymphocyte count (ALC):
 - Multiply WBC × percent lymphocytes
 - If ALC >2,000, likely CD4 >200, if ALC <1,000, likely CD4 <200

Imaging

- CXR:
 - Bilateral interstitial infiltrates: PCP
 - Reticulonodular infiltrates: TB, KS, or fungal pneumonia
 - Hilar lymphadenopathy with infiltrate: TB, cryptococcosis, histoplasmosis, neoplasm
 - Lobar consolidation: Bacterial pneumonia
 - Cavitation: TB, necrotizing bacterial pneumonia, coccidioidomycosis
 - Normal x-ray does not rule out PCP or TB
- Head CT with and without IV contrast:
 - Multiple ring-enhancing lesions with edema in basal ganglia or cortex: Toxoplasmosis or CNS lymphoma
 - Subcortical nonenhancing lesions: PML
- Abdominal/pelvic CT:
 - Splenomegaly: CMV, TB
 - Intestinal perforation or bowel obstruction: CMV colitis, lymphoma, histoplasmosis, MAC, appendicitis, ulcer disease, KS
 - Cholecystitis or cholangitis: Cryptosporidium, Microsporidium, CMV
 - Pancreatitis: Medication-related, neoplasm, infectious

DIFFERENTIAL DIAGNOSIS

- For pulmonary symptoms with HIV:
 - Pulmonary emboli
 - Pulmonary HTN
 - TB
 - Pneumonia: Bacterial, fungal, viral
 - Pulmonary malignancies
 - Lymphocytic interstitial pneumonitis
- For CNS symptoms with HIV:
 - Neurosyphilis
 - CMV or HSV encephalitis
 - Toxoplasmosis
 - CNS lymphoma
 - Meningitis (bacterial, coccidioidal, etc.)

- Subarachnoid hemorrhage
- Cerebral infarction
- HIV or metabolic encephalitis
- Progressive multifocal leukoencephalopathy
- Cardiac symptoms with HIV:
 - Cardiomyopathy
 - Pericarditis/myocarditis
 - Endocarditis
 - Acute coronary syndrome
 - Pericardial effusion
- Oral symptoms with HIV:
 - Fungal infection (i.e., candidiasis)
 - Viral lesions (HSV, CMV, hairy leukoplakia)
 - Bacterial lesions (TB, periodontal disease)
 - Autoimmune (salivary gland disease, aphthous ulcers)
 - Neoplasm (KS, lymphoma)
- Esophageal symptoms with HIV:
 - Infectious esophagitis (candida, CMV, HSV)
 - Reflux esophagitis
- Diarrhea with HIV:
 - Medication side effect
 - Parasites (Cryptosporidium, Giardia, Isospora)
 - Bacteria
 - Viral (CMV, HSV, HIV)
 - Fungi (histoplasmosis, cryptococcus)
 - HIV-associated enteropathy
- Hepatomegaly with HIV:
 - Hepatitis
 - Opportunistic infection (CMV, MAC, TB)
- Renal disease with HIV:
 - Drug nephrotoxicity
 - HIV nephropathy
 - Vasculitis
 - Obstruction



TREATMENT

ED TREATMENT/PROCEDURES

- Patients who appear to have bacterial infections, appear toxic, or have rapidly progressive symptoms should receive their 1st dose of antibiotics in the ED.
- Begin HIV treatment if: Low CD4 (< 350) or high viral load, pregnancy, AIDS defining illness or HIV-associated nephropathy (in general, patients with

documented primary HIV infection also undergo resistance testing after the diagnosis has been established)

- Triple therapy (HAART):
 - _ 1 non-nucleoside reverse transcriptase inhibitors (NNRTI) and 2 nucleoside reverse transcriptase inhibitors (NRTI)
 - _ 1 PI and 2 NRTIs
 - _ Triple NRTI
- Postexposure prophylaxis:
 - _ Start therapy within 2 hr if possible and continue for 4 wk
 - _ 2-drug regimen for most exposures:
 - Zidovudine + lamivudine (combivir)
 - Lamivudine + stavudine
 - Stavudine + didanosine
 - _ 3-drug expanded regimen for very high-risk exposure
- Toxoplasmosis: Pyrimethamine:
 - _ Sulfadiazine
 - _ Leucovorin
 - _ Steroids for cerebral edema
 - _ Treat for at least 6 wk
- Cryptococcal meningitis:
 - _ Amphotericin B
 - _ Flucytosine
 - _ Treat with above for 2 wk, then fluconazole for 8 wk
- CMV retinitis: Ganciclovir
- Esophageal candidiasis:
 - _ Fluconazole for 14–21 days
- MAC: Clarithromycin:
 - _ Ethambutol
 - _ May add rifabutin if severe immunosuppression
- PCP:
 - _ Trimethoprim/sulfamethoxazole
 - _ Pentamidine or dapsone for sulfa-allergic patients
 - _ If PaO₂ < 70 mm Hg or A-a gradient > 35 mm Hg, add prednisone 40 mg PO BID for 5 days, then taper
- Oral candidiasis: Clotrimazole troches
- HIV acute demyelinating polyneuropathy: Plasmapheresis

MEDICATION

- Common medication complications:
 - _ Hypersensitivity reaction: Abacavir
 - _ Pancreatitis:
 - Dideoxyinosine
 - Dideoxycytidine

- Didanosine
- Lamivudine
- Cotrimoxazole
- Pentamidine
- Ritonavir
- Stavudine
- Zalcitabine
- _ Peripheral neuropathy:
 - Didanosine
 - Isoniazid
 - Linezolid
 - Stavudine
 - Zalcitabine
- _ Kidney stones: Indinavir and Atazanavir
- _ Hepatotoxicity: All agents to some degree:
 - Nevirapine
 - Didanosine
 - Stavudine
- _ Lactic acidosis: Stavudine:
 - Didanosine
- _ Stevens–Johnson syndrome:
 - Nevirapine
 - Atazanavir
 - Delavirdine
 - Efavirenz
 - Cotrimoxazole
 - Trimethoprim/sulfamethoxazole
- _ Hemolytic anemia:
 - Dapsone (used for treatment of TB)
 - Zidovudine with ribavirin
- _ Psychosis: Efavirenz
- _ Hypoglycemia: Pentamidine
- _ Postural hypotension: Maraviroc
- _ Hyperlipidemia, truncal obesity, and atherosclerosis: Stavudine:
 - Protease inhibitors
- _ Dilated cardiomyopathy: Zidovudine
- _ Benign increase in unconjugated bilirubin: Atazanavir and indinavir
- _ Macrocytic anemia: Zidovudine
- _ Many cause some hematologic effects, GI upset, and rash



DISPOSITION

Admission Criteria

- Unexplained fever with CNS involvement or suspected endocarditis
- Neutropenic fever
- Hypoxemia ($\text{PaO}_2 < 70$ mm Hg)
- Cardiac symptoms suggestive of ACS
- Pericardial effusion
- Suspected bacterial pneumonia or TB
- A change in neurologic status
- New-onset seizures
- Hemodynamic instability
- Inability to ambulate or tolerate oral intake
- Intractable diarrhea with dehydration

Discharge Criteria

The patient can maintain adequate oral intake, provide self-care, and ambulate.

Issues for Referral

- Patient should be referred to a primary HIV care provider for initiation of HAART therapy regimen and ongoing care.
- Be alert for signs of depression and refer for counseling or psychiatric treatment as this may inhibit treatment compliance.
- HIV patients are at higher risk for many malignancies—refer those with concerning symptoms for follow-up.

PEARLS AND PITFALLS

- Immune reconstitution inflammatory syndrome usually manifests within 8 wk of initiation of HAART as symptoms of opportunistic or autoimmune disease.
- For occupation exposures, there is a low risk of seroconversion (0.3% for significant percutaneous exposure and 0.09% for mucocutaneous).
- HIV patients on HAART should be considered at higher risk for insulin resistance and acute coronary syndrome/CAD, independent of other risk factors.
- Measure oxygen saturation after walking in patients with a normal CXR and symptoms of pneumonia to help diagnose PCP.
- HIV is an independent risk factor for COPD, pulmonary hypertension, CVA, venous thromboembolic disease, TTP, osteoporosis, and osteonecrosis of the hip.

ADDITIONAL READING

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CODES

ICD9

- 042 Human immunodeficiency virus [HIV] disease
- V08 Asymptomatic human immunodeficiency virus [HIV] infection status
- 007.4 Cryptosporidiosis

ICD10

- A07.2 Cryptosporidiosis
- B20 Human immunodeficiency virus [HIV] disease
- Z21 Asymptomatic human immunodeficiency virus infection status

HORDEOLUM AND CHALAZION

Shari Schabowski

BASICS

DESCRIPTION

- Result from inflammatory processes involving the glands of the eyelid along the lash line:
 - Hordeolum—acute glandular obstruction resulting in inflammation and abscess formation
 - Chalazion—end result of inspissation of glandular contents and chronic granulomatous inflammation
- Hordeolum:
 - Develops owing to outflow obstruction in 1 or more of the glands of the eyelid
 - Obstructed glands may become secondarily infected.
 - May progress to localized abscess formation or may be complicated by periorbital cellulitis
- Chalazion:
 - Chronic granulomatous inflammation in the meibomian gland:
 - Originates from inspissated secretions
 - Blockage of the gland's duct at the eyelid margin may result in release of the contents of the gland into the surrounding eyelid soft tissue.
 - A lipogranulomatous reaction ensues
 - Occasionally, chalazia become secondarily infected.
 - May evolve from incompletely drained internal hordeolum

ETIOLOGY

Hordeolum:

- May become secondarily infected:
 - Staphylococcus most common
- Predisposing conditions:
 - Meibomian gland dysfunction
 - Blepharitis
 - Rosacea
 - Previous hordeolum

DIAGNOSIS

SIGNS AND SYMPTOMS

- Hordeolum:

- Develops acutely when glandular outflow is obstructed
- Red, tender, painful, swollen mass along the eyelid margin
- Typically solitary, rarely may be multiple
- May be recurrent
- Well-localized inflammation
- Presentation depends on which gland is affected:
 - External hordeolum (stye):
 - Originates from obstruction of the superficial sebaceous or sweat glands whose ducts are located between the eye lashes
 - Exquisitely tender small mass that typically points anteriorly
 - Internal hordeolum:
 - Originates from obstruction of the sebaceous glands whose ducts are located on the inner aspect of the lid margin
 - Painful small mass that is palpable through the eyelid
 - May cause a foreign body sensation in the eye and visual disturbance
 - Typically more inflamed, larger, and more painful
 - May point internally or through skin
- Nonsystemic process
- May be complicated by:
 - Conjunctivitis
 - Periorbital cellulitis
- Chalazion:
 - Firm, circumscribed, nontender, or minimally tender nodule:
 - Typically long standing
 - Noninflamed
 - Symptoms most commonly owing to physical properties:
 - Disrupts natural contour of eye
 - Obstructs visual field/peripheral vision
 - Pressure on globe
 - Corneal desiccation or injury due to exposure
 - Nonacute, nonemergent process, which requires no urgent or emergent intervention unless secondary corneal or significant globe pressure is present.

History

Hordeolum—sudden, well localized, painful mass along the margin of eyelid:

- No systemic symptoms

Physical-Exam

Focal, tender, inflammation of an external or internal gland of the eyelid:

- Minimal surrounding edema may be seen
- Abscess may point within lash line, from palpebral conjunctiva or externally via

skin of the lid

ESSENTIAL WORKUP

- Complete ophthalmologic exam including slit lamp exam and corneal evaluation
- Evaluation for evidence of associated cellulitis and/or systemic findings
- Hordeolum:
 - Identify the origin of the abscess
- Chalazion:
 - Determine whether physical properties of chalazion result in corneal exposure and injury.

DIAGNOSIS TESTS & INTERPRETATION

Lab

Cultures of any drainage rarely aids in management

DIFFERENTIAL DIAGNOSIS

- Blepharitis
- Dacryocystitis
- Dacryoadenitis
- Pyogenic granuloma
- Sebaceous cell carcinoma
- Basal cell carcinoma
- Squamous cell carcinoma



TREATMENT

ED TREATMENT/PROCEDURES

- Hordeolum—relieve obstruction and prevent abscess formation
 - Warm compresses for 15 min 4–6 times per day
 - Gently massage the nodule to express obstructed material
 - Rarely, in severe cases, incision and drainage of internal hordeolum may be necessary:
 - Typically done by ophthalmologist
 - If pointed toward the conjunctiva, vertical incision is made to avoid injury to the meibomian glands and reduce corneal injury from inadvertent scarring.
 - External skin incision is very rarely indicated.
 - When necessary, horizontal incision is used
 - Removing single involved eyelash may be helpful in rare more severe cases of external hordeolum
 - Botox

- Chalazion—complaints typically reflect nonemergent aesthetic and cumbersome physical properties of the mass:
 - Referral to ophthalmology for incision and curettage or steroid injection
 - Lubricating eye drops may provide symptomatic relief

MEDICATION

Ophthalmologic moisturizing drops as needed for comfort.



FOLLOW-UP

DISPOSITION

Discharge Criteria

No indication for admission unless secondary complication is present (i.e., marked periorbital cellulitis with systemic symptoms)

Issues for Referral

- Urgent consultation with ophthalmologist should be considered if incision and drainage of internal hordeolum is deemed indicated.
- Chalazia should be referred to ophthalmologist for definitive treatment options.

FOLLOW-UP RECOMMENDATIONS

- Follow-up with ophthalmology in 1–2 days to evaluate response to conservative management.
- Symptoms should completely resolve in 1–2 wk

PEARLS AND PITFALLS

- Conservative treatment of hordeola with warm compresses and gentle massage is the standard:
 - Majority of cases respond without further intervention
 - Emergent incision and drainage is rarely indicated and should only be considered in extreme cases
 - Incision and drainage may result in long-term complications including corneal injury, fistula formation, and aesthetic complications
 - Consult ophthalmology for incision and drainage if possible
- Chalazia do not require emergent intervention:
 - Referral is the standard management

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Dacryoadenitis
- Dacryocystitis
- Red Eye



CODES

ICD9

- 373.2 Chalazion
- 373.11 Hordeolum externum
- 373.12 Hordeolum internum

ICD10

- H00.019 Hordeolum externum unspecified eye, unspecified eyelid
- H00.19 Chalazion unspecified eye, unspecified eyelid
- H00.029 Hordeolum internum unspecified eye, unspecified eyelid

HORNER'S SYNDROME

Richard S. Krause



BASICS

DESCRIPTION

Unilateral sympathetic denervation of the eye produces signs of Horner's syndrome:

- Relaxation of retracting muscles in upper and lower lids:
 - *Ptosis* (drooping of the lids)
- Loss of pupillary dilator innervation:
 - *Miosis* (unopposed pupillary constriction)
- Loss of sympathetic stimulation of sweat glands:
 - Anhidrosis

ETIOLOGY

- 40% unknown (in 1 large series)
- Tumors of lung or metastases to cervical nodes:
 - May interrupt preganglionic sympathetic fibers (between thoracic sympathetic trunk and superior cervical ganglion)
- Trauma: Penetrating neck wounds directly injure sympathetic fibers
- Pneumothorax:
 - Tension pneumothorax may cause traction on sympathetic fibers owing to shift of mediastinal structures.
- Infiltration or infection of cervical nodes:
 - Sarcoidosis, tuberculosis
- Vascular disorders:
 - Migraine or cluster headaches
 - Carotid artery dissection
- Lateral medullary infarction produces Horner's syndrome as part of the Wallenberg syndrome:
 - Presents with vertigo and ataxia, which may overshadow the Horner's syndrome.
- Cavernous sinus thrombosis may present with some of the features of Horner's syndrome:
 - The condition typically causes headache and/or eye pain.
 - Ocular signs include ocular palsies, pain, chemosis, and proptosis.

Pediatric Considerations

- Hereditary Horner's syndrome:
 - Blue iris (or irregular coloration) on affected side
 - Brown on unaffected side (heterochromia iridis)

- Birth trauma:
 - May cause damage to sympathetic chain
- New Horner's syndrome in a child should prompt workup for tumor (neuroblastoma).



DIAGNOSIS

SIGNS AND SYMPTOMS

- Horner's syndrome is characterized by:
 - *Ptoxis*: Drooping of eyelid on affected side, usually slight
 - *Miosis*: Decrease in pupillary size on involved side (pupillary asymmetry ≥ 1 mm)
 - *Anhidrosis*: Lack of sweating on involved side of face
- The importance of Horner's syndrome is its association with certain disease states.

History

Focus on pre-existing conditions that predispose to Horner's syndrome or are risk factors for these conditions:

- Tumors, vascular disease, trauma:
 - Minor trauma often precedes carotid dissection.
- Cardiovascular risks
- Exposures:
 - For pseudo-Horner
- Pain:
 - Suggests carotid dissection

ALERT

- Acute Horner's syndrome with neck or facial pain:
 - Presume carotid dissection until proven otherwise:
 - 50% of internal carotid artery dissections present with a painful Horner's syndrome.

Physical-Exam

Concentrate on a focused neurologic exam looking to confirm Horner's syndrome and exclude other neurologic deficits:

- General physical exam should focus on identifying signs of other suspected conditions, such as tumor.

ESSENTIAL WORKUP

- History and physical exam focused on neurologic findings
- CXR to screen for tumor or pneumothorax

DIAGNOSIS TESTS & INTERPRETATION

Provocative testing:

- Pharmacologic (cocaine) testing confirms diagnosis of sympathetic ocular lesion:
 - 1 drop of 5% ocular cocaine solution is instilled *into each eye*.
 - Failure of pupil on involved side to dilate as much as other pupil (increase in amount of anisocoria) in 1 hr is confirmatory (positive test).

Lab

Not useful for Horner per se:

- Often needed as part of the workup of causative or associated conditions.

Imaging

- CXR is usually indicated because of the association of chest pathology and Horner's syndrome.
- CT or MRI of head, neck, or chest may be indicated depending on signs and symptoms.
- For suspected carotid dissection:
 - MRA or CTA of the head and neck:
 - Either test is appropriate
 - The lesion is expected to be ipsilateral to the Horner's syndrome.
- Choice of neuroimaging in suspected stroke depends heavily on local protocols and resources.
 - At a minimum, if stroke is suspected CT of the brain is indicated to rule out hemorrhagic stroke.
 - If acute stroke (< 3 hr) is suspected and patient is a candidate for thrombolysis image on STAT basis

Diagnostic Procedures/Surgery

Ocular tonometry is indicated if acute glaucoma is suspected.

DIFFERENTIAL DIAGNOSIS

- Increased intracranial pressure (ICP):
 - Almost always associated with altered level of consciousness (LOC), headache
- *Simple anisocoria (pseudo-Horner's syndrome)*:
 - 15–20% of the population has anisocoria and 3–4% also has miosis and ptosis.
 - Cocaine test is negative (both pupils dilate equally).
 - Inspect photo ID for pre-existing anisocoria.
- Topical medications or exposures are a common cause of miosis.
- Migraine or cluster headache
- Glaucoma, inflammatory ocular diseases, or ocular trauma



TREATMENT

PRE HOSPITAL

Cautions:

- The importance of Horner's syndrome is its association with more serious underlying conditions.
- Patients with increased ICP or tension pneumothorax must be recognized and treated as soon as possible.
- If acute stroke suspected transport to designated stroke center when possible

INITIAL STABILIZATION/THERAPY

- If increased ICP is suspected, initiate measures to control ICP:
 - _ Intubation, osmotic diuretics
- Tension pneumothorax:
 - _ Needle thoracostomy followed by chest tube

ED TREATMENT/PROCEDURES

Horner's syndrome per se requires no ED treatment:

- Causative or associated conditions may require treatment.

MEDICATION

Cocaine: 5% (adult), 2.5% (peds) ophthalmic solution: 1 drop in each eye is diagnostic.



FOLLOW-UP

DISPOSITION

Admission Criteria

Admission for isolated Horner's syndrome is not needed:

- Admission may be needed for underlying condition.

Discharge Criteria

- Stable patients with isolated Horner's syndrome may be discharged with appropriate follow-up arranged for continued workup as outpatient:
 - _ When Horner's syndrome is suspected, emergencies such as carotid dissection or stroke should be ruled out prior to discharge.

FOLLOW-UP RECOMMENDATIONS

Neurologists and ophthalmologists must often be involved in the workup of Horner's syndrome.

PEARLS AND PITFALLS

Must consider underlying etiology

ADDITIONAL READING

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- Proctor C, Chavis PS. Horner's syndrome. *J Neuroophthalmol*. 2013;33(1):88–89
- Thanvi B, Munshi SK, Dawson SL, et al. Carotid and vertebral artery dissection syndromes. *Postgrad Med*. 2005;81:383–388.
- Wilhelm H, Ochsner H, Kopycziok E, et al. Horner's syndrome: A retrospective analysis of 90 cases and recommendations for clinical handling. *Ger J Ophthalmol*. 1992;1(2):96–102.



CODES

ICD9

- 337.9 Unspecified disorder of autonomic nervous system
- 374.30 Ptosis of eyelid, unspecified
- 379.42 Miosis (persistent), not due to miotics

ICD10

- G90.2 Horner's syndrome
- H02.409 Unspecified ptosis of unspecified eyelid
- H57.03 Miosis

HUMERUS FRACTURE

G. Richard Bruno

BASICS

DESCRIPTION

- Proximal humeral fractures:
 - Typically described as nondisplaced, displaced, and/or fracture/dislocation
 - Account for 5% of all fractures
 - Increased incidence with age
 - 4:1 female to male
 - Vast majority of patients are >60 yr old
 - 3rd most common osteoporotic fracture, after hip and distal radius fractures
 - *Neer classification*: A system that identifies the number of fragments and their location:
 - Fractures consist of 2–4-part fractures; the locations include the anatomic neck, surgical neck, greater tuberosity, and lesser tuberosity.
 - Fracture/dislocations also part of the Neer classification
- Humeral shaft fractures:
 - Account for <3% of fractures
 - May be spiral, oblique, or transverse
 - Humeral shaft fractures (AO classification):
 - Simple
 - Wedge
 - Comminuted (complex)

ETIOLOGY

- Proximal humerus fractures:
 - Most often a history of a fall (low energy)
 - Most common is fall on outstretched hand
 - Less common is violent muscle contraction from shock or seizure or higher-energy injury
- Humeral shaft fractures:
 - High-energy direct trauma (penetrating or blunt) or bending force
 - Less common from fall
 - Stress fractures from throwing injury

DIAGNOSIS

SIGNS AND SYMPTOMS

- Pain, swelling, and tenderness
- Difficulty in initiating active motion
- Arm often closely held against chest
- Shortening of the extremity
- Crepitus may be present
- Ecchymosis
- Neurovascular compromise

History

- Mechanism of injury
- Contributory comorbid factors (age, fall risk, malignancy)
- Associated injuries

Physical-Exam

- Complete exam of the affected extremity:
 - _ Inspect shoulder and humerus for obvious deformity, shortening, and open injuries
 - _ Assess ROM at shoulder, elbow
 - _ Neurovascular exam

ESSENTIAL WORKUP

- Assess individual nerves:
 - _ Radial (special attention in midshaft humeral fractures)
 - _ Median
 - _ Ulnar
 - _ Axillary (sensation to the lateral aspect of the shoulder)
 - _ Musculocutaneous nerve (sensation to the extensor aspect of the forearm)
- Assess vascular supply:
 - _ Presence of radial, ulnar, and brachial pulses
 - _ Good capillary refill in all digits
- Radiology exams to define injury

Pediatric Considerations

- Most common in age <3 or >12 yr
- Neonatal fractures from delivery trauma:
 - _ Most common in neonates >4.5 kg and breech births
 - _ May see pseudoparalysis
- Older children: Same injury mechanisms as adults
- Periosteum thicker in children and limits displacement of humeral shaft fractures
- Proximal humeral fractures:
 - _ Salter I fractures should be considered when films of proximal humerus are normal but significant pain is present

- Salter II most common in younger children
- Consider abuse (especially <3 yr):
 - Injury patterns:
 - Transverse (direct blow)
 - Oblique/spiral (traction and humeral rotation)
 - Metaphyseal fractures (bucket-handle fractures)

DIAGNOSIS TESTS & NTERPRETATION

Imaging

- Plain films:
 - Proximal humeral fractures (at least 3 views):
 - Anteroposterior (AP), lateral, and axillary views
 - Axillary view to assess tuberosity displacement, glenoid articular surface, and relationship of the humeral head to glenoid
 - Humeral shaft fractures:
 - AP and lateral views of entire humerus are mandatory.
 - Include shoulder and elbow views to exclude associated joint involvement.
- CT scan:
 - Helpful in proximal humeral fractures to define complex/comminuted injures and plan surgery
 - Help define relationship of humeral head to glenoid fossa in suspected fracture/dislocations

Diagnostic Procedures/Surgery

Not applicable

DIFFERENTIAL DIAGNOSIS

- Acute hemorrhagic bursitis
- Traumatic rotator cuff tear
- Dislocation
- Acromioclavicular separation
- Calcific tendinitis
- Contusion
- Tendon rupture
- Neurapraxia
- Pathologic fracture



TREATMENT

PRE HOSPITAL

Cautions:

- Avoid excessive movement of the arm, which may produce further neurovascular injury.
- Immobilize with sling and swath and transport.
- Rapid transport in the presence of neurologic or vascular deficits

INITIAL STABILIZATION/THERAPY

- Primary and secondary survey for associated injuries
- Immobilization:
 - _ For comfort
 - _ Prevent fracture displacement
 - _ Prevent neurovascular injury
- Axillary pad may also be used for comfort.
- Pain control
- Application of ice to limit swelling
- Open fractures:
 - _ Cover with a sterile dressing
 - _ Tetanus prophylaxis
 - _ Parenteral antibiotics

ED TREATMENT/PROCEDURES

- Patient should receive adequate analgesia during diagnosis and treatment of injury:
 - _ Narcotics PO/IM/IV are first-line therapy
- Proximal humeral fractures:
 - _ Single-part proximal humeral fractures:
 - >80% of proximal humeral fractures
 - Can be treated nonoperatively
 - Treatment is sling and swath
 - Early ROM exercises often employed
 - _ Displaced, multipart proximal humeral fractures:
 - Use Neer classification to describe
 - >1 cm separation or >45° are considered displaced
 - Orthopedic review and referral is appropriate for 2-part or higher fractures
 - Many 2-part fractures can be reduced and managed nonoperatively
 - 3- and 4-part fractures may need ORIF/hemiarthroplasty
 - Surgical options depend not only on type of fracture, but also patient's age, comorbidities, and patient's functional expectations of the extremity
 - _ Indications for emergent orthopedic consult for proximal humeral fractures:
 - Open fracture
 - Fracture/dislocation that cannot be reduced in ED

- Vascular compromise
- Humeral shaft fractures:
 - Most humeral shaft fractures can be managed nonoperatively and do not require reduction (>90%)
 - 20° of anterior angulation and 30° of varus angulation are well tolerated by the musculature around the humerus.
 - Humerus can tolerate up to 3 cm of shortening with little functional deficit
- Nondisplaced humeral shaft fractures:
 - ED can treat with a coaptation splint
 - Except transverse fractures
 - Functional brace may be utilized by orthopedist
- Transverse fractures:
 - ED treatment should be sling and swath
 - Higher incidence of nonunion
- Displaced humeral shaft fractures:
 - Orthopedist may utilize hanging cast to treat and reduce displaced or shortened fractures.
- Indications for emergent orthopedic consult for humeral shaft fractures:
 - Neurovascular compromise
 - Segmental fractures
 - Fractures extending into articular surface
 - Open fractures
 - “Floating elbow” (fractures with concurrent ipsilateral forearm fractures)

Pediatric Considerations

In children nearing skeletal maturity, it is essential to determine the degree of displacement or separation of the proximal humeral epiphysis, as exact reduction is important to prevent later disturbance of growth.

MEDICATION

- Pain medications:
 - Narcotics (first line)
 - NSAIDS (second line)
- Procedural sedation with closed reductions (see Procedural Sedation)



FOLLOW-UP

DISPOSITION

Admission Criteria

- Open fractures for operative management and parenteral antibiotic therapy
- Fractures associated with vascular compromise

- Displaced fractures that cannot be treated adequately through closed reduction
- Significant associated injuries that require admission and observation

Discharge Criteria

- Nondisplaced fracture
- Fracture treated with closed reduction
- Most closed humeral shaft fractures without other injuries
- More complicated proximal humeral fractures, (i.e., proximal humeral fracture of Neer 3 and 4 type), that may require nonemergent surgery. Discharge should be done in consultation with orthopedist.

Pediatric Considerations

- Pediatric patients are often less compliant with immobilization and less able to verbalize complaints; they may benefit from admission.
- Assess safety of environment in cases suspicious of NAT.

Issues for Referral

- Most humeral fractures should have outpatient orthopedic referral.
- Complicated proximal humeral fractures (Neer classification 2–4) should be reviewed with orthopedist to develop outpatient plan and possible nonemergent intervention.
- Some single-part nondisplaced proximal humeral fractures may be managed by the PCP.
- Displaced humeral shaft fractures require orthopedic referral for definitive care (functional bracing, hanging cast, ORIF, etc.).

FOLLOW-UP RECOMMENDATIONS

- Most patients should be seen in close follow-up for repeat exam of the injured extremity, to verify adequate pain control, and to review treatment plan soon after ED visit.
- Proximal humeral fractures that are stable should be evaluated for early ROM therapy to minimize risk of adhesive capsulitis.

PEARLS AND PITFALLS

- All humeral fractures should have diligent neurovascular exams:
 - Neurovascular exam should be repeated after manipulation.
 - Radial nerve injuries are the most common deficits seen in humeral shaft fractures.
 - Most radial nerve deficits will resolve spontaneously over time (months).
- Avascular necrosis is a risk in proximal humeral fractures that involves the surgical neck or articular surface.
- Patients with multiple-part proximal humeral fractures (Neer 2 or higher) may

often be discharged from the ED, but a plan must be developed with the orthopedist because surgical intervention and/or hemiarthroplasty are possible.

ADDITIONAL READING

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- Rothberg D, Higgins T. Fractures of the proximal humerus. *Orthop Clin North Am.* 2013;44:9–19.
- Walker M, Palumbo B, Badman B, et al. Humeral shaft fractures: A review. *J Shoulder Elbow Surg.* 2011;20:833–844.



See Also (Topic, Algorithm, Electronic Media Element)

- Conscious Sedation
- Elbow Fracture
- Shoulder Dislocation



CODES

ICD9

- 812.01 Closed fracture of surgical neck of humerus
- 812.02 Closed fracture of anatomical neck of humerus
- 812.20 Closed fracture of unspecified part of humerus

ICD10

- S42.213A Unsp disp fx of surgical neck of unsp humerus, init
- S42.296A Oth nondisp fx of upper end of unsp humerus, init
- S42.309A Unsp fracture of shaft of humerus, unsp arm, init

HYDATIDIFORM MOLE

Emi M. Latham

BASICS

DESCRIPTION

- Noninvasive localized tumors arising from trophoblastic tissue
- Can be associated with malignancy
- Twinning with normal pregnancy possible:
 - Higher risk for persistent maternal disease and metastasis
 - Possible to have normal infant
- Complete mole:
 - Estimated in 1/1,500 pregnancies
 - Fetal tissue not present
 - Diffuse chorionic villi swelling
 - Diffuse trophoblastic hyperplasia
 - Malignancy associated in 15–20%, usually lung
 - Genetics:
 - Karyotype: 46,XX (90%); 46,XY (10%)
 - Paternal DNA expressed
 - Enucleate egg fertilized by 2 sperms or by a haploid sperm that duplicates
- Partial mole:
 - Estimated in 1/750 pregnancies
 - Fetal or embryonic tissue often present
 - Focal chorionic villi swelling
 - Focal trophoblastic hyperplasia
 - Malignancy associated in 4–12%
 - Genetics:
 - Karyotype: 90% are triploid 69XXX, 69XXY, rarely 69XYY
 - Maternal and paternal DNA
 - Haploid ovum duplicates and is fertilized by normal sperm, or haploid ovum fertilized by 2 sperms

ETIOLOGY

- Largely unknown
- Extremes of maternal age best estimated risk factor:
 - > 35 yr old carries 5–10-fold risk
 - < 20 yr old
- Previous molar pregnancy carries 1–2% risk in future pregnancies
- Frequency multiple times greater in Asian and Latin American countries:

- 1 per 1,000–1,500 live births in US and Western Europe
- Reported up to 1 per 12–500 live births in other countries
- Deficiency in animal fat and vitamin A
- Smoking (> 15 cigs/day)
- Maternal blood type AB, A, or B
- History of infertility, nulliparity
- Finding in 1 of 600 therapeutic abortions

DIAGNOSIS

SIGNS AND SYMPTOMS

- Usually exaggerated subjective symptoms of pregnancy
- Complete mole:
 - Vaginal bleeding, most common (97%):
 - Late 1st trimester
 - Usually painless and like “prune juice”
 - May also have vaginal tissue passage:
 - Often described as grapelike vesicles
 - Usually occurs in 2nd trimester < 20 wk
 - Hyperemesis from high levels β -hCG
 - Preeclampsia (27%):
 - Visual changes
 - HTN
 - Proteinuria
 - Hyperreflexia
 - Possibly convulsions
 - Hyperthyroidism (7%):
 - Marked tachycardia, tremor
 - Due to high levels of β -hCG or thyroid stimulating substance (thyrotropin)
 - Acute respiratory distress (2%):
 - Tachypnea, diffuse rales, tachycardia, mental status changes
 - Possible embolism of trophoblastic tissue
 - May also be due to cardiopulmonary changes from preeclampsia, hyperthyroidism, or iatrogenic fluid replacement
- Partial mole:
 - Usually does not exhibit dramatic clinical features of complete mole
 - Frequently presents with symptoms similar to patients with threatened or spontaneous abortion:
 - Vaginal bleeding
 - May have fetal heart tones
 - Often presents at more advanced gestational age

History

Similar to that of pregnancy:

- Missed menstrual periods
- Positive pregnancy test
- Nausea, vomiting, vaginal bleeding

Physical-Exam

- Uterine size/date discrepancy occurs in 50–66% of cases
 - Complete mole usually larger than dates would indicate
 - Partial mole can be smaller than dating suggests
- Ovarian masses:
 - Present in complete moles, rarely in partial moles
 - Usually from ovarian enlargement
 - Multiple bilateral theca lutein cysts due to high levels of β -hCG, usually found by US

ESSENTIAL WORKUP

- hCG
 - Complete mole β -hCG > 100,000 mIU/mL, but can be normal
 - Partial mole: Usually lower than that seen with normal pregnancy
 - β -hCG > 40,000 mIU/mL carries poor prognosis
- US:
 - Complete mole:
 - Characteristic “snowstorm” vesicular pattern
 - Absence of fetal tissue and swelling of chorionic villi with anechoic spaces
 - No amniotic fluid
 - Theca lutein cysts
 - Bilateral, multiloculated
 - Large at 6–12 cm
 - Partial mole:
 - “Swiss-cheese” appearance
 - Cystic changes in placenta with scalloping of villa and in shape of gestational sac
 - Fetus may be present

DIAGNOSIS TESTS & INTERPRETATION

Lab

- β -hCG
- Blood type, Rh, and cross-match
- CBC to assess for anemia and thrombocytopenia
- Coagulation profile to assess for disseminated intravascular coagulation

- Electrolytes with BUN and creatinine
- LFTs
- TSH and thyroxin (free T₄) if hyperthyroidism suspected
- Urinalysis to evaluate for protein if preeclampsia suspected

Imaging

- US:
 - May be performed at bedside
- CXR:
 - Assess for pulmonary edema in acute respiratory distress
 - Check for metastatic disease
 - For baseline study

Diagnostic Procedures/Surgery

Pathology/histology:

- All conception products should be sent for formal evaluation
- Products may be the only way to diagnose a partial molar pregnancy
- Complete mole:
 - Edematous chorionic villi
 - Hyperplasia of trophoblasts
- Partial mole:
 - Fetal tissue and vessels
 - Amnion
 - Edematous chorionic villi

DIFFERENTIAL DIAGNOSIS

- Threatened abortion
- Missed abortion
- Incomplete abortion
- Ectopic pregnancy
- Hyperthyroidism
- Hyperemesis gravidarum
- Hypertension
- Preeclampsia



TREATMENT

PRE HOSPITAL

- Ensure patent airway, provide oxygen
- IV access
- Treat convulsions appropriately with benzodiazepine
- Save passed tissue for histologic evaluation

INITIAL STABILIZATION/THERAPY

- IV access
- Cardiac monitoring
- Type and cross-match for blood, especially if patient requires uterine extraction

ED TREATMENT/PROCEDURES

- Acute respiratory distress:
 - Intubation and mechanical ventilation
 - CXR
- Hyperthyroidism:
 - β -adrenergic blockers:
 - Administer before molar evacuation
 - Stress of anesthesia or surgery may precipitate thyroid storm
- Preeclampsia/eclampsia:
 - Convulsions
 - Administer benzodiazepine (diazepam)
 - Administer magnesium sulfate
 - Hypertension:
 - Administer hydralazine or labetalol
- Coagulopathy:
 - Transfuse with blood products as needed
 - Human anti-D immunoglobulin (RhoGAM):
 - Although fetal blood not present in complete mole, may be delay in distinguishing partial vs. complete
- Suction curettage:
 - Done by obstetrician, possibly in ED
 - Curative in 80% of cases
 - Method of choice in women wishing to preserve fertility
 - Oxytocin infusion to induce myometrial tone, may require other uterotonic formulations
- Chemoprophylaxis:
 - Very controversial
 - Prescribed by obstetrician only for patients with follow-up
 - Usually used in high-risk complete mole or if hormonal monitoring is unavailable
- Hysterectomy:
 - Patients in older age group
 - Patients not interested in keeping fertility
 - High-risk disease
 - Does not prevent possible metastasis

MEDICATION

- Diazepam: 0.2–0.4 mg/kg IV, or 0.3–0.5 mg PR, up to 5–10 mg, for max. 30 mg

- Hydralazine: 5–10 mg IV q20min, up to 60 mg.
- Labetalol: 20 mg IV with doubled dosing q10m for max. 300 mg
- Magnesium sulfate: 4–6 g IV over 15–20 min then maintain 1–2 g/h
- Oxytocin: Postpartum bleeding, 10 U IM
- Propranolol: 1 mg IV increments q2m
- RhoGAM: 300 µg within 72 hr



FOLLOW-UP

DISPOSITION

Admission Criteria

- Enlargement of uterus beyond 16 wk of gestation size:
 - The larger the uterus, the greater the risk for uterine perforation during suction curettage, hemorrhage, and pulmonary complications due to embolism
- Clinical evidence of preeclampsia hyperthyroidism, respiratory distress
- Hysterectomy
- Partial molar pregnancy
- Hemodynamic instability

Discharge Criteria

- Uncomplicated dilation and curettage of low-risk and small-size mole in reliable patient
- Stress importance of follow-up
- Pelvic rest for 4–6 wk after uterine evacuation
- Recommend no pregnancies for 12 mo
- Future pregnancies should have early sonographic evaluation due to increased risk in future pregnancies

FOLLOW-UP RECOMMENDATIONS

- Close follow-up and monitoring by OB-GYN
- Serial hCG levels:
 - Obtained weekly for at least 4 wk, then monthly intervals
 - Levels should consistently drop and never increase
 - If increase is noted, evaluation for metastatic disease should ensue
- Use contraception
- US:
 - Early in all future pregnancies
 - Increased risk for future molar pregnancies (1–1.5% with 2nd, 20% after 2 moles)

PEARLS AND PITFALLS

- Missed diagnosis in conjunction with:
 - Normal pregnancy
 - Preeclampsia, especially < 24 wk gestation
 - Hyperemesis with normal pregnancy
- The importance of follow-up must be stressed:
 - If hCG is not followed, may lead to undiagnosed metastatic disease
 - 20% can develop malignancy

ADDITIONAL READING

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- Sebire NJ, Seckl MJ. Gestational trophoblastic disease: Current management of hydatidiform mole. *BMJ*. 2008;337:a2076.
- Soper JT, Mutch DG, Schink JC, et al. Diagnosis and treatment of gestational trophoblastic disease: ACOG Practice Bulletin No. 53. *Gynecol Oncol*. 2004;93:575–585.



See Also (Topic, Algorithm, Electronic Media Element)

- Preeclampsia/Eclampsia
- Pregnancy



CODES

ICD9

630 Hydatidiform mole

ICD10

- O01.0 Classical hydatidiform mole
- O01.1 Incomplete and partial hydatidiform mole
- O01.9 Hydatidiform mole, unspecified

HYDROCARBON POISONING

James W. Rhee

BASICS

DESCRIPTION

- Main complication from hydrocarbon exposure is aspiration:
 - Hydrocarbon aspiration primarily affects central nervous and respiratory systems.
- Physical properties determine type and extent of toxicity:
 - Viscosity (resistance to flow):
 - Higher aspiration risk from products with lower viscosity
 - Volatility (ability of a substance to vaporize):
 - Hypoxia from aromatic hydrocarbons displacing alveolar air
 - Surface tension (ability to adhere to itself at liquid's surface):
 - Low surface tension allows easy spread from oropharynx to trachea, promoting aspiration (e.g., mineral oil, seal oil).
- Volatile-substance abuse:
 - Common solvents abused:
 - Typewriter correction fluid
 - Adhesive
 - Gasoline
 - Cigarette-lighter fluid
 - *Sniffing*: Product inhaled directly from container
 - *Huffing*: Product inhaled through a soaked rag held to face
 - *Bagging*: Product poured into bag and multiple inhalations taken from bag
- Major classes of hydrocarbons:
 - Aliphatics:
 - Include kerosene, mineral oil, seal oil, gasoline, solvents, and paint thinners
 - Pulmonary toxicity via aspiration
 - Asphyxiation from gaseous methane and butane by displacement of alveolar oxygen
 - Halogenated hydrocarbons:
 - Include carbon tetrachloride and trichloroethane
 - Found in industrial settings as solvents
 - Well absorbed by lungs and gut
 - High toxicity
 - Liver and renal failure associated with ingestion
 - Cyclics or aromatic compounds include toluene and xylene:
 - Highly volatile and well absorbed from gut

- Death from benzene reported with 15 mL ingestion
- _ Terpenes or wood distillates include turpentine and pine oil:
 - Significant GI tract absorption
 - Significant CNS depression

ETIOLOGY

- Accidental exposures typical in young children
- Inhalation abuse of volatile hydrocarbons
- Suicide attempts in adolescents and adults

DIAGNOSIS

SIGNS AND SYMPTOMS

- Often asymptomatic at presentation
- Odor of hydrocarbons on breath
- Early: Euphoria:
 - _ Disinhibition
- Late: Dysphoria:
 - _ Ataxia
 - _ Confusion
 - _ Hallucination
- Sudden sniffing death:
 - _ Cardiac arrest in volatile-substance abusers secondary to hypersensitization of myocardium leading to malignant dysrhythmias on adrenergic stimulation
- Pulmonary:
 - _ Mild to severe respiratory distress
 - _ Cyanosis
 - _ Aspiration (primary complication)
- CNS:
 - _ Intoxication
 - _ Euphoria
 - _ Slurred speech
 - _ Lethargy
 - _ Coma
- GI tract:
 - _ Local mucosal irritation
 - _ Gastritis
 - _ Diarrhea
- Cardiac:
 - _ Tachycardia
 - _ Dysrhythmias (volatile-substance abuse)
- Dermal:

- Local erythema
- Maculopapular or vesicular eruptions
- Defatting dermatitis from chronic skin exposure
- Huffer face rash in chronic abusers

History

- Route, type, quantity, and time of exposure:
 - Determine intentionality and coingestions
- Symptoms:
 - Vomiting, respiratory distress, mental status change or pain
- Bystander actions or pre-hospital interventions

Physical-Exam

- Evaluate for airway compromise in patients with decreased level of consciousness and vomiting
- Respiratory symptoms generally occur within 30 min but are frequently delayed several hours
- Monitor for hypoxia, hypotension, and cardiac dysrhythmias
- Cyanosis and hypoxia suggest respiratory failure but may result from methemoglobinemia
- Temperature may be elevated at presentation following aspiration and indicates pneumonitis:
 - Fever after 48 hr suggests bacterial superinfection

ESSENTIAL WORKUP

Obtain information on the following:

- Product: Exact name on label, manufacturer, and ingredients
- Nature of ingestion or exposure: Accidental or intentional
- Estimated amount ingested
- In industrial settings, Material Safety Data Sheets (MSDSs)

DIAGNOSIS TESTS & NTERPRETATION

ECG for intoxicated volatile-substance abusers

Lab

- Pulse oximetry:
 - If abnormal, follow with arterial blood gases.
- Electrolytes; BUN, creatinine, and glucose levels; and liver function tests:
 - For halogenated and aromatic hydrocarbon exposure
 - Metabolic acidosis
 - Hypokalemia
- Carboxyhemoglobin levels for methylene chloride exposure:
 - Methylene chloride metabolized to carbon monoxide in vivo

Imaging

CXR:

- Abnormalities visible 20 min–24 hr after exposure (usually by 6 hr)
- Increased bronchovascular marking and bibasilar and perihilar infiltrates (typical)
- Lobar consolidation (uncommon)
- Pneumothorax, pneumomediastinum, and pleural effusion (rare)
- Pneumatoceles resolve over weeks
- Repeat chest radiograph if worsening respiratory symptoms

DIFFERENTIAL DIAGNOSIS

- Caustic, pesticide, or toxic alcohol ingestions
- Accidental vs. intentional:
 - Psychiatric evaluation for all intentional ingestions
- Child neglect:
 - Poor supervision or unsafe home environment



TREATMENT

PRE HOSPITAL

- Decontaminate clothes, skin, and hair of any hydrocarbon exposure
- Do not induce emesis.
- Ipecac contraindicated owing to increased risk of aspiration
- Keep volatile-substance abusers calm and avoid interventions that cause anxiety or distress.
- Management of *accidental* hydrocarbon exposures at home controversial:
 - < 1% require physician intervention.
 - For asymptomatic or quickly asymptomatic after ingestion with reliable observer available
 - Applies only when exact product and its components are known and there is no indication for gastric decontamination or possibility for delayed organ toxicity

INITIAL STABILIZATION/THERAPY

- ABCs
- IV access and fluid resuscitation if hypotensive or ongoing fluid losses
- Oxygen
- Cardiac monitor
- Naloxone, thiamine, D₅₀W (or Accu-Chek) if altered mental status

ED TREATMENT/PROCEDURES

- Supportive care
- Treat respiratory symptoms:

- Oxygen
- Nebulized
- β_2 -agonist for bronchospasm (albuterol)
- Endotracheal intubation and mechanical ventilation for respiratory failure
- Steroids not indicated for bronchospasm
- Avoid using epinephrine in volatile-substance abusers as it may precipitate dysrhythmias

ALERT

- Gastric evacuation not indicated for vast majority of hydrocarbon ingestions:
 - Increases risk of aspiration which can cause significant chemical pneumonitis
 - Aspiration risk higher than risk of systemic absorption for aliphatic hydrocarbon mixtures, which account for most ingestions
 - Contraindicated if spontaneous emesis has occurred
 - Small-bore nasogastric tube aspiration of stomach contents may be indicated for some hydrocarbon (CHAMP) ingestions that have systemic toxicity:
 - CHAMP: Camphor, *halogenated hydrocarbons*, *aromatic hydrocarbons*, *metals* (e.g., lead, mercury), *pesticides*
 - Only for very recent ingestions (60 min)
 - Benefit of doing this procedure needs to be weighed heavily against risk of aspiration and subsequent pneumonitis.
 - Cuffed-tube endotracheal intubation for airway protection during lavage if no gag reflex or altered mental status
- Activated charcoal does not bind to hydrocarbons well, and is not indicated except for significant life-threatening coingestants
- Cathartics not indicated:
 - Diarrhea common with hydrocarbon

MEDICATION

- Albuterol 2.5–5 mg NEB (peds: 0.15–0.3 mg/kg) for bronchospasm
- Dextrose: D₅₀W 1 ampule of 50 mL or 25 g (peds: D₂₅W 2–4 mL/kg) IV
- Naloxone (Narcan): 2 mg (peds: 0.1 mg/kg) IV or IM initial dose
- Thiamine (vitamin B₁): 100 mg (peds: 50 mg) IV or IM



FOLLOW-UP

DISPOSITION

Admission Criteria

- Symptomatic patients
- Patients with potential delayed organ toxicity (carbon tetrachloride or other toxic additives)

Discharge Criteria

- Observe for 6 hr then discharge:
 - Asymptomatic patients with normal chest radiograph and pulse oximetry findings
 - Asymptomatic patients with abnormal CXR and normal oxygenation and respiratory rate may be discharged if reliable follow-up is ensured.
 - Symptomatic patients on presentation who quickly become asymptomatic
- Observe volatile-substance abusers until mental status clears.

Issues for Referral

Psychiatry consultation as needed

FOLLOW-UP RECOMMENDATIONS

- Follow up in 24 hr for patients who remain asymptomatic after a minimum of 6 hr observation
- Asymptomatic patients with an abnormal CXR should have a repeat study in 24 hr

PEARLS AND PITFALLS

- Main complication from hydrocarbon exposure is aspiration:
 - Aspiration risk is inversely related to viscosity and surface tension and directly related to volatility
- Provide external decontamination
- Gastric decontamination is rarely indicated
- Avoid induced emesis and cathartics
- Observe patients a minimum of 6 hr post ingestion for evidence of toxicity
- Admit symptomatic patients to hospital
- Admit when there is potential for delayed organ toxicity
 - CHAMP

ADDITIONAL READING

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- Dice WH, Ward G, Kelly J, et al. Pulmonary toxicity following gastrointestinal ingestion of kerosene. *Ann Emerg Med*. 1982;11:138–142.
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CODES

ICD9

- 982.3 Toxic effect of other chlorinated hydrocarbon solvents
- 987.1 Toxic effect of other hydrocarbon gas
- 989.2 Toxic effect of chlorinated hydrocarbons

ICD10

- T53.91XA Toxic effect of unspecified halogen derivatives of aliphatic and aromatic hydrocarbons, accidental (unintentional), initial encounter
- T59.891A Toxic effect of other specified gases, fumes and vapors, accidental (unintentional), initial encounter

HYDROCELE

Jessica L. Osterman



BASICS

DESCRIPTION

- Most common cause of painless scrotal swelling.
- Classified as congenital or acquired (secondary):
 - Congenital result from a patent process vaginalis and communication between tunica vaginalis and peritoneal cavity:
 - Normally occurs spontaneously and most are closed by 2 yr of age
 - Acquired occur secondary to interscrotal infection, neoplasm, inguinal or scrotal surgery, or regional or systemic disease.
- Communicating hydrocele:
 - Patent processus vaginalis
 - Scrotum fills and empties with peritoneal fluid depending on body position and intraperitoneal pressures.
- *Noncommunicating hydrocele* is due to production of serous fluid by a disease process or impaired absorption within the scrotum itself

ETIOLOGY

- Imbalance between production and resorption of fluid within the space between tunica vaginalis and tunica albuginea.
- Disease processes causing adult noncommunicating hydrocele include:
 - Epididymitis
 - Hypoalbuminemia
 - TB
 - Trauma
 - Mumps
 - Spermatic vein ligation
 - In developing world, hydrocele is primarily caused by infections such as *Wuchereria bancrofti* or *Loa Loa* (filariasis is the cause of most hydroceles worldwide)
 - Rarely malignancy (1st-degree testicular neoplasm or lymphoma)
- Rare etiology is the abdominoscrotal hydrocele that may cause hydroureter or unilateral limb edema owing to compression:
 - US reveals single sac extending from scrotum into abdominal cavity via the deep inguinal ring.

Pediatric Considerations

- Congenital in 6% of newborn boys

- Usually diagnosed in newborn nursery
- Caused by patent processus vaginalis, a structure that remains patent in 85% of newborns
- May vary in size owing to position or crying:
 - Patients may present with history of scrotal mass that has resolved.
- Most close by the age of 2 yr

DIAGNOSIS

SIGNS AND SYMPTOMS

Painless scrotal swelling with a sensation of pulling, dragging, or heaviness.

History

History and exam with special attention to identifying torsion of testicle.

Physical-Exam

- Mass may be soft and doughy or firm depending on the amount of fluid present.
- Initial evaluation includes transillumination of affected side (looking for a homogeneous area without internal shadows):
 - This is rapidly being replaced as diagnostic test of choice by bedside US.

ESSENTIAL WORKUP

- Bedside US:
 - Allows visualization of hydrocele as well as of testicle
 - Especially in cases of massive fluid collection, bedside US should be the diagnostic test of choice.
 - May help to identify an underlying mass
- Because of possibility in adults that a hydrocele may be owing to a primary neoplasm, the testicle must be palpated in its entirety.

DIAGNOSIS TESTS & INTERPRETATION

Lab

No specific lab testing is indicated unless underlying cause demands it (UA, AFP, hCG).

Imaging

US is diagnostic and allows visualization of testicular anatomy:

- Appears as large anechoic fluid-filled space surrounding the anterolateral testicle

DIFFERENTIAL DIAGNOSIS

- Epididymitis
- Indirect inguinal hernia

- Orchitis
- Testicular neoplasm
- Testicular torsion
- Varicocele



TREATMENT

INITIAL STABILIZATION/THERAPY

Stabilization should focus on underlying cause (e.g., trauma).

ED TREATMENT/PROCEDURES

Appropriate exam of testicle to exclude primary neoplasm and referral.

MEDICATION

Treat underlying cause.



FOLLOW-UP

DISPOSITION

Admission Criteria

Patients with secondary hydrocele may need admission for further evaluation of underlying pathology (e.g., neoplasm, trauma).

Discharge Criteria

- Otherwise healthy patients without comorbid illness may be referred for further evaluation to urologist.
- Hydrocele is usually repaired if cosmesis is a factor or in cases where it causes discomfort.
- Repair can be:
 - _ Surgical:
 - Aspiration or sclerotherapy are alternatives to open hydrocelectomy.
 - _ Medical:
 - Aspiration of hydrocele contents and sclerotherapy to prevent recurrence.

Pediatric Considerations

- Most hydroceles in infant population will spontaneously resolve by 12 mo of age:
 - _ Referral and observation are appropriate once diagnosis is made.
- After the age of 12–18 mo, refer for surgical repair as communicating hydroceles usually have hernia that needs repair.

FOLLOW-UP RECOMMENDATIONS

Patients should be referred to Urology.

PEARLS AND PITFALLS

The mass may fail to transilluminate due to thickening of the tunica vaginalis.

- Bedside US should visualize both the fluid-filled mass and the testicle.

ADDITIONAL READING

- Cokkinos DD, Antypa E, Tserotas P, et al. Emergency ultrasound of the scrotum: A review of the commonest pathologic conditions. *Curr Probl Diagn Radiol*. 2011;40(1):1–14.
- Hoerauf A. Filariasis: New drugs and new opportunities for lymphatic filariasis and onchocerciasis. *Curr Opin Infect Dis*. 2008;21:673–681.
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- Wampler SM, Llanes M. Common scrotal and testicular problems. *Prim Care*. 2010;37(3):613–626.



See Also (Topic, Algorithm, Electronic Media Element)

- Epididymitis/Orchitis
- Hernia
- Testicular Torsion



CODES

ICD9

- 603.1 Infected hydrocele
- 603.9 Hydrocele, unspecified
- 778.6 Congenital hydrocele

ICD10

- N43.1 Infected hydrocele
- N43.3 Hydrocele, unspecified
- P83.5 Congenital hydrocele

HYDROCEPHALUS

Richard S. Krause



BASICS

DESCRIPTION

- Increased volume of CSF in cranial cavity
- Cerebral atrophy also leads to increased CSF in the cranial vault but CSF pressure is not increased
- *Obstructive hydrocephalus* is the most common form:
 - Obstruction is within ventricular system or in subarachnoid space
- Acute *obstructive* hydrocephalus may cause rapid rise in intracranial pressure (ICP), rapidly leading to death or permanent cerebral damage
- *Nonobstructive hydrocephalus* causes subacute symptoms and is a potentially treatable form of dementia
- Also described as “communicating” and “noncommunicating”:
 - Communicating hydrocephalus: Flow of CSF is blocked after it exits the ventricles (ventricles still “communicate”)
 - Noncommunicating hydrocephalus: Flow of CSF blocked along 1 or more of the passages connecting the ventricles (ventricles do not “communicate”)

ETIOLOGY

- Obstructive hydrocephalus:
 - Obstruction of:
 - Aqueduct of Sylvius (most common, both lateral ventricles and 3rd ventricle dilated, 4th ventricle is spared)
 - Aqueductal stenosis can be congenital or acquired (tumor, subarachnoid hemorrhage, post meningitis, idiopathic)
 - Foramen of Monro (lateral ventricles dilated, usually both but may be unilateral)
 - Foramina of Luschka and Magendie (4th ventricle blocked followed by 3rd and lateral ventricles)
 - Subarachnoid space around brainstem (postinfectious or postsubarachnoid hemorrhage [post-SAH] entire system dilated)
 - Acute presentations usually secondary to CSF shunt blockage, SAH, or severe head trauma
- Nonobstructive hydrocephalus:
 - Normal pressure hydrocephalus:
 - Increased intracranial volume of CSF without intracranial hypertension
 - Increased ventricular size on CT (without volume loss as in atrophy)

- Sometimes called “chronic hydrocephalus”
- Usually occurs due to inadequate CNS absorption
- Pediatric hydrocephalus:
 - Congenital hydrocephalus owing to neonatal hemorrhage, congenital malformations, or acquired post meningitis secondary to subarachnoid scarring around brainstem

DIAGNOSIS

SIGNS AND SYMPTOMS

- Obstructive hydrocephalus:
 - Headache
 - Nausea and vomiting
 - Decreased level of consciousness
 - Urinary incontinence
 - Ocular palsies
 - Papilledema, decreased vision
 - Pupillary dilation
 - Cushing response:
 - Raised systolic pressure and bradycardia secondary to increased ICP
 - Pediatric patients:
 - Full fontanelle, irritability, and lethargy
 - BP often elevated
 - May present like nonobstructive hydrocephalus if obstruction develops slowly
- Nonobstructive hydrocephalus:
 - Progressive dementia, somnolence
 - Gait disturbance
 - Urinary incontinence
 - Impaired upward gaze
 - Generalized weakness and lethargy
 - Dementia is often insidious with subacute onset of progressive intellectual deterioration
 - No headache or papilledema

Pediatric Considerations

- Pediatric patients increase CSF volume slowly:
 - Craniomegaly
 - Retardation
 - Prominent scalp veins
 - Impaired upward gaze (setting sun sign)

History

- Onset of symptoms
- History of CSF shunt
- Nausea/vomiting
- Headache
- Weakness
- Confusion
- Visual changes
- Incontinence of urine

Physical-Exam

- Thorough neurologic exam:
 - _ Motor
 - _ Sensation
 - _ Deep tendon reflexes
 - _ Gait
 - _ Cranial nerve exam
 - _ Papilledema may be seen
- Confusion
- Decreased level of consciousness
- Palpate CSF shunt if present
 - _ Malfunction indicated by failure to compress (distal shunt malfunction) or failure to refill (proximal shunt obstruction)
- Full anterior fontanelle in children:
 - _ Other findings as noted in Signs and Symptoms

ESSENTIAL WORKUP

CT scan of the head w/o contrast will allow assessment of ventricular size and symmetry:

- Aid in diagnosis of cerebral edema, mass lesions, and hemorrhage

DIAGNOSIS TESTS & NTERPRETATION

Lab

Lumbar puncture (LP) is typically performed after head CT (for nonobstructive causes):

- Opening pressure on LP will reflect increased ICP in nonobstructive hydrocephalus.
- CSF should be sent for routine tests if infection is suspected.
 - _ Gram stain, culture, protein, glucose, and cell count

Imaging

MRI of brain reveals ventricular size and symmetry and may allow for better visualization of masses than CT

Diagnostic Procedures/Surgery

LP may be indicated

DIFFERENTIAL DIAGNOSIS

- Acute cerebral infarction or hemorrhage
- Intracranial infection
- Mass effect from fast-growing tumor or hematoma
- Dementia or delirium of other cause
- Toxic or metabolic encephalopathies

Pediatric Considerations

- Suspect hydrocephalus in an infant whose head circumference is increasing excessively, has progressive lethargy, persistent vomiting, impaired upward gaze, etc.
- Congenital anomalies:
 - Dandy–Walker malformation
 - Arnold–Chiari malformation
 - Meningomyelocele
 - Choroid plexus papilloma
 - Hypoplasia/dysfunction of arachnoid villi
- Infections:
 - Rubella
 - Cytomegalovirus (CMV)
 - Toxoplasmosis
 - Syphilis
 - Bacterial meningitis
 - Reye syndrome
- Tumors, especially posterior fossa tumors:
 - Medulloblastoma
 - Astrocytoma
 - Ependymoma
- Hemorrhage:
 - Intraventricular
 - Subarachnoid



TREATMENT

PRE HOSPITAL

Cautions:

- Elevated ICP/hydrocephalus cannot be definitively diagnosed in the field
- When it is suspected, supplemental O₂ and airway management (if needed) are indicated

- Patients should be transported with head elevated at $\sim 30^\circ$
 - HOB should not be elevated if patient is hypotensive for concern about decreased cerebral perfusion
 - Initial treatment for hypotension is usually volume expansion with normal saline

INITIAL STABILIZATION/THERAPY

- Signs of impending herniation:
 - Rapid-sequence intubation (RSI)
 - Thiopental or etomidate for induction
 - Paralytic choice is controversial
 - Depolarizing agents (succinylcholine) may transiently increase ICP, this effect may not be clinically significant
 - Nondepolarizing agents (rocuronium, vecuronium) may be preferable
 - Controlled ventilation to maintain PaCO₂ at ~ 35 mm Hg
 - Maintain systolic BP > 100 mm Hg (adult) with fluids or pressors.
 - Mannitol
- If a CSF shunt is present and there are signs of impending herniation:
 - Forced pumping of shunt chamber:
 - Flush device with 1 mL saline to remove distal obstruction
 - Slow drainage of CSF from reservoir to achieve pressure < 20 cm H₂O

ED TREATMENT/PROCEDURES

- Hydrocephalus does not generally require ED treatment unless:
 - Signs of impending herniation
 - Acute shunt malfunction
- Definitive treatment involves either placement (or revision) of shunting device or treatment of underlying cause (e.g., tumor)
- Neurologic symptoms (gait disturbance) or severe headache associated with normal pressure hydrocephalus may respond to removal of CSF by LP (20–30 mL)
- If intraventricular hemorrhage (usually from trauma or SAH) causes acute obstructing hydrocephalus a ventriculostomy may be placed in the lateral ventricle
- Patients who are agitated or intubated should be sedated
- Maintain elevation of the head unless hypotensive
- Mannitol may be used
- Consider seizure prophylaxis with fosphenytoin

MEDICATION

- Atropine: 0.02 mg/kg IV (max. 0.1 mg)
- Etomidate: 0.2–0.3 mg/kg
- Lidocaine: 1 mg/kg IV
- Mannitol: 0.5–1.5 g/kg
- Rocuronium: 0.6 mg/kg IV

- Succinylcholine: 1–1.5 mg/kg IV
- Vecuronium: 0.1 mg/kg
- Fosphenytoin: 15–20 mg/kg loading dose



FOLLOW-UP

DISPOSITION

Admission Criteria

Evidence of increased ICP or shunt malfunction requires admission

Discharge Criteria

Patients with presumed normal pressure hydrocephalus may be discharged for follow-up

Issues for Referral

Involvement of a neurosurgeon may be needed in acute obstructive hydrocephalus or for acute shunt malfunction

- Consider transfer if a neurosurgeon is not available at presenting hospital
- Airway management prior to transfer should be considered in acute cases

FOLLOW-UP RECOMMENDATIONS

If patient is stable for discharge, follow-up with neurologist and/or neurosurgeon is essential

PEARLS AND PITFALLS

- LP should not be performed in obstructive hydrocephalus (risk of herniation)
- Suspect hydrocephalus in children whose head circumference is growing rapidly
- Consider hydrocephalus in patients with CSF shunts and any neurologic complaint

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Ventricular Peritoneal Shunts



CODES

ICD9

- 331.3 Communicating hydrocephalus
- 331.4 Obstructive hydrocephalus
- 331.5 Idiopathic normal pressure hydrocephalus (INPH)

ICD10

- G91.0 Communicating hydrocephalus
- G91.2 (Idiopathic) normal pressure hydrocephalus
- G91.9 Hydrocephalus, unspecified

HYPERBARIC OXYGEN THERAPY

Trevonne M. Thompson



BASICS

DESCRIPTION

- Administration of 100% oxygen at > 1 atm (typically 2–3 atm)
- Mechanisms of action:
 - Increases oxygen availability at the cellular level:
 - Breathing 100% oxygen at 3 atm supplies enough dissolved oxygen to support life without hemoglobin.
 - Compresses formed gas bubbles (in cases of air embolism or decompression sickness)
- 2 types of hyperbaric oxygen chambers:
 - Monoplace:
 - Accommodates 1 supine patient
 - Technician outside the chamber for monitoring
 - Compressed with 100% oxygen
 - Multiplace:
 - Holds multiple patients
 - Holds attendants who “dive” with the patients
 - Airlocks available for medication/equipment transfer outside of the chamber
 - Compressed with air—patients breath oxygen by face mask, endotracheal tube, or face hood.



DIAGNOSIS

SIGNS AND SYMPTOMS

Indications for hyperbaric oxygen therapy:

- Arterial gas embolism
- Decompression sickness
- Carbon monoxide toxicity
- Soft tissue infections:
 - Clostridial myonecrosis
 - Necrotizing fasciitis
 - Refractory osteomyelitis
 - Chronic nonhealing wounds
- Wound care:
 - Radiation-induced tissue injury
 - Crush injuries

- Thermal burns
- Compromised skin grafts and flaps

ALERT

The ED physician should focus on arterial embolism, decompression sickness, and carbon monoxide toxicity as uses for hyperbaric oxygen.

ESSENTIAL WORKUP

- Determine need for hyperbaric oxygen therapy as described above.
- Perform a comprehensive physical exam to screen for contraindications to therapy and to establish a pretreatment baseline exam.
- Contraindications to therapy:
 - Untreated pneumothorax is the absolute contraindication:
 - May convert to a tension pneumothorax
 - Cardiovascular instability:
 - Unstable patient cannot be treated in a monoplace chamber.
 - Such a patient may be treated in multiplace chamber if benefit outweighs risk.

DIAGNOSIS TESTS & INTERPRETATION

Lab

Arterial blood gas:

- To evaluate for hypoxia in appropriate cases

Imaging

Chest radiography:

- To evaluate for occult pneumothorax



TREATMENT

INITIAL STABILIZATION/THERAPY

- Manage ABCs
- Establish IV access.
- 100% oxygen
- Cardiac monitor (when appropriate)

ED TREATMENT/PROCEDURES

- Determine need for hyperbaric oxygen therapy.
- Fill any devices with balloons (Foley catheters, endotracheal tubes) with fluid to avoid rupture during therapy.
- Pretreat patients with any sinus complaints with decongestants.
- Place myringotomy tubes in obtunded or mechanically ventilated patients or in

patients with middle ear pathology (e.g., otitis media).

ALERT

Complications of hyperbaric oxygen therapy:

- Sinus/ear pain
- Barotrauma:
 - _ Ruptured tympanic membranes
 - _ Tension pneumothorax
- Seizures:
 - _ May be a result of oxygen toxicity
- Decompression sickness:
 - _ When decompression is too rapid
 - Inability to access an unstable patient when using a monoplace chamber



FOLLOW-UP

DISPOSITION

Admission Criteria

- Arterial gas embolism
- Decompression sickness
- Significant carbon monoxide toxicity

Discharge Criteria

Stable patient with resolved symptoms

Issues for Referral

- May need to transfer to a facility that has a hyperbaric oxygen chamber
- Evaluate risks and benefits when considering the transfer of a potentially unstable patient.
- Divers Alert Network:
 - _ 24 hr emergency hotline for consultation of dive-related injuries
 - _ Referral source for hyperbaric oxygen chambers
 - _ Telephone number:
 - 919-684-9111
 - _ Website:
 - www.diversalertnetwork.org

FOLLOW-UP RECOMMENDATIONS

Hyperbaric follow-up for repeat recompression therapy.

PEARLS AND PITFALLS

- Fill any devices with balloons (Foley catheters, endotracheal tubes) with fluid to avoid rupture during therapy.
- Check for occult pneumothorax.

ADDITIONAL READING

- Buckley NA, Juurlink DN, Isbister G, et al. Hyperbaric oxygen for carbon monoxide poisoning. *Cochrane Database Syst Rev*. 2011;(4):CD002041.
- Guzman, JA. Carbon monoxide poisoning. *Crit Care Clin*. 2012;28(4):537–548.
- Weaver LK. Carbon monoxide poisoning. *N Engl J Med*. 2009;360:1217–1225.



See Also (Topic, Algorithm, Electronic Media Element)

- Carbon Monoxide Toxicity
- Decompression Sickness



CODES

ICD9

- 958.0 Air embolism
- 986 Toxic effect of carbon monoxide
- 993.3 Caisson disease

ICD10

- T58.94XA Toxic effect of carb monx from unsp source, undet, init
- T70.3XXA Caisson disease [decompression sickness], initial encounter
- T79.0XXA Air embolism (traumatic), initial encounter

HYPERCALCEMIA

Matthew A. Wheatley • Ryan A. Stroder



BASICS

DESCRIPTION

- Severity depends on serum calcium level and rate of increase
- 0.1–1% of patients on routine screening
- Most cases mild (< 12 mg/dL) and asymptomatic
- Hypercalcemic crisis, usually > 14 mg/dL, causes serious signs and symptoms
- Calcium in bloodstream in 3 forms:
 - Ionized: 45%
 - Bound to protein (primarily albumin): 40%
 - Bound to other anions: 15%
- Ionized calcium—only physiologically active form

ETIOLOGY

- Primary hyperparathyroidism
- Malignancy
- Miscellaneous



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Neurologic:
 - Headache
 - Fatigue, lethargy
 - Weakness
 - Difficulty concentrating
 - Confusion
 - Depression, paranoia
- Renal:
 - Polyuria, polydipsia
 - Complaints related to oliguric renal failure
 - Chronic, complaints related to:
 - Renal calculi
 - Nephrocalcinosis
 - Interstitial nephritis
- GI:

- Anorexia
- Nausea, vomiting
- Abdominal pain
- Constipation
- Chronic, complaints related to:
 - Peptic ulcer disease
 - Pancreatitis
- Dermatologic:
 - Pruritus
 - Mnemonic: “Stones, Bones, Groans, Thrones and Psychiatric Overtones,” “bones” refers to bone pain and “thrones” refers to polyuria.

Pediatric Considerations

- Failure to thrive
- Slow development
- Mental retardation may ensue

Physical-Exam

- Neurologic:
 - Irritability
 - Lethargy
 - Stupor
 - Coma
 - Hyporeflexia
- Cardiovascular:
 - Hypotension, if severely volume depleted, or HTN
 - Sinus bradycardia
 - Cardiac arrest with severe hypercalcemia (rare)
- Renal:
 - Signs of dehydration
- Dermatologic:
 - Band keratopathy
 - Ectopic calcification

Pediatric Considerations

- Characteristic facies: Pug nose, fat nasal bridge, “cupid’s bow” upper lip
- Hypotonia

ESSENTIAL WORKUP

- Ionized and total serum calcium levels, albumin levels:
 - Normal total calcium level is <10.5 mg/dL
 - Must correct for calcium that is protein bound, primarily to albumin

- Corrected total calcium (mg/dL) = measured total calcium (mg/dL) + 0.8 × [4.0 – albumin concentration (g/dL)]
- Electrolytes, BUN/creatinine, glucose
 - Possible oliguric renal failure
- ECG:
 - Shortening of QT interval
 - Prolongation of PR interval
 - QRS widening
 - Accentuated side effects of digoxin
 - Sinus bradycardia, bundle branch block, AV block, cardiac arrest with severe hypercalcemia (rare)
 - Can cause Osborn J-wave at the end of QRS complex that is usually associated with hypothermia

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Phosphate
- Protein
- Urinalysis
- Parathyroid hormone (PTH) level:
 - If elevated or high normal, likely primary hyperparathyroidism.
 - If <20 pg/mL, consider testing PTH-related peptide and vitamin D metabolites.
- Vitamin D metabolites, if suspected
 - 25-hydroxy vitamin D (calcidiol):
 - If elevated, consider exogenous source (i.e., meds, vitamins, supplements).
 - 1,25-dihydroxy vitamin D (calcitriol):
 - If elevated, consider lymphoma or sarcoid
- Digoxin level, if taking
- Thyroid function tests

Imaging

- CT head for altered mental status
- Chest x-ray and workup for occult malignancy, if no other cause for hypercalcemia

Diagnostic Procedures/Surgery

Parathyroidectomy:

- For primary hyperparathyroidism resulting in symptomatic or severe hypercalcemia
- Some patients require urgent parathyroidectomy.

DIFFERENTIAL DIAGNOSIS

- Primary hyperparathyroidism:
 - Most common cause among outpatients
 - Parathyroid adenoma 80%; hyperplasia 15%; carcinoma 5%
 - Usually mild, <11.2 mg/dL
 - Patients can be asymptomatic or have chronically elevated calcium
 - Increased bone resorption, relative decrease in calcium excretion, increased intestinal calcium absorption
- Malignancy:
 - Most common cause in hospitalized patients
 - Usually a rapid rise in serum calcium
 - Patients are more often symptomatic
 - Higher serum calcium concentrations
 - Most common paraneoplastic complication of cancer
 - Common tumors causing hypercalcemia: Breast, lung, colon, stomach, cervix, uterus, ovary, kidney, bladder, head and neck, multiple myeloma, and lymphoma
 - Most commonly from production of PTH-related protein with similar actions
 - May result from production of other bone-resorbing substances by tumor
 - May result from local effects of osteolytic skeletal metastasis
- Miscellaneous:
 - Hypercalcemia associated with granulomatous diseases
 - Excessive calcium supplements
 - Thiazide diuretics causing increased renal reabsorption
 - Familial hypocalciuric hypercalcemia
 - Acute vitamin A intoxication
 - Exogenous vitamin D intake
 - Milk-alkali syndrome from excessive ingestion of calcium and nonabsorbable antacids, such as milk or calcium carbonate
 - Long-term lithium therapy
 - Renal transplantation
 - Hyperthyroidism
 - Acute tubular necrosis

Pediatric Considerations

Differential diagnosis: Differences from adults:

- Primary hyperparathyroidism:
 - Less common than in adults
- Infantile hypercalcemia:
 - Uncertain cause
 - Possibly hypersensitivity and in utero excessive exposure to vitamin D
- Immobilization hypercalcemia:
 - Typically adolescent who is growing rapidly

- Prolonged immobilization, especially in traction, leads to hypercalciuria and then hypercalcemia
- Presumably from increased bone resorption with decreased or arrested bone mineralization



TREATMENT

PRE HOSPITAL

Routine stabilization techniques

INITIAL STABILIZATION/THERAPY

- ABCs, IV access, oxygen, cardiac monitor
- 0.9% NS 1 L bolus (20 mL/kg) for hypotension or severe dehydration
- Naloxone, thiamine, D₅₀W (or stat serum glucose measurement) for altered mental status

ED TREATMENT/PROCEDURES

- General:
 - Immediate therapy for severe hypercalcemia (corrected total > 14 mg/dL) regardless of symptoms, or for symptomatic hypercalcemia
 - Asymptomatic, mild hypercalcemia does not require emergency treatment
- Restoration of IV volume:
 - Isotonic saline:
 - 200–300 mL/hr adjusted to maintain urine output 100–150 mL/hr
 - Often need 2–5 L/day
 - Bedside vigilance necessary to prevent fluid overload
 - Correct other electrolyte abnormalities
 - Cardiovascular status of patient may necessitate central venous pressure monitoring to adjust fluid administration rates
- Renal elimination:
 - After volume expansion and if needed to avoid overload, administer loop diuretics (furosemide)
 - Avoid thiazide diuretics
 - May need peritoneal or hemodialysis against a low calcium dialysate in renal failure
- Inhibition of osteoclastic activity:
 - Reduce mobilization of calcium from bone
 - Administer drug therapy when corrected calcium level > 14 mg/dL or signs or symptoms
 - First-line drug therapy:
 - Bisphosphonates: Pamidronate (more potent and possibly less toxic), etidronate

- Calcitonin: Rapid onset but modest decrease in levels
- Other potential drug therapy:
 - Plicamycin: Efficacious but numerous side effects
 - Hydrocortisone: Especially useful with malignancies, granulomatous disorders, or vitamin D intoxication
- Encourage ambulation in appropriate patients
- Treat underlying disorder:
 - Parathyroidectomy for primary hyperparathyroidism resulting in symptomatic or severe hypercalcemia
 - Discontinue medication if cause of hypercalcemia

MEDICATION

First Line

- Calcitonin: 4 IU/kg IM/SC q12h
- Etidronate: 7.5 mg/kg over 4 hr daily for 3–7 days IV
- Furosemide: 10–40 mg q6–8h (peds: 1–2 mg/kg) IV
- Pamidronate: Single 2–24 hr infusion of 60–90 mg IV (peds: Consult pediatrician)

Second Line

- Gallium nitrate: Continuous infusion of 200 mg/m²/d for 5 days IV
- Hydrocortisone: 200–400 mg/d IV for 3–5 days (peds: Consult pediatrician)
- Plicamycin: 25 µg/kg/d over 4–6 hr IV for 3–8 doses

Pediatric Considerations

- In infants, loop diuretics are rarely necessary and possibly harmful as they may decrease glomerular filtration rate and worsen hypercalcemia
- Bisphosphonates have not been extensively studied in pediatrics but do appear to be safe



FOLLOW-UP

DISPOSITION

Admission Criteria

- Corrected total calcium level > 13 mg/dL
- Signs or symptoms attributed to hypercalcemia, especially EKG changes
- Monitored bed or ICU for corrected level > 14 or serious signs and symptoms

Discharge Criteria

Corrected calcium level < 13 mg/dL and no signs or symptoms of hypercalcemia

Issues for Referral

- Rapid follow-up arranged to determine cause and long-term therapy
- Consultation with endocrinologist should be considered

FOLLOW-UP RECOMMENDATIONS

- Fluid hydration
- Watch for mental status changes

PEARLS AND PITFALLS

- Make decisions based on symptoms or corrected Ca levels
- All patients with serum Ca > 14 mg/dL require treatment regardless of symptoms
- Pay careful attention to EKG changes
- Careful monitoring is required for patients receiving IV volume repletion:
 - They often require a large volume of fluid but care must be taken to avoid volume overload

ADDITIONAL READING

- Ariyan CE, Sosa JA. Assessment and management of patients with abnormal calcium. *Crit Care Med.* 2004;32(suppl 4):S146–S154.
- Inzucchi SE. Management of hypercalcemia. Diagnostic workup, therapeutic options for hyperparathyroidism and other common causes. *Postgrad Med.* 2004;115:27–36.
- Lietman SA, Germain-Lee EL, Levine MA. Hypercalcemia in children and adolescents. *Curr Opin Pediatr.* 2010;22(4):508–515.
- Marx JA, Hockberger RS, Walls RM, eds. *Rosen's Emergency Medicine.* Philadelphia, PA: Elsevier; 2009.



See Also (Topic, Algorithm, Electronic Media Element)

- Hyperparathyroidism
- Hypocalcemia
- Hypoparathyroidism



CODES

ICD9

- 275.42 Hypercalcemia
- 275.49 Other disorders of calcium metabolism
- 588.89 Other specified disorders resulting from impaired renal function

ICD10

- E83.52 Hypercalcemia
- E83.59 Other disorders of calcium metabolism
- N25.89 Oth disorders resulting from impaired renal tubular function

HYPEREMESIS GRAVIDARUM

David Della-Giustina



BASICS

DESCRIPTION

- Hyperemesis gravidarum is the most severe form along the continuum of nausea and vomiting of pregnancy
- Also known as pernicious vomiting of pregnancy
- Characterized by unexplained intractable vomiting and dehydration
- Occurs in 0.3–2% of pregnancies
- Diagnosis of exclusion

ETIOLOGY

- Exact cause unknown
- Possible causes include the following:
 - Elevated gestational hormone levels of human chorionic gonadotropin (hCG) and/or estradiol
 - Thyrotoxicosis
 - Upper GI motility dysfunction
 - Hepatic abnormalities
 - Autonomic nervous system dysfunction
 - Psychological factors
 - *Helicobacter pylori* infection
 - Genetic predisposition



DIAGNOSIS

SIGNS AND SYMPTOMS

- Nausea and vomiting during pregnancy affects between 50% and 90%
- Onset of symptoms by the 4th–10th wk of pregnancy with resolution by the 20th:
 - Symptoms after the 20th wk should raise one's suspicion of another process
- Peak onset is at 8–12 wks
- Hyperemesis gravidarum is a clinical diagnosis defined by the following:
 - Persistent and severe nausea and vomiting
 - Dehydration
 - Weight loss of > 5% of total body weight
 - Lab findings: Increased urine specific gravity, ketonuria, electrolyte disturbances, ketonemia

History

- Onset of vomiting
- Gestational history:
 - Similar symptoms in prior pregnancies
- Last menstrual period
- Oral intake
- Urine output
- Bloody or bilious vomiting
- Abdominal pain
- Vaginal bleeding
- Risk factors include the following:
 - History of motion sickness
 - Younger age
 - Migraine headaches
 - Symptoms earlier in the day
 - Low prepregnancy body mass index
 - More common in nulliparous women
 - 15% recurrence rate if manifested in previous pregnancy

Physical-Exam

- Observe for signs of dehydration
- Abdominal tenderness

ESSENTIAL WORKUP

- History and physical exam with special attention to state of hydration and abdominal exam for other diagnoses associated with vomiting (appendicitis, cholecystitis, etc.)
- Obtain an uncontaminated urinalysis
- If patient has unremitting vomiting for >24 hr, obtain a CBC, electrolytes, renal function, liver enzymes, bilirubin, and lipase

DIAGNOSIS TESTS & NTERPRETATION

Lab

- Urinalysis:
 - Increased specific gravity and ketonuria
 - Presence of glucose mandates checking serum glucose to rule out diabetes
 - Presence of bilirubin mandates a search to rule out hepatobiliary cause for the vomiting
- CBC:
 - May have an elevated hematocrit owing to dehydration
 - WBC is usually normal
- Electrolytes:
 - Elevated BUN indicating volume depletion; elevated creatinine if renal

failure present

- Hyponatremia, hypokalemia, hypochloremia, and metabolic alkalosis from loss of HCl in emesis
- Liver function tests:
 - Mild increases in bilirubin may occur, but should be < 4 mg/dL.
 - AST and ALT may also be mildly elevated, but not > 100 IU/L
- Amylase/lipase:
 - In 1 study, amylase was elevated in 24% of patients with hyperemesis gravidarum; however, the amylase was salivary in origin; use lipase rather than amylase to evaluate for pancreatitis
- TSH
- Serum hCG levels are not indicated if known intrauterine pregnancy

Imaging

- US when 1st trimester US has not been performed to evaluate for:
 - Molar pregnancy
 - Multiple gestations

DIFFERENTIAL DIAGNOSIS

- Pyelonephritis; most commonly missed
- Gastroenteritis; gastroparesis; intestinal obstruction; Mallory–Weiss tear
- Hepatobiliary disease; hepatitis, cholecystitis, fatty liver of pregnancy, achalasia
- Pancreatitis
- Appendicitis
- Diabetic ketoacidosis
- Hyperthyroidism; hyperparathyroidism
- Uremia; persistent nausea and vomiting are seen with severe renal dysfunction.
- Pseudotumor cerebri



TREATMENT

PRE HOSPITAL

- IV, monitor if signs of significant volume depletion
- IV hydration

INITIAL STABILIZATION/THERAPY

IV hydration using a crystalloid solution (LR or NS)

ED TREATMENT/PROCEDURES

- IV hydration using LR or NS
- Dextrose may be added to help break cycle of ketosis
- Treat until patient is no longer symptomatic from hypovolemia

- Antiemetics administered IV are given to break the vomiting cycle
- Most commonly used medications:
 - Metoclopramide:
 - FDA category B
 - Promethazine and prochlorperazine:
 - Both FDA category C
 - Recent FDA warning regarding complications of IV promethazine administration
 - Ondansetron:
 - FDA category B with recent warning about the risk of prolonged QT syndrome and the recommendation for ECG monitoring of the patient with electrolyte abnormalities such as hypokalemia or hypomagnesemia
 - These have been used extensively in pregnancy, and there is little or no evidence associated with increased risk of congenital anomalies
 - Antiemetics are preferable to the risk of prolonged ketosis and hypovolemia
- Oral rehydration in the ED after the initial fluid resuscitation and antiemetics
- Thiamine 100 mg IV/IM/PO in the patient who requires IV rehydration due to case reports of Wernicke encephalopathy
- Antihistamines have been shown to be effective
- Methylprednisolone may be effective for patients with hyperemesis gravidarum:
 - Last resort
 - Avoid if < 10 wks gestation

MEDICATION

First Line

- Metoclopramide (category B): 10–20 mg IV
- Ondansetron (category B): 4–8 mg IV or 4 mg PO or ODT every 8 hr
- Prochlorperazine (category C): 5–10 mg IV not to exceed 40 mg/d
- Promethazine (category C): 12.5–25 mg IM
- Discharge outpatient medications:
 - Meclizine (category B): 25 mg PO q6h PRN
 - Metoclopramide (category B): 10 mg PO q6–8h PRN
 - Prochlorperazine (category C): 5–10 mg PO q6h or 25 mg PR q12h PRN
 - Promethazine (category C): 12.5–25 mg PO or PR q4–6h PRN
 - Pyridoxine (vitamin B₆; category A): 25 mg PO TID (OTC)
 - Ginger (*Zingiber officinale*): 500–1500 mg div. bid/tid
 - Doxylamine (Unisom—OTC) 12.5 mg PO q6–8h usually with pyridoxine (vitamin B₆)
 - Thiamine: 50 mg PO per day for symptoms > 3 wks

Second Line

Methylprednisolone (category C): 16 mg IV or PO q8h × 3 days and then taper. Should be prescribed in consultation with obstetrician.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Inability to tolerate oral intake after treatment
- Inability to control the emesis despite treatment
- Severe electrolyte or metabolic disturbances
- At highest risk < 8 wk gestation

Discharge Criteria

- Most patients can be discharged as long as they are able to tolerate oral intake and have adequate follow-up
- Correction of dehydration and associated symptoms
- Decreased ketonuria
- Reassure patient that their symptoms are common and usually self-limited
- Patients should be counseled that frequent, small meals may be helpful:
 - Meals should contain simple carbohydrates and be low in fats
 - Avoid irritant or spicy foods
- Home IV therapy can be arranged if indicated

FOLLOW-UP RECOMMENDATIONS

- All patients with diagnosis should take at least 3 mg thiamine/day to help prevent Wernicke encephalopathy; a supplement of 50 mg/day PO is recommended
- Risk for 1st trimester fetal loss is less in women with hyperemesis

PEARLS AND PITFALLS

- Other diagnoses should be explored in patients presenting after 9 wk gestation with nausea and vomiting as initial symptoms
- The use of PICC lines has been shown to carry significantly increased risk of maternal morbidity when compared to patients managed with either NG tube or medications alone
- Be aware of the risk for central pontine myelinosis in hyponatremia patients when replacing sodium
- Wernicke encephalopathy is the most devastating maternal complication:
 - Patients may not have the classic triad of ataxia, nystagmus, and dementia. Be concerned for any evidence of apathy or confusion

- Be sure to give patients thiamine 100 mg IV for any patient who presents with apathy or confusion

ADDITIONAL READING

- Bottomley C, Bourne T. Management strategies in hyperemesis. *Best Prac Res Clin Obstet Gynaecol.* 2009;23:549–564.
- Goodwin TM. Hyperemesis gravidarum. *Obstet Gynecol Clin North Am.* 2008;35(3):401–417.



CODES

ICD9

- 643.00 Mild hyperemesis gravidarum, unspecified as to episode of care or not applicable
- 643.10 Hyperemesis gravidarum with metabolic disturbance, unspecified as to episode of care or not applicable

ICD10

- O21.0 Mild hyperemesis gravidarum
- O21.1 Hyperemesis gravidarum with metabolic disturbance

HYPERKALEMIA

Christopher B. Colwell



BASICS

DESCRIPTION

- Potassium distribution:
 - Extracellular space: 2%
 - Intracellular space: 98%
- Potassium excretion:
 - Renal: 90%
 - GI: 10%
- Renal (80–90%) and extrarenal mechanisms maintain normal plasma concentration between 3.5 and 5 mmol/L.
- Renal excretion of potassium affected by:
 - Dietary intake
 - Distal renal tubular function
 - Acid–base balance
 - Mineralocorticoids
- Regulation between intracellular and extracellular potassium balance is affected by:
 - Acid–base balance
 - Insulin
 - Mineralocorticoids
 - Catecholamines
 - Osmolarity
 - Drugs

ETIOLOGY

- Decreased potassium excretion:
 - Most common cause: Renal failure (acute or chronic)
 - Distal tubular diseases:
 - Acute interstitial nephritis
 - Renal transplant rejection
 - Sickle cell nephropathy
 - Renal tubular acidosis (diabetes)
 - Mineralocorticoid deficiency:
 - Addison disease
 - Hypoaldosteronism
 - Drugs:
 - ACE inhibitors/angiotensin receptor blockers

- β -blockers
- Potassium-sparing diuretics
- NSAIDs
- Cyclosporine
- High-dose trimethoprim
- Lithium toxicity
- Intracellular to extracellular potassium shifts:
 - Metabolic acidosis:
 - Serum K^+ rises 0.2–1.7 mmol/L for each 0.1 U fall in arterial pH.
 - Hyperosmolar states
 - Insulin deficiency
 - Cell necrosis
 - Rhabdomyolysis
 - Hemolysis
 - Chemotherapy
 - Drugs:
 - Digitalis toxicity
 - Depolarizing muscle relaxants (e.g., succinylcholine)
 - β -blockers
 - α -agonists
 - Hyperkalemic periodic paralysis
- Excess exogenous potassium load:
 - Cellular breakdown:
 - Trauma
 - Tumor lysis
 - Salt substitutes
 - Oral potassium
 - Potassium penicillin G
 - Rapid transfusions of banked blood
- Pseudohyperkalemia:
 - Traumatic venipuncture with hemolysis
 - Postvenipuncture release of potassium can occur in the setting of:
 - Thrombocytosis (platelets $> 800,000/\text{mm}^3$)
 - Extreme leukocytosis (WBC $> 100,000/\text{mm}^3$)
 - Prolonged tourniquet time

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Hyperkalemia is often asymptomatic, even at high levels.

- Neuromuscular symptoms, predominantly weakness, which can progress to paralysis.
- Dyspnea owing to respiratory muscle weakness.
- Cardiac dysrhythmias may be the initial manifestation, so patients could also present with chest pain, palpitations, or syncope.

Physical-Exam

- Muscular weakness (rare except in severe cases)
 - _ Paralysis has been described
- Cardiac dysrhythmias (see ECG Changes)

ESSENTIAL WORKUP

- Serum potassium > 5 mmol/L
- Collect in heparinized tube if pseudohyperkalemia suspected.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Electrolytes, BUN, creatinine, glucose:
 - _ Elevated BUN, creatinine in renal failure
 - _ Hyponatremia with mineralocorticoid deficiency
 - _ Mild metabolic acidosis with type IV renal tubular acidosis
- Arterial blood gases:
 - _ Assess acid–base status
- Creatinine kinase:
 - _ Rhabdomyolysis can lead to renal insufficiency and result in hyperkalemia
- Ca^{2+}
- For hyperkalemia in face of normal renal function, calculate transtubular potassium gradient (TTKG):
 - _ $\text{TTKG} = \text{urine K} \times \text{Posm} / \text{plasma K} \times \text{Uosm}$
 - _ Posm, plasma osmolality; Uosm, urine osmolality
 - _ $\text{TTKG} > 8$ suggests extrarenal cause; $\text{TTKG} < 6$ indicates renal excretory defect.

Diagnostic Procedures/Surgery

- ECG: Changes correlate with degree of hyperkalemia:
 - _ > 5–6.5: Peaking of T-waves; shortening of QT_c interval
 - _ > 6.5–8: PR prolongation; loss of P-waves; widening of QRS complexes
 - _ > 8: Intraventricular blocks; bundle branch blocks; QRS axis shifts; sine wave complex
- Serum potassium cannot be reliably predicted by ECG:
 - _ Some patients (particularly those with chronic renal failure) will tolerate

higher potassium levels than others.

- Potassium effects (as seen on ECG) are more important than potassium level
- While unusual, the ECG can be normal in the setting of severe hyperkalemia

DIFFERENTIAL DIAGNOSIS

Pseudohyperkalemia

ALERT

Most common cause of hyperkalemia reported by lab is pseudohyperkalemia owing to hemolysis of red blood cells.



TREATMENT

PRE HOSPITAL

- Treatment of hyperkalemic-induced dysrhythmias/cardiac arrest involves different drugs from usual advanced cardiac life support (ACLS) measures (see Treatment, below):
 - Inhaled albuterol can lower potassium temporarily by 1 mmol/L.
 - β -agonists can also be administered by metered-dose inhaler or intravenously
 - Sodium bicarbonate can be effective in the setting of a metabolic acidosis.
 - Calcium chloride or gluconate is available in some prehospital systems and should be considered in the unstable patient when hyperkalemia is suspected.
- Diagnosis suggested by prehospital rhythm strip or in at-risk populations (renal failure)

INITIAL STABILIZATION/THERAPY

- ABCs
- IV access
- Cardiac monitor

ED TREATMENT/PROCEDURES

- Hyperkalemia with ECG changes (widened QRS complexes): Antagonize potassium-mediated cardiotoxicity:
 - Administer calcium gluconate (in awake patient) or calcium chloride (in patient without pulse):
 - Onset 1–3 min
 - 30–60 min duration
 - No effect on total serum potassium levels
- Severe (>7) or moderate (6–7) with ECG changes (heightened T-waves or loss of P-wave): Shift potassium intracellularly:
 - Administer combination of insulin and glucose:

- Onset 20–30 min
- 2–4 hr duration
- IV sodium bicarbonate:
 - Much more effective in patient who is acidotic
 - Onset 20 min
 - 2 hr duration
 - Caution in patients at risk for volume overload
 - Worsens concomitant hypocalcemia and hypernatremia
- Inhaled albuterol:
 - Onset within 30 min
 - 2–4 hr duration
- Can also be given by metered-dose inhaler or intravenously
- Calcium should be administered if the patient is unstable
- Enhanced excretion for $K^+ > 6$ without ECG changes:
 - Administer cation exchange resin:
 - Calcium or sodium polystyrene sulfonate PO or per rectum (PR)
 - Avoid in patients with suspected ileus or bowel obstruction.
- All patients:
 - Limit exogenous potassium and potassium-sparing drugs.
 - Treat underlying cause.

Special Situations

- Renal failure:
 - Hemodialysis immediately effective at removing potassium
 - Furosemide:
 - Effective in the absence of oliguric renal failure
 - Causes potassium-losing diuresis
- Cardiac arrest:
 - Administer $CaCl_2$ and $NaHCO_3$ with known or suspected hyperkalemia.
- Digoxin toxicity:
 - Avoid calcium if possible
 - When necessary, administer small doses very slowly.
 - Consider Digibind for $K^+ > 5.5$ mmol/L.
- Mineralocorticoid deficiency:
 - Administer hydrocortisone

MEDICATION

- Albuterol: 10–20 mg (peds: 2.5 mg if < 25 kg; 5 mg if ≥ 25 kg) nebulized over 10 min
- Calcium chloride 10%: 10 mL amp (peds: 0.2 mL/kg per dose) IV over 2–5 min
- Calcium gluconate 10%: 10 mL amp (peds: 0.6–1 mL/kg) IV over 2–5 min
- Furosemide: 40–80 mg (peds: 1 mg/kg) IV—modify dose to achieve appropriate

diuresis

- Hydrocortisone: 100 mg (peds: 1–2 mg/kg) IV
- Insulin and glucose: 10 U (peds: 0.1 U/kg) regular insulin + 50 mL 50% (peds: 0.5–1 g/kg) dextrose IV
- Sodium bicarbonate: 1–3 amps (44 mEq per amp) IV over 20–30 min (peds: 1–2 mEq/kg per dose)
- Sodium polystyrene sulfonate (Kayexalate) or calcium polystyrene sulfonate (preferred with volume overload):
 - Oral: 15 g mixed with water or 50 mL of sorbitol q2h to total of 5 doses
 - Rectal enema: 50 g in 200 mL of sorbitol q4–6h
 - Peds: 1 g/kg orally or rectally

First Line

- Calcium (under appropriate situations)
- Insulin and glucose

Second Line

- Sodium bicarbonate
- Kayexalate
- Albuterol



FOLLOW-UP

DISPOSITION

Admission Criteria

Admit most cases:

- Process of potassium removal is relatively slow.
- Most potassium is intracellular and, therefore, not measured on serum electrolytes.
- Significant changes in levels will take time.
- Levels may continue to rise.

Discharge Criteria

Mild hyperkalemia (< 5.5 mmol/L) provided that:

- Response to treatment has been demonstrated
- Known correctable cause
- Further rises in serum potassium not anticipated
- Early follow-up possible

Issues for Referral

Follow-up to address the underlying cause is important. In many cases, the underlying cause is renal insufficiency and the potassium will become elevated again if this is not

addressed. Often this will mean regular hemodialysis.

FOLLOW-UP RECOMMENDATIONS

Many patients with hyperkalemia will be admitted. For those who are not, close follow-up and in many cases access to hemodialysis will be important.

PEARLS AND PITFALLS

- Potassium effect is more important than absolute potassium level. The ECG is the most important determinate of need to treat acutely.
- Do not wait for confirmation of the potassium level to treat when the ECG indicates a hyperkalemic emergency
- Hyperkalemia is often asymptomatic until late: Obtain an ECG early in patients at risk.
- Sodium bicarbonate administration can easily lead to volume overload if not careful.

ADDITIONAL READING

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- Weisberg LS. Management of severe hyperkalemia. *Crit Care Med*. 2008;36(12):3246–3251.



See Also (Topic, Algorithm, Electronic Media Element)

- Dialysis Complications
- Renal Failure



CODES

ICD9

- 276.7 Hyperpotassemia
- 583.89 Nephritis and nephropathy, not specified as acute or chronic, with other specified pathological lesion in kidney
- 586 Renal failure, unspecified

ICD10

- E87.5 Hyperkalemia
- N05.9 Unsp nephritic syndrome with unspecified morphologic changes
- N19 Unspecified kidney failure

HYPERNATREMIA

Linda Mueller

BASICS

DESCRIPTION

Hypernatremia definition: Sodium > 145 mEq/L:

- Mild hypernatremia: Serum sodium 146–155 mEq/L
- Severe hypernatremia: Serum sodium > 155 mEq/L

ETIOLOGY

Divided into 3 categories

hypovolemic Hypernatremia

- Most common
- Loss or deficiency of water and sodium with water losses being greater than sodium losses
- Examples:
 - Renal failure
 - Medications (e.g., diuretics, lactulose)
 - Osmotic diuresis (mannitol, glucosuria, high protein feedings)
 - Insensible losses (burns, sweating)
 - Respiratory loss
 - Defective thirst mechanism
 - Lack of access to water
 - Diarrhea/vomiting
 - Intubated patients

Isovolemic Hypernatremia

- Water deficiency without sodium loss; free water loss
- Examples:
 - Fever
 - Hypothalamic diabetes insipidus (DI):
 - Head trauma
 - Tumor
 - Congenital
 - Infection (TB, syphilis, mycoses, toxoplasmosis, encephalitis)
 - Granulomatous disease (sarcoid, Wegner)
 - Cerebrovascular accident
 - Aneurysm
 - Nephrogenic DI:

- Congenital
- Drugs (lithium, amphotericin B, foscarnet, demeclocycline)
- Obstructive uropathy
- Chronic tubulointerstitial disease (sickle cell nephropathy, multiple myeloma, amyloidosis, sarcoidosis, systemic lupus erythematosus, polycystic kidney)
- Electrolyte disorders (hypercalcemia, potassium depletion)

Hypervolemic Hypernatremia

- Gain of water and sodium, with sodium gain greater than water gain.
- Examples:
 - Iatrogenic—most common cause:
 - Sodium bicarbonate administration
 - NaCl tablets
 - Hypertonic parenteral hyperalimentation
 - Hypertonic IV fluid (IVF)
 - Hypertonic dialysis
 - Hypertonic medicine preparations such as ticarcillin and carbenicillin
 - Cushing disease
 - Adrenal hyperplasia
 - Primary aldosteronism
 - Sea water drownings

Pediatric Considerations

- More prone to iatrogenic causes
- More likely to die or to have permanent neurologic sequelae
- Morbidity ranges from 25% to 50%.
- May present with high-pitched cry, lethargy, irritability, muscle weakness
- Poor breast feeding and inappropriate formula preparations are a potential cause in neonates
- If hypernatremia is due to DKA, follow pediatric DKA protocols for fluid resuscitation
- DDAVP dose for 3 mo–12 yr is 5–30 µg/day intranasally
 - Consider NG hydration

Geriatric Considerations

- Most commonly affected group due to impaired renal concentrating ability and reduced thirst mechanism
- Consider neglect if underlying etiology is dehydration alone

Pregnancy Considerations

- May encounter transient DI of pregnancy

- Vasopressin and desmopressin are category B drugs in pregnancy
- Hydration status much more difficult to evaluate accurately by exam



DIAGNOSIS

SIGNS AND SYMPTOMS

- Most symptoms attributed to underlying cause (e.g., dehydration)
- More marked with acute changes
- Death likely to occur with sodium of ≥ 185 mEq/L
- May see the following symptoms, usually at levels ≥ 160 mEq/L:
 - _ Neurologic:
 - Headache
 - Tremulousness
 - Irritability
 - Ataxia
 - Mental confusion
 - Delirium
 - Seizures
 - Coma
 - Hyperreflexia
 - Asterixis
 - Chorea
 - Subarachnoid, intracerebral, and subdural hemorrhages
 - Dural sinus thrombosis
 - _ Musculoskeletal:
 - Spasticity
 - Muscle weakness
 - Muscle twitching
 - _ Other:
 - Anorexia
 - Tachypnea
 - Poor skin turgor
 - Nausea/vomiting

Hypovolemic Hypernatremia

- Tachycardia
- Orthostasis
- Dry mucous membranes
- Oliguria
- Azotemia

Hypervolemic Hypernatremia

- Pulmonary edema
- Peripheral edema

Physical-Exam

- Evaluate for hydration status
- Look at mucous membranes, neck veins, and skin turgor
- Perform a complete neurologic exam and repeat throughout ED stay
- Obtain orthostatic vital signs

ESSENTIAL WORKUP

Serum Na⁺ level

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Electrolytes, BUN/creatinine, glucose
- CBC
- Urinalysis:
 - Specific gravity
 - Urine/serum osmolality
 - Urine Na⁺

Imaging

- CXR:
 - For infection/aspiration
 - Pulmonary edema with hypervolemic hyponatremia
- CT brain:
 - For altered mental status
 - Venous sinus thrombosis
 - Subarachnoid hemorrhages
 - Subdural hematoma

Diagnostic Procedures/Surgery

Consider Foley catheter to accurately monitor input and output

DIFFERENTIAL DIAGNOSIS

- Diabetic ketoacidosis
- Hyperosmolar coma
- Primary CNS lesions



TREATMENT

PRE HOSPITAL

Volume resuscitation if hypovolemic or evidence of hemodynamic compromise

INITIAL STABILIZATION/THERAPY

- ABCs
- 0.9% NS IV bolus for severe hypotension
- Naloxone, thiamine, D₅₀W (or Accu-Chek) for altered mental status

ED TREATMENT/PROCEDURES

General:

- Calculate water deficit:
 - _ Water deficit = $0.6 (\text{weight in kg}) \times (1 - \text{desired sodium/actual sodium})$
- Do not rapidly correct hypertonicity to normal serum osmolality:
 - _ Rapid correction may cause seizures.
 - _ Reduce serum sodium level by $< 0.5\text{--}0.7 \text{ mEq/L/hr}$.

Hypovolemic Hypernatremia

- Replace volume contraction with 0.9% NS IV bolus.
- Change to D₅W or hypotonic saline once volume replenished and hemodynamically stable.

Isovolemic Hypernatremia

- Calculate water deficit.
- Correct water deficit with D₅W or hypotonic saline:
 - _ Replace half of deficit in 1st 24 hr, then remainder over 1–2 days.

Hypervolemic Hypernatremia

- Remove excess water with diuretics or dialysis.
- When euvolemic, replace water deficit with D₅W.
- Avoid hypertonic saline solutions because patient already has excess of total body sodium.

Diabetes Insipidus Hypernatremia

- Sodium restriction
- Desmopressin:
 - _ Aqueous vasopressin (DDAVP)
 - _ Best therapeutic agent
- Chlorpropamide (Diabinese) enhances effect of vasopressin at renal tubule.
- Carbamazepine causes release of vasopressin.
- Hydrochlorothiazide enhances sodium excretion.
- Discontinue DI-inducing drugs.

MEDICATION

- Chlorpropamide (Diabinese): 100–500 mg/d
- Vasopressin (DDAVP): 1–2 µg IV/SC q12h or 5–20 µg intranasally

First Line

Volume correction starting initially with NS

Second Line

Correct the underlying cause.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Newly diagnosed sodium > 150 mEq/L for monitoring and treatment
- Admit sodium > 160 mEq/L or symptomatic patients to ICU.

Discharge Criteria

- Sodium < 150 mEq/L in asymptomatic patient
- Sodium > 150 mEq/L in patients with history of chronically elevated sodium who are at their baseline and asymptomatic

FOLLOW-UP RECOMMENDATIONS

Repeat serum sodium levels within a week.

PEARLS AND PITFALLS

- Up to 30% of acute hypernatremia patients will have permanent neurologic sequelae, a complete and well-documented neurologic exam is a must.
- Patients at extreme ages and with chronic conditions are most susceptible to neurologic complications:
 - On going fluid losses may require recalculation of fluid needs
 - Repeat lab work to confirm controlled correction of sodium

ADDITIONAL READING

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- Lin M, Lu S, Lim I. Disorders of water imbalance. *Emerg Med Clin North Am.* 2005;23:749–770, ix.

- Pfennig CL, Slovis CM. Sodium disorders in the emergency department: A review of hyponatremia and hypernatremia. *Emerg Med Pract.* 2012;14(10):1–20.
- Ranadive SA, Rosenthal SM. Pediatric disorders of water balance. *Endocrinol Metabol Clin North Am.* 2009;38(4):663–672.



See Also (Topic, Algorithm, Electronic Media Element)

- Diabetic Ketoacidosis
- Hyperosmolar Coma
- Hyponatremia



CODES

ICD9

- 276.0 Hyperosmolality and/or hypernatremia
- 775.5 Other transitory neonatal electrolyte disturbances

ICD10

- E87.0 Hyperosmolality and hypernatremia
- P74.2 Disturbances of sodium balance of newborn

HYPEROSMOLAR SYNDROME

Matthew T. Robinson

BASICS

DESCRIPTION

- Results from a relative insulin deficiency in the undiagnosed or untreated diabetic
- Sustained hyperglycemia creates an osmotic diuresis and dehydration:
 - Extracellular space maintained by the osmotic gradient at the expense of the intracellular space
 - Eventually profound intracellular dehydration occurs.
- Total body deficits of H_2O , Na^+ , Cl^- , K^- , PO_4^- , Ca^{2+} , and Mg^{2+}
- In contrast to diabetic ketoacidosis (DKA), severe ketoacidosis does not occur:
 - Circulating insulin levels are higher.
 - The elevation of insulin counter-regulatory hormones is less marked.
 - The hyperosmolar state itself inhibits lipolysis (the release of free fatty acids) and subsequent generation of keto acids

Geriatric Considerations

- Most commonly seen in elderly type II diabetics who experience a stressful illness that precipitates worsening hyperglycemia and reduced renal function
- In the elderly, 30–40% of cases are associated with the initial presentation of diabetes.

Pediatric Considerations

Hyperosmolar hyperglycemic states (HHS) rare in pediatric patients

ETIOLOGY

- Hyperosmolar state precipitated by factors that:
 - Impair peripheral insulin action
 - Increase endogenous or exogenous glucose
 - Decrease patient's ability to replace fluid loss
- Infection is the most common precipitating factor in 32–60% of cases.
- Other precipitating causes include:
 - Inadequate diabetes therapy
 - Medication omission
 - Diet indiscretion
 - Infections
 - Pneumonia
 - UTI
 - Sepsis

- Medications/drugs
- Diuretics
- β -blockers
- Calcium channel blockers
- Phenytoin
- Cimetidine
- Amphetamines
- Ethanol
- Myocardial infarction
- Stroke
- Renal failure
- Heat stroke
- Pancreatitis
- Intestinal obstruction
- Endocrine disorders
- Burns
- Heat stroke



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Progression of signs and symptoms typically occur over days to weeks.
- Polyuria/polydipsia/weight loss
- Dizziness/weakness/fatigue
- Blurred vision
- Leg cramps

Physical-Exam

- Dehydration
- Tachycardia
- Sunken eyes
- Hypotension
- Orthostasis
- Dry mucous membranes
- Decreased skin turgor
- Collapsed neck veins
- Coma/lethargy/drowsiness
- Urinary output maintained until late
- Seizures/focal neurologic deficits
- Concurrent precipitating medical illness

ESSENTIAL WORKUP

Diagnostic criteria:

- Serum glucose ≥ 600 mg/dL (usually $> 1,000$ mg/dL)
- Minimal ketosis
- pH ≥ 7.30 , $\text{HCO}_3^- \geq 15$ mEq/L
- Effective serum osmolality > 320 mOsm/kg:
 - _ $= 2 \times \text{Na}^+ + \text{glucose}/18$
 - _ BUN not included because it is freely permeable between fluid compartments

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Broad testing indicated to evaluate hyperosmolar syndrome and for precipitating causes
- Electrolytes:
 - _ K^+ may be elevated even in the presence of total body deficit owing to shift from intracellular space to extracellular space.
 - _ Mild anion gap metabolic acidosis owing to lactic acid, β -hydroxybutyric acid, or renal insufficiency
 - _ Increased sodium—correct for hyperglycemia: Corrected $[\text{Na}^+] = [\text{Na}^+] + 1.6 \times [(\text{glucose in mg/dL}) - 100]/100$
- BUN, creatinine:
 - _ Azotemia with elevated BUN/creatinine ratio owing to prerenal and intrarenal causes
- Venous blood gas (VBG) or arterial blood gas (ABG) to rapidly determine pH:
 - _ ABG necessary to evaluate mixed acid–base disorders
- Serum ketones, β -hydroxybutyrate, and lactate level if pH < 7.3 or significantly elevated anion gap to evaluate mixed acid–base disorder
- Serum osmolality
- CBC:
 - _ Leukocytosis due to infection, stress, or hemoconcentration
 - _ Increased hemoglobin and hematocrit due to hemoconcentration
- Lipase and amylase:
 - _ Pancreatitis common
 - _ Elevated amylase and lipase with no evidence of pancreatitis common
 - _ May be due to increased salivary secretion, hemoconcentration, or decreased renal clearance
- Urinalysis:
 - _ Check for ketones/glucose.
 - _ Assess for UTI.
- Magnesium, calcium, phosphate

- Blood cultures in sepsis
- Creatine kinase for rhabdomyolysis:
 - Incidence as high as 17%
- Urine pregnancy test in females of childbearing years
- Cardiac enzymes and troponin for myocardial infarction

Imaging

- CXR to evaluate for possible underlying pneumonia
- Head CT: When indicated for AMS or with focal neurologic deficit

Diagnostic Procedures/Surgery

ECG:

- Evaluate for electrolyte abnormalities causing conduction impairment
- Evaluate for signs of ischemia as triggering event

DIFFERENTIAL DIAGNOSIS

Differentiate from DKA:

- If acidosis or significant anion gap present, must determine cause (i.e., ketosis, DKA, lactic acidosis, [hypoperfusion, sepsis, or postictal], or other causes of metabolic acidosis)
- Mixed disorder of HHS and DKA present in up to 33% of patients



TREATMENT

PRE HOSPITAL

IV fluid resuscitation and initial stabilization

INITIAL STABILIZATION/THERAPY

ABCs:

- Secure airway in comatose patients.
- Cardiac monitor and 18G IV
- Naloxone, thiamine, and blood glucose for coma of unknown cause
- Restore hemodynamic stability with IV fluids.
- 0.9% NS 1–2 L over the 1st hr
- Larger volumes of fluid may be needed to normalize the vital signs and establish urine output.

ED TREATMENT/PROCEDURES

- General strategy:
 - Frequent reassessment of volume and mental status
 - Electrolyte assessment difficult:
 - Serum levels of Na^+ , K^+ , PO_4^- do not accurately reflect the total

body solute deficits or the intracellular environment.

- Repeat electrolyte and glucose levels hourly.

– Search for a precipitating illness.

- Fluids:

– Begin resuscitation with 0.9% NS 1–2 L over 1–2 hr to restore intravascular volume and achieve hemodynamic stability.

– Use 0.45% saline after initial resuscitation

– Calculate total body water (TBW) deficit using corrected serum sodium:

- $TBW \text{ deficit} = 0.6 \times \text{weight (kg)} \times (1 - 140/\text{corrected Na}^+)$

– Average fluid deficit is 9 L.

– Replace 50% of the fluid deficit over the next 12 hr.

– Change fluid to D5 1/2 NS when serum glucose is < 250 mg/dL.

- Potassium:

– Anticipate hypokalemia:

- Total body deficit of $\sim 5\text{--}10$ mEq/kg body weight (replace over 3 days)

– Begin potassium repletion after urine output is established. Do not start in anuric patients or if initial K^+ level is > 5 mEq/L.

- If the initial K^+ is normal (4–5 mEq/L), give 20–30 mEq KCl in the 1st L of fluids, then give 20 mEq/hr.

- If the initial K^+ is low (3–4 mEq/L), give 40 mEq in 1st L

- If serum K^+ is < 3 mEq/L hold insulin and give 10–20 mEq/h until $K^+ > 3.3$, then add 40 mEq to each liter

- Follow repeat serum K^+ levels q1–2h and adjust treatment accordingly.

- Insulin:

– No role in the early resuscitation

– Earlier use of insulin may cause rapid correction of hyperglycemia with collapse of the intravascular space, hypotension, and shock or hypokalemia and dysrhythmias.

– Some patients will not require insulin.

– Use insulin as sole therapy in patients with fluid overload (i.e., acute renal failure [ARF]).

– Begin only after achieving hemodynamic stability and evaluating for hypokalemia:

- Do not use unless serum $K^+ > 3.3$ mEq/L

– SC or IM insulin not recommended due to erratic absorption

– Titrate drip to optimally decrease serum glucose by 50–90 mg/dL/hr. More rapid correction places the patient at risk for developing cerebral edema.

– Decrease drip rate by 1/2 when serum glucose < 250 mg/dL.

– Adjust insulin drip to maintain serum glucose between 150–200 mg/dL, and

continue until serum bicarbonate is >18 mg/dL and pH > 7.3

- Phosphate:
 - Routine replacement not recommended
 - If serum levels <1 mg/dL, give 20–30 mmol potassium phosphate over 24 hr
 - Monitor serum calcium levels closely
- Magnesium:
 - 0.35 mEq/kg magnesium in fluids for 1st 3–4 hr (2.5–3 g MgSO_4 in 70 kg patient)
 - Caution in ARF
- Anticoagulation:
 - Arterial thrombosis may complicate hyperosmolar state:
 - Consider SC heparin as prophylaxis.
 - Remain vigilant to detect thrombotic complications (e.g., MI, pulmonary embolus, mesenteric ischemia).

MEDICATION

- Insulin: Begin with 0.05–0.1 U/kg/h; modify after assessing clinical response.
- MgSO_4 (magnesium sulfate): 50% (5 g/10 mL; dilute to at least 20% before IV use)
- Naloxone: 2 mg (peds: 0.1 mg/kg) IV push (IVP)
- Potassium phosphate IV: Phosphorous serum level <0.5 mg/dL: 0.5 mmol/kg IV infused over 4–6 hr; phosphorous serum level 0.5–1 mg/dL: 0.25 mmol/kg IV infused over 4–6 hr
- Potassium phosphate PO: Phosphorus 250 mg per tablet and potassium 1.1 mEq per tablet
- Thiamine: 100 mg (peds: 10–25 mg) IVP



FOLLOW-UP

DISPOSITION

Admission Criteria

- All but the mildest cases should be admitted to ICU:
 - Frequent serial labs for the 1st 24 hr
 - Rapid shifts in fluids and electrolytes and the potential for deterioration in mental status and arrhythmias mandate close monitoring.
- Mild cases may be managed in an observation unit over 12–24 hr.

Discharge Criteria

- Patients meeting the diagnostic criteria for hyperosmolar syndrome should not be discharged.
- Mild hyperglycemia patients with mild volume deficits and normal serum

osmolarity can be discharged after hydration and correction of hyperglycemia.

Issues for Referral

Patient should follow-up with endocrinology and with their primary physician within 1 wk postdischarge for long-term blood glucose monitoring and insulin therapy.

PEARLS AND PITFALLS

- Failure to look for precipitating event or cause
- Too rapid correction of glucose—may lead to hypotension
- Continuing isotonic fluids after volume resuscitation—may lead to hypernatremia
- Continuing hypotonic fluids without frequent electrolytes—may lead to cellular edema, cerebral edema
- Failure to prevent hypokalemia: Respiratory depression, dysrhythmias
- Avoid phenytoin in the event of seizure activity:
 - _ Inhibits the endogenous release of insulin

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Diabetic Ketoacidosis



CODES

ICD9

- 250.20 Diabetes with hyperosmolarity, type II or unspecified type, not stated as uncontrolled
- 250.21 Diabetes with hyperosmolarity, type I [juvenile type], not stated as uncontrolled
- 276.0 Hyperosmolality and/or hypernatremia

ICD10

- E11.01 Type 2 diabetes mellitus with hyperosmolarity with coma
- E87.1 Hypo-osmolality and hyponatremia

HYPERPARATHYROIDISM

Rami A. Ahmed • Brad D. Gable



BASICS

DESCRIPTION

- Parathyroid hormone (PTH) excess with symptoms owing to PTH actions:
 - _ Decreases urinary Ca^{2+} loss
 - _ Increases urinary PO_4^{2-} loss
 - _ Stimulates vitamin D conversion from 25(OH)-D to 1,25(OH)-D in kidney
 - _ Liberates Ca^{2+} and PO_4^{2-} from bone
 - _ Hypercalcemia is the primary metabolic finding
- Hypercalciuria from hypercalcemia (despite decreased urinary loss) produces increased magnesium loss in urine
- Magnesium (negative feedback to prevent hypercalcemia causes hypomagnesaemia):
 - _ Cofactor in the production of PTH
 - _ Essential for action of PTH in target tissues
- Genetics:
 - _ Associated with multiple endocrine neoplasia type 1:
 - Hyperparathyroidism
 - Pancreatic islet disease
 - Pituitary disease
 - _ Associated with multiple endocrine neoplasia type 2:
 - Hyperparathyroidism (type 2A, rare in 2B)
 - Medullary carcinoma of the thyroid (type 2A and 2B, less virulent in type 2A)
 - Pheochromocytoma (type 2A and 2B)
 - Mucosal neuroma (type 2B)

ETIOLOGY

- Excess secretion of PTH owing to:
 - _ Primary hyperparathyroidism (adenoma 85%, hyperplasia 14%, carcinoma <1%)
 - _ Secondary hyperparathyroidism (response to vitamin D deficiency or chronic renal failure with hyperphosphatemia):
 - Calcium is low or normal, but PTH levels are elevated



DIAGNOSIS

SIGNS AND SYMPTOMS

Stones, bones, abdominal groans, and psychiatric moans

ALERT

- Hypercalcemic crisis:
 - _ Anorexia, nausea, vomiting
 - _ Mental obtundation

History

Depends on the severity and rapidity of hypercalcemia

Pediatric Considerations

- Neonate:
 - _ Hypotonia, weakness, and listlessness
 - _ Following delivery to hypoparathyroid mothers
- Hypercalcemic infants:
 - _ Broad forehead
 - _ Epicanthal folds
 - _ Underdeveloped nasal bridge
 - _ Prominent upper lip

Physical-Exam

- Dehydration
- Cardiac:
 - _ Hypertension (even in the face of dehydration)
 - _ Cardiac conduction abnormalities (*not* proportional to degree of hypercalcemia)
 - _ Bradydysrhythmia
 - _ Bundle branch blocks
 - _ Complete heart block
 - _ Asystole
 - _ Short QT interval (shortened ST segment)
 - _ Potentiation of digitalis effects (Hypercalcemia + digoxin = digitalis toxicity)
- Neurologic:
 - _ Headaches
 - _ Decreased reflexes
 - _ Proximal muscle weakness
 - _ Dementia
 - _ Lethargy
 - _ Coma
- Psychiatric:

- Personality changes
- Depression
- Inability to concentrate
- Anxiety
- Psychosis
- GI:
 - Anorexia, nausea, vomiting
 - Constipation
 - Peptic ulcer disease
 - Pancreatitis
- General:
 - Fatigue
 - Weight loss
 - Polyuria and polydipsia
- Musculoskeletal:
 - Gout/pseudogout
 - Bone pain, bone cysts (osteitis cystica)
 - Arthralgias
 - Chondrocalcinosis
- Renal:
 - Kidney stones
 - Nephrocalcinosis
 - Decreased renal concentrating ability

ESSENTIAL WORKUP

- Calcium level
- Albumin:
 - Elevated albumin—falsely elevated calcium level
 - Low albumin—falsely lowered calcium level
- Evaluate for symptoms of hypercalcemia, especially impending parathyroid storm (hypercalcemic crisis—anorexia, nausea, vomiting, obtundation progressing to coma).
- Review history for medication ingestion (see Differential Diagnosis below)
- No further ED workup if:
 - Asymptomatic
 - Normal ECG
 - Calcium level < 14 mg/dL when corrected for albumin
- If symptomatic with $\text{Ca}^{2+} < 14$ mg/dL or any patient with $\text{Ca}^{2+} \geq 14$ mg/dL, check:
 - Ionized calcium
 - Chest radiograph (for CHF/malignancy)
 - Phosphorus
 - Electrolytes, BUN, creatinine

- Sedimentation rate
- Alkaline phosphatase
- Magnesium
- Thyroid-stimulating hormone (TSH)
- CBC

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Calcium correction for albumin:
 - Corrected Ca^{2+} (mg/dL) = measured Ca^{2+} (mg/dL) + 0.8 [4 – albumin (g/dL)]
 - Acidosis:
 - Decreases affinity to albumin—increases ionized (metabolically active) Ca^{2+}
 - Decrease of 0.1 pH unit increases the ionized Ca^{2+} by 3–8%
- Phosphorus:
 - Low in primary hyperparathyroidism
 - Usually high in secondary hyperparathyroidism
 - Normal or high in malignancy-related hypercalcemia
- Chloride/ PO_4^{2-} ratio:
 - > 33—hyperparathyroidism
 - < 30—malignancy
- Alkaline phosphatase:
 - Increased in 50% of patients with hyperparathyroidism
 - Normal with vitamin D excess
- Erythrocyte sedimentation rate (ESR):
 - Normal in hyperparathyroidism
 - Elevated in malignancy or granulomatous diseases
- Anemia:
 - Present with malignancy or granulomatous disease
 - Absent in hyperparathyroidism
- Magnesium:
 - Low or low normal
- PTH:
 - Elevated in primary and secondary hyperparathyroidism
- PTH-related peptide:
 - Secreted by squamous cell carcinomas of lung, head, neck; renal carcinomas, bladder carcinomas, adenocarcinomas, and lymphomas

Imaging

- Chest radiograph:
 - To assess CHF risk during IV hydration
 - Granulomatous disease or malignancy if cause of hypercalcemia is uncertain

Diagnostic Procedures/Surgery

Definitive treatment is parathyroidectomy to treat and establish cause of hyperparathyroidism

DIFFERENTIAL DIAGNOSIS

- PTH related:
 - Primary or secondary hyperparathyroidism
 - Familial hypocalciuric hypercalcemia
- Malignancy related:
 - PTH-related peptide or Ca^{2+} release from osteolytic tumor
- Vitamin D related:
 - Excess vitamin D intake or vitamin D production by granulomas
- Immobilization:
 - Associated with Paget disease
- Drug induced:
 - Thiazide diuretics
 - Lithium
 - Aluminum-containing antacids
 - Tamoxifen
 - Estrogens
 - Androgens
 - Vitamin A



TREATMENT

PRE HOSPITAL

May present as a primarily psychiatric disorder

INITIAL STABILIZATION/THERAPY

- Cardiac monitor if:
 - Symptomatic hypercalcemia
 - Ca^{2+} level > 14 mg/dL
- Hydrate with IV 0.9% NS.
- Correct acidosis

ED TREATMENT/PROCEDURES

- Treat hypercalcemia:
 - Vigorous hydration with 0.9% NS at minimum of 250 mL/hr unless CHF:

- Lowers calcium 1.5–2 mg/dL in 24 hr
- Achieve urine output 100 mL/hr
- Administer furosemide or other loop diuretic (calciuric) after adequate volume replacement or in the presence of CHF:
 - Common error: Administration of furosemide before adequate hydration
 - If urinary sodium losses exceed replacement sodium, then renal conservation measures impede calcium excretion
- Avoid thiazide diuretics (impede calcium excretion)
- Consider glucocorticoid administration (decreases gut absorption and increases renal excretion of Ca^{2+}); most effective with vitamin D intoxication or granulomatous diseases
- Start bisphosphonates (pamidronate or etidronate) in conjunction with primary physician (inhibits calcium mobilization from bone)
- Treat cardiac dysrhythmias in standard fashion:
 - Correct acidosis
- Determine the cause of the hypercalcemia.
- Stop all medications that may contribute to hypercalcemia
- Exercise extreme caution in the use of digoxin.
- Anticipate CHF and electrolyte imbalance with frequent reassessment of patient and monitoring of serum electrolytes and magnesium levels
- Calcitonin if unable to use hydration
- Emergent dialysis with renal failure

MEDICATION

First Line

- NS hydration: Initial 250–300 mL/h depending on patient's propensity to CHF
- Furosemide: 40 mg IV q2–4h after assurance of adequate hydration
- Prednisone: 40–60 mg PO OR Hydrocortisone: 100 mg (peds: 1–2 mg/kg) IV

Second Line

- IN CONSULTATION WITH ENDOCRINOLOGIST
- Calcitonin salmon 4 U/kg SC if saline hydration contraindicated
 - Test dose: Intradermal 0.1 mL of 10 U/mL solution recommended
 - Initial dose: 4 U/kg SC q12h
- Pamidronate:
 - If albumin-corrected Ca^{2+} level 12–13.5 mg/dL: 60 mg IV infused over 2 hr
 - If albumin-corrected Ca^{2+} level > 13.5 mg/dL: 90 mg IV over 4 hr
 - Dosage should be reduced in renal impairment and infusion time may be extended to reduce nephrotoxic potential but no formal recommendations exist (pregnancy category D – maternal benefit may outweigh fetal risk)

- Zoledronic acid: 4 mg IV over 15–30 min (first-line agent due to efficacy and convenience, but less preferred due to lack of less expensive available generic)
- Cinacalcet (Sensipar): 30 mg PO daily or BID (calcimimetic for secondary hyperparathyroidism or parathyroid carcinoma)



FOLLOW-UP

DISPOSITION

Admission Criteria

- Corrected calcium > 14 mg/dL
- Symptomatic hypercalcemia
- Evidence of abnormal cardiac rhythm or conduction

Discharge Criteria

- Not meeting admission criteria
- Able to maintain adequate hydration

Issues for Referral

If diagnosis is suspected, referral to check PTH levels and response to therapy

FOLLOW-UP RECOMMENDATIONS

- If hyperparathyroidism is suspected arrange follow-up and send a PTH level
- Patient needs to be instructed to maintain hydration and stop medications associated with hypercalcemia (see the list in Differential Diagnosis)

PEARLS AND PITFALLS

- The hypercalcemia of hyperparathyroidism is rarely symptomatic and Ca^{2+} level rarely > 14. (Higher levels are most frequently attributable to neoplastic disease)
- The importance of diagnosis is to prevent long-term complications
- Calcium level should be measured as ionized Ca^{2+} , or corrected for albumin level
- Administration of loop diuretics prior to adequate saline hydration will worsen hypercalcemia; some experts suggest that loop diuretics may be no longer warranted for this indication

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See Also (Topic, Algorithm, Electronic Media Element)

Hypoparathyroidism



CODES

ICD9

- 252.00 Hyperparathyroidism, unspecified
- 252.01 Primary hyperparathyroidism
- 252.02 Secondary hyperparathyroidism, non-renal

ICD10

- E21.0 Primary hyperparathyroidism
- E21.1 Secondary hyperparathyroidism, not elsewhere classified
- E21.3 Hyperparathyroidism, unspecified

HYPERTENSIVE EMERGENCIES

David F. M. Brown • Eva Tovar Hirashima



BASICS

DESCRIPTION

- *Hypertensive crisis:*
 - Severely elevated BP defined by a SBP > 179 mm Hg or a DBP > 109 mm Hg
- *Hypertensive urgency:*
 - Severely elevated BP without end-organ damage
- *Hypertensive emergency:*
 - Severely elevated BP associated with acute end-organ damage
- Loss of autoregulation of blood flow in hypertensive emergency:
 - Arterioles vasoconstrict to counter pressure
 - High pressures overwhelm arterioles and endothelial damage occurs
 - Endothelial injury leads to increase permeability, activation of the coagulation cascade and platelets, and deposition of fibrin
- Activation of the renin–angiotensin system and the sympathetic nervous system:
 - Leads to further vasoconstriction and production of proinflammatory cytokines
- End-organ ischemia:
 - Renewed release of vasoconstrictors
 - Worsened by pressure natriuresis
 - Triggers a vicious cycle
- Organs affected:
 - Brain (encephalopathy, CVA, ICH)
 - Retina (hemorrhage, papilledema)
 - Heart (MI, aortic dissection, acute HF)
 - Kidneys (acute renal failure)
 - Placenta (preeclampsia/eclampsia)

ETIOLOGY

- Essential HTN
- Renal:
 - Vascular disease
 - Parenchymal disease
- Coarctation of the aorta
- CNS disorders:
 - Head trauma
 - CVA/ICH

- Brain tumor
- Spinal cord injury
- Endocrine:
 - Pheochromocytoma
 - Cushing syndrome
 - Primary hyperaldosteronism
 - Renin-secreting tumor
- Drugs:
 - Cocaine, phencyclidine, amphetamines
 - Erythropoietin, tacrolimus, cyclosporine, corticosteroids, oral contraceptives
 - MAOI interactions
 - Antihypertensive medication withdrawal
 - Lead intoxication
- Autonomic hyperreactivity:
 - Guillain–Barré syndrome
 - Acute intermittent porphyria
- Postop pain and/or anesthesia complications
- Pregnancy related:
 - Preeclampsia/eclampsia



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Inquire about:
 - Use of any prescribed and OTC medication
 - Duration and control of pre-existing HTN
 - Prior end-organ damage
 - Details of antihypertensive therapy
 - Comorbid conditions (obesity, CAD, DM)
 - Recreational drug use
- Assess for end-organ compromise in decreasing order of frequency:
 - Dyspnea
 - Chest pain
 - Headache
 - Altered mental status/confusion
 - Focal neurologic symptoms

Physical-Exam

- BP measured in both arms
 - Use proper cuff size

- Assess for end-organ compromise:
 - Neurologic:
 - Level of consciousness
 - Visual fields
 - Focal motor/sensory deficits
 - Ophthalmologic:
 - Funduscopic exam (retinal hemorrhages, papilledema)
 - Cardiovascular:
 - Elevated JVP
 - Lung crackles
 - Aortic insufficiency murmur
 - S3
 - Asymmetrical pulses

ESSENTIAL WORKUP

- 12-lead EKG:
 - Ischemic changes, LV hypertrophy
- Assess kidney function
 - Acute renal failure may be asymptomatic

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC
 - Anemia and thrombocytopenia are present in thrombotic microangiopathy
- Standard hospital protocols for chest pain
- BUN, creatinine
- Electrolytes
 - Hypokalemia present in primary mineralocorticoid excess
- Urinalysis:
 - Proteinuria, hematuria, and casts
- Urine toxicology screen:
 - If recreational drugs are suspected
- HCG

Imaging

- Chest x-ray:
 - If cardiopulmonary symptoms are present
- Head CT:
 - If headache, confusion, neurologic findings
- CTA chest and abdomen:
 - If concern for aortic dissection

Diagnostic Procedures/Surgery

- Arterial line
- Lumbar puncture:
 - Exclude subarachnoid hemorrhage

DIFFERENTIAL DIAGNOSIS

- Acute coronary syndrome (ACS)
- Acute heart failure (AHF)
- Aortic dissection
- Intracerebral hemorrhage (ICH)
- CVA (ischemic or hemorrhagic)
- Preeclampsia/eclampsia
- Withdrawal syndromes:
 - β -blockers
 - Clonidine (central α 2-agonist)
- States of catecholamine excess:
 - Pheochromocytoma
 - Cocaine/sympathomimetic drug intoxication
 - Tyramine ingestion when on MAOIs



TREATMENT

PRE HOSPITAL

- ABCs
- Consider gentle BP reduction.

INITIAL STABILIZATION/THERAPY

- ABC, cardiac monitoring, pulse oximetry
- Oxygen administration
- IV access

ED TREATMENT/PROCEDURES

- Hypertensive urgency:
 - No need to treat, but close follow-up
 - Use oral agents only
 - Give any missed home dose
 - Goal: Lower the BP gradually over 24–48 hr
- Hypertensive emergency:
 - Treat end-organ damage, not absolute BP
 - Reduce MAP by ≤ 20 –25% in the 1st hr
 - Goal: Systolic ~ 160 mm Hg, diastolic ~ 100 mm Hg in 2–6 hr
 - Once BP stable with IV therapy, transition to oral therapy within 6–12 hr
 - More gradual reduction recommended in:

- Acute ongoing injury to CNS
 - More rapid reduction recommended in:
 - Aortic dissection
- *Hypertensive encephalopathy:*
 - Goal: MAP lowered by max. 20% or to DBP 100–110 mm Hg within 1st hr then gradual reduction in BP to normal over 48–72 hr
 - Drug of choice: Nicardipine, clevidipine, or labetalol
- *Ischemic stroke:*
 - CPP = MAP – ICP
 - Decreased CPP from hypotension (low MAP) or cerebral edema (high ICP) may extend infarct
 - Treat only SBP > 220 mm Hg or DBP > 120 mm Hg
 - Lytic candidates should have BP lowered to < 185/110 mm Hg
 - Goal: MAP lowered by no more than 15–20%, DBP not < 100–110 mm Hg in first 24 hr
 - Goal post tPA: BP < 180/105 mm Hg
 - Drug of choice: Nicardipine, clevidipine, or labetalol
- *Hemorrhagic CVA or SAH:*
 - Treat if SBP > 180 mm Hg/DBP > 100 mm Hg
 - Goal: MAP lowered by 20–25% within the 1st hr or SBP 140–160 mm Hg
 - Drug of choice: Nicardipine, clevidipine, or labetalol
 - Avoid dilating cerebral vessels with nitroglycerin or nitroprusside
- *ACS:*
 - Goal: MAP to 60–100 mm Hg
 - Drug of choice: Labetalol or esmolol in combination with nitroglycerin
 - Avoid: Hydralazine (reflex tachycardia) and nitroprusside (“coronary steal”)
- *AHF:*
 - Goal: MAP to 60–100 mm Hg
 - Drug of choice nitroprusside or NTG with ACEI and/or loop diuretic
- *Acute renal failure/microangiopathic anemia:*
 - Goal: MAP lowered by 20–25% within 1st hr
 - Drug of choice: Nicardipine, clevidipine, or fenoldopam. For scleroderma renal crises ACEI are drugs of choice.
- *Aortic dissection:*
 - Reduce shear force (dP/dT) by reducing both BP and HR
 - β -blockade must precede any drug that may cause reflex tachycardia
 - Goal: SBP 100–120 mm Hg and HR < 65 bpm within 1st 20 min
 - Drug of choice: Esmolol in combination with dihydropyridine CCB or nitroprusside
 - Consult vascular surgery if type A
- *Sympathomimetics (pheochromocytoma, cocaine, amphetamines):*
 - Goal: MAP lowered by 20–25% within 1st hr
 - Avoid pure β -blockade (α is left unopposed)

- Drug of choice: Phentolamine or calcium channel blocker with benzodiazepine. Use clonidine in cases of clonidine withdrawal

Pregnancy Considerations

- ***Preeclampsia:***
 - Definition: SBP > 140 or DBP > 90 mm Hg with proteinuria (> 300 mg/24 hr or a urine protein/creatinine > 0.3 or dipstick 1+)
 - Occurs > 20 wk gestation – 4 wk postpartum
 - Headache, vision changes, peripheral edema, RUQ pain
 - Complications: Eclampsia, HELLP
 - Goal: SBP 130–150 mm Hg and DBP 80–100 mm Hg
 - Drug of choice: Labetalol, nicardipine, hydralazine, magnesium
 - Consult Obstetrics
- ***Esmolol:***
 - β 1-blockade
 - Onset 60s, duration 10–20 min
 - Avoid in AHF, COPD, heart block
- ***Labetalol:***
 - Combined α - and β -blocker
 - Onset 2–5 min, duration 2–6 hr
 - No reflex tachycardia due to β -blockade
 - Avoid in: COPD, AHF, bradycardia
- ***Clevidipine:***
 - 3rd generation dihydropyridine CCB
 - Onset 2–4 min, duration 5–15 min
 - Elimination independent of liver/renal function
 - Avoid in allergies to soy or egg products, defective lipid metabolism, AFib
- ***Nicardipine:***
 - 2nd generation dihydropyridine CCB
 - Onset 5–15 min, duration 4–6 hr
 - Avoid in: AHF, coronary ischemia
- ***Nitroglycerin:***
 - Venous > arteriolar dilation
 - Onset 2–5 min, duration 10–20 min
 - Perfuses coronaries, decreasing ischemia
 - Causes reflex tachycardia, tachyphylaxis, methemoglobinemia
- ***Nitroprusside:***
 - Short-acting arterial and venous dilator
 - Onset 3 s, duration 1–2 min
 - Complications:
 - Reflex tachycardia, “coronary steal”, increase ICP
 - Cyanide toxicity after prolonged use
 - Avoid in pregnancy, renal failure (relative)

- Hydralazine:
 - Arteriolar dilator
 - Onset 5–15 min, duration 3–10 hr
 - Hypotensive effect may be less predictable
 - Safe in pregnancy
- Enalaprilat:
 - ACE inhibitor
 - Onset 0.5–4 hr, duration 6 hr
 - Avoid in: Pregnancy, AMI
- Fenoldopam:
 - Selective postsynaptic dopaminergic receptor agonist (DA1)
 - Onset 5–15 min, duration 1–4 hr
 - No reflex tachycardia
 - Maintains renal perfusion
 - Avoid in: Glaucoma
- Phentolamine:
 - α 1-blocker, peripheral vasodilator
 - Onset 1–2 min, duration 10–30 min

MEDICATION

- Clevidipine: 1–16 mg/h IV infusion
- Enalaprilat: 1.25–5 mg q6h IV bolus
- Esmolol: 80 mg IV bolus, then 150 μ g/kg/min infusion
- Fenoldopam: 0.1–0.6 μ g/kg/min IV infusion
- Hydralazine: 10–20 mg IV bolus
- Labetalol: 20–80 mg IV bolus q10min (total 300 mg); 0.5–2 mg/min IV infusion
- Nicardipine: 2–15 mg/h IV infusion
- Nitroglycerin: 5–100 μ g/min IV infusion; USE NON-PVC tubing
- Nitroprusside: 0.25–10 μ g/kg/min IV infusion
- Phentolamine: 5–15 mg q5–15min IV bolus



FOLLOW-UP

DISPOSITION

Admission Criteria

- All patients with end-organ damage
- ICU for cardiac and BP monitoring

Discharge Criteria

- Absence of end-organ damage
- Likely to be compliant with primary care

- Known history of HTN
- Reversible precipitating cause (e.g., medication noncompliance)
- Able to resume previous medication regimen
- Return with chest pain or headache

FOLLOW-UP RECOMMENDATIONS

Initiation of a suitable medication regimen under care of a primary care provider

PEARLS AND PITFALLS

- Avoid IV agents for hypertensive urgency
- BP goal in hypertensive emergency is a reduction of the MAP by 20–25% within the 1st hr except in ischemic CVA and aortic dissection
- Avoid excessive or precipitous decrease in BP because it may exacerbate end-organ damage
- Avoid reflex tachycardia in aortic dissection
- Avoid unopposed α in catecholamine excess

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See Also (Topic, Algorithm, Electronic Media Element)

- Acute Coronary Syndrome
- Acute Stroke
- Aortic Dissection
- Congestive Heart Failure
- Preeclampsia/Eclampsia
- Subarachnoid Hemorrhage



CODES

ICD9

- 401.9 Unspecified essential hypertension

- 437.2 Hypertensive encephalopathy

ICD10

- I10 Essential (primary) hypertension
- I67.4 Hypertensive encephalopathy

HYPERTHERMIA

Michelle J. Sergel • Emily Singer



BASICS

DESCRIPTION

- Range of progressively more severe illnesses due to increasingly overwhelming heat stress
- Begins with dehydration and electrolyte abnormalities and progresses to thermoregulatory dysfunction and multisystem organ failure
- Body temperature is maintained within a narrow range by balancing heat production with heat dissipation
- Oxidative phosphorylation becomes uncoupled and essential enzymes cease to function above 42°C (108°F)

Heat Stroke

- Core body temp > 105°F (40.5°C)
- Failure of thermoregulatory function leads to severe CNS dysfunction and multisystem organ failure
- Classic heat stroke (nonexertional)
 - Occurs in patients with compromised thermoregulation or an inability to remove themselves from a hot environment (e.g., extremes of age, debilitated)
 - Develops over days to weeks, usually during heat waves
 - Severe dehydration, skin warm and dry
- Exertional heat stroke
 - Younger, athletic patients with combined environmental and exertional heat stress (e.g., military recruits)
 - Develops over hours
 - Internal heat production overwhelms dissipating mechanisms, often despite persistent sweating

Heat Exhaustion

- Core temp moderately elevated but usually < 104°F (40°C)
- Fluid and/or salt depletion occurs secondary to heat stress
- Thermoregulatory function is maintained and CNS function is preserved
- Variable nonspecific symptoms including malaise, headache, fatigue, and nausea
- If left untreated, progresses to heat stroke

ETIOLOGY

- Pre-existing conditions that hinder the body's ability to dissipate heat predispose

for heat-related illness

- Age extremes
- Dehydration (incl. gastroenteritis, inadequate fluid intake)
- Cardiovascular disease (incl. CHF, CAD)
- Obesity
- Diabetes mellitus, hyperthyroidism, pheochromocytoma
- Febrile illness
- Skin diseases that hinder sweating (incl. psoriasis, eczema, cystic fibrosis, scleroderma)
- Pharmacologic contributors
 - Sympathomimetics
 - LSD, PCP, cocaine
 - MAO inhibitors, antipsychotics, anxiolytics
 - Anticholinergics
 - Antihistamines
 - β -blockers
 - Diuretics
 - Laxatives
 - Drug or alcohol withdrawal
- Environmental factors
 - Excessive heat/humidity
 - Prolonged exertion
 - Lack of mobility
 - Lack of air conditioning
 - Lack of acclimatization
 - Occlusive, nonporous clothing

Pediatric Considerations

Children are at increased risk of heat illness due to increased body surface area to mass ratio and lower sweat production

DIAGNOSIS

SIGNS AND SYMPTOMS

Heat Stroke

- Classic triad: Hyperthermia, CNS dysfunction, hot skin (often with anhidrosis)
- Core temp: $> 105^{\circ}\text{F}$ (40.5°C)
- CNS:
 - Severe confusion/delirium
 - Lethargy or coma
 - Seizure

- Ataxia
- CV:
 - Tachycardia
 - Wide pulse pressure
 - Low peripheral vascular resistance
 - Hypotension
 - Conduction disturbances
- Pulmonary:
 - Tachypnea
 - Rales due to noncardiac pulmonary edema
 - Respiratory alkalosis (may be substantial enough to cause tetany)
 - Hypoxemia (due to aspiration, pneumonitis, pulmonary edema, and high metabolic demand)
- GI:
 - Nausea/vomiting
 - Diarrhea
- Skin:
 - Cutaneous vasodilation → Hot skin
 - Usually dry, though sweating may be present if not dehydrated
- Acute oliguric renal failure due to dehydration +/- rhabdomyolysis
- Hepatic failure with elevation of transaminases in the tens of thousands
- Coagulopathy, including DIC (poor prognostic sign) → purpura, melena, hematochezia, hematuria, CNS hemorrhage

Heat Exhaustion

- Core temp moderately elevated, usually <104°F (40°C) and never >40.5°C
- CNS:
 - Frontal headache
 - Fatigue/malaise
 - Impaired judgment
 - Vertigo
 - Agitation
 - No severe CNS dysfunction
- CV:
 - Mild tachycardia
 - Dehydration
- Pulmonary: Tachypnea
- GI: Nausea, vomiting
- Skin: Perspiration present, often profuse

Heat Cramps

- Cramps in heavily worked muscles after exercise

- Occurs after profuse sweating and rehydration with hypotonic fluid (i.e., water)
- Results in hyponatremia and hypochloremia without rhabdomyolysis or renal damage
- Treat with oral salt solutions if minor or NS IV if severe

Heat Edema

- Swelling of feet/ankles from environmental heat in nonacclimatized people
- Due to vasodilatation and orthostatic pooling and increased aldosterone
- Resolves after acclimatization. Treatment with elevation or compression stockings.

Heat Syncope

- Unexplained syncope during heat exposure with prolonged standing, especially in elderly
- Cutaneous vessels dilate in an effort to dissipate heat → decreased central blood volume → syncope
- Self-limited illness. Resolves when the patient lays flat.

Prickly Heat

- Pruritic maculopapular/vesicular rash over clothed areas after profuse sweating in tight clothing
- Due to blockage of pores and secondary staphylococcus infection

ESSENTIAL WORKUP

- Accurate core temperature
- History of heat exposure
- Heat exhaustion is a diagnosis of exclusion
- Core temperature > 105°F (40.5°C) and CNS dysfunction required to make diagnosis of heat stroke

DIAGNOSIS TESTS & NTERPRETATION

Lab

For Heat Stroke and Heat Exhaustion

- CBC
 - Leukocytosis, hemoconcentration
- Electrolytes, BUN, Cr, glucose
 - Hypernatremia with severe dehydration
 - Hyponatremia can occur if drinking copious free water
 - Acute renal failure
- UA
 - Myoglobin present in rhabdomyolysis

- Blood and urine cultures to rule out septic etiology
- Toxicology screen
- Serum creatinine kinase to rule out rhabdomyolysis
- ABG
 - Acidosis is common with exertional heat stroke, and lactate is usually elevated

For Heat Stroke

- PT/PTT/DIC panel – coagulopathy implies poor prognosis
- Liver function tests
- Troponin I – poor prognosis if elevated
- Consider lumbar puncture to distinguish from meningitis/encephalitis

Imaging

- EKG in elderly or patients at cardiac risk
- CT head for altered mental status
- CXR for ARDS, aspiration pneumonia, and to rule out septic etiology

DIFFERENTIAL DIAGNOSIS

- Febrile illness/sepsis
- Thyroid storm
- Pheochromocytoma
- Cocaine/PCP
- Anticholinergics
- MAO inhibitors
- Meningitis/encephalitis
- Cerebral falciparum malaria
- Delirium tremens
- Neuroleptic malignant syndrome
- Malignant hyperthermia
- Serotonin syndrome



TREATMENT

PRE HOSPITAL

- Initiate cooling measures for severe heat illness
 - Remove from heat stress
 - Disrobe patient
 - Cover body with wet sheet

INITIAL STABILIZATION/THERAPY

- ABCs

- Continuous core temperature monitoring with a rectal or esophageal probe
- Rapid cooling if temperature $> 104^{\circ}\text{F}$ (40°C)
- Start with IV 0.9% NS 500 cc fluid bolus if hypotensive
- If altered mental status, administer glucose (or Accu-Chek), thiamine, naloxone

ED TREATMENT/PROCEDURES

Cooling Measures

- Initiate for body temperature $> 104^{\circ}\text{F}$ (40°C)
- Evaporative cooling
 - Extremely effective ($0.05\text{--}0.3^{\circ}\text{C}/\text{min}$)
 - Spray disrobed patient with fine mist of warm water (prevents shivering)
 - Airflow with fans blowing over patient
- Conductive cooling
 - Ice packs to groin/axilla. Combine with evaporative cooling treatment above
 - Iced or cold water immersion—effective but impractical
- Iced peritoneal lavage, cardiopulmonary bypass, or HD with cold dialysate for refractory cases – not well studied
- Stop cooling therapy at 102°F (39°C) to avoid overshooting and hypothermia
- Antipyretic agents are not helpful because underlying mechanism does not involve a change in the hypothalamus set point
- Avoid alcohol sponge baths. Toxicity can occur due to dilated cutaneous vessels.

Supportive Measures

- Rehydration for heat stroke/heat exhaustion
 - Initial rehydration with 0.5–1.0 L 0.9% NS
 - Aggressive fluid resuscitation until BP $> 90/60$ or central venous pressure (CVP) > 12 mL H_2O
 - Avoid overhydration, which can contribute to pulmonary edema and ARDS
 - Peds: Start with 20 cc/kg bolus
 - Place Foley catheter to monitor urine output for heat stroke victims and CVP monitor if feasible. Maintain UOP > 2 mL/kg/hr if rhabdomyolysis is present
 - Rehydrate to hemodynamic stability with NS then slowly administer free water if needed for correction of hypernatremia
- Benzodiazepines for seizure, agitation, or to stop shivering
- Tachyarrhythmias can develop, which usually resolve with cooling. Avoid electricity or α -adrenergics until after the myocardium is cooled
- Heat cramps: Analgesics and oral or IV hydration with electrolyte-containing fluid
- Heat edema: Lower extremity elevation + compression stockings
- Prickly heat: Chlorhexidine cream/lotion +/- salicylic acid 1% TID

MEDICATION

- Diazepam: 5–10 mg (peds: 0.2–0.4 mg/kg) IVP
- Lorazepam: 1–2 mg (peds 0.05–0.1 mg/kg) IVP
- Naloxone (Narcan): 2 mg (peds: 0.1 mg/kg) IVP



FOLLOW-UP

Admission Criteria

- Heat stroke – admit to the ICU
- Heat exhaustion – admit to general or monitored floor if:
 - Severe electrolyte abnormalities
 - Renal failure or evidence of rhabdomyolysis
 - Elderly

Discharge Criteria

All patients except those with heat stroke or severe heat exhaustion may be discharged

PEARLS AND PITFALLS

- Cannot make diagnosis of heat stroke without temp $> 40.5^{\circ}\text{C}$ and severe CNS dysfunction.
- Management of heat stroke requires management of ABCs and rapid cooling.
- Continuous core monitoring with a rectal or esophageal probe is standard of care.
- Evaporative cooling is the cooling method of choice.

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CODES

ICD9

- 992.0 Heat stroke and sunstroke
- 992.2 Heat cramps
- 992.5 Heat exhaustion, unspecified

ICD10

- T67.0XXA Heatstroke and sunstroke, initial encounter
- T67.2XXA Heat cramp, initial encounter
- T67.5XXA Heat exhaustion, unspecified, initial encounter

HYPERTHYROIDISM

Rita K. Cydulka • Christopher S. Campbell



BASICS

DESCRIPTION

- Excessive thyroid hormone production results in a continuum of disease caused by both the direct physiologic effect of thyroid hormones as well as increased catecholamine sensitivity:
 - Subclinical or mild hyperthyroidism
 - Thyrotoxicosis
 - Thyroid storm or thyrotoxic crisis with life-threatening manifestations:
 - 1–2% of patients with hyperthyroidism
- Regulation of thyroid hormone:
 - Thyrotropin-releasing hormone (TRH) from hypothalamus acts on the anterior pituitary
 - Thyroid stimulating hormone (TSH) released by anterior pituitary gland and results in increased T_3 and T_4 from the thyroid gland:
 - Most of circulating hormone is T_4 , which is peripherally converted to T_3
 - T_3 is much more biologically active than T_4 although it has a shorter half-life
- Genetics:
 - Interplay between genetics and environment
 - Graves disease is associated with HLA-B8 and HLA-DR3
 - Autosomal dominant inheritance seen in some families with nontoxic goiter

ETIOLOGY

- Primary hyperthyroidism:
 - Toxic diffuse goiter (Graves disease)
 - Toxic multinodular (Plummer disease) or uninodular goiter
 - Excessive iodine intake (Jod-Basedow disease)
- Thyroiditis:
 - Postpartum thyroiditis
 - Radiation thyroiditis
 - Subacute thyroiditis (de Quervain)
 - Chronic thyroiditis (Hashimoto/lymphocytic)
- Metastatic thyroid cancer
- Ectopic thyroid tissue (struma ovarii)
- Pituitary adenoma

- Drug induced:
 - Amiodarone
 - Lithium
 - α -interferon
 - Interleukin-2
 - Iodine (radiographic contrast agents)
 - Excessive thyroid hormone (factitious thyrotoxicosis)
 - Aspirin overdose

DIAGNOSIS

ALERT

Thyroid storm is a life-threatening condition, which may be precipitated by:

- Infection
- Trauma
- Diabetic ketoacidosis
- Organophosphate intoxication
- Cytotoxic chemotherapy
- Myocardial infarction
- Cerebrovascular accident
- Surgery
- Abrupt withdrawal of antithyroid medication or acute ingestion of thyroid medication

SIGNS AND SYMPTOMS

- Signs and symptoms reflect end-organ responsiveness to thyroid hormone:
 - Signs:
 - Fever
 - Tachycardia, wide pulse pressure
 - Diaphoresis/sweating
 - Congestive heart failure (CHF)
 - Shock
 - Tremor
 - Disorientation/psychosis
 - Goiter/thyromegaly
 - Thyrotoxic stare/exophthalmos/lid lag
 - Hyperreflexia
 - Pretibial myxedema
 - Symptoms:
 - Weight loss despite increased appetite
 - Dysphagia or dyspnea secondary to obstruction by a goiter
 - Rash/pruritus/hyperhidrosis
 - Palpitations/chest pain

- Diarrhea and vomiting
- Myalgias and weakness
- Nervousness/anxiety
- Menstrual irregularities
- Heat intolerance
- Insomnia and fatigue
- Thyroid storm involves exaggerated signs and symptoms of thyrotoxicosis:
 - Extreme tachycardia/dysrhythmias
 - CHF
 - Shock
 - Disorientation and mental status changes including coma and seizure
 - Thromboembolic events

Geriatric Considerations

Apathetic hyperthyroidism:

- Owing to multinodular goiter, often have history of nontoxic goiter
- Subtle clinical findings that often reflect single-organ system dysfunction:
 - CHF
 - Refractory atrial fibrillation (AFib)
 - Weight loss
 - Depression, emotional lability, flat affect
 - Tremor
 - Hyperactivity

History

Gradual onset of aforementioned signs and symptoms

Physical-Exam

- Vital signs:
 - Fever
 - Tachycardia
 - Elevation of systolic blood pressure
 - Widened pulse pressure
 - Tachypnea/hypoxia
- Alopecia
- Exophthalmos or lid lag
- Thyromegaly or goiter, thyroid bruit
- Fine, thin, diaphoretic skin
- Irregularly irregular heartbeat
- Lung rales (CHF)
- Right upper quadrant tenderness/jaundice
- Muscular atrophy/weakness

- Tremor
- Mental status changes/coma

ESSENTIAL WORKUP

- Find underlying cause/precipitating factors.
- Plasma TSH is the initial ED test of choice:
 - _ Normal level usually rules out hyperthyroidism:
 - TSH may be low with normal T₄. Get T₃ level to rule out T₃ thyrotoxicosis
 - _ If TSH levels unavailable, clinical suspicion should prompt initiation of therapy

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Thyroid function tests for:
 - _ Symptoms of hyperthyroidism
 - _ Elderly patient with new-onset CHF
 - _ New AFib/supraventricular tachycardia (SVT)
- TSH (usually decreased)
- Free T₄ (usually elevated):
 - _ If free T₄ is unavailable, total T₄ and resin T₃ uptake
 - _ 5% will have T₃ thyrotoxicosis, if low TSH with normal T₄, send T₃ to rule out
- Lab studies are often not helpful/nonspecific, get as needed to look for underlying precipitants:
 - _ CBC to rule out anemia
 - _ Chemistry panel:
 - BUN, creatinine may be elevated secondary to dehydration
 - Hypokalemia, hyperglycemia
- Liver function tests (increased transaminases)
- ABG for hypoxemia and acidosis
- Cardiac markers

Imaging

CXR (in CHF or sepsis)

Diagnostic Procedures/Surgery

EKG:

- Most commonly sinus tachycardia
- Rule out MI as precipitant of thyroid storm

- New-onset AFib

DIFFERENTIAL DIAGNOSIS

- Pheochromocytoma
- Sepsis
- Sympathomimetic ingestion
- Psychosis
- Heat stroke
- Delirium tremens
- Malignant hyperthermia
- Neuroleptic malignant syndrome
- Hypothalamic stroke
- Hypothyroidism (may mimic apathetic hyperthyroidism)
- Factitious thyrotoxicosis



TREATMENT

PRE HOSPITAL

Stabilization and supportive care

INITIAL STABILIZATION/THERAPY

- Airway, breathing, and circulation management
- Cardiac monitor
- Supplemental oxygen
- IV fluids
- Initiate cooling measures:
 - _ Acetaminophen for fever:
 - Avoid aspirin (displaces thyroid hormone from thyroglobulin, elevates free T₄)
 - _ Cooling blanket

ED TREATMENT/PROCEDURES

- Identify and treat the precipitating event
- For thyroid storm, initiate treatment sequence outlined below based on clinical suspicion
- Inhibit hormone synthesis using thioamides:
 - _ Propylthiouracil (PTU):
 - Drug of choice
 - Decreases hormone synthesis and reduces peripheral conversion of T₄
 - _ Methimazole (MMI)
- Block hormone release using iodine *only after hormone synthesis is inhibited as above*:
 - _ Oral Lugol solution (saturated potassium iodide solution), or

- Iopanoic acid (Telepaque)
- Give iodine at least 1 hr after thioamides to prevent increased hormone production
- Consider lithium in patient allergic to iodine
- Block peripheral effects of thyroid hormone:
 - β -blockade:
 - Propranolol is first line as it also inhibits T_4 conversion to T_3
 - Esmolol, β -1 selective so may be used in patient with active CHF, asthma, etc.
 - Reserpine, guanethidine
 - Albumin solution
 - Cholestyramine to reduce enteric reabsorption of thyroid hormone
- Dexamethasone/hydrocortisone:
 - Prevents peripheral T_4 to T_3 conversion
- Treatment of thyrotoxicosis, secondary thyroiditis:
 - β -blockade
 - Anti-inflammatory medications
- General thyrotoxicosis support:
 - Acetaminophen for hyperpyrexia
 - Treat CHF with usual methods
 - Manage dehydration with 10% dextrose solution (D 10) to restore depleted hepatic glycogen
- Identify and treat associated and underlying conditions (infection, ketoacidosis, pulmonary thromboembolism, stroke, etc.)

MEDICATION

- Cholestyramine: 4 g PO QID
- Dexamethasone: 2 mg IV q6h (peds: 0.15 mg/kg q6h)
- Esmolol: 500 μ g/kg IV over 1 min followed by 50 μ g/kg/min IV; titrate to effect
- Guanethidine: 30–40 mg PO q6h for 1–3 days
- Hydrocortisone: 100 mg IV initially, followed by 100 mg IV q8h for first 24–36 hr
- Iopanoic acid: 1 g IV q8h for first 24 hr, then 500 mg IV BID
- Lithium carbonate: 300 mg PO QID (peds: 15–60 mg/kg/d div. TID–QID)
- Lugol solution: 5 drops (250 mg) PO q6h
- MMI: 60–80 mg/d PO (peds: 0.4 mg/kg) (peds: 0.2 mg/kg/d) in 3 div. doses
- Propranolol: 0.5–1 mg IV + subsequent 2–3 mg doses over 10–15 min q several hours, *or* 60–80 mg PO q4h
- PTU: 100–150 mg PO q8h initially then 200–250 mg PO q4h (peds: 5–7 mg/kg/d in 3 div. doses)
- Reserpine: 1–5 mg IM, then 0.07–0.3 mg/kg in the 1st 24 hr

First Line

- PTU
- Propranolol
- Iodine therapy (Lugol), 1 hr after PTU

Second Line

- MMI
- Esmolol
- Lithium (only with iodine allergy)
- Guanethidine (for patients with bronchospasm), reserpine

Pregnancy Considerations

- Physiologic changes associated with pregnancy may resemble many symptoms of hyperthyroidism
- Poorly controlled hyperthyroidism during pregnancy may result in:
 - Hyperemesis gravidarum
 - Premature labor
 - Preeclampsia
 - Low birth weight
 - Spontaneous abortion
 - Stillbirth
- Thyroid storm often precipitated by stressors including infection, labor, birth
- Treatment:
 - Initial stabilization as in the nonpregnant patient (ABCs, supportive measures)
 - PTU considered safer than MMI. Both cross the placenta. PTU should be \leq 200 mg/day
 - Propranolol may be safely used
 - Radioactive iodine absolutely contraindicated when pregnant or nursing
 - Thyroidectomy is the only other option if unable to tolerate PTU while pregnant
- Postpartum thyroiditis:
 - 5–10% of patients within 6 mo of delivery
 - May require antithyroid medications
 - 50% affected become euthyroid within 1 yr
 - Transient hypothyroidism may follow



FOLLOW-UP

DISPOSITION

Admission Criteria

- Thyroid storm

- Requiring IV medications to control heart rate
- Significantly symptomatic or unstable patients

Discharge Criteria

Minimal symptoms that respond well to PO therapy

FOLLOW-UP RECOMMENDATIONS

- Should have PCP follow-up within a few weeks depending on symptoms
- May benefit from endocrinology referral

PEARLS AND PITFALLS

- Thyroid storm can be fatal. Diagnosis requires a high level of suspicion and treatment often needs to be started presumptively
- Radioactive iodine is never a treatment option in the pregnant patient with hyperthyroidism
- Never give iodine before blocking hormone synthesis with PTU or MMI in thyroid storm

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Hypothyroidism



CODES

ICD9

- 242.20 Toxic multinodular goiter without mention of thyrotoxic crisis or storm
- 242.90 Thyrotoxicosis without mention of goiter or other cause, and without mention of thyrotoxic crisis or storm
- 242.91 Thyrotoxicosis without mention of goiter or other cause, with mention of thyrotoxic crisis or storm

ICD10

- E05.01 Thyrotoxicosis w diffuse goiter w thyrotoxic crisis or storm
- E05.20 Thyrotoxicosis w toxic multinod goiter w/o thyrotoxic crisis
- E05.90 Thyrotoxicosis, unspecified without thyrotoxic crisis or storm

HYPERVENTILATION SYNDROME

Robert F. McCormack

BASICS

DESCRIPTION

- Hyperventilation syndrome describes a constellation of symptoms:
 - Most commonly: Dyspnea, chest pain, lightheadedness, and paresthesias
- Produced by a nonphysiologic increase in minute ventilation:
 - Minute ventilation may be increased by increasing respiratory rate or tidal volume (sighs).
- Pathologic or physiologic causes of hyperventilation must be excluded before the diagnosis of hyperventilation syndrome can be assigned.
- Prevalence:
 - 10–15% in the general population
 - More common in women (may be related to progesterone)

ETIOLOGY

- Etiology of symptoms is unclear:
 - Usually a response to psychological stressors
- Controversy exists regarding underlying disorders that may contribute to hyperventilation:
 - Hypocapnia
 - Hypophosphatemia
 - Hypocalcemia

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Past episodes
 - Duration
 - Triggers
 - Past treatment
 - Typical time point of onset during the day
- Cardiac:
 - Chest pain
 - Dyspnea
 - “Air hunger”
 - Palpitations

- Neurologic:
 - Dizziness
 - Lightheadedness
 - Syncope
 - Paresthesias
 - Headache
 - Carpopedal spasm
 - Tetany
- Psychiatric:
 - Intense fear, anxiety
 - Giddiness
 - Feeling of unreality
- General:
 - Fatigue
 - Weakness
 - Malaise

Physical-Exam

- Clinical signs are rare and varied:
 - Tachypnea most common
 - However, tachypnea may not be present. Patient may increase tidal volume rather than respiratory rate.
- Carpopedal spasm:
 - May be dramatic
- Chvostek sign may be present

ESSENTIAL WORKUP

- Diagnosis of exclusion:
 - Primary pathologic or physiologic causes of hyperventilation must be investigated and excluded.
- Clinical diagnosis based on the history and physical exam
- Vital signs including pulse oximetry
- Hyperventilation syndrome will not result in hypoxia.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Consider an ABG in any hypoxic patient.
- Electrolytes, BUN, creatinine, and glucose levels for suspected acidosis/diabetic ketoacidosis
- EKG if chest pain present

Imaging

CXR of any patient with hypoxia or focal findings on lung exam

Diagnostic Procedures/Surgery

- Hyperventilation provocation test after resolution of symptoms:
 - Forced overbreathing for 3 min may be attempted to reproduce the symptoms.
 - Diagnostic accuracy is controversial.
 - Reproducibility of the symptoms may help the patient understand the role of overbreathing and help manage future attacks.

DIFFERENTIAL DIAGNOSIS

- Pathologic
- Hypoxia:
 - Asthma
 - CHF
 - Pulmonary embolus
 - Pneumonia
- Severe pain
- CNS lesions
- Acidosis (DKA)
- Pulmonary HTN
- Pulmonary embolus
- Hypoglycemia
- Mild asthma
- Drugs:
 - Aspirin intoxication
 - Withdrawal syndrome (e.g., alcohol, benzodiazepines)
- Physiologic
- Pregnancy
- Pyrexia
- Altitude



TREATMENT

PRE HOSPITAL

- Patients with abnormal vital signs require IV access and pulse oximetry.
- Supplemental oxygen if hypoxic

INITIAL STABILIZATION/THERAPY

- Patients with abnormal vital signs require IV access and pulse oximetry.
- Initiate therapy for pathologic or physiologic cause of hyperventilation.

ED TREATMENT/PROCEDURES

- Initiate treatment of hyperventilation syndrome if initial workup does not support a pathologic or physiologic cause, and history and physical exam findings suggest the diagnosis of hyperventilation syndrome.
- Reassurance, calming, and explanation of the voluntary component of the patient's symptoms often have immediate dramatic results.
- Do not use paper bag rebreathing to increase the PCO₂. This has not been supported in the literature:
 - It may be dangerous in patients with hypoxia or a pathologic or physiologic cause for hyperventilation.
- Clarification of the psychological stressors helps the patient avoid further attacks.
- Assess for need of psychiatric evaluation (i.e., suicidal ideation).
- Anxiolytics:
 - Benzodiazepine if symptoms persist to break the cycle of anxiety and hyperventilation
 - Short course of anxiolytics may benefit patients with definable temporary stressors.

MEDICATION

- Alprazolam 0.25–0.5 mg PO
- Lorazepam: 1–2 mg PO or IV
- Diazepam: 2–5 mg PO or IV
- Outpatient treatment:
 - Buspirone: 5 mg PO TID
 - Diazepam: 2–5 mg PO BID–QID



FOLLOW-UP

DISPOSITION

Admission Criteria

Hyperventilation syndrome does not require admission.

Discharge Criteria

- Exclusion or successful treatment of primary pathologic or physiologic causes of hyperventilation
- No acute psychiatric issues
- Adequate follow-up with a primary care physician

FOLLOW-UP RECOMMENDATIONS

- Follow-up with primary care physician
- Assess the need for psychiatric follow-up.

PEARLS AND PITFALLS

- Exclude pathologic or physiologic causes of hyperventilation.
- Hyperventilation syndrome will not result in hypoxia.

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- Saisch SG, Wessely S, Gardner WN. Patients with acute hyperventilation presenting to an inner-city emergency department. *Chest*. 1996;110(4):952–957.



CODES

ICD9
306.1 Respiratory malfunction arising from mental factors

ICD10

F45.8 Other somatoform disorders

HYPERVISCOSITY SYNDROME

Matthew B. Mostofi

BASICS

DESCRIPTION

- Hyperviscosity syndrome (HVS) is the clinical consequence of increased blood viscosity.
- The classic clinical symptoms are the triad of mucosal bleeding, visual disturbances, and neurologic signs.
- Viscosity is the resistance a material has to change in form.
- The higher the blood viscosity, the more the internal resistance to blood flows.
- Increased cardiac output is required to provide adequate perfusion of hyperviscous blood.
- Oxygen delivery is impaired as transit through the microcirculatory system slows. This impaired microcirculatory oxygenation gives rise to the clinical symptoms of this syndrome.

ETIOLOGY

- Hyperviscosity occurs when there is elevation of either the cellular or acellular components of circulating blood.
- Acellular (protein) hyperviscosity:
 - The most common cause (85–90%) of hyperviscosity is increased concentration of γ globulins:
 - Monoclonal gammopathies: From malignant diseases like Waldenstrom macroglobulinemia and multiple myeloma
 - Polyclonal gammopathies: Usually rheumatic diseases (very rare)
- Cellular (blood cell) hyperviscosity:
 - Much less common (10–15%)
 - Increased numbers of RBC, as in polycythemia vera
 - Increased concentration ($> 100,000$) of WBC, as in acute and chronic leukemia
 - Thrombocytosis

DIAGNOSIS

SIGNS AND SYMPTOMS

- Classic triad:
 - Mucosal bleeding
 - Visual disturbances
 - Neurologic

- Hematologic:
 - Bleeding is the most common manifestation. Mechanism thought to be platelet dysfunction.
 - Epistaxis
 - Gingival, rectal, uterine bleeding
 - Prolonged postprocedural bleeding
 - Blood dyscrasias
 - Pruritus owing to red cell breakdown products
 - Splenic enlargement
- Ocular:
 - Change in visual acuity:
 - Blurring
 - Diplopia
 - Visual loss
 - Characteristic “link-sausage effect” on funduscopy
 - Alternating bulges and constrictions within the retinal veins
 - Retinal hemorrhage, detachment
 - Exudate, microaneurysm formation
 - Papilledema
- Renal:
 - Nephritic or nephrotic syndrome
 - Hematuria
 - Sterile pyuria
- Neurologic:
 - Headache
 - Ataxia
 - Mental status changes/coma
 - Dizziness/vertigo
 - Nystagmus
 - Tinnitus, hearing loss
 - Paresthesia, peripheral neuropathy
 - Seizure
 - Intracranial hemorrhage
- Cardiovascular:
 - Angina or myocardial infarction
 - Dysrhythmias
 - CHF
- Dermatologic:
 - Raynaud phenomenon
 - Livedo reticularis
 - Palpable purpura
 - Eruptive spider nevus–like lesions
 - Digital infarcts

- Peripheral gangrene

History

HVS should be considered in the following patient:

- Any patient presenting with the classic symptom triad of bleeding, visual disturbance, and neurologic dysfunction.
- Any patient with an established immunoglobulin-producing hematologic disease that presents with signs or symptoms of microvascular end-organ damage or cardiac decompensation.
- Any patient with an established hypercellular hematologic disease who presents with signs or symptoms of microvascular end-organ damage or cardiac decompensation.

Physical-Exam

There are no specific physical exam findings unique to HVS. However, patient will exhibit findings based on the affected end organs. Mucosal bleeding, petechial rash or bruising, focal neurologic findings, signs of decompensated heart failure, and fundoscopic abnormalities have all been reported.

ESSENTIAL WORKUP

- Evaluate end-organ ischemia and bleeding.
- Measure serum or whole blood viscosity.
- Suspect diagnosis if the lab evaluation is hampered by serum stasis and increased viscosity causing analyzer blockage

DIAGNOSIS TESTS & NTERPRETATION

Lab

- CBC with WBC differential:
 - Anemia or erythrocytosis can be seen in HVS.
 - Anemia usually normocytic and normochromic
 - Rouleaux of erythrocytes on the peripheral smear is an important diagnostic clue
 - WBC for leukemia
- Electrolyte, BUN, creatinine, and glucose levels:
 - Renal dysfunction is commonly noted in HVS.
 - Hypercalcemia and pseudohyponatremia in multiple myeloma
- Urinalysis:
 - Proteinuria
 - Hematuria
 - Sterile pyuria
- Coagulation profile
- Serum and urine protein electrophoresis

- Measurement of serum viscosity (not routinely available in ED setting):
 - _ Ostwald viscosimeter
 - _ Normal range for the serum viscosity relative to water is 1.4–1.8.
 - _ Minimal viscosity at which symptoms develop is 4 centipoise (cp).
- Elevated leukocyte alkaline phosphatase, lactate dehydrogenase, and serum vitamin B₁₂ levels

Imaging

One should consider CT of head in patients with signs or symptoms of central neurologic dysfunction to exclude intracranial hemorrhage.

DIFFERENTIAL DIAGNOSIS

- Bleeding and clotting disorders:
 - _ Platelet disorders (qualitative and quantitative)
 - _ Hereditary factor deficiencies
 - _ Acquired disorders (vitamin K deficiency, liver disease)
 - _ Disseminated intravascular coagulation



TREATMENT

PRE HOSPITAL

IV fluid resuscitation with hemorrhage

INITIAL STABILIZATION/THERAPY

- Rehydrate with 0.9% NS IV fluid.
- Bleeding or end-organ ischemia may not be controlled by any treatment except plasmapheresis.
- In patients with anemia and a leukemic picture, avoid blood transfusion until plasmapheresis is performed to avoid exacerbation of HVS.

ED TREATMENT/PROCEDURES

- Hydration, supportive care, and early hematologist consultation are initial ED management.
- Phlebotomy or emergent plasma exchange: This temporizing measure can be performed in a patient with HVS and severe neurologic findings like coma or seizures:
 - _ Easily performed in the ED and is useful in acute severe cases if plasmapheresis not readily available
 - _ Simply draw off (100–200 mL) of whole blood and replace volume with isotonic saline.
 - _ Should be performed in consultation with hematologist when possible.
 - _ Treatment of choice in patients with polycythemia vera.

- Plasmapheresis/leukapheresis:
 - In stable patients: 40 mL/kg of body weight
 - In critical patients: 60 mL/kg of body weight
 - Side effects include hypocalcemia with use of a citrate-containing anticoagulant and dysrhythmia (rare).
 - Many patients require more than 1 plasmapheresis.
 - Definitive treatment for HVS. Should be performed in consultation with plasmapheresis/hematology team.
 - Leukapheresis is reserved as the initial treatment in patients with hyperleukocytosis (usually WBC > 100,000)
- ED physician can help in urgent situations by establishing or facilitating the establishment of large-bore central dialysis catheter, caution should be taken to avoid bleeding complications of this procedure



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients with hyperviscosity and significant symptoms or any evidence of end-organ ischemia or hemorrhage should be admitted for treatment of the underlying hematologic disorder.
- ICU admission for the following:
 - Hemorrhage
 - Altered mental status
 - Acute MI

Discharge Criteria

Discharge after definitive treatment of the underlying disorder.

Issues for Referral

All patients with HVS should be referred to hematologist.

PEARLS AND PITFALLS

- Avoid diuretics in patients with HVS because they can increase blood viscosity.
- The classic triad of symptoms of HVS includes visual disturbances, bleeding, and neurologic manifestations.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Disseminated Intravascular Coagulation



CODES

ICD9

- 273.3 Macroglobulinemia
- 289.0 Polycythemia, secondary

ICD10

- C88.0 Waldenstrom macroglobulinemia
- D75.1 Secondary polycythemia

HYPHEMA

Jamil D. Bayram • Sami H. Uwaydat

BASICS

DESCRIPTION

- Blood in anterior chamber (AC) of the eye (between iris and cornea).
- Hyphema: Grossly visible layering of blood.
- Microhyphema: Suspended RBCs visible by slit-lamp only.
- Genetics:
 - Genetic predisposition is related to hereditary blood dyscrasias (see below).

ETIOLOGY

- Blunt trauma: Most common (70–80%).
- Anteroposterior compression of the globe with simultaneous equatorial globe expansion causing rupture of iris stromal/ciliary body vessels
- Penetrating trauma: Direct injury to stromal vessels or sudden ocular decompression.
- Spontaneous: Less common, lower incidence of complications:
 - Tumors:
 - Melanoma
 - Retinoblastoma
 - Xanthogranuloma
 - Metastatic tumors
 - Blood dyscrasias:
 - Hemophilia
 - Leukemia
 - Thrombocytopenia
 - Von Willebrand disease
 - Blood thinners: Aspirin, Coumadin, heparin
 - Neovascularization of iris: In proliferative diabetic retinopathy, retinal vein occlusion, carotid stenosis.
 - Postsurgical: Cataract extraction, trabeculectomy, pars plana vitrectomy.

ALERT

In children with no history of trauma, suspect child abuse.

DIAGNOSIS

SIGNS AND SYMPTOMS

- Photophobia

- Blurring of vision
- Decreased visual acuity
- Ocular pain
- Nausea/vomiting

History

- Previous visual acuity
- Prior eye surgery
- Prior glaucoma treatment.
- Past medical history (blood disorders including sickle cell disease).
- Mechanism of trauma.
- Exact time of injury and of visual loss.
- History of excessive tearing after injury.

ALERT

History of excessive tearing may indicate open globe injury.

Physical-Exam

- General physical exam with emphasis on associated bodily injuries.
- Periorbital ecchymosis
- Eyelid lacerations
- Enophthalmos (depression of the globe within the orbit)
- Limited ocular movement with diplopia (may indicate orbital floor fracture)
- Proptosis (may indicate retro-orbital hemorrhage)
- Ocular exam:
 - Visual acuity
 - Rule out open globe (positive Seidel sign, corneal laceration, diffuse subconjunctival hemorrhage, decreased ocular motility, prolapse of intraocular structures)
 - Pupillary reaction to light (check for afferent pupillary defect prior to using dilating drops)
 - Tonometry for intraocular pressure (IOP) measurement

ALERT

Exclude open globe injury before measuring IOP

- Slit-lamp exam; look for layer of blood in AC:
 - 4 grades of hyphema depending on percentage of AC occlusion by blood:
 - Grade I: $< 1/3$
 - Grade II: $1/3-1/2$
 - Grade III: $> 1/2$
 - Grade IV: Total (called 8-ball hyphema; blood is dark and filling 100% of AC)
 - High-grade hyphemas are:

- More likely to rebleed (25% of grade I compared with 67% of grade III)
- More likely to develop glaucoma and corneal staining
- Less likely to recover visual acuity
- _ Dilated fundus exam (avoid pressure on globe)

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Lab tests should be individualized depending on the case.
- Platelet count, PT/PTT, bleeding time if bleeding disorder is suspected, or if the patient is on anticoagulants.
- BUN, creatinine, and pregnancy test if aminocaproic acid is to be used (see below)
- Factor VIII assay if family history of hemophilia
- Sickle cell screen especially in African Americans and Mediterranean descent

Imaging

- CT orbits (1 mm cuts) if open globe injury, intraocular foreign body, or orbital wall fracture is suspected
- US biomicroscopy (B scan) if total hyphema and intraocular structures cannot be visualized.

ALERT

Do not perform B scan if open globe injury is suspected (pressure applied during this procedure may cause extrusion of intraocular contents).

ESSENTIAL WORKUP

- Exam: Visual acuity, status of globe, IOP, associated ocular/bodily injuries
- Labs: Platelet count, PT/PTT, and sickle cell screen if indicated
- Imaging: B scan or CT orbits if indicated

DIFFERENTIAL DIAGNOSIS

- Uveitis
- Endophthalmitis



TREATMENT

PRE HOSPITAL

Place eye shield in case of corneal perforation or suspected open globe injury.

INITIAL STABILIZATION/THERAPY

- Keep head upright to allow blood in AC to settle down.
- Limit activity; avoid bending, straining, or exertion.

- Place metal or plastic shield over involved eye until integrity of globe is confirmed.
- Do not patch affected eye (if eye is patched, patient cannot notice sudden loss of vision).
- Note that metal and plastic shields have holes that let patient see through whereas patch completely blocks patient's vision.

ED TREATMENT/PROCEDURES

- Mild analgesics (avoid NSAIDs because of antiplatelet effect)
- Antiemetics (associated N/V may worsen hyphema by increasing IOP)
- Cycloplegics decrease pain from iritis:
 - Atropine 1% or cyclopentolate 1% eye drops: BID–TID until hyphema resolves.
- Topical steroids may decrease inflammation from iritis:
 - Prednisolone acetate: 1% eye drops (or equivalent) 4–8 times per day until hyphema resolves (usually 7–10 days).
- Aminocaproic acid (antifibrinolytic):
 - Use in consultation with ophthalmologist:
 - Not commonly used because of frequent systemic side effects.
 - Stabilizes fibrin clot in AC and decreases incidence of rebleed, but has no effect on final visual outcome.
 - 50 mg/kg PO q4h for total of 5 days (do not exceed 30 g/day). Dose should be adjusted in renal failure.
 - May cause postural hypotension, nausea, vomiting, diarrhea.
 - New topical form is not yet FDA approved.
 - Do not use in pregnant women or in patients prone to thrombosis. It can also cause acute renal failure in patients with hemophilia.
- Oral prednisone:
 - Indications:
 - Hemophilia
 - Uncooperative children
 - Total hyphema
 - History of thrombotic disease
 - Dose: 0.6–0.75 mg/kg/24 hr in div. doses, up to 60 mg/day for 5 days
- For increased IOP:
 - For non-sickle cell patients, treat if IOP > 30 mm Hg.
 - For sickle cell patients, treat if IOP > 24 mm Hg.
 - Treat until IOP is controlled as indicated above.
 - Always start with 1 medication. Add another if unsuccessful in controlling pressure:
 - β -blockers—drug of choice: Timolol or Levobunolol 0.5% BID
 - α -agonist: Brimonidine 0.2% or apraclonidine 0.5% TID
 - Topical carbonic anhydrase inhibitors (CAI): Dorzolamide 2% or

brinzolamide 1% TID

- Oral CAI: Acetazolamide 500 mg PO q12h (peds: 8–30 mg/kg/24 hr q6–8h) or methazolamide 50 mg q8h.
- Mannitol (1–2 mg/kg IV over 45 min q24h) when all other eye drops fail to lower IOP to acceptable level.
- Avoid CAI and Mannitol in sickle cell patients, as they may cause acidosis and induce sickling.

– Allow 25–30 min for each eye drop to work. If after using all the drops and mannitol, IOP is still high, then surgical evacuation of blood clot is warranted (AC tap or washout).

- Drugs to avoid:

- Pilocarpine: Constricts pupil and prevents visualization of lens and retina
- Prostaglandin eye drops (e.g., Latanoprost): Increase inflammatory response

ALERT

Criteria for immediate consultation with ophthalmologist from the ED (If possible, consultation should be arranged within 24 hr.):

- Visual acuity worse than 20/200 at presentation.
- Sickle cell disease/trait with high IOP
- Large hyphema (filling >1/3 of AC).
- Medically uncontrolled IOP.
- Suspected open globe injury

MEDICATION

First Line

- Atropine: 1% TID
- Prednisolone acetate: 1% QID

Second Line

- Timolol 0.5% or levobunolol 0.5% BID
- Brimonidine 0.2% or apraclonidine 0.5% TID
- Dorzolamide 2% or brinzolamide 1% TID
- Acetazolamide: 500 mg PO q12h.



FOLLOW-UP

DISPOSITION

- Discharge patient on atropine, prednisolone, and any appropriate IOP-lowering medications.

- Continue aminocaproic acid if decision was made to start it in ED.
- Antiemetics if needed.
- Stool softeners to minimize straining during bowel movements.

Admission Criteria

- Hyphema size is not a criterion for discharge or admission; IOP control is the most important factor.
- Medically uncontrolled IOP requiring surgical intervention
- Ruptured globe
- Noncompliant patients
- Associated ocular or orbital injuries
- Children <7 yr of age:
 - Age group is usually at risk of amblyopia (also called lazy eye, which is irreversible visual loss secondary to visual deprivation in early childhood).
- Patients at risk of complications (sickle cell disease, hemophilia).

Discharge Criteria

Absence of any admission criteria with IOP <30 mm Hg for non-sickle patients and <24 mm Hg for patients with sickle cell disease/trait.

FOLLOW-UP RECOMMENDATIONS

Arrange for follow-up with ophthalmologist:

- Daily slit-lamp exam for 3 days after initial trauma to monitor for rebleeding, corneal staining, and increased IOP.
- Follow-up exam will determine length of treatment with atropine, prednisone acetate, and IOP-lowering eye drops.

PEARLS AND PITFALLS

- Rule out ruptured globe prior to checking IOP and prior to initiating treatment.
- IOP control is not immediate. Allow at least 30 min for any treatment to take effect:
 - Always check for sickle cell disease in African Americans.

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See Also (Topic, Algorithm, Electronic Media Element)

- Endophthalmitis
- Globe Rupture
- Uveitis
- Vitreous Hemorrhage



CODES

ICD9

- 364.41 Hyphema of iris and ciliary body
- 921.3 Contusion of eyeball

ICD10

- H21.00 Hyphema, unspecified eye
- S05.10XA Contusion of eyeball and orbital tissues, unsp eye, init
- S05.12XA Contusion of eyeball and orbital tissues, left eye, init

HYPOCALCEMIA

Michelle J. Sergel • Damali N. Nakitende



BASICS

DESCRIPTION

- Hypocalcemia is defined as a total plasma calcium level < 8.7 mg/dL:
 - Ionized calcium may be normal and, therefore, have no clinical manifestations.
- Normal total serum calcium concentrations are 8.7–10.5 mg/dL.

ETIOLOGY

- Incidence in the general population is 0.6%.
- Mechanism:
 - From either increased loss of calcium from the circulation or decreased entry into the circulation
 - Intravascular calcium circulates in 3 forms:
 - Bound to proteins (mainly albumin): 45–50%
 - Bound to complexing ions (citrate, phosphate, carbonate): 5–10%
 - Ionized (free) calcium (physiologically active form): 45–50%
 - Serum levels of calcium are primarily controlled by 3 hormones:
 - Parathyroid hormone (PTH)
 - Decrease in calcium levels leads to an increase in PTH secretion (increasing bone resorption, renal absorption, intestinal absorption, urinary phosphate excretion).
 - Vitamin D (1,25-dihydroxyvitamin D):
 - Decrease in calcium level activates vitamin D (increasing bone resorption and intestinal absorption).
 - Calcitonin:
 - Causes a direct inhibition of bone resorption with increased calcium levels
- Hypoalbuminemia—the most common cause:
 - Each 1 g/dL decrease in serum albumin decreases protein-bound serum calcium by 0.8 mg/dL.
 - Ionized (free) calcium levels do not change.

Pediatric Considerations

- Children have higher values of normal calcium (9.2–11 mg/dL).
- Neonatal hypocalcemia: Total serum calcium concentrations < 7.5 mg/dL or serum-ionized calcium levels < 4 mg/dL
- Symptoms of hypocalcemia in infancy:

- Hyperactivity, jitteriness
- Tachypnea
- Apneic spells with cyanosis
- Vomiting



DIAGNOSIS

SIGNS AND SYMPTOMS

- Occur when ionized calcium < 3.2 mg/dL
- Dependent upon absolute calcium concentration and rate at which it falls
- Neuromuscular:
 - Paresthesias
 - Hyperreflexia
 - Muscle spasm
 - Tetany:
 - Neuromuscular irritability
 - Uncommon unless ionized calcium < 4.3 mg/dL
 - Latent tetany
 - Chvostek sign (finger taps of parotid gland over the facial nerve causes facial muscle spasm)
 - Trousseau signs (an inflated blood pressure cuff over the arm causes carpopedal spasm)
 - Laryngeal stridor
 - Seizures
 - Choreoathetosis
- Cardiovascular:
 - Dysrhythmias:
 - Torsades de pointes
 - Heart block
 - Hypotension
 - Impaired contractility (heart failure)
 - ECG changes:
 - Bradycardia
 - QT and ST prolongation
 - T-wave abnormalities
- Psychiatric:
 - Irritability/anxiety
 - Psychosis
 - Depression
 - Confusion
 - Delusions
 - Chorea

- Parkinsonisms
- Ocular:
 - Papilledema
 - Cataracts
 - May occur in patients with acute onset hypocalcemia

ESSENTIAL WORKUP

Serum-ionized calcium level confirms the diagnosis

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Arterial blood gas:
 - Change from normal pH of 0.1 U equals a reciprocal change in ionized calcium of ~ 1.7 mg/dL.
- Serum albumin
- Electrolytes, BUN/creatinine, glucose
- Magnesium
- Phosphate:
 - Increase in phosphate associated with hypoparathyroidism
 - Decrease in phosphate associated with vitamin D deficiency
- PTH:
 - Very high levels of PTH associated with pseudohypoparathyroidism
 - High levels of PTH associated with vitamin D deficiency
 - Low levels of PTH associated with hypoparathyroidism
- Serum calcidiol or calcitriol

Diagnostic Procedures/Surgery

ECG:

- Prolonged QT interval
- Heart block

DIFFERENTIAL DIAGNOSIS

- Impaired PTH action or secretion:
 - Parathyroid or thyroid surgery or radical neck surgery and/or irradiation for head and neck cancer
 - Autoimmune disease (typically presents in childhood)
 - Congenital hypoparathyroidism
 - Neonatal secondary to maternal hyperparathyroidism
 - Pseudohypoparathyroidism (resistance to PTH)
 - Infiltrative (amyloidosis, sarcoidosis, metastases, iron overload)
 - HIV infection
- Impaired vitamin D synthesis or action:

- Nutritional malabsorption or poor intake
- Renal disease
- Pronounced hypophosphatemia
- Sepsis or severe burns:
 - Impaired secretion of PTH and calcitriol
 - End-organ resistance to the action of PTH
- Calcium complex formation or sequestration:
 - Hyperphosphatemia
 - Ethylene glycol, ethylenediaminetetraacetic acid (EDTA), citrate (from transfusion)
 - Pancreatitis, rhabdomyolysis
 - Alkalosis (i.e., hyperventilation)
- Hypomagnesemia:
 - Causes end-organ PTH resistance
 - Decreased PTH secretion
 - Seen in chronic and/or critical illness
 - Must give magnesium to correct hypocalcemia
- Medications:
 - Mithramycin, plicamycin, phosphate, calcitonin, bisphosphonates
 - Phenobarbital, phenytoin
 - Cisplatin
 - Cadmium, colchicine
 - Fluoride, citrate, PPI
- Malignancies:
 - Prostate cancer
 - Breast cancer
 - Lung cancer
 - Chondrosarcoma
- “Hungry bone syndrome”:
 - After parathyroid removal
 - Rapid accretion of calcium as bone is remineralized



TREATMENT

INITIAL STABILIZATION/THERAPY

ABCs:

- Establish IV catheter access.
- Cardiac monitor

ED TREATMENT/PROCEDURES

- Acute management:
 - Treat symptomatic hypocalcemia as a medical emergency with parenteral

calcium administration.

- Calcium IV bolus:
 - Calcium gluconate 1–2 g in 50 mL of 5% dextrose
 - Infuse over 20 min
 - Faster IV rates can cause cardiac dysrhythmias
 - Calcium salts are irritating to veins.
 - IM calcium gluceptate or calcium gluconate if IV access not available
 - Bolus dose increases ionized calcium for only 1–2 hr, therefore, must be followed by an infusion
- Calcium infusion:
 - Calcium infusion rate: 0.5–1.5 mg/kg/hr
 - Do not mix with bicarbonate or phosphate or precipitation of salts may form.
 - Administer cautiously in patients taking digitalis—may initiate and exacerbate digitalis toxicity
- Response to therapy:
 - Individual responses vary.
 - Monitor calcium concentrations q1–4h during therapy.
 - Titrate treatment to symptoms or ECG changes.
 - Consider hypomagnesemia if the patient fails to respond to calcium therapy—correct hypomagnesemia with Mg 2 g IVPB 10% solution over 10 min
 - In the setting of acidosis, correct calcium 1st; alkalosis will further reduce ionized calcium.
 - Side effects of IV calcium include: Nausea, vomiting, hypotension, and dysrhythmias
- Chronic management:
 - Oral calcium supplementation
 - 1.5–2 g/day of Calcium in div. doses. May need up to 4 g/day in patients with malabsorption.
 - Vitamin D:
 - Enhances intestinal absorption
 - Initiate with calcium supplementation—alone not sufficient to restore calcium levels.
 - 600 IU for ages 19–50 yr
 - 600 IU for ages 51–70 yr
 - 800 IU for ages 71 and older
 - Multivitamins contain variable amounts of vitamin D
 - Vitamin D preparations:
 - Ergocalciferol: 125 µg/day
 - Dihydroxycholesterol: 100–400 µg/day
 - Calcifediol: 50–200 µg/day
 - Calcitriol: 0.25–0.5 µg/day: Rapid onset (preferred). Most active

Pregnancy Considerations

Calcitriol requirements may double or triple toward the end of pregnancy.

MEDICATION

- IV calcium:
 - Calcium chloride: 1 g in 10 mL (1 g = 360 mg [13.6 mEq] elemental calcium)
 - Calcium gluceptate (IV/IM): 1 g in 5 mL (1 g = 90 mg [4.5 mEq] elemental calcium)
 - Calcium gluconate: 1 g in 10 mL (1 g = 90 mg [4.5 mEq] elemental calcium)
- Oral calcium:
 - Calcium carbonate: 350–1,500 mg tablets (1 g = 400 mg)
 - Calcium citrate: 950 mg tablets (1 g = 211 mg elemental calcium)
 - Calcium glubionate: 18 g/5 mL of syrup (1 g = 65 mg elemental calcium)
 - Calcium gluconate: 500—1,000 mg tablets (1 g = 90 mg elemental calcium)
 - Calcium lactate: 350–1,000 mg tablets (1 g = 130 mg elemental calcium)

Pediatric Considerations

- Initial calcium bolus with 10% calcium gluconate should be 9–18 mg of elemental calcium/kg or 1–2 mL/kg not to exceed 5 mL in premature infants or 10 mL in term infants.
- Calcitriol dose in children ranges from 0.1–3 µg/day.
- MISCELLANEOUS:
 - Calcium content of common foods:
 - Milk or yogurt, 8 oz = 300 mg
 - Cheddar cheese, 1 oz = 200 mg
 - Calcium-fortified cereal, 1 cup = 300 mg
 - Calcium-fortified orange juice, 1 cup = 270 mg
 - Shrimp, 3 oz = 50 mg
 - Peanuts = 130 mg
 - Orange = 50 mg



FOLLOW-UP

DISPOSITION

Admission Criteria

- Symptomatic or severe ionized hypocalcemia (<3.2 mg/dL)
- Continuous IV calcium preparations necessary to maintain calcium levels

Discharge Criteria

- Asymptomatic hypocalcemia
- Ionized calcium > 3.2 mg/dL in healthy patients with no comorbid illness

FOLLOW-UP RECOMMENDATIONS

Close follow-up with an endocrinologist may be necessary for impaired PTH or vitamin D action or synthesis.

PEARLS AND PITFALLS

- Hypocalcemia has many causes
- Treatment of hypocalcemia varies with its severity and underlying cause
- Patients who are severely symptomatic require rapid correction with IV calcium therapy
- To effectively treat hypocalcemia with concurrent magnesium deficiency, magnesium must first be normalized

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Hypercalcemia
- Hyperparathyroidism
- Hypoparathyroidism



CODES

ICD9

- 775.4 Hypocalcemia and hypomagnesemia of newborn
- 275.41 Hypocalcemia
- 275.5 Hungry bone syndrome

ICD10

- E83.51 Hypocalcemia
- E83.81 Hungry bone syndrome
- P71.1 Other neonatal hypocalcemia

HYPOGLYCEMIA

Chadi I. Kahwaji • Matthew N. Graber



BASICS

DESCRIPTION

- Deficiency in counterregulatory hormones (glucagon, epinephrine, cortisol, growth hormone) or excessive insulin response
- Serum glucose < 70 mg/dL

RISK FACTORS

- Strict glycemic control with insulin
- Prior hypoglycemia episodes
- Hypoglycemia unawareness
- Decreased counterregulation
- <5 years of age or elderly
- Comorbid conditions:
 - Renal disease
 - Malnutrition
 - Coronary artery disease
 - Liver disease

Genetics

- Congenital metabolic and endocrine disorders that decrease gluconeogenic ability (e.g., hereditary fructose intolerance)
- Congenital hyperinsulinism
- Neonatal diabetes mellitus (often a mutation effecting an ATP-dependent potassium channel)

ETIOLOGY

- Increased insulin levels:
 - Overdose of oral hypoglycemic agent or insulin
 - Oral antihyperglycemics (i.e., α -glucosidase inhibitors, biguanides, and thiazolidinediones) do not cause hypoglycemia alone, but may enhance the risk when used with insulin or sulfonylureas.
 - Sepsis
 - Insulinoma
 - Autoimmune hypoglycemia
 - Alimentary hyperinsulinism
 - Renal failure (partially responsible for insulin metabolism)
 - Liver cirrhosis (responsible for significant insulin metabolism)

- Underproduction of glucose:
 - Alcohol (inhibitory effect on glycogen storage and gluconeogenesis)
 - Drugs
 - Salicylates
 - β -blockers (including eye drops)
 - SSRIs
 - Some antibiotics (e.g., sulfonyleureas, pentamidine)
 - Adrenal insufficiency
 - Malnutrition
 - Dehydration
- Cerebral edema
- Extremes of age
- Congestive heart failure
- Counterregulatory hormone deficiency
- Hypothyroidism or hyperthyroidism

Pregnancy Considerations

- 3rd-trimester pregnant patients risk relative substrate deficiency–induced hypoglycemia.
- The fetus is less likely to become hypoglycemic during mother’s hypoglycemic episode secondary to active glucose transport across placenta:
 - Oral hypoglycemic use in pregnancy may lead to profound and prolonged neonatal hypoglycemia.

Pediatric Considerations

Most common cause of hypoglycemia in the 1st 3 mo of life is persistent hyperinsulinemic hypoglycemia of infancy (PHHI) in mothers with diabetes.

DIAGNOSIS

SIGNS AND SYMPTOMS

- Adrenergic caused by excessive counter-regulatory hormones (i.e., epinephrine):
 - Diaphoresis
 - Anxiety
 - Tachycardia/palpitations
 - Hunger
 - Paresthesias
 - Chest pain
 - Ischemic ECG changes
- Neuroglycopenic:
 - CVA mimic
 - Any focal or general neurologic change

- Dizziness
- Confusion
- Mood changes
- Hyperactive or psychotic behavior
- Slurred speech
- Cranial nerve palsies
- Seizures
- Hemiplegia
- Decerebrate posturing
- Neonatal presentation:
 - Asymptomatic
 - Limp
 - Bradycardia
 - Irritable
 - Tremulous
 - Seizures
 - Poor feeding

ALERT

Patients with “hypoglycemia unawareness” have reduced warning signals, do not recognize that their blood sugar is low, and instead may present with only late findings such as seizure, focal neurologic findings, altered mental status, and coma.

History

- Underlying diseases or conditions: Diabetes, renal failure, liver failure, alcohol use.
- Certain medications—long-acting insulin and oral hypoglycemic agents—are more concerning.
- Possible insulin or oral hypoglycemic overdose.

Physical-Exam

See Signs and Symptoms

ESSENTIAL WORKUP

- Diagnosis requires:
 - Demonstration of neuroglycopenic signs and symptoms as defined above
 - Lab evidence of hypoglycemia
 - Clearing of symptoms following glucose administration

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Blood glucose (initial and post-treatment)
- Electrolytes, BUN, creatinine

- Prothrombin time
- Urinalysis for possible infection
- Urine and other cultures as appropriate in evaluation for infection
- C-peptide if concern for exogenous insulin overdose

Imaging

CXR for:

- Possible aspiration during hypoglycemic episode
- Pneumonia as source of sepsis

Diagnostic Procedures/Surgery

- ECG if MI/ischemia owing to hypoglycemia or as cause of hypoglycemia suspected.
- Hypoglycemia may affect cardiac electrical conduction.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis is extensive; see Altered Mental Status for a complete list.

Major concerns include:

- Neurologic:
 - Cerebral vascular accident/transient ischemic attack (CVA/TIA)
 - Seizure disorder
- Drug or alcohol intoxication
- Hypoxia
- Sepsis
- Metabolic derangements
- Endocrine derangements
- Environmental stressors
- Psychosis, depression, or anxiety

Pediatric Considerations

- Growth hormone deficiency
- Inborn errors of metabolism
- Ketotic hypoglycemia
- Reye syndrome
- Salicylate ingestion



TREATMENT

PRE HOSPITAL

- Diagnosis with finger stick glucose
- IV dextrose preferred
- Oral glucose-containing fluids in awake patient if unable to obtain IV

- Glucagon if unable to give IV glucose or oral glucose

INITIAL STABILIZATION/THERAPY

- ABCs with aspiration and seizure precautions
- Glucose:
 - Dextrose IV push (IVP)—this should always be given if possible.
 - Oral glucose in awake patient (with no IV) without risk of aspiration
 - Glucagon IM if unable to establish IV access

ED TREATMENT/PROCEDURES

- Administer D₅₀W 50 mL for decreased level of consciousness:
 - 2nd or 3rd amp may be necessary.
 - Complications include volume overload and hypokalemia.
- Administer octreotide:
 - If hypoglycemia refractory to glucose administration
 - If hypoglycemia secondary to sulfonylureas
 - Initiate continuous IV infusion of 5–20% glucose solution for persistent mild hypoglycemia or if patient cannot eat.
- Administer glucagon:
 - If hypoglycemia refractory to glucose
 - If IV access delayed
 - Ineffective in alcohol-induced hypoglycemia and significant liver disease
 - May repeat twice q20–30min
 - Administer hydrocortisone with glucagon for adrenal insufficiency.
 - Effective in 10–20 min

Geriatric Considerations

Elderly patients often have less hypoglycemic awareness and require significant time for resolution of symptoms, even after appropriate treatment of hypoglycemia.

MEDICATION

First Line

- D₅₀W: 1–2 amps (25 g) of 50% dextrose IVP
 - Zimmerman rule of 50: Adult 1 mL/kg of D₅₀W; child: 2 mL/kg D₂₅W; infants: 5 mL/kg D₁₀W)

Second Line

- Octreotide: 50 µg IV bolus then 50 µg IV/hr drip or 50 µg q12hSC/IV
- Glucagon: 0.5–2 mg IV/IM/SC:
 - Child: 0.03–0.1 mg/kg IV/IM/SC
 - Infant: 0.3 mg/kg IV/IM/SC

- May repeat in 4 hr
- Hydrocortisone: 100 mg (peds: 1–2 mg/kg) IV
- Oral glucose: 20 g orally equals ~12 oz nondiet fruit juice, 14 oz nondiet cola
 - Carbohydrate without fat or protein preferred



FOLLOW-UP

DISPOSITION

Admission Criteria

- Overdose of long-acting oral hypoglycemic agent (e.g., sulfonylureas) or long-acting insulin mandate observation for at least 24 hr.
- Failure of neuroglycopenic symptoms to improve with glucose administration suggests neurologic injury, pre-existing neurologic condition, or another cause for these symptoms.
- Recurrent hypoglycemic state in ED
- Patients unable to tolerate oral fluids or food
- Suicidal intentions
- Older patients may require several days for complete recovery from severe or prolonged hypoglycemia.

Discharge Criteria

- Discharge mild unintentional insulin overusage or failure to take oral calories if blood glucose normal, symptoms resolved, tolerating oral intake, and can be observed.
- Families of patient with recurrent hypoglycemia should be instructed in IM glucagon administration.
- Monitor blood glucose for at least 3 hr prior to discharge.

Issues for Referral

Refer to primary physician for consideration of medication or diet changes if recurrent hypoglycemic episodes.

FOLLOW-UP RECOMMENDATIONS

PMD follow-up for medication re-evaluation within 48 hr

PEARLS AND PITFALLS

- Administration of PO glucose or food may initially further decrease glucose level; therefore, IV dextrose always preferred if possible
- Multiple amps of D₅₀W commonly required
- Do not over rely on D10/D20 as even these concentrations contain relatively small

amounts of glucose.

- Hypoglycemia should be in the differential for all neurologic and psychiatric presentations.
- Recurrent hypoglycemia patients often require hours to days for full neurologic recovery

ADDITIONAL READING

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- Stanley CA, Baker L. The causes of neonatal hypoglycemia. *N Engl J Med.* 1999;340:1200–1201.



See Also (Topic, Algorithm, Electronic Media Element)

- Altered
- Mental
- Status



CODES

ICD9

- 251.1 Other specified hypoglycemia
- 251.2 Hypoglycemia, unspecified
- 775.1 Neonatal diabetes mellitus

ICD10

- E16.1 Other hypoglycemia
- E16.2 Hypoglycemia, unspecified
- P70.2 Neonatal diabetes mellitus

HYPOGLYCEMIC AGENT POISONING

Timothy J. Meehan



BASICS

DESCRIPTION

- Oral or parenteral agents that may cause hypoglycemia or other metabolic imbalances
- Hypoglycemic poisoning may be intentional or unintentional (accidental)

ETIOLOGY

- Insulin:
 - Enhances glucose uptake into cells
 - Limits glucose availability to the brain (most sensitive to hypoglycemia)
 - Influences potassium redistribution (hypokalemia)
- Sulfonylurea and Meglitinide agents:
 - Enhance insulin release from pancreatic β cells, reduce hepatic glucose production, and increase peripheral insulin sensitivity
 - Hypoglycemic effect enhanced by:
 - Polypharmacy (drug interactions)
 - Alcohol use and hepatic dysfunction (poor nutritional stores)
 - Renal insufficiency (decreased clearance)
- GLP1 modulators:
 - Exenatide is an analog of glucagon-like peptide 1 (GLP1)
 - Gliptins (sitagliptin and saxagliptin) inhibit DDP4 which normally inactivates GLP1
 - Net effects: Enhanced insulin secretion, delayed gastric emptying, and increased satiety
 - Unclear effects on glucose metabolism in overdose (data are lacking at this time)
- Biguanide agents (metformin):
 - Antihyperglycemic agents:
 - Decrease elevated serum glucose concentrations
 - Generally do not cause hypoglycemia in isolation.
 - In the presence of insulin, biguanides do the following:
 - Increase glucose uptake into cells
 - Limit glucose availability to the brain (most sensitive to hypoglycemia)
 - Influence potassium redistribution (hypokalemia)
 - Decrease GI glucose absorption
 - Decrease hepatic gluconeogenesis

- Metabolize glucose to lactate in intestinal cells, which may accumulate and lead to profound lactic acidosis
- Thiazolidinediones:
 - In the presence of insulin, thiazolidinediones increase glucose uptake and use and decrease gluconeogenesis
- α -glucosidase inhibitors:
 - Lower systemic glucose by decreasing GI absorption of carbohydrates

DIAGNOSIS

SIGNS AND SYMPTOMS

- Insulin or sulfonylureas:
 - Overdose causes hypoglycemia
 - Symptoms most often occur when glucose < 40–60 mg/dL (may occur at higher levels in diabetics)
 - Symptoms blunted by β -antagonists
 - Facial flushing, diaphoresis, pallor, piloerection
 - Hunger, nausea, abdominal cramping
 - Labored respirations, apnea
 - Headache, blurred vision
 - Paresthesias, weakness, incoordination, tremor
 - Anxiety, irritability, bizarre behavior, confusion, stupor, coma, seizures
 - Palpitations, tachycardia, bradycardia (late)
 - Hypertension
 - Hypothermia
- Biguanides:
 - Toxicity primarily owing to lactic acid accumulation
 - Nausea, vomiting, abdominal pain
 - Agitation, confusion, lethargy, coma
 - Kussmaul respirations
 - Hypotension, tachycardia

Pediatric Considerations

- Neonatal hypoglycemia may occur after maternal use of sulfonylureas during labor
- Ingestion of 1 sulfonylurea tablet may cause hypoglycemia in a child:
 - Death has been reported after ingestion of a single tablet
- Onset of symptomatic hypoglycemia may be delayed up to 8 hr

History

- Diagnosis of diabetes in patient
- Access to diabetic medications:
 - If occurring in a medical setting (hospital, nursing home), consider:

- Dosing error
- Malicious intent

Physical-Exam

- Vital signs:
 - Tachycardia (may be blunted if on β -blockers)
- Neurologic:
 - Confusion, obtundation, coma
 - Ataxia, other cerebellar signs

ESSENTIAL WORKUP

- Diagnosis based on clinical presentation and an accurate history
- Monitor serum glucose concentration
- Monitor vital signs and neurologic status
- Obtain serum electrolytes and lactate for biguanide ingestion
- Obtain liver function tests for thiazolidinedione ingestion

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Serum glucose before and after treatment
- Electrolytes:
 - Check for hypokalemia
 - Anion gap acidosis
- BUN, creatinine:
 - May reveal renal insufficiency, causing drug accumulation
- CBC
- Ethanol level
- Lactate level (especially if biguanide medications involved)
- Liver function tests
- Arterial blood gas
- Assays for immunoreactive insulin and C-peptide levels:
 - Confirms administration of exogenous insulin if insulin level is high and C-peptide is low in the setting of hypoglycemia
 - Do not correlate with severity of clinical symptoms

Imaging

- ECG: Sinus tachycardia, premature ventricular contractions (PVCs), atrial dysrhythmias
- EEG: Diffuse slowing without focal abnormalities
- CT scan: Cerebral edema if prolonged hypoglycemia
- Chest radiograph: Aspiration pneumonia or pulmonary edema

DIFFERENTIAL DIAGNOSIS

- Addison disease
- Panhypopituitarism
- Sepsis
- Insulinoma
- Neuroendocrine tumors
- Cirrhosis
- Chronic ethanol abuse
- Ethanol ingestion
- Salicylate ingestion
- β -antagonist ingestion
- Ackee fruit poisoning



TREATMENT

PRE HOSPITAL

Transport all medications, pills, and pill bottles involved in overdose for identification in ED

INITIAL STABILIZATION/THERAPY

- ABCs:
 - Airway control essential
 - Administer supplemental oxygen
 - IV access
 - Cardiac monitor and pulse oximetry
- Naloxone, thiamine, D₅₀ (or Accu-Chek) if altered mental status

ED TREATMENT/PROCEDURES

- Hypoglycemia:
 - D₅₀ bolus, then:
 - IV infusion D₅W or D₁₀W to maintain euglycemia or mild hyperglycemia
 - Food (if mental status improves or normalizes)
- Neuroglycopenia:
 - May persist shortly after serum glucose corrected
 - Persistent symptoms require further dextrose administration
- Decontamination:
 - Consider administration of activated charcoal for recent or large ingestion of oral agent (sulfonylurea or biguanide)
- Provide supportive care
- Hypotension:
 - 0.9% NS IV fluid bolus

- Pressor support with dopamine or norepinephrine as needed:
 - Pressors may increase lactate production
 - Use cautiously with biguanide-induced lactic acidosis
- Administer sodium bicarbonate for biguanide-induced lactic acidosis if pH < 7
- Administer benzodiazepines for seizures
- Inhibit insulin secretion for sulfonylurea overdose with recurrent hypoglycemia with:
 - Octreotide
 - Diazoxide (watch for hypotension)
- Early hemodialysis may be beneficial in cases of biguanide-induced lactic acidosis:
 - Corrects acid–base abnormalities
 - Enhances elimination of the drug

MEDICATION

- Activated charcoal: 1 g/kg PO
- Dextrose: 50–100 mL D₅₀ (peds: 2 mL/kg of D₂₅ over 1 min) IV; repeat if necessary
- Diazepam: 5–10 mg (peds: 0.2–0.5 mg/kg) IV q10–15min
- Diazoxide: 200 mg PO or 1–3 mg/kg IV (infant: 8–15 mg/kg/24 h q8–12hPO/IV; child: 3–8 mg/kg/24 h q8h PO/IV)
- Glucagon: 1–2 mg (peds: 0.03–0.1 mg/kg) IM/SC/IV
- Lorazepam: 2–4 mg (peds: 0.03–0.05 mg/kg) IV q10–15min
- Octreotide: 50–100 µg q8–12h SC/IV
- Thiamine (vitamin B₁): 100 mg (peds: 50 mg) IV or IM



FOLLOW-UP

DISPOSITION

Admission Criteria

- Hypoglycemia owing to sulfonylurea agents (may require several days of monitoring) or long-acting insulin preparations
- Any patient requiring a constant infusion of dextrose to maintain euglycemia
- Intentional overdose or self-injection of insulin warrants admission for 24 hr glucose monitoring
- All children with accidental ingestion of sulfonylureas
- Metabolic alterations owing to biguanide ingestion or accumulation

Discharge Criteria

- Accidental hypoglycemia owing to short-acting insulin injection in the setting of dietary insufficiency:
 - Must be tolerating oral intake
 - Ensure return to baseline mental status

- Discharge after glucose correction and a 4 hr period of observation

Issues for Referral

- Patients with unintentional (accidental) poisoning require poison prevention counseling
- Patients with intentional (e.g., suicide) poisoning require psychiatric evaluation

FOLLOW-UP RECOMMENDATIONS

Close primary care follow-up to help monitor blood sugar and adjust medication dosages

PEARLS AND PITFALLS

- Sulfonylureas can have markedly prolonged half-lives and long elimination times:
 - Delayed hypoglycemia and refractory hypoglycemia are common
 - Admit for observation, at a minimum
- Metformin must be held for 48 hr after any study requiring IV contrast media:
 - IV contrast can prolong renal clearance of biguanides
 - Can induce metformin-associated lactic acidosis

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See Also (Topic, Algorithm, Electronic Media Element)

Hypoglycemia



CODES

ICD9

962.3 Poisoning by insulins and antidiabetic agents

ICD10

T38.3X1A Poisoning by insulin and oral hypoglycemic drugs, acc, init

HYPOKALEMIA

David N. Zull



BASICS

DESCRIPTION

- Defined as a serum potassium level < 3.5 mEq/L:
 - _ Mild: 3–3.5 mEq/L
 - _ Moderate: 2.5–3 mEq/L
 - _ Severe: < 2.5 mEq/L
- Frequency:
 - _ Up to 20% of inpatients have documented hypokalemia (5% have levels < 3 mEq/L).
 - _ Up to 14% of outpatients are mildly hypokalemic (most are related to diuretics or GI loss).
 - _ 5% of geriatric patients have $K < 3$ mEq/L.
- Potassium is the major intracellular cation:
 - _ Gradient is maintained by Na–K ATPase activity (enhanced by insulin and β -agonists) and mineralocorticoids.
- Total body potassium is ~ 55 mEq/kg of body weight (98% ICF, 2% ECF).
- Electrophysiologic effects of hypokalemia:
 - _ Increase in the normal intracellular to extracellular potassium gradient:
 - Alters the depolarization threshold for muscles and nerves
 - Inhibits the termination of action potentials
 - _ Alterations in intracellular potassium directly affect cellular function.

ETIOLOGY

Renal Losses

- Diuretics (thiazides, loop diuretics, carbonic anhydrase inhibitors), usually associated with loss of other cations (Mg^{2+} , Ca^{2+} , P^{3+} , Na^{+})
- Renal tubular damage:
 - _ Primary renal tubular disorders (RTA type I and II)
 - _ Interstitial nephritis, analgesic nephropathy, drug toxicity (amphotericin, gentamicin, toluene, cisplatin), myeloma kidney
 - _ Overdose toxicity: Acetaminophen, NSAIDs, hydroxychloroquine
- Hyperaldosteronism:
 - _ Primary (primary hyperaldosteronism, Cushing, pituitary tumor-producing ACTH, congenital adrenal hyperplasia)
 - _ Secondary (volume depletion, CHF, cirrhosis, nephrotic)
 - _ Exogenous (steroids; fludrocortisone, glycyrrhizic acid [licorice]) hyperrenin

state in renal artery stenosis

- Hypomagnesemia (increased secretion)
- Polyuria:
 - Osmotic diuresis (mannitol, hyperglycemia)
 - Psychogenic polydipsia
- Congenital disorders:
 - Bartter and Gitelman syndromes—hypokalemic metabolic alkalosis and low BP
 - Liddle syndrome is the same but with hypertension.
- Delivery of nonreabsorbable anions such that sodium is reabsorbed and potassium is exchanged out and excreted:
 - Bicarbonate in metabolic alkalosis
 - β -hydroxybutyrate in DKA
 - Hippurate in toluene abuse
 - Penicillins—high dose IV therapy

GI Losses

- Diarrhea:
 - Proportional to volume and duration
 - Villous adenomas
 - Laxative abuse
- Vomiting and nasogastric suction result in volume depletion and metabolic alkalosis, which increases renal losses of potassium from bicarbonaturia and hyperaldosteronism.
- Ureterosigmoidostomy
- Intestinal fistulae, ileostomy
- Cystic fibrosis

Intracellular Shift of Potassium

- Alkalosis (metabolic or respiratory)
- Insulin:
 - Insulin administration
 - Stimulation of insulin release by IV glucose or massive sweetened beverage intake
 - Refeeding in prolonged starvation
- Adrenergic excess:
 - Severe stress (trauma, MI, sepsis)
 - Treatment of asthma (frequent β -agonists and theophylline toxicity)
 - Cocaine, amphetamines, caffeine excess
 - Dobutamine, dopamine, pseudoephedrine
- Hypokalemic periodic paralysis:
 - Familial

- Thyrotoxic
- B₁₂ administration in severely deficient patient
- Hypothermia
- Drugs: GM-CSF, quetiapine, risperidone

Poor Intake (Rare as a Sole Cause)

- Nutritional (poverty, pica, dementia)
- Eating disorders
- Dental problems/oral lesions
- Esophageal disease



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Neuromuscular:
 - Severe weakness (K < 2.5 mEq/L):
 - Begins in the lower extremities and progresses cephalad
 - May progress to paralysis if K < 2 mEq/L and rapid development
 - Muscle cramps, tetany, and tenderness
 - Rhabdomyolysis
 - Paresthesias
 - Generalized fatigue and malaise
- GI:
 - Constipation
 - Ileus
- Cardiovascular (heart disease increases risk):
 - Ventricular and atrial premature beats
 - AV block, atrial or junctional tachycardias
 - Ventricular tachycardia (VT) or fibrillation
 - Potentiation of digoxin toxicity
- Renal:
 - Impaired urinary concentrating ability resistant to ADH (polyuria, polydipsia)
 - Increased renal bicarbonate reabsorption and ammonia production (worsens alkalosis)

Physical-Exam

- HTN—renal artery stenosis, primary hyperaldosteronism, licorice, congenital adrenal hyperplasia, Liddle syndrome, glucocorticoid use
- Hypotension—GI losses, diuretic use, Bartter and Gitelman syndromes

- Neuromuscular—muscle weakness, decreased reflexes, muscle tenderness

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Electrolytes, BUN, creatinine, glucose:
 - High HCO_3 suggests diuretic abuse, vomiting, mineralocorticoid excess, Bartter, Gitelman.
 - Low HCO_3 suggests renal tubular disease or diarrhea
 - Low serum sodium suggests diuretic use or marked volume depletion from GI losses
 - High serum sodium suggests nephrogenic diabetes insipidus or primary hyperaldosteronism
- Urine K (spot sample):
 - < 20 mEq/L suggests GI loss, potassium shift into cells, poor intake.
 - > 20 mEq/L suggests renal loss.
- Urine K to creatinine ratio is more precise
 - < 13 mEq/g or 1.5 mEq/mmol (nonrenal)
 - > 13 mEq/g or > 1.5 mEq/mmol (renal loss)
- Urine Na:
 - < 20 mEq/L with elevated urine K suggests secondary hyperaldosteronism.
- Plasma renin if hypertensive:
 - High renin: Secondary hyperaldosteronism, renal artery stenosis
 - Low renin: Primary hyperaldosteronism
- TSH and free T4 if Asian male

ECG Findings

- Low-voltage T-waves
- Sagging of the ST segments
- U-waves:
 - In severe hypokalemia, the T disappears and the U-wave predominates, giving the illusion of dramatic QT prolongation.
- Diminutive P-waves (appears nodal)
- Dysrhythmias (very prevalent if underlying cardiomyopathy or digoxin toxic):
 - Atrial: Premature atrial contractions (PACs), atrial fibrillation (Afib)
 - Ventricular: Premature ventricular contractions (PVCs), VT, torsade

DIFFERENTIAL DIAGNOSIS

- Intrinsic cardiac disease with dysrhythmias
- Causes of muscular weakness:
 - Neuromuscular junction disease (myasthenia gravis, organophosphate poisoning, botulism)

- Spinal cord disease
- Polyneuropathies
- Primary acute myopathies
- Cataplexy



TREATMENT

INITIAL STABILIZATION/THERAPY

- Establish IV access/volume resuscitation
- ABCs
- Cardiac monitoring

ED TREATMENT/PROCEDURES

- Total body deficit is 200–300 mEq per 1 mEq/L decrement in serum potassium level.
- Rate of replacement and route dependent on presence of symptoms, severity of hypokalemia, and comorbidities.
- Complete replacement over several days
- Oral potassium preferable to IV therapy whenever possible
- Identify and prevent ongoing K losses:
 - Hold diuretics or laxatives
 - Treat vomiting or diarrhea
 - Minimize nasogastric suction losses by administering H2 blockers or PPIs
 - Avoid glucose-containing fluids

MEDICATION

- Oral potassium chloride:
 - Preferred replacement in almost all cases
 - Liquid (or powder dissolved in water or juice) is more bioavailable, but nausea may occur:
 - 10–40 mEq per dose
 - Rapid rise in K, but will drop after 4 hr from transcellular shift
 - Tablets (wax matrix and microencapsulated):
 - More palatable, more sustained effect
 - Slowly absorbed
 - Potential for small bowel ulceration.
 - Dosage for hypokalemia:
 - Mild to moderate: 10–20 mEq q6–12h
 - Moderate to severe: 40–60 mEq q8–12h
 - Continue until K remains 3–3.5 mEq/L
- Oral potassium gluconate or citrate:
 - Use in acidotic patients (e.g., RTA)

- Ineffective if accompanying metabolic alkalosis
- Less effective than KCl
- Can be used as prophylaxis of calcium oxalate renal stones or may dissolve uric acid stones
- IV potassium:
 - Recommended if neuromuscular symptoms, cardiac arrhythmias, ongoing GI losses, or severe hypokalemia
 - Potassium chloride is the preferred replacement:
 - Potassium phosphate is used only if accompanying severe hypophosphatemia.
 - Administration:
 - A potassium rider at 10 mEq/h piggybacked into maintenance 0.9 NS is safest and best tolerated (peds: 0.1–0.2 mEq/kg/h)
 - 15–20 mEq/h are feasible by peripheral vein but not recommended due to risk of phlebitis and pain
 - If K is added to maintenance fluids, the concentration should not be >40 mEq/L and dextrose solutions should be avoided
 - If sustained life-threatening dysrhythmias, 20–40 mEq/h by central line or 2 peripheral lines can be considered.
 - If cardiac arrest occurs in a patient with known severe hypokalemia, 20 mEq could be given IV over 2–3 min
 - Monitor serum potassium after every 40 mEq IV
 - Hypokalemic periodic paralysis and other situations in which there is significant transcellular K shifts (adrenergic excess):
 - Small amounts of K are effective (20 mEq IV).
 - More zealous administration may lead to rebound hyperkalemia.
- Electrolyte corrections:
 - Magnesium:
 - Consider if hypokalemia is resistant to K replacement.
 - Magnesium sulfate 2 g slow IV infusion
 - Chloride:
 - Hypokalemia with alkalosis is resistant to replacement unless volume depletion and hyponatremia is corrected by saline administration.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Need of IV potassium repletion
- Cardiac dysrhythmias
- Profound muscle weakness

- Ongoing K losses
- Serum potassium < 2.5 mEq/L
- Associated with significant hypotension or severe HTN
- Significant comorbidities or geriatric

Discharge Criteria

- Asymptomatic
- Able to replete deficiency with oral potassium
- Early follow-up available and reliable patient
- Repeat electrolyte determination in 2–3 days with the primary care doctor.
- Nephrology referral or consult if suspicion of renal wasting.
- Continue K replacement for 2–3 days if acute, self-limited loss, but ongoing therapy if the cause is not corrected (e.g., diuretic therapy, chronic diarrhea).

PEARLS AND PITFALLS

- If hypokalemia is accompanied by acidosis, correct hypokalemia 1st before treating the acidosis so as to avoid life-threatening hypokalemia from transcellular shifts.
- Minimize glucose administration when treating hypokalemia, since glucose will stimulate insulin release, which will lead to K movement into cells.
- Large doses of oral potassium can be given safely in patients with normal renal function, limited only by GI tolerance.
- Check for hypomagnesemia if hypokalemia is severe or resistant to replacement therapy.
- Relatively small amounts of IV potassium are required to reverse hypokalemia in periodic paralysis and states of adrenergic excess since transcellular shifts are transient.

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See Also (Topic, Algorithm, Electronic Media Element)

Hyperkalemia



CODES

ICD9

- 255.13 Bartter's syndrome
- 276.3 Alkalosis
- 276.8 Hypopotassemia

ICD10

- E26.81 Bartter's syndrome
- E87.3 Alkalosis
- E87.6 Hypokalemia

HYPONATREMIA

Linda Mueller

BASICS

DESCRIPTION

- Sodium < 136 mEq/L
- Most common electrolyte disturbance (1–4% of hospitalized patients)

ETIOLOGY

Pseudohyponatremia

- Low measured serum sodium but normal measured serum osmolarity
- Occurs secondary to the displacement of sodium to aqueous phase of serum
- Seen with elevated lipids or proteins
- Lab or blood raw error
- Disease examples include:
 - Multiple myeloma
 - Hyperlipidemia

Hyponatremia with Normal Osmolarity and Fluid Overload

- Inappropriate retention of water
- Disease examples include:
 - CHF
 - Cirrhosis
 - Renal failure
 - Nephrotic syndrome

Hyponatremia with Normal Osmolarity and Euvolemia

- Patients tend to have increased total body water without marked edema
- Purest form of dilutional hyponatremia
- Disease examples include:
 - Endocrine abnormalities:
 - Hypothyroid
 - Stress
 - Syndrome of inappropriate antidiuretic hormone (SIADH)
 - Diseases that cause SIADH:
 - Pulmonary disease (tuberculosis, Legionella, Aspergillosis, COPD)
 - CNS disorders (malignancy, sarcoid, infection)
 - Cancer (small cell lung, pancreas, duodenum)
 - HIV infection

- Water intoxication (3–7% of institutionalized psychotic patients), can also occur in marathon runners
- Mineralocorticoid abnormalities
- Postoperative hyponatremia (particularly after transurethral prostatectomy)
- Consumption of large amounts of beer (beer potomania)
- MDMA (Ecstasy)

Hyponatremia with Normal Osmolarity and Hypovolemia

- Deficits in total body water and total body sodium
- Sodium deficits exceed water deficits
- Possible etiologies include:
 - GI losses
 - Sweating
 - Cerebral salt wasting (occurs after head injury or neurosurgical procedures)
 - Burns
 - Cystic fibrosis
 - Salt-wasting nephropathies
 - Diuretics

Drug Induced

- Drugs may stimulate antidiuretic hormone (ADH) and cause hyponatremia:
 - Amiodarone
 - Barbiturates
 - Bromocriptine
 - Carbamazepine
 - Clofibrate
 - Cyclophosphamide
 - Opiates
 - Oxytocin
 - Vincristine, vinblastine
- Drugs may increase sensitivity to ADH and cause hyponatremia:
 - Chlorpropamide
 - NSAIDs
- Drugs may stimulate thirst and cause hyponatremia:
 - Amitriptyline
 - Ecstasy
 - Fluoxetine
 - Fluphenazine
 - Haloperidol
 - Sertraline
 - Thiothixene

Hyponatremia with Hyperosmolarity

- Due to excessive osmotically active substances
- Possible etiologies include:
 - Elevated glucose (most common cause of hyponatremia)
 - Corrected $\text{Na}^+ = 0.016 \times (\text{measured glucose} - 100) + \text{measured sodium}$
 - Mannitol infusion
 - Maltose and glycine

Pediatric Considerations

- More prone to water intoxication
- High incidence of iatrogenic hyponatremia due to dilute formula or rehydration with water only
- If hyponatremia secondary to DKA, follow hydration per pediatric DKA recommendations

Pregnancy Considerations

Conivaptan and Tolvaptan are class C drugs in pregnancy.

Geriatric Considerations

- Tend to develop more symptoms
- Hyponatremia more common due to impaired water secretion and low sodium diets

DIAGNOSIS

SIGNS AND SYMPTOMS

- Mild: $\text{Na}^+ > 120 \text{ mEq/L}$:
 - Headache
 - Nausea
 - Vomiting
 - Weakness
 - Anorexia
 - Muscle cramps
 - Rhabdomyolysis
- Moderate: Na^+ between 110 and 120 mEq/L:
 - Impaired response to verbal stimuli
 - Decreased response to painful stimuli
 - Visual/auditory hallucinations
 - Bizarre behavior
 - Incontinence
 - Hyperventilation

- Gait disturbance
- Severe: $\text{Na}^+ < 110 \text{ mEq/L}$:
 - Signs of herniation
 - Decorticate/decerebrate posturing
 - Bradycardia
 - HTN
 - Altered temperature regulation
 - Dilated pupils
 - Seizure activity
 - Respiratory arrest
 - Coma/unresponsive

Chronic

May be asymptomatic

History

Review patient medication list.

Physical-Exam

- Assess volume status including skin turgor, neck veins, peripheral edema, and signs of ascites
- Perform a complete neurologic exam.

ESSENTIAL WORKUP

Serum sodium level:

- Recheck sodium to verify.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Electrolytes, BUN/creatinine
- Glucose:
 - Correct sodium value accordingly if severe hyperglycemia (add 1.6 Na for each 100 mg/dL of glucose above normal)
- Calculate osmolality:
 - Plasma osmolality = $[2 \times \text{NA (mEq L)} + \text{Glucose}/18 + \text{BUN}/2.8]$
- Urine sodium
- Serum and urine osmolality
- Thyroid function test
- Adrenal function tests
- CPK for possible rhabdomyolysis

Imaging

- CXR to rule out CHF, infection, and tumor
- CT of head, particularly if patient has AMS

DIFFERENTIAL DIAGNOSIS

- Pseudohyponatremia due to:
 - Hyperglycemia
 - Hyperlipidemia
 - Hyperproteinemia
 - Radiocontrast dye particularly in chronic renal insufficient patients



TREATMENT

PRE HOSPITAL

- Establish IV
- Supportive care

INITIAL STABILIZATION/THERAPY

- ABCs
- Initiate IV fluid with 0.9% NS.
- Naloxone, thiamine, D₅₀W (or Accu-Chek) for altered mental status

ED TREATMENT/PROCEDURES

- Depends on severity and chronicity of hyponatremia and underlying etiology
- Chronic hyponatremia is to be corrected slowly to minimize osmotic demyelination syndrome. Correction should be limited to 10–12 mmol/L in 24 hr
- Acute hyponatremia with severe CNS symptoms/actively seizing:
 - Goal:
 - Raise serum sodium by 8–10mEq/L in 4–6 hr or to level > 120–125 mEq/L with administration of hypertonic saline, slow or discontinue when seizure subsides.
 - 200–400 mL of 3% saline solution will be the approximate amount needed in most adults over the 1st 2 hr
 - OR may dose 1–2 mL/kg/hr of 3% saline solution
 - Calculate sodium deficit:
 - Na^+ deficit = 0.6 (weight in kg) (140 – Na^+)
 - Sodium contents:
 - 1 L 0.9% NS = 154 mEq of sodium
 - 1 L 3% saline = 513 mEq of sodium
- Hypovolemic hyponatremia:
 - Correct underlying cause
 - Replete volume with 0.9% NS IV.

- Primary goals to restore:
 - Extracellular fluid
 - Cardiac output
 - Organ perfusion
- Hypervolemic/euvolemic hyponatremia:
 - Water restriction to <1 L/day with high dietary salt intake
 - For faster correction of sodium:
 - Administer IV 0.9% NS with loop diuretic (furosemide).
 - Maximum rate of correction = 0.5 mEq/L/hr

MEDICATION

- Furosemide: 20–40 mg IV push
- Sodium replacement:
 - Calculate Na⁺ deficit
 - Replace no more than 1/2 of requirement over 8–12 hr

First Line

500 mL–1 L of saline for a fluid challenge

Second Line

- Conivaptan: Arginine vasopressin antagonist
- 20 mg IV loading dose over 30 min followed by 20 mg continuous IV infusion over 24 hr
- Tolvaptan: Selective vasopressin V2 receptor antagonist dose 15 mg/d PO and may increase in 24 hr to 30 mg
- Conivaptan and tolvaptan are for the treatment of euvolemic and hypervolemic hyponatremia only



FOLLOW-UP

DISPOSITION

Admission Criteria

- Symptomatic hyponatremia
- Sodium <120 mEq/L
- Asymptomatic, mild hyponatremia (Na⁺ 120–127 mEq/L), with comorbid factors

Discharge Criteria

- Sodium >130 mEq/L and asymptomatic
- Known chronic history of hyponatremia with no acute changes
- Asymptomatic, mild hyponatremia (Na⁺ 120–129 mEq/L) with no comorbid

factors; however, must have close outpatient follow-up.

FOLLOW-UP RECOMMENDATIONS

Have repeat serum sodium within a week, particularly if related to thiazide diuretics

PEARLS AND PITFALLS

- Too rapid correction may cause osmotic demyelination syndrome
- Females, alcoholics, malnourished patients, hypokalemia, and history of liver transplant are risk factors for osmotic demyelination syndrome.
- Repeat and document neurologic exam during correction.
- Beware of falsely low sodium when blood is drawn near an IV site with hypotonic fluid.
- Thiazide diuretics may cause persistent hyponatremia up to 2 wk after discontinuation.

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See Also (Topic, Algorithm, Electronic Media Element)

Hypernatremia



CODES

ICD9

- 253.6 Other disorders of neurohypophysis
- 276.1 Hyposmolality and/or hyponatremia
- 276.69 Other fluid overload

ICD10

- E22.2 Syndrome of inappropriate secretion of antidiuretic hormone
- E87.1 Hypo-osmolality and hyponatremia

- E87.79 Other fluid overload

HYPOPARATHYROIDISM

Rami A. Ahmed • Brad D. Gable

BASICS

DESCRIPTION

- Hypoparathyroidism occurs secondary to a deficiency in parathyroid hormone (PTH)
- Pseudohypoparathyroidism occurs secondary to end-organ unresponsiveness to PTH
- PTH:
 - _ Decreases urinary Ca^{2+} loss
 - _ Increases urinary PO_4 loss
 - _ Stimulates vitamin D conversion from 25(OH)-D to 1,25(OH)₂-D in kidney
 - _ Liberates Ca^{2+} and PO_4 from bone
- Hypocalcemia is the major metabolic derangement
- Calcitonin:
 - _ Promotes deposition of Ca^{2+} and PO_4 into bone (produced primarily in C cells in thyroid)
- Magnesium:
 - _ Cofactor in production of PTH
 - _ Essential for action of PTH in target tissues
- Hypoparathyroidism:
 - _ Primary failure of the parathyroid gland (may have associated Addison disease)
- Pseudohypoparathyroidism:
 - _ Tissue unresponsiveness with elevated PTH levels
 - _ Associated with hypothyroidism and hypogonadism
- Genetics:
 - _ Congenital absence
 - _ DiGeorge syndrome:
 - Hypoparathyroidism
 - Thymic dysplasia
 - Severe immunodeficiency
 - _ Wilson disease:
 - Destruction of gland owing to copper deposition
 - _ Autoimmune polyglandular syndrome type I
 - Hypoparathyroidism
 - Adrenal insufficiency

- Mucocutaneous candidiasis
- Albright syndrome (hereditary osteodystrophy):
 - Short stature
 - Obesity
 - Round face
 - Short neck
 - Short 4th and 5th metacarpals and metatarsals (type I pseudohypoparathyroidism)

ETIOLOGY

- Failure of parathyroid gland:
 - Autoimmune destruction
 - Surgical interruption of blood supply or gland removal
 - Radiation damage
 - Hypomagnesemia (PTH cofactor)
- End-organ unresponsiveness to PTH



DIAGNOSIS

SIGNS AND SYMPTOMS

ALERT

The most common symptomatic presentation is postoperatively after parathyroidectomy.

Pediatric Considerations

Neonates/infants:

- Transient hypoparathyroidism in 1st yr of life
- Below normal intelligence proportional to duration of hypocalcemia
- Dental hypoplasia

History

- Most common presentation is in the postoperative period after parathyroidectomy or thyroidectomy
- Prolonged severe hypomagnesemia, in the alcoholic or high-dose diuretic patient, is the next most common presentation and can be slow in onset; usually less symptomatic

Physical-Exam

- Related to severity, rapidity of onset, and duration of hypocalcemia
- General:
 - Weakness

- Malaise
- Neuromuscular:
 - Paresthesias (especially circumoral and extremities)
 - Carpal pedal spasm
 - Latent spasm elicited by:
 - Chvostek sign (twitching of circumoral muscles after tapping facial nerve in front of the tragus)
 - Trousseau sign (spasm after inflating BP cuff 20 mm above patient's systolic BP for 3 min)
 - Laryngospasm/bronchospasm
 - Blepharospasm
 - Muscle cramps
 - Tetany
 - Seizures (presenting symptom of 1/3 with hypoparathyroidism)
 - Increased intracranial pressure (ICP) with papilledema
 - Parkinson syndrome and other extrapyramidal disorders
 - Myelopathy
- Cardiovascular:
 - Prolonged QT interval (owing to ST-segment prolongation)
 - Heart block
 - CHF
 - Ventricular fibrillation (VFib)
 - Vasoconstriction
- Psychiatric:
 - Impaired memory
 - Confusion
 - Hallucinations
 - Dementia
- Dermatologic:
 - Brittle hair and nails
 - Psoriasis
- Hyperpigmentation:
 - Lenticular cataracts

ESSENTIAL WORKUP

- If *no hypocalcemic symptoms* with hypocalcemia, check albumin level:
 - If still low after correcting for hypoalbuminemia, check ionized Ca^{2+}
- If *hypocalcemic symptoms* with normal total Ca^{2+} , check pH for alkalosis:
 - If not alkalotic, check ionized Ca^{2+} (active form)
 - Metabolic or respiratory alkalosis increases the binding to albumin reducing the ionized Ca^{2+}
- If *hypocalcemic symptoms* with low ionized Ca^{2+} , check a PTH level:

- Low in primary hypoparathyroidism and in vitamin D deficiency
- Elevated in pseudohypoparathyroidism and hypocalcemia from renal failure

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Calcium: Correct for albumin using formula:
 - Corrected Ca^{2+} (mg/dL) = measured Ca^{2+} (mg/dL) + 0.8[4.0 – albumin (g/dL)]
- Ionized Ca^{2+} if symptomatic with low total calcium
- Electrolytes, BUN, creatinine, glucose
- Magnesium
- Arterial blood gas (ABG) if symptomatic with normal total Ca^{2+}
 - Elevation of 0.1 pH unit decreases the ionized Ca^{2+} by 3–8%.
- Phosphorus:
 - Elevated except when hypocalcemia caused by vitamin D deficiency
 - Metastatic calcification can cause hypocalcemia by tissue deposition when the calcium/phosphorus product is > 60.

Diagnostic Procedures/Surgery

ECG:

- Prolonged QT interval:
 - Owing to ST-segment prolongation from hypocalcemia

DIFFERENTIAL DIAGNOSIS

- Must differentiate from a variety of causes of hypocalcemia
- Lab artifact:
 - Low total calcium that is normal when corrected for albumin level with no symptoms of hypocalcemia
- Alkalosis:
 - Symptomatic hypocalcemia with a normal total calcium
- Hypomagnesemia (needed for PTH secretion)
- PTH resistance (congenital)
- Vitamin D deficiency (low Ca^{2+} + low PO_4):
 - Anticonvulsant use (decreased vitamin D absorption)
 - Liver disease
 - Resistance to vitamin D
 - Malabsorption or dietary deficiency
- Gram-negative sepsis
- Renal failure or nephrotic syndrome
- Chelation:

- Pancreatitis (fatty acids chelate calcium)
- Ammonium bifluoride (tire cleaner spray)
- Hydrofluoric acid
- Citrated blood
- Acute hyperphosphatemia:
 - o Fleet enemas
 - o Rhabdomyolysis
 - o Acute renal failure



TREATMENT

PRE HOSPITAL

- Administer calcium in refractory VFib or status epilepticus in addition to usual medications if known hypoparathyroidism or suspected hypocalcemia
- Stridor may herald laryngospasm

INITIAL STABILIZATION/THERAPY

- Airway, breathing, and circulation management (ABCs):
 - Manage airway if laryngospasm
- Administer IV calcium bolus (chloride or gluconate) if unstable cardiac rhythm or tetany:
 - Slow infusion much safer unless patient markedly symptomatic
- Prepare for ventricular dysrhythmias including VFib.
- Seizure precautions

ED TREATMENT/PROCEDURES

- Calcium replacement:
 - Calcium chloride 10% (27.2 mg elemental Ca^{2+} /mL):
 - o For life-threatening conditions: 10 mL (1 g) IV over 5 min OR
 - Calcium gluconate 10% (9 mg elemental Ca^{2+} /mL):
 - o For life-threatening conditions: 20–30 mL (2–3 g) over 3–5 min
 - For non-life-threatening conditions, administer calcium via slow infusion of 500–1,000 mg elemental Ca^{2+} over 6–24 hr (peds: 100 mg elemental Ca^{2+} /kg/24 hr)
 - Continuous cardiac monitoring
 - Stop infusion if bradycardia develops
 - Perform frequent checks of serum Ca^{2+} levels
 - Calcium administration may precipitate digitalis toxicity
 - Supplement to lowest possible Ca^{2+} level keeping the patient asymptomatic, then switch to oral replacement:
 - o Soft tissue calcification may occur with calcium/phosphorus product

of 60 (Ca × PO₄)

- Replace magnesium if low
- Bind phosphorus:
 - Aluminum hydroxide–containing antacids (Maalox, Mylanta, or Gelusil) if creatinine < 2
 - Calcium acetate (Phoslo) or calcium carbonate when concurrent renal failure if creatinine > 2
 - Sevelamer HCl or carbonate (Renagel, Renvela)
- Vitamin D supplementation
- Avoid carbonated beverages (high in phosphorus)
- Assess for associated endocrinopathies

MEDICATION

First Line

- Calcium gluconate: 10% (9 mg elemental Ca²⁺/mL): 20–30 mL over 3–5 min if life-threatening condition; otherwise, slow infusion (peds: 20 mg/kg of calcium gluconate 10% or 2 mg/kg elemental Ca):
 - Follow with slow infusion: Calcium gluconate 10 g in liter of 5% dextrose infused at 1–3 mg/kg/h in adults
 - Calcium gluconate has lower risk of venous irritation or extravasational injury compared to calcium chloride
- Magnesium sulfate: 2 g IV (peds: 25–50 mg/kg up to 2 g) over 2 hr—if severe, 6 g over 6 hr
- Calcium chloride 10% (27.2 mg elemental Ca²⁺/mL): 10 mL (1 g) IV over 5 min if life-threatening condition; otherwise, slow infusion

Second Line

- Calcium acetate: 667 mg (169 mg elemental Ca): 1 or 2 tabs TID with meals
- Calcium carbonate: 1,250 mg (500 mg elemental Ca): 1 or 2 tabs QID (2–4 g/d) (peds: 45–65 mg elemental Ca mg/kg/d div. QID)
- Sevelamer (Renagel, Renvela) 800 mg: 1 or 2 tabs TID with meals
- Magnesium oxide 400 mg: 1 tab daily or BID
- Vitamin D: 400 IU PO daily for supplement (if not responsive to standard supplement, then consider calcitriol (1,25(OH)₂-D) 0.25 µg daily; titrate to 0.5–2 µg/d):
 - Preferred over other long-acting vitamin D analogues due to patient availability and lower cost, quicker onset and offset of action
- Thiazide diuretics: HCTZ 25 100 mg daily



FOLLOW-UP

DISPOSITION

Admission Criteria

- Symptomatic hypocalcemia
- Abnormal ECG
- Inability to take vitamin D or calcium orally
- Corrected calcium < 5 mg/dL

Discharge Criteria

- Asymptomatic hypocalcemia
- Not meeting any admission criteria

FOLLOW-UP RECOMMENDATIONS

- Any patient requiring therapy or needing follow-up lab studies
- Repeat of calcium, phosphorus, magnesium levels in 1–2 days

PEARLS AND PITFALLS

- Rapid onset of symptoms following surgical excision of the parathyroid glands is the most common symptomatic presentation
- Symptoms often confused with hyperventilation or anxiety
- In the absence of surgery or severe hypomagnesemia, be sure that hypocalcemia is not due to sepsis or rhabdomyolysis
- With the exception of life-threatening presentations, avoid rapid IV administration of calcium salts

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See Also (Topic, Algorithm, Electronic Media Element)

Hyperparathyroidism



CODES

ICD9

- 252.1 Hypoparathyroidism
- 275.49 Other disorders of calcium metabolism
- 279.11 Digeorge's syndrome

ICD10

- E20.1 Pseudohypoparathyroidism
- E20.9 Hypoparathyroidism, unspecified
- D82.1 Di George's syndrome

HYPOTHERMIA

Jordan Moskoff

BASICS

DESCRIPTION

- Body temperature $< 35^{\circ}\text{C}$
- Risk factors:
 - Poor temperature regulation:
 - Very young
 - Advanced age
 - Comorbid condition
 - Intoxication
- Pathophysiology:
 - Loss of heat:
 - Radiation: Most rapid, 50% of heat loss
 - Conduction
 - Convection
 - Evaporation
 - Respiration
 - Heat production:
 - Shivering
 - Nonshivering thermogenesis
 - Increased thyroxine
 - Increased epinephrine

ETIOLOGY

- Dermal disease:
 - Burn
 - Exfoliative dermatitis
 - Severe psoriasis
- Drug induced:
 - Ethanol
 - Phenothiazines
 - Sedative–hypnotics
- Environmental:
 - Immersion
 - Nonimmersion
- Iatrogenic:
 - Aggressive fluid replacement
 - Heat stroke treatment

- Metabolic:
 - Hypoadrenalism
 - Hypopituitarism
 - Hypothyroidism
- Neurologic:
 - Acute spinal cord transection
 - Head trauma
 - Stroke
 - Tumor
 - Wernicke disease
- Neuromuscular inefficiency:
 - Age extreme
 - Impaired shivering
 - Lack of acclimatization
- Sepsis

Pediatric Considerations

Infants have a large body surface to mass ratio Child abuse.

DIAGNOSIS

SIGNS AND SYMPTOMS

- Mild (35–32.2°C/95–90°F):
 - Initial excitation phase to combat cold:
 - HTN
 - Shivering
 - Tachycardia
 - Early tachycardia followed by bradycardia
 - Tachypnea
 - Vasoconstriction
 - Over time with onset of fatigue:
 - Apathy
 - Ataxia
 - Cold diuresis
 - Defect in distal tubular reabsorption of sodium and water
 - Impaired judgment
- Moderate (32.2–28°C/90–82.4°F):
 - Atrial dysrhythmias
 - Bradycardia:
 - Decreased spontaneous depolarization of pacemaker cells
 - Refractory to atropine
 - Decreased level of consciousness

- Decreased respiratory rate:
 - Progressive respiratory depression with CO₂ retention
- Dilated pupils
- Diminished gag reflex
- Extinction of shivering
- Hyporeflexia
- Hypotension
- J-wave (Osborn wave) on ECG
- Severe (< 28°C / < 82.4°F):
 - Apnea
 - Coma
 - Decreased or no activity on EEG (electroencephalography)
 - Nonreactive pupils
 - Oliguria:
 - Renal blood flow depressed 50%
 - Pulmonary edema
 - Ventricular dysrhythmias/asystole:
 - Cardiac cycle lengthens, resulting in increased intervals

History

Time of submersion for near drowning in cold water.

Physical-Exam

- May not be able to palpate pulse
- May not be able to obtain BP
- Pupils dilate < 26°C

ESSENTIAL WORKUP

Accurate core temperature confirms diagnosis.

DIAGNOSIS TESTS & NTERPRETATION

Lab

- Finger stick glucose
- ABG:
 - Temperature correction not needed
- CBC:
 - Hematocrit rises owing to decreased plasma volume.
 - Leukopenia does not imply absence of infection:
 - High-risk groups (e.g., neonate, immunocompromised) should receive empiric antibiotics.
- Electrolytes, BUN, creatinine:

- Vary during rewarming; recheck frequently, especially creatine phosphokinase (CPK) and potassium (K⁺)
- Serum lactate
- PT, PTT, and platelets:
 - Prolonged clotting times, thrombocytopenia common
- Toxicology screen:
 - Alcohol/drug ingestion common

Imaging

- CXR:
 - Pneumonia common complication
- EKG:
 - Tachycardia to bradycardia
 - Atrial fibrillation with slow response
 - Ventricular fibrillation
 - Asystole
 - Prolonged PR, QRS, QT intervals
 - J-wave (Osborn waves)
 - ST-elevation mimicking acute coronary syndrome

DIFFERENTIAL DIAGNOSIS

- Environmental
- Sepsis
- Primary CNS disorder
- Metabolic
- Drug induced



TREATMENT

PRE HOSPITAL

- Patient is not dead until “Warm and Dead”:
 - CPR recommended during transport:
- Prolonged palpation/auscultation for cardiac activity: 30–45 sec
 - Apparent cardiovascular collapse may be depressed cardiac output, often sufficient to meet metabolic demands.

INITIAL STABILIZATION/THERAPY

- ABCs:
 - Supplemental oxygen
 - Oral and nasotracheal intubation are safe.
 - Place nasogastric (NG) tube postintubation.
 - Cardiac monitor

- Warmed D5.9 NS preferred over lactated Ringer:
 - Shivering depletes glycogen.
- Remove wet clothing and begin passive external rewarming.
- Administer Narcan, D₅₀W (or Accu-Chek), and thiamine to a patient with altered mental status.
- Stress-dose steroids (Solu-Cortef 100 mg IV) for known adrenal insufficiency or treatment failure.
- Obtain accurate core temperatures using rectal thermometer.

ED TREATMENT/PROCEDURES

- Cardiac arrest resuscitation:
 - Most dysrhythmias correct with rewarming alone.
 - Ventricular fibrillation induction occurs with rough handling, chest compressions, hypoxia, and acid–base changes.
 - CPR is less effective owing to decreased chest wall elasticity.
 - Defibrillation is rarely successful at temperatures <28–30°C
 - Defibrillate 1–3 times and then again post rewarming.
 - Once >30°C, if ventricular fibrillation persists consider amiodarone.
 - Direct current results in myocardial damage.
- Dysrhythmia management:
 - Atrial fibrillation:
 - Commonly <32°C
 - Usually converts spontaneously
 - Malignant ventricular dysrhythmias:
 - Amiodarone drug of choice though limited proof of effectiveness.
- Rewarming techniques:
 - Faster rewarming rates (1–2°C/hr) generally have better prognosis than slower rewarming rates (<0.5°C/hr).
 - Active rewarming is necessary at core temperature of <32°C:
 - Internal thermogenesis insufficient to increase body temperature
 - Shivering extinguished
- Passive external rewarming:
 - Ideal technique for most healthy patients with mild hypothermia
 - Must have intact thermoregulatory mechanisms, normal endocrine function, and adequate energy stores
 - Cover the patient with dry insulating material.
 - Endogenous thermogenesis must generate an acceptable rate of rewarming:
 - Must increase 0.5–2°C/hr
 - Disadvantage: Core rises very slowly.
- Active external rewarming:
 - Delivers heat directly to the skin
 - Safe in previously healthy, young, acutely hypothermic victims
 - Requires intact circulation to remove peripherally rewarmed blood to core

- Associated with core temperature afterdrop
- Rewarming shock: Venous pooling in warmed extremities secondary to vasodilatation
- Cover trunk preferentially.
- Bair Hugger device provides forced warm air: Prevents shock or afterdrop.
- Active core rewarming techniques:
 - Airway rewarming (complete humidification at 40–45°C):
 - Administer to all patients.
 - Heated IV (40–42°C) D5.9 NS:
 - Administer to all patients.
 - High flow rates must be maintained.
 - Use blood warmer or calibrated microwave.
 - Heated gastric irrigation via NG or orogastric tubes:
 - Not recommended
 - Low amount of surface area
 - Aspiration risk if airway not secured
 - Pleural irrigation (0.9 NS at 30–42°C):
 - Use in severe hypothermia without cardiac activity.
 - 1–2 chest tubes; midaxillary and midclavicular bilaterally
 - Contraindicated in patients with cardiac rhythm because the chest tube may induce ventricular fibrillation
 - Heated peritoneal lavage (0.9 NS at 40–45°C):
 - Use in unstable hypothermic patients or stable patients with severe hypothermia whose rewarming rates are $< 1^{\circ}\text{C/hr}$.
 - 1–2 catheters
 - Advantageous in patients with overdose or rhabdomyolysis
- Extracorporeal rewarming:
 - Most effective rewarming method
 - Hemodialysis:
 - Initiate for patients with drug overdoses or severe electrolyte disturbances.
 - Continuous arteriovenous rewarming:
 - BP must be > 60 mm Hg.
 - Blood circulated through warmer from percutaneously inserted femoral arterial and contralateral venous catheters
 - Extracorporeal venovenous rewarming:
 - Blood is removed via central venous catheter, heated to 40°C, and returned via 2nd central or large peripheral venous catheter.
 - Cardiopulmonary bypass:
 - Treatment of choice in severe hypothermia with cardiac arrest
- Additional therapy:
 - Methylprednisolone or hydrocortisone for suspicion of adrenocortical insufficiency or steroid dependence

- Empiric treatment with levothyroxine only for myxedematous patients

MEDICATION

- Amiodarone: 300 mg IV push (IVP) for ventricular fibrillation followed by 1 mg/min infusion
- Dextrose: D₅₀W 1 amp—50 mL or 25 g (peds: D₂₅W 2–4 mL/kg) IV
- Hydrocortisone: 250 mg IVP
- Levothyroxine: 50–500 µg IV over several minutes
- Methylprednisolone: 30 mg/kg IVP
- Naloxone (Narcan): 2 mg (peds: 0.1 mg/kg) IV or IM initial dose
- Thiamine (vitamin B₁): 100 mg (peds: 50 mg) IV or IM



FOLLOW-UP

DISPOSITION

Admission Criteria

- Moderate to severe hypothermia ($< 32^{\circ}\text{C}$)
- Young, healthy patients with no comorbid illness who have mild accidental hypothermia ($> 32^{\circ}\text{C}$) that responds well to warming:
 - Admit to an observation area.
 - Discharge if asymptomatic after 8–12 hr and they remain asymptomatic.

Discharge Criteria

- Young, healthy patients with no comorbid illness
- Very mild accidental hypothermia ($> 35^{\circ}\text{C}$) that responds well to warming
- Safe, warm environment to go to after discharge

FOLLOW-UP RECOMMENDATIONS

Social work follow-up for homeless patients with cold exposure and hypothermia

PEARLS AND PITFALLS

- Defibrillation is rarely successful at temperatures $< 28\text{--}30^{\circ}\text{C}$:
 - Defibrillate 1–3 times and then again post rewarming.
- Atrial fibrillation usually converts spontaneously.
- Faster rewarming rates (1–2°C/hr) generally have better prognosis than slower rewarming rates ($< 0.5^{\circ}\text{C/hr}$).
- Afterdrop is the continued decline in core temp after removed from cold
 - Ongoing conduction of heat from core warming periphery prior to the core
- Rewarming shock
 - Hypovolemic shock secondary to failure to replete volume during

resuscitation.

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See Also (Topic, Algorithm, Electronic Media Element)

Frostbite



CODES

ICD9

- 778.3 Other hypothermia of newborn
- 780.65 Hypothermia not associated with low environmental temperature
- 991.6 Hypothermia

ICD10

- P80.9 Hypothermia of newborn, unspecified
- R68.0 Hypothermia, not associated w low environmental temperature
- T68.XXXA Hypothermia, initial encounter

HYPOTHYROIDISM

Rita K. Cydulka • Tammy L. Weiner

BASICS

DESCRIPTION

- Decreased level of effective circulating thyroid hormone leads to decreased metabolic rate and decreased sensitivity to catecholamines.
- More common in woman and the elderly
- Myxedema coma is a rare, extreme form of hypothyroidism characterized by altered mental status and defective thermoregulation triggered by a precipitating event in a patient with hypothyroidism.

ETIOLOGY

- Primary:
 - _ Idiopathic
 - _ Congenital
 - _ Autoimmune:
 - Thyroiditis
 - Hashimoto disease
 - _ Iatrogenic:
 - Postsurgical
 - External radiation
 - Radioiodine therapy
 - Drugs (iodides, lithium, amiodarone, sunitinib, bexarotene, interferons, narcotics, sedatives)
 - _ Neoplasm: Primary (carcinoma) or secondary (infiltration)
 - _ Infection: Viral (rarely aerobic or anaerobic bacteria)
 - _ Iodine deficiency (most common cause worldwide)
- Central (very rare):
 - _ Pituitary or hypothalamic disorder induced by drugs or severe illness
 - _ May have other associated hormone deficiencies
- Myxedema coma:
 - _ Critical decompensation of a patient with hypothyroidism due to a stress, often during winter months. Stressors include:
 - Infection
 - Hypothermia
 - Intoxication
 - Drugs
 - Cerebrovascular accident
 - Heart failure

- Trauma

Pregnancy Considerations

- Hypothyroid women require increased exogenous thyroid hormone replacement during pregnancy above baseline
- Postpartum thyroiditis occurs in up to 10% of women:
 - Usually 3–6 mo postpartum
 - Typically resolves without treatment



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Exhaustion
- Cold intolerance
- Headaches
- Diminished hearing
- Myalgias and muscle weakness
- Menorrhagia
- Infertility
- Carpal tunnel syndrome
- Constipation
- Weight gain
- Depression, hallucinations, or paranoia
- Cognitive impairment

Physical-Exam

- Periorbital edema
- Sparse, coarse hair and brittle nails
- Absent lateral 1/3 of eyebrows (Queen Anne sign)
- Husky or hoarse voice
- Goiter
- Prolonged relaxation phase of deep tendon reflexes (DTRs)
- Yellow, dry, pale, cool, coarse skin
- Myxedema (dry, waxy swelling of skin)
- Nonpitting edema of hands and feet
- Myxedema coma:
 - Altered mental status
 - Hypotension
 - Hypothermia
 - Respiratory failure

- Bradycardia

Pediatric Considerations

- Undiagnosed hypothyroidism in infants has largely been eliminated via universal screening at birth
- Hypothyroidism in childhood is usually due to Hashimoto disease.
- Children may manifest with retardation of mental developmental, linear growth, and sexual maturation

Geriatric Considerations

Typical symptoms of hypothyroidism may be confused with changes associated with aging.

ESSENTIAL WORKUP

Lab confirmation of the diagnosis of hypothyroidism/myxedema coma may not be available in the ED, and therapy should be initiated based on clinical suspicion.

DIAGNOSIS TESTS & INTERPRETATION

Search for the underlying cause of myxedema coma.

Lab

- Thyroid function studies:
 - Low total and free thyroxine (T₄)
 - Low total and free triiodothyronine (T₃)
 - Thyroid stimulating hormone (TSH):
 - Increased in primary hypothyroidism but normal or decreased in central pathology
- Anemia
- Hyponatremia
- Hypoglycemia
- Hypoxemia
- Hypercapnia
- Respiratory acidosis
- Elevated lactate dehydrogenase (LDH), creatine kinase (CK), cholesterol, creatinine

Imaging

CXR:

- Enlarged cardiac silhouette due to pericardial effusion

Diagnostic Procedures/Surgery

ECG:

- Sinus bradycardia, low voltage, PR interval prolongation, bundle branch blocks, QT interval prolongation and nonspecific ST-T-wave changes
- May see Osborn wave if profoundly hypothermic

DIFFERENTIAL DIAGNOSIS

- Chronic nephritis
- Chronic renal disease
- Heart failure
- Depression
- Hypoalbuminemia
- Pernicious anemia
- Nephrotic syndrome
- Sepsis

ALERT

- Euthyroid sick syndrome:
 - Illness, surgery, fasting may produce abnormal thyroid function test results
 - Thyroid function tests performed during acute nonthyroid illness may be abnormal and should be interpreted with caution



TREATMENT

INITIAL STABILIZATION/THERAPY

- ABCs:
 - Intubation and ventilation may be necessary
- Cardiac monitor
- Blood pressure support
- Supplemental oxygen to meet metabolic needs
- Correct hypothermia:
 - Initiate passive warming measures
 - Aggressive rewarming may precipitate hypotension from vasodilation

ED TREATMENT/PROCEDURES

- Mild hypothyroidism:
 - Refer for oral thyroid hormone replacement as an outpatient
- Myxedema coma:
 - Life-threatening condition
 - Initiate thyroid hormone replacement therapy if a high index of suspicion:
 - Prompt IV replacement improves survival
 - Controversy over regimen exists
 - Thyroxine (T₄) and triiodothyronine (T₃)
 - Reassess 4 hr after initial dose

- Use smaller doses of T₄ and avoid T₃ in the elderly or patients with cardiac disease to avoid precipitating ischemia
- Hydrocortisone to prevent Addisonian crisis
- Dextrose for hypoglycemia
- IV fluid bolus for hypotension:
 - Avoid pressors if possible, may precipitate dysrhythmias
 - Response to pressors is poor until thyroid replacement initiated
 - Thyroid hormone augments pressors
- Consider hypertonic saline for severe hyponatremia
- Correct the underlying precipitant

MEDICATION

First Line

Thyroid hormone therapy:

- Administer T₄, T₃, or a combination:
 - Combination therapy:
 - Thyroxine (T₄): 2 µg/kg (ideal body mass) load IV followed by 10–40 µg IV/PO daily
 - **PLUS**
 - Triiodothyronine (T₃): 10 µg load IV followed by 10 µg IV q8–12h until able to tolerate PO T₄
 - Thyroxine (T₄): 10–40 µg IV or IM daily
 - Triiodothyronine (T₃): 10–20 µg load IV followed by 10 µg IV q4h for 24 hr, then 10 µg IV q6h for 24–48 hr

Second Line

- Hydrocortisone: 100 mg (peds: 4 mg/kg/24h) IV q6–8h
- Dextrose: 50–100 mL D₅₀ (peds: 5 mL/kg of D₁₀) IV



FOLLOW-UP

DISPOSITION

Admission Criteria

All patients with myxedema coma require ICU admission.

Discharge Criteria

Hypothyroidism is managed in the outpatient setting.

Issues for Referral

- Primary care providers can generally manage hypothyroidism.
- Pregnant patients, elderly patients, and those with ischemic heart disease require special consideration when initiating thyroid hormone replacement.

FOLLOW-UP RECOMMENDATIONS

- Patients should be referred to a primary care provider for initiation of oral thyroid hormone replacement therapy.
- Severe untreated maternal hypothyroidism can negatively impact fetal brain development and cause obstetrical complications.

PEARLS AND PITFALLS

- Signs and symptoms of hypothyroidism are nonspecific and may be confused with other mental or physical disorders.
- Response to treatment for hypothyroidism may take weeks and is best initiated by the primary care physician.
- Consider myxedema coma in patients with altered mental status and underlying hypothyroidism.
- Myxedema coma has a high mortality rate and requires aggressive treatment. However, avoid parenteral T₃ in cardiac and elderly patients.

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See Also (Topic, Algorithm, Electronic Media Element)
Hyperthyroidism



CODES

ICD9

- 243 Congenital hypothyroidism
- 244.8 Other specified acquired hypothyroidism
- 244.9 Unspecified acquired hypothyroidism

ICD10

- E03.1 Congenital hypothyroidism without goiter
- E03.5 Myxedema coma
- E03.9 Hypothyroidism, unspecified

IDIOPATHIC THROMBOCYTOPENIC PURPURA

Matthew T. Keadey • Richard D. McCormick

BASICS

DESCRIPTION

- Idiopathic thrombocytopenic purpura (ITP) is thrombocytopenia without apparent cause or abnormalities in other celllines.
- Incidence is ~2–5/100,000/yr—may be an underestimate owing to undetected, subclinical cases.
- Acute ITP:
 - 1/2 of cases involve children.
 - 80% of children recover within 8 wk with or without therapy.
 - Adult recovery is delayed and requires specific therapy to achieve remission.
- Chronic ITP:
 - Occurs mostly in adults
 - Young women are most susceptible in adult onset ITP
 - Characterized by variable response to corticosteroids and other immune suppressants
 - 60–70% respond to splenectomy
- Chronic refractory ITP:
 - Platelet counts may often wax and wane.
 - Often do not respond to therapy
 - No clear optimal course of treatment
- Genetics:
 - ITP appears to run in families, as do variations in response to corticosteroids for treatment.

ETIOLOGY

- Autoantibodies produced by B cells and plasma cells cause immune-mediated destruction of circulating platelets
- Macrophages in spleen and liver mediate destruction of platelets via IgG autoantibodies
- IgM and IgA rarely have been seen
- Some patients do not possess autoantibodies, suggesting a role for T cell-mediated cytotoxicity.
- C3 and C4 complements have also been shown to play a role in patients that lack autoantibodies
- Poor platelet production may also play a role especially in chronic or refractory cases of ITP
- Eradication of *Helicobacter pylori* can sometimes be associated with platelet

recovery (unclear mechanism).



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Bleeding is the most common complaint:
 - Common:
 - Mucous membrane: Gingiva, epistaxis, conjunctival, menorrhagia
 - Rare (more common in coagulopathies):
 - GI bleeding, hemarthrosis, hematuria, and hematomas
- 84% of pediatric cases are 2–3 wk following a viral illness:
 - Small percentage found following vaccinations
- Most adult cases have an insidious onset:
 - Up to 28% of patients are asymptomatic and diagnosed on routine CBC
 - <5% present with life-threatening bleeding

Physical-Exam

- Mucosal bleeding often apparent in symptomatic cases
- Commonly may follow dental procedures, extraction or trauma
- Petechiae (nonblanching)
- Nonpalpable purpura
- Distinguishes ITP from Henoch–Schönlein purpura (HSP)
- Melena, bright red blood per rectum, or hemocult positive stools
- Spleen normal size in ITP:
 - Enlarged spleen may be found in leukemia or other platelet sequestration syndromes
- Neurologic deficits from intracranial hemorrhage (ICH):
 - ICH is the most common cause of death in ITP.
 - Risk of ICH in ITP increases with age:
 - Age <40 yr: 2%
 - Age >60 yr: 48%

ESSENTIAL WORKUP

- Diagnosis of exclusion—other causes of thrombocytopenia must be ruled out.
- CBC with differential
- PT and PTT if actively bleeding to exclude other forms of coagulopathy
- BUN and creatinine to evaluate renal function
- Liver function to exclude liver disease
- Type and screen if actively bleeding
- Pregnancy test if of childbearing age

- HIV testing should be considered

DIAGNOSIS TESTS & INTERPRETATION

Lab

CBC with differential and peripheral smear:

- Thrombocytopenia
- Increased mean platelet volume
- Normal WBC and RBC morphology and size
- Liver function, coagulation studies, and kidney function should all be within normal limits

Imaging

CT head without contrast to evaluate for ICH if clinically indicated by focal findings, headache, or head trauma.

Diagnostic Procedures/Surgery

Bone marrow biopsy:

- All adults > 60 yr to evaluate for malignancy
- Atypical symptoms and cases refractory to treatment
- Patients considering splenectomy
- Children with persistent thrombocytopenia > 6 mo
- Children unresponsive to intravenous immunoglobulin (IVIG)
- Antibody testing is of no clinical value

DIFFERENTIAL DIAGNOSIS

- Impaired bone marrow production:
 - Bone marrow fibrosis
 - Bone marrow infiltration owing to malignancy
 - Cytotoxic drugs used in chemotherapy
 - Congenital/acquired bone marrow abnormalities
- Splenic sequestration:
 - Portal hypertension
 - Neoplastic infiltration
 - Sickle cell disease
- Accelerated destruction of platelets:
 - Vasculitis
 - TTP/HUS
 - Disseminated intravascular coagulation (DIC)
 - HELLP syndrome
 - Cardiac valvular disease
- Drug induced:
 - Decreased platelet production:

- Chemotherapy
- Thiazide diuretics
- Ethanol
- Estrogen
- _ Increased destruction of platelets:
 - Aspirin
 - Heparin
 - Chlorpropamide
 - Chloroquine
 - Gold salts
 - Sulfonamides
 - Insecticides



TREATMENT

PRE HOSPITAL

Stabilize ABCs

- Significant oral or laryngeal bleeding may affect airway.
- CNS event may affect ability to control airway
- Establish IV access for cases of significant bleeding
- Control any bleeding with direct pressure

INITIAL STABILIZATION/THERAPY

- ABCs
- Stabilize life-threatening bleeding:
 - _ ICH:
 - Airway control
 - Neurosurgery consultation: Craniotomy not typically possible until platelet counts > 75 K
 - _ Hemorrhagic shock:
 - 2 large-bore IVs
 - Direct pressure for hemostasis
 - Resuscitation with blood transfusions and isotonic crystalloid
 - Medications and platelets for acute life-threatening episodes of bleeding:
 - IV high-dose dexamethasone or methylprednisolone
 - IVIG infusion
 - Platelet transfusions: 2–3 times the normal amount
 - _ Platelets typically infused after steroids and/or IVIG
 - _ Mucous membrane bleeding:
 - Topical agents
 - Other:

- IV Aminocaproic acid may be considered
- In rare cases, plasmapheresis has been proven helpful

ED TREATMENT/PROCEDURES

- Initial treatment options for ITP are based on:
 - _ Degree of thrombocytopenia
 - _ Severity of illness
 - _ Duration of symptoms
 - _ Age
 - _ Risk factors for bleeding (hypertension, peptic ulcer disease, vigorous lifestyle)
 - _ Hematologist preference
- Efficacy of treatment is demonstrated in terms of platelet recovery time and not morbidity or mortality.
- Specific treatment options:
 - _ Observation is recommended for children without bleeding complications or profound thrombocytopenia (< 20–30 K)
 - _ Profound thrombocytopenia (< 20 K):
 - High-dose corticosteroids: 75% response
 - IVIG: 80% response, but costly, so reserved in time critical emergencies
 - Anti-D IG: 70% response (used only in Rh⁺ patients)
 - _ Splenectomy:
 - 2nd-line therapy if inadequate response to a course of glucocorticoid therapy
 - Two-thirds of adult patients respond, three-fourths of pediatric patients respond.
 - No specific indications for emergent splenectomy

Pregnancy Considerations

- Differential diagnosis of thrombocytopenia during pregnancy:
 - _ Gestational thrombocytopenia (75%):
 - Usually of no clinical significance
 - Does not cause neonatal thrombocytopenia
 - Remits 1–2 wk after delivery
 - Platelets typically > 50 K
- HELLP syndrome (*hemolysis, elevated liver enzymes, and low platelets*)
- ITP (15%):
 - _ Platelets typically < 50 K
 - _ Maternal platelet count does not correlate to neonatal thrombocytopenia.
 - _ Treatment with steroids or IVIG does not alter incidence of neonatal thrombocytopenia.

- Degree of thrombocytopenia should not alter decision of vaginal birth vs. C-section.
- Measure neonatal CBC and check brain ultrasound for ICH.
- Treat neonatal thrombocytopenia with IVIG and steroids

MEDICATION

First Line

- Glucocorticoids (high dose over 2–3 wk):
 - Dexamethasone: 40 mg PO daily
 - Prednisone: 1–2 mg/kg/24 hr PO/day
 - Methylprednisolone: 1 g IV q8h (30 mg/kg/24 hr pediatric dose)
- IVIG: 1–2 g/kg IV × 1 dose and possibly repeated in 24 hr:
 - Used only for critical bleeding or when the need to acutely raise platelets is required such as emergency surgery
- Anti-D immunoglobulin: 50 µg/kg/24 hr IV:
 - Used only presplenectomy and in Rh⁺ patients

Second Line

- Chronic long-term suppression and steroid bolus therapy
- Immunosuppressive agents:
 - Azathioprine
 - Cyclosporin
 - Mycophenolate
 - Chemotherapeutic agents:
 - Vinca alkaloids
 - Cyclophosphamide
 - Combinational chemotherapy
- Other:
 - Rituximab: Monoclonal antibody directed against B cell antigens
 - Danazol: Antiandrogen
- Experimental:
 - Other monoclonal antibodies directed at B cells
 - Stem cell transplants
 - Thrombopoietin and thrombopoietin-like agonists



FOLLOW-UP

DISPOSITION

Admission Criteria

- Life-threatening bleeding regardless of platelet count

- Any bleeding with platelet count < 20 K
- Asymptomatic patient with platelet count < 20 K with issues of noncompliance or poor follow-up

Discharge Criteria

- Asymptomatic patients
- Patients with minor bleeding and platelets > 30 K

FOLLOW-UP RECOMMENDATIONS

Hematology referral is indicated in all cases (either outpatient or inpatient consultation)

PEARLS AND PITFALLS

- Before low platelet counts are evaluated, pseudothrombocytopenia should be excluded.
- Spontaneous bleeding usually does not occur until platelet counts are < 10 K.

ADDITIONAL READING

- British Committee for Standards in Haematology General Haematology Task Force. Guidelines for the investigation and management of idiopathic thrombocytopenic purpura in adults, children and in pregnancy. *Br J Haematol.* 2003;120:574–596.
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- Neunert C, Lim W, Crowther M, et al. The American Society of Hematology 2011 evidence-based practice guideline for immune thrombocytopenia. *Blood.* 2011;117(16):4190–4207.
- Schipperus M, Fijnheer R. New therapeutic options for immune thrombocytopenia. *Neth J Med.* 2011;69(11):480–485.



See Also (Topic, Algorithm, Electronic Media Element)

HELLP Syndrome; Thrombotic Thrombocytopenic Purpura



CODES

ICD9

287.31 Immune thrombocytopenic purpura

ICD10

D69.3 Immune thrombocytopenic purpura

IMMUNIZATIONS

Garth D. Meckler

BASICS

DESCRIPTION

- Immunization enhances or initiates resistance to infectious diseases.
- Protection from immunization occurs through several mechanisms:
 - Passive immunization: Administration of purified antibodies or passive transfer of maternal antibodies through the placenta/breast milk.
 - Active immunization: Stimulates immune system, producing IgM antibodies after 7–10 days followed by IgG antibodies, peaking between 2 and 6 wk.
- Oral and nasal vaccines induce mucosal secretory IgA antibodies while parenteral vaccines may not. Improper administration (route, dose, bad storage, etc.) may result in decreased immunity.

ETIOLOGY

- Several types of vaccines are available:
 - Live attenuated (weakened) viruses (e.g., varicella [VZV]; measles, mumps, rubella [MMR]; rotavirus) replicate in the host and induce an immune response:
 - May cause serious infections in the immunocompromised.
 - Inactivated (or killed) vaccines (e.g., polio [IPV], hepatitis A [HepA], some influenza, pertussis) are safe in patients with compromised immune system.
 - Toxoid, subunit, or conjugate vaccines (e.g., diphtheria, tetanus, *Haemophilus influenzae b* [Hib], Human papilloma virus [HPV], Pneumococcus, Meningococcus) use antigenic portions of toxins, proteins, or carbohydrates from viruses or bacteria to induce immune response
 - Hepatitis B (HepB) vaccine uses recombinant DNA technology
- Several combination vaccines are also available but have an increased cost:
 - Pediarix (diphtheria and tetanus toxoids and acellular pertussis adsorbed [DTaP], HepB, and inactivated poliovirus vaccine [IPV] combined)
 - Comvax (HepB and Hib).
 - Pentacel (DTaP, IPV, and Hib).
 - Twinrix (HepA and HepB).
 - MMRV (MMR and varicella).

EPIDEMIOLOGY

- The incidence of several life-threatening illnesses has been markedly reduced with widespread immunization use:
 - Polio caused by wild-type viruses has been eliminated from the Western Hemisphere.

- Hib, diphtheria, and tetanus vaccines have nearly eliminated these invasive diseases among children in North America.
- The incidence of measles, rubella, and varicella has also declined; sporadic in unimmunized communities and foreign travelers to US.
- 7-valent and 13-valent conjugate pneumococcal vaccines (Prevnar 7 and Prevnar 13) have reduced invasive disease from 100–21 cases/100,000 for all types of pneumococcal infection and from 80–0.2/100,000 for vaccine serotypes (99% reduction).
- Rotavirus is a live oral vaccine
- Respiratory syncytial virus immune globulin given to high-risk patients
- Global surveillance of influenza activity allows for annual production of vaccines against seasonal influenza. Inactivated vaccines are available for IM administration and live attenuated virus vaccines can be given via the nasal route.
- Immunization recommendations and schedules are based on epidemiology, individual risks for disease and exposure, as well as vaccine safety and efficacy:
 - Infants and young children are vaccinated against common childhood diseases, but some vaccines are not immunogenic (e.g., pneumococcal capsular polysaccharide antigen vaccine) or may be dangerous (e.g., MMR, VZV) in infants.
 - Pregnant women are recommended to receive Tdap and inactivated influenza, but should not receive other live virus vaccines.
 - Specific recommendations exist for other at-risk groups including international travelers, the elderly, health care workers, and immunocompromised individuals.



DIAGNOSIS

SIGNS AND SYMPTOMS

- Concern for vaccine-associated adverse events: The most common events associated with vaccination are mild and include local reactions (pain, swelling, erythema) and/or fever (usually within 7–10 days):
 - Local reactions can occur with almost any injected vaccine, but are particularly common with tetanus, diphtheria, and pertussis (particularly after repeat doses), Hib (can cause sterile abscesses in young infants), VZV, HPV, and pneumococcus.
 - Fever may follow immunization with rotavirus (40–43%), conjugate pneumococcus (24–35%), HPV (10–13%), MMR or MMRV (more common in the latter), and meningococcal vaccine. Influenza vaccine may cause fever 6–24 hr after administration.
 - Rash may be seen as a rare side effect of VZV and MMRV, MMR may cause transient rash or fever 6–12 days after immunization.

- Neurologic symptoms are rare but most commonly seen after DTP vaccines (fussiness, inconsolable crying, drowsiness, brief seizures without fever, hypotonic–hypo-responsive episodes, encephalopathy, and Guillain–Barré syndrome). Headache may follow vaccination with HepB or meningococcus; and MMR and MMRV are associated with febrile seizures in children and rare reports of encephalopathy. Guillain–Barré has also been linked to influenza vaccine in adults.
- Vomiting and diarrhea are rare adverse effects of rotavirus vaccine in infants.
- MMR has been associated with arthralgias in adult women, and rare hematologic adverse events such as thrombocytopenia.
- Live attenuated influenza (nasal) has been associated with mild respiratory symptoms in adults and asthma exacerbations in children with a history of asthma.

ESSENTIAL WORKUP

- ED visits for any reason present an opportunity to review immunization status and provide appropriate follow-up. Take a history of status of immunizations:
 - If incomplete, take a good history as to the reason why immunizations have not been administered.
- True contraindications to vaccination:
 - Anaphylactic reaction to a previous dose of the vaccine or:
 - Anaphylaxis to baker’s yeast is a contraindication to HepB vaccine.
 - Anaphylaxis to chicken or egg protein is a contraindication to influenza vaccine (but not MMR).
 - Anaphylaxis to neomycin or gelatin is a contraindication to MMR vaccine.
 - Anaphylaxis to neomycin, streptomycin, or polymyxin is a contraindication to IPV vaccine.
 - Specific reactions within 48 hr of vaccine of a previous vaccine:
 - Severe, inconsolable screaming for 3 hr
 - Distinctive high-pitched cry
 - Hypo-responsive episode
 - Temperature >40.5°C unexplained by other cause
 - Severe local reaction involving the circumference of the injected limb unless owing to inadvertent SC injection
 - Encephalopathy within 7 days of vaccine:
 - Severe acute neurologic illness with prolonged seizures and/or unconsciousness and/or focal signs
 - Progressive neurologic disease excluding epilepsy
- Reasons to defer vaccine administration:
 - Moderate or severe acute disease regardless of fever.
 - Congenital or acquired immunodeficiency (e.g., HIV,

malignancy/chemotherapy associated): Possible risk from live attenuated vaccines such as VZV, MMR, and influenza. Caution should be used when considering these vaccines for healthy individuals in close contact with the immunocompromised.

- Pregnancy is a contraindication to live attenuated virus vaccines including VZV and HPV; inactivated virus (influenza) and conjugate vaccines (DTaP) are thought to be safe.
- Recent convulsion is a relative contraindication to pertussis.
- Recent administration of immune globulin may lessen the efficacy of vaccinations
- Vaccines may be given with the following:
 - Mild acute illness with or without fever
 - Mild to moderate local reaction (i.e., swelling, redness, soreness), low-grade or moderate fever after previous dose
 - Current antimicrobial therapy
 - Convalescent phase of illness
 - Premature birth (HepB vaccine is an exception)
 - Recent exposure to an infectious disease
 - History of penicillin allergy, other nonvaccine allergies, relative with allergies, receiving allergen extract immunotherapy
 - HIV-infected children who are either asymptomatic or not severely immunocompromised should be vaccinated.



TREATMENT

PRE HOSPITAL

Attention should be focused on the airway, breathing, and circulation.

INITIAL STABILIZATION/THERAPY

Initial medications for anaphylactic reaction to vaccines include IM or IV epinephrine, diphenhydramine, albuterol for wheezing, and IV fluids for hypotension.

ED TREATMENT/PROCEDURES

- Treat anaphylaxis with epinephrine, antihistamines, albuterol and IV fluids as indicated.
- Treatment of potential exposure to infectious disease or contaminated wounds follows specific guidelines for active or passive immunization.
- Treatment of adverse reactions depend on symptoms:
 - Local reactions at the injection site can be treated with cold compresses, analgesics, or antipruritics. Control bleeding with a pressure dressing.
 - Treat fever, headaches, myalgias, and arthralgias with acetaminophen or ibuprofen.

- Treat ongoing seizures with benzodiazepines
- Consider prophylaxis with acetaminophen at the time of injection of vaccines and again 4–8 hr later:
 - Children who receive varicella vaccine should avoid salicylates for 6 wk post vaccination because of the association of varicella infection and salicylates to Reye syndrome.
- Specific discussion with the parents is required to review the risks and benefits of tetanus vaccination, particularly given the frequent occurrence of trauma and the need to provide both passive and active immunity at that time:
 - Document in the chart that the risks and benefits have been thoroughly discussed. A formal informed consent is used in some settings.
 - The National Childhood Vaccine Injury Act requires that a copy of the Vaccine Information Statements be provided before administering each dose of the vaccine.

MEDICATION

- Acetaminophen 15 mg/kg/dose q4–6h PO
- Ibuprofen 10 mg/kg/dose q6–8h PO



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients with serious adverse reactions following immunization should be admitted.
- Patients with anaphylaxis and encephalopathy may require admission to a pediatric ICU.
- Unexpected adverse events should be reported to the Vaccine Adverse Event Reporting System.

Discharge Criteria

Patients may be discharged home after routine immunizations unless an immediate adverse reaction occurs. It is essential that follow-up with the primary care physician be arranged to complete immunizations.

PEARLS AND PITFALLS

- Failure to continue diphenhydramine for 48 hr following an allergic reaction. Steroids may also be considered.
- Failure to recognize egg allergy as a contraindication to influenza vaccine.

ADDITIONAL READING

- Advisory Committee on Immunization Practices, Centers for Disease Control and Prevention. Recommended Guidelines. Available at: <http://www.cdc.gov/vaccines/recs/acip/>
- American Academy of Pediatrics: Report of the Committee on Infectious Diseases. 29th ed. Elk Grove, CA: ILL; 2012.
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- Payne DC, Boom JA, Staat MA, et al. Effectiveness of pentavalent and monovalent rotavirus vaccines in concurrent use among US children <5 years of age, 2009–2011. *Clin Infect Dis*. 2013;57(1):13–20.
- Swamy GK, Garcia-Putnam R. Vaccine-preventable diseases in pregnancy. *Am J Perinatol*. 2013;30(2):89–97.



See Also (Topic, Algorithm, Electronic Media Element)

- Anaphylaxis
- Encephalitis
- Hepatitis
- Influenza
- Measles
- Mumps
- Pertussis
- Polio
- Rabies
- Rubella
- Seizure, Adult
- Seizure, Pediatric
- Tetanus
- Varicella



CODES

ICD9

- V07.2 Need for prophylactic immunotherapy
- V15.83 Personal history of underimmunization status

ICD10

- Z23 Encounter for immunization
- Z28.3 Underimmunization status

IMMUNOSUPPRESSION

Lara K. Kulchycki

BASICS

DESCRIPTION

Congenital or acquired deficiency in the ability to fight infection:

- Antibody production (B cell)
- Cellular immunity (T cell)
- Phagocytic dysfunction
- Complement deficiency
- Breach of skin/mucosal barriers

ETIOLOGY

- Congenital disorders
- Immunosuppressive medications
- Aging:
 - Immunosenescence
 - Poor circulation and wound healing
- Chronic (lung, kidney, or heart) disease
- HIV infection:
 - CD4 count determines susceptibility to pathogens
- Diabetes:
 - Hyperglycemia impairs immune response
 - Vascular insufficiency
- Malnutrition:
 - Poverty
 - Alcoholism and drug abuse
 - Eating disorders
- Asplenia:
 - Functional asplenia (sickle cell disease) or surgical splenectomy increases risk of infection with encapsulated organisms
- Organ transplantation:
 - Antirejection medications suppress immune response
 - Infections may be donor derived, recipient derived, or nosocomial
 - Increased risk of viral pathogens, such as cytomegalovirus, Epstein–Barr virus, and human herpes viruses
 - Time elapsed since transplantation is crucial, as different patterns of infection arise in early, intermediate, and late posttransplantation periods
- Malignancy
- Chemotherapy:

- Increased risk of infection with pyogenic bacteria and fungi
- Infection risk related to length and severity of neutropenia
- Neutropenia:
 - Defined as absolute neutrophil count (ANC) $< 500/\text{mm}^3$ or $< 1,000/\text{mm}^3$ with an anticipated nadir of $< 500/\text{mm}^3$
 - In US, gram-positive organisms are the leading etiology of infection
 - Gram-negative organisms are somewhat less common but often virulent
 - Polymicrobial infections are increasingly frequent
 - Anaerobic isolates remain relatively rare
 - The risk of fungal pathogens increases with prolonged neutropenia (> 1 wk), prior use of broad-spectrum antibiotics, or intense chemotherapy

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Fever may be the only symptom of a life-threatening infection in an immunocompromised host
- Perform a careful review of systems to identify any localizing symptoms
- Identify risk factors for nosocomial infections, such as recent hospitalization or nursing home residence
- Ask about close contacts with transmissible illnesses, such as influenza
- Review medications for the presence of immunosuppressive agents, such as steroids
- Recognize that prophylactic medicines, such as trimethoprim/sulfamethoxazole or fluconazole, may alter both the spectrum of likely pathogens and their resistance patterns

Physical-Exam

- Examine the patient from head to toe
- Some clinicians advise avoiding digital rectal exams in patients with febrile neutropenia
- Inflammation may be subtle or absent:
 - Surgical abdomen without peritoneal signs
 - Meningitis without nuchal rigidity
 - Infected wounds or indwelling lines without induration, erythema, or purulent discharge

ESSENTIAL WORKUP

- Choice of studies must be tailored to the patient and the presenting complaint
- Test interpretation may be difficult since inflammatory responses are often

blunted in immunosuppressed patients:

- Pneumonia without radiographic infiltrates
- UTIs without pyuria
- Meningitis without CSF pleocytosis

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC with differential:
 - Identify leukocytosis, left shift, bandemia, or neutropenia
 - Risk of infection begins to increase once ANC $< 1,000/\text{mm}^3$
- Blood cultures:
 - 2 sets of bacterial cultures
 - Draw 1 culture from an indwelling line, if present
 - Obtain fungal cultures if indicated
- Urinalysis/urine culture:
 - Obtain by clean catch, if possible, as catheterization may introduce infection
- Serum lactate:
 - Useful for identifying occult hypoperfusion in sepsis
- Arterial blood gas:
 - Useful in determining the need for steroids in suspected cases of *Pneumocystis jirovecii* pneumonia (PCP)
- Pregnancy testing in women of childbearing age

Imaging

- Chest x-ray recommended if patient is neutropenic, hypoxic, or has abnormal pulmonary signs
- Further imaging, such as CT or MRI, can be tailored to the patient's presentation and risk factors

Diagnostic Procedures/Surgery

- Lumbar puncture should be performed if there is a clinical suspicion for meningitis:
 - Check platelet counts and coagulation studies prior to procedure if thrombocytopenia or coagulopathy is suspected
 - Consider cryptococcal antigen testing even in the absence of CSF pleocytosis

DIFFERENTIAL DIAGNOSIS

- Infection:
 - Oropharynx
 - Sinuses
 - Lung

- GI tract
- Perineum/anus
- Urinary tract
- Skin/soft tissue
- Bone
- Indwelling catheters/devices
- Noninfectious etiology of fever:
 - Drug fever
 - Allograft rejection
 - Malignancy
 - Vasculitis
 - Rheumatologic disease
 - Pulmonary embolism
 - Thyroid dysfunction
 - Blood product transfusion



TREATMENT

PRE HOSPITAL

- Establish IV access
- IV fluid bolus

INITIAL STABILIZATION/THERAPY

- Aggressive fluid resuscitation for patients with hypovolemia
- Goal-directed therapy for patients with sepsis
- Ultrasound can be used to evaluate the IVC (caval index) to estimate volume status as well as screen for malignant pericardial tamponade
- Administer pressors for hypotension that fails to respond to IV fluids:
 - Dopamine 5–20 $\mu\text{g}/\text{kg}/\text{min}$ IV
 - Norepinephrine 2–12 $\mu\text{g}/\text{min}$ IV

ED TREATMENT/PROCEDURES

- Institute appropriate infection control precautions, such as neutropenic or contact precautions
- Rapidly collect appropriate cultures and administer broad-spectrum antibiotics
- Most patients with febrile neutropenia are admitted, but low-risk patients with fever may be candidates for outpatient treatment
- Low risk:
 - Age < 60 yr
 - Outpatient status at time of fever
 - ANC > 100 cells/mm³
 - Duration of neutropenia < 7 days

- Expected resolution of neutropenia < 10 days
- Well appearing
- Stable vital signs
- No change in mental status
- No dehydration
- Lack of significant comorbid conditions:
 - o Chronic pulmonary disease
 - o Diabetes
 - o Organ failure
- Disease in remission
- No history of fungal infections
- Normal chest x-ray

MEDICATION

- Treatment regimens should, if possible, be tailored to the patient
- Empiric therapy with broad-spectrum agents must be rapidly administered in febrile neutropenia or sepsis
- Oral antibiotic therapy:
 - Produces comparable results in low-risk adults with febrile neutropenia
 - Ciprofloxacin 750 mg PO BID + amoxicillin–clavulanate 875 mg PO BID
- Parenteral monotherapy options:
 - Ceftazidime: 2 g IV q8h (peds: 50 mg/kg IV q8h)
 - Cefepime: 2 g IV q8h (peds: 50 mg/kg IV q8h)
 - Imipenem–cilastatin: 500 mg IV q6h (peds: Dose based on age/weight)
 - Meropenem: 1 g IV q8h (peds: Dose based on age/weight)
 - Piperacillin–tazobactam: (Less well studied in neutropenia) 4.5 g IV q6h (peds: Dose based on age)
- For high-risk patients, consider adding an aminoglycoside (AG) for synergism:
 - Gentamicin: Dose based on Cr clearance (peds: Dose based on age)
 - AG use increases risk of adverse events, such as acute renal failure and ototoxicity
- Empiric vancomycin is usually not indicated:
 - Consider adding if suspected line sepsis or history of methicillin-resistant *Staphylococcus aureus*
 - Vancomycin: 1 g IV q12h (peds: Dose based on age/weight)
- Anaerobic coverage may be added if there is concern for oral or abdominal/perianal infections:
 - Clindamycin: 600–900 mg IV q8h (peds: Dose based on age)



FOLLOW-UP

DISPOSITION

Admission Criteria

- ANC < 100 cells/mm³
- Immunocompromised patients with infection who do not meet low-risk criteria
- Patients with inadequate access to outpatient medical care
- Maintain lower admission criteria for:
 - Elderly
 - Diabetics
 - Children

Discharge Criteria

- Low-risk patients that are well appearing and can tolerate oral antibiotics and fluids may be considered for outpatient management
- Discuss the disposition with the responsible hematology/oncology, infectious disease, or transplant physician prior to discharge

FOLLOW-UP RECOMMENDATIONS

24-hr follow-up must be available in order to reassess the patient and monitor culture results

PEARLS AND PITFALLS

- Failure to learn institutional/regional infection and antibiotic resistance patterns
- Failure to recognize that a vague symptom or isolated fever may be the sole warning sign of serious infection in an immunocompromised host
- Failure to administer broad-spectrum antibiotics rapidly in febrile neutropenia or sepsis
- Failure to review the patient's previous microbiology results
- Failure to involve the appropriate primary care and specialty physicians who are familiar with the patient and can help tailor therapy and ensure follow-up

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Sepsis



CODES

ICD9

- 279.2 Combined immunity deficiency
- 279.3 Unspecified immunity deficiency
- 279.8 Other specified disorders involving the immune mechanism

ICD10

- D83.8 Other common variable immunodeficiencies
- D84.1 Defects in the complement system
- D84.9 Immunodeficiency, unspecified

IMPETIGO

Irving Jacoby



BASICS

DESCRIPTION

- Impetigo is a common infection of the skin
- Primary infection:
 - Infection of minor breaks in the skin
- Secondary infection:
 - Infection of previously existing skin lesions, known as “impetiginization”
- Most prevalent in children aged 2–5 yr
- More common in summer months and warm and humid climates
- Predisposing factors:
 - Minor trauma, esp. around nose area
 - Burns
 - Insect bites
 - HIV infection
 - Diabetes mellitus
 - Existing skin disease
 - Varicella infection
- Complications:
 - Acute poststreptococcal glomerulonephritis
 - 1–5% in patients with nonbullous impetigo
 - Sepsis
 - Cellulitis
 - Endocarditis
 - Toxic shock syndrome
 - Staphylococcal scalded skin syndrome (SSSS)

ETIOLOGY

- Classic impetigo:
 - The result of bacteria entering through traumatic skin portal from scratch, abrasion, or insect bite
 - Caused by *Staphylococcus aureus*, group A β -hemolytic streptococci, or both
 - Often associated with poor hygiene
 - Treatment of both streptococci and *S. aureus*
- Bullous impetigo:
 - Caused by *S. aureus*, phage group II
 - Epidermal cleavage is caused by staphylococcal exfoliative toxins A, B, and D, which are serine proteases that bind and cleave desmoglein 1, an

Pregnancy Considerations

Zinc supplementation to mother during pregnancy may lead to decreased incidence in infants

DIAGNOSIS

SIGNS AND SYMPTOMS

- **Classic (nonbullous) impetigo:**
 - Begins as a single 2–4 mm erythematous macule or papule that may evolve into a vesicle or pustule, on a red base
 - Rupture of the vesicle, usually within 24 hr, leaves a honey-colored, dark brown, or reddish-black exudative crust
 - Highly contagious
 - Often pruritic, may be spread from the original site of infection by scratching
 - Mild lymphadenopathy may be seen, usually not lymphadenitis
 - Systemic manifestations are rare
 - Rheumatic fever does not occur following streptococcal skin infection
 - Skin infections with nephritogenic strains of group A streptococci are major antecedents of poststreptococcal glomerulonephritis
- **Bullous impetigo:**
 - Occurs most commonly in the neonate, but can occur at any age
 - Lesions begin as vesicles that turn into flaccid bullae with clear yellow fluid
 - Nikolsky sign is absent
 - Large, fragile bullae rupture quickly, leaving only a shiny, erythematous base with peeling edges

History

Fever and constitutional symptoms are uncommon

Physical-Exam

- **Common sites of infection:**
 - Face
 - Extremities
 - Scalp
- The diagnosis is made based on observation of the classic exam findings, especially appearance, and distribution

ESSENTIAL WORKUP

Cultures of fluid from bullae or pustules may be considered in those cases refractory to

traditional therapy or if methicillin-resistant *S. aureus* (MRSA) is of particular concern during an outbreak

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Antistreptolysin O titer after streptococcal impetigo is scant
- AntiDNase B response readily occurs; 90% of patients with nephritis complicating streptococcal skin infections have elevated titers
- Urinalysis to evaluate for hematuria or proteinuria which might suggest onset of poststreptococcal glomerulonephritis

Imaging

Not usually indicated

Diagnostic Procedures/Surgery

- Biopsy is generally not needed for diagnosis
- If biopsy is performed:
 - Subcorneal epidermal cleavage plane
 - Inflammatory infiltrate of neutrophils and lymphocytes in the upper dermis
 - Subcorneal blisters with occasional acantholytic cells
- Gram stain of blister fluid may show PMNs and gram-positive cocci in chains or clusters

DIFFERENTIAL DIAGNOSIS

- Herpes simplex
- Varicella zoster (shingles)
- Atopic dermatitis
- Contact dermatitis
- Dermatophytosis
- Erysipelas
- Candidiasis
- Scabies
- Folliculitis
- Pediculosis
- Pemphigus vulgaris
- Bullous pemphigoid
- Seborrheic dermatitis
- Thermal burns
- Stevens–Johnson syndrome
- Bullous erythema multiforme
- SSSS, caused by systemic spread of exfoliatin in susceptible individuals
- Pemphigus neonatorum (Ritter disease), or SSSS in the newborn

- Toxic epidermal necrolysis
- Cutaneous anthrax



TREATMENT

PRE HOSPITAL

- Apply dressings to cover for transport
- Gloves must be worn, as agents can be transmitted person to person
- Cautions:
 - _ Maintain universal precautions
 - _ Siblings of affected children in same household should be checked for lesions

INITIAL STABILIZATION/THERAPY

In healthy children or adults, classic or bullous impetigo is not a life-threatening condition and does not require resuscitative measures

ED TREATMENT/PROCEDURES

- Small nonbullous lesions may be treated with topical therapy alone
- Larger, widespread lesions, or presence of bullous impetigo, or presence of lymphadenopathy should be treated with systemic therapy
- Systemic treatment should include a β -lactamase-resistant penicillin, cephalosporin, or macrolide antimicrobial for 10 days:
 - _ If no response, check for MRSA and switch antibiotic to cover for MRSA
- Systemic antibiotic advisable during epidemics of acute poststreptococcal glomerulonephritis or in communities with widespread MRSA
- Local care should include cleansing, removal of crusts, and application of wet dressings to the affected areas

MEDICATION

- All treatment regimens are 10 days, except for topical retapamulin, which is used for 5 days and may enhance compliance, and oral azithromycin, which lasts 9 days when taken for 5 days
- Avoid use of erythromycin if high incidence of erythromycin resistance of streptococci or staphylococci in the community
- Oral:
 - _ Amoxicillin/clavulanic acid: 250 mg PO q8h (peds: 30 mg/kg/d PO in div. doses q8h)
 - _ Azithromycin: 500 mg PO on day 1; 250 mg PO days 2–5 (peds: 10 mg/kg PO on day 1; 5 mg/kg PO days 2–5)
 - _ Cephalexin: 500 mg PO QID (peds: 25–50 mg/kg/d PO in div. doses q8–12h)
 - _ Clarithromycin: 250 mg PO q12h (peds: 15 mg/kg in div. doses q12h)
 - _ Clindamycin: 150 mg PO TID (peds: 5 mg/kg TID)
 - _ Dicloxacillin: 250 mg PO q6h (peds: 25–50 mg/kg/d PO in div. doses q6h)

- Doxycycline: 100 mg PO q12h when MRSA suspected: (Peds over 8 yr and under 45 kg, give 4.5 mg/kg/d in div. doses q12h). Not recommended for children under 8 yr
- Erythromycin ethylsuccinate: 250 mg PO q6h (peds: 40 mg/kg/d PO in div. doses q6h)
- Trimethoprim–sulfamethoxazole DS: 1 tab PO BID for 10 days (peds: >2 mo; 4 mg/kg of trimethoprim component PO q12h for 10 days); Useful when MRSA suspected
- Linezolid: 600 mg PO BID—expensive, used only for multiallergic patients or MRSA (peds: Not approved for children)
- Topical:
 - Mupirocin (2% ointment [Bactroban]): Adult and peds: Apply topically to affected area TID (nonbullous impetigo only) for 10 days.
 - Retapamulin (1% ointment) (Altabax): Adult and peds >9 mo: Apply topically to affected areas BID for 5 days.

First Line

Topical:

- Mupirocin: (up to 46% of US 300 strain of Community-acquired MRSA carry gene encoding for resistance to mupirocin)

Second Line

• Topical:

- Retapamulin (package insert says not for MRSA due to not enough cases studied, but has shown effect on mupirocin-resistant MRSA)

• Oral antibiotics:

- Amoxicillin/clavulanic acid
- Cephalosporins
- Dicloxacillin
- Erythromycin
- Doxycycline



FOLLOW-UP

DISPOSITION

Admission Criteria

- Admission for impetigo alone is rarely necessary
- Patients with disease that is widespread, especially widespread bullae, or with larger areas of denuded skin, and dehydration, or refractory to outpatient therapy
- Toxic, ill-appearing, or immunocompromised patients require admission, as do neonates suspected of having sepsis

- Nephritis may already be present at time patients present for care if presentation is delayed > 4–5 days
- More typically, nephritis, if seen, occurs 2–4 wk after a streptococcal skin infection

Discharge Criteria

- Patients should not be toxic appearing
- Patients/caregivers should be able to comply with the recommended treatment regimen
- Follow-up for re-evaluation

Issues for Referral

Periorbital edema, leg swelling, or hematuria or proteinuria should suggest poststreptococcal glomerulonephritis and referral to nephrologist

FOLLOW-UP RECOMMENDATIONS

- Follow-up with primary care physician should be arranged to assure resolution without complications
- Return for failure of lesions to respond
- Return for development of hematuria, periorbital edema, or leg swelling

PEARLS AND PITFALLS

- Treat with systemic antibiotics in the presence of bullous impetigo or if lymphadenopathy is present
- Increasing antibiotic resistance continues to limit ability to use historic standard antibiotic protocols
- Mupirocin resistance exists, should be suspected in failures to respond and switch to retapamulin
- Cultures and sensitivity must be checked for recalcitrant lesions
- Relapse, representing reinfection, may occur if other affected family members are not treated at the same time

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- Wolfson AB, Hendey GW, Ling LJ, et al., eds. *Harwood Nuss' Clinical Practice of Emergency Medicine*. 5th ed. Philadelphia, PA: Lippincott; 2010.



See Also (Topic, Algorithm, Electronic Media Element)

- [Cellulitis](#)
- [Erysipelas](#)
- [Toxic Epidermal Necrolysis](#)



CODES

ICD9

- [684 Impetigo](#)
- [694.3 Impetigo herpetiformis](#)
- [704.8 Other specified diseases of hair and hair follicles](#)

ICD10

- [L01.00 Impetigo, unspecified](#)
- [L01.01 Non-bullous impetigo](#)
- [L01.03 Bullous impetigo](#)

INBORN ERRORS OF METABOLISM

David A. Perlstein • David H. Rubin

BASICS

DESCRIPTION

- Defect in the type, amount, and toxicity of metabolites that accumulate due to an inherited abnormal pathway in children; result in a variety of clinical findings; > 400 human diseases are caused by inborn errors of metabolism
- Epidemiology:
 - Incidence:
 - Variable: 1:10,000–1:200,000 births
- Genetics:
 - Common inherited metabolic diseases:
 - Amino acid disorders
 - Urea cycle defects
 - Organic acidemias
 - Defects in fatty acid oxidation
 - Mitochondrial fatty acid defects and carnitine transport defects
 - Mitochondrial disease
 - Carbohydrate disorders
 - Mucopolysaccharidoses
 - Sphingolipidoses
 - Peroxisomal disorders
 - Protein glycosylation disorders
 - Lysosomal disorders
 - Rhizomelic chondrodysplasia punctata
- Pathophysiology:
 - Related to defect in a metabolic pathway

ETIOLOGY

Diverse group of disorders involving genetic deficiency of an enzyme of an intermediary metabolite or a membrane transport system.

DIAGNOSIS

SIGNS AND SYMPTOMS

- Disorders may present with either a rapid decompensation or a chronic indolent course
- Neonates, initial presentation:
 - Asymptomatic

- Hypothermia (mitochondrial defects)
- Hypotonia/hypertonia (peroxisomal disorders)
- Apnea (urea cycle defects, organic acidosis)
- Seizures (peroxisomal disorders, glucose transporter defects)
- Coma (numerous)
- Vomiting (numerous)
- Poor feeding, growth (numerous)
- Jaundice (galactosemia, Niemann–Pick C)
- Hypoglycemia (galactosemia, maple syrup urine)
- Dysmorphic features (lysosomal storage disorders, congenital adrenal hyperplasia, Smith–Lemli–Opitz)
- Older children, untreated:
 - Failure to thrive (urea cycle defects)
 - Dehydration (organic acidosis)
 - Vomiting (urea cycle defects and others)
 - Diarrhea (numerous)
 - Food intolerance (lipid defects, amino acid defects)
 - Lethargy (urea cycle defects)
 - Ataxia (urea cycle defects)
 - Seizures (numerous)
 - Mental retardation (phenylketonuria and others)

History

Complete history of current and concomitant illness:

- Newborn screening
- Dietary
- Family
- Consanguinity
- Other

Physical-Exam

- Abnormal odor
- Altered mental status
- Tachypnea
- Abnormal facies
- Cataract
- Cardiomyopathy
- Hepatomegaly
- Splenomegaly
- Dermatitis
- Jaundice

ESSENTIAL WORKUP

Key is to consider in differential diagnosis:

- Deteriorating neurologic status
- Unexplained failure to thrive, with dehydration, persistent vomiting, or acidosis
- Shock unresponsive to conventional resuscitative measures

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Bedside glucose determination
- Electrolytes, BUN/creatinine, glucose
- CBC with differential
- Calcium level
- LFTs, fractionated bilirubin, PTT
- Arterial or venous blood gas
- Lactate and pyruvate level
- Uric acid
- Urinalysis
- Chemistries, as indicated:
 - Ammonia level
 - Quantitative serum amino acids
 - Urine organic and amino acids
- Cultures:
 - Blood
 - CSF

Imaging

- CT scan of head for altered mental status
- CXR

Diagnostic Procedures/Surgery

Lumbar puncture

DIFFERENTIAL DIAGNOSIS

- Often misdiagnosed as sepsis, dehydration, failure to thrive, toxic ingestion, or nonaccidental trauma
- Infection:
 - Sepsis
 - Meningitis
 - Encephalitis
- Metabolic:
 - Reye syndrome
 - Hepatic encephalopathy
 - Hyperinsulinemia

- Hormonal abnormality
- Renal:
 - Renal failure
 - Renal tubular acidosis
- Toxic ingestion
- CNS mass lesions
- Nonaccidental trauma



TREATMENT

PRE HOSPITAL

ALERT

- ABCs
- Bedside glucose
- IV glucose infusion takes precedence over fluid boluses unless patient in shock. Correction can occur concurrently.
- Avoid lactated Ringer solution.
- Keep child NPO.

INITIAL STABILIZATION/THERAPY

For altered mental status, administer Narcan, glucose (ideally after Accu-Chek and thiamine)

ED TREATMENT/PROCEDURES

- Establish airway, breathing, and circulation.
- For fluid boluses, use normal saline and avoid lactated Ringer and avoid hypotonic fluid.
- Initiate IV glucose at rate of 8–10 mg/kg/min to prevent catabolism:
 - Corresponds to D₁₀ at 1.5 times maintenance.
 - Do not delay glucose infusion to give a “bolus” of isotonic saline; may be given concurrently in a child in shock.
 - If patient is severely hypoglycemic, give IV glucose bolus of D₂₅.
- Rehydrate if patient is hypoglycemic:
 - Restore normal acid–base balance.
- Administer bicarbonate if pH is < 7.0:
 - Initiate dialysis if severe acidosis does not improve quickly.
- Increase urine output to help in removal of some toxins.
- Initially, stop all oral intake; amino acid metabolites may be neurotoxic.
- Treat severe hyperammonemia (≥ 500 –600 mmol/L) with immediate dialysis or with ammonia-trapping drugs such as:
 - Arginine hydrochloride

- Sodium benzoate
- Sodium phenylacetate
- Sodium phenylbutyrate
- Doses vary with disease; consult metabolic physician before use.
- Identify and treat intercurrent or precipitating infection/illness.
- Consult metabolic physician when any child presents with suspected inherited metabolic disease.

MEDICATION

- D₂₅: 2–4 mL/kg IV
- Sodium bicarbonate: 1–2 mEq/kg IV
- Other disease-specific drugs, including pyridoxine and levocarnitine as indicated

First Line

Glucose:

- 0.9% NS at 20 mL/kg

Second Line

Bicarbonate therapy for pH <7.0:

- Hemodialysis as needed



FOLLOW-UP

DISPOSITION

Admission Criteria

- Infants and children presenting with new onset of suspected inherited metabolic disease
- Significant urinary ketones or not tolerating oral intake
- ICU:
 - Significant altered mental status
 - Severe or persistent acidosis
 - Unresponsive hypoglycemia
 - Hyperammonemia
- Transfer to specialized pediatric center may be indicated.

Discharge Criteria

- Normal mental status
- Normal hydration with unremarkable labs
- No evidence of significant intercurrent illness
- Close follow-up arranged with primary care physician

Issues for Referral

Neurodevelopment:

- Diet
- Medications

FOLLOW-UP RECOMMENDATIONS

- Primary care physician
- Metabolic disease specialist

PEARLS AND PITFALLS

Watch for dehydration:

- Treat dehydration with normal saline fluid bolus:
 - _ Follow glucose level carefully; avoid hypoglycemia.
 - _ Use bicarbonate cautiously and only consider if pH < 7.0.
 - _ Hemodialysis may be necessary for hyperammonemia.

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CODES

ICD9

- 270.6 Disorders of urea cycle metabolism
- 270.9 Unspecified disorder of amino-acid metabolism

- 277.9 Unspecified disorder of metabolism

ICD10

- E72.9 Disorder of amino-acid metabolism, unspecified
- E72.20 Disorder of urea cycle metabolism, unspecified
- E88.9 Metabolic disorder, unspecified

INFLAMMATORY BOWEL DISEASE

Shayle Miller



BASICS

DESCRIPTION

- Idiopathic, chronic inflammatory diseases of intestines, which can involve extraintestinal sites as well.
- Differentiation between ulcerative colitis (UC) and Crohn's is not always clear; intermediate forms of inflammatory bowel disease (IBD) exist.
- May present as initial onset of disease or exacerbation of existing disease.
- Maintain high index of suspicion owing to frequent, subtle presentation of Crohn's disease.
- Pediatric considerations:
 - Can occur in 1st few years of life.
 - Extraintestinal manifestations may predominate.
- Differences between Crohn's and UC:
 - Rectum almost always involved in UC with continuous inflammation proximally.
 - Small intestine is not involved in UC.
 - Crohn's can occur anywhere from mouth to anus, often with normal GI tract segments between affected areas.
 - Crohn's involves transmural inflammation, whereas UC is confined to submucosa.
- Similarities between Crohn's and UC:
 - Higher rate of colon cancer with disease > 10 yr.
 - Bimodal age distribution, with early peak between teens and early 30s and 2nd peak about age 60 yr.
- Crohn's disease clinical pattern:
 - Ileocecal: ~ 40%
 - Small bowel: ~ 30%
 - Colon: ~ 25%
 - Other: ~ 5%
- UC clinical pattern on presentation:
 - Pancolitis: 30%:
 - Most severe clinical course
- Proctitis or proctosigmoiditis: 30%:
 - Relatively mild clinical course
- Left-sided colitis (up to splenic flexure): 40%:
 - Moderate clinical course

ETIOLOGY

- Unknown
- Crohn's disease and UC are separate entities with common genetic predisposition.
- A positive family history is very common.
- Multifactorial origin involving interplay among the following factors:
 - Genetic
 - Environmental
 - Immune
- Pathogenesis:
 - Gut wall becomes unable to downregulate its immune responses, ultimately resulting in chronic inflammation.
- There is no definitive evidence for the etiologic role of infectious agents.
- Psychogenic factors may play a role in some symptomatic exacerbations.

DIAGNOSIS

SIGNS AND SYMPTOMS

- Crohn's disease can present with any clinical correlates of chronic inflammatory, fibrostenotic, or fistulizing illness.
- UC may begin subtly or as catastrophic illness.
- Constitutional, GI, and extraintestinal manifestations are common with both Crohn's and UC.

History

- Constitutional:
 - Crohn's:
 - Low-grade fever
 - Night sweats
 - Weight loss
 - Fatigue
 - Pediatric: Growth or pubertal delay
 - UC:
 - Fever usually only in fulminant disease
 - Weight loss/fatigue
- GI:
 - Abdominal pain/tenderness—Crohn's disease:
 - Episodic
 - Periumbilical; may localize to right lower quadrant (RLQ) with ileal disease
 - Generalized with more diffuse intestinal involvement
 - Can localize to area of intra-abdominal abscesses or fistulous involvement

- Tenderness and distension suggest obstruction or toxic megacolon
- Abdominal pain/tenderness—UC:
 - More generalized than Crohn's disease
 - Often limited to predefecatory period
 - Tenderness with distension—suspect toxic dilation
- Stool:
 - Crohn's disease:
 - Mild, loose stool, rarely >5/day
 - ~50% bloody
 - UC:
 - Diarrhea is variable, can be severe.
 - Vast majority are bloody, sometimes with severe hemorrhage.
 - Mucus
 - Tenesmus and urgency are common.
- Nausea/vomiting:
 - Crohn's disease:
 - Obstruction common with ileocolonic disease
 - UC:
 - Obstruction rare
 - Diminished bowel sounds with toxic dilation
- Liver:
 - Sclerosing cholangitis can be seen.
 - Cholelithiasis can be seen in 35–60% of Crohn's.
- Renal:
 - Nephrolithiasis
 - Obstructive hydronephrosis
- Musculoskeletal:
 - Peripheral arthritis/artralgias—follows disease activity.
 - Pediatric—may be confused with juvenile rheumatoid arthritis, idiopathic growth failure, anorexia nervosa.

Physical-Exam

- Perianal:
 - Crohn's disease:
 - Perianal abscesses
 - Fissures—characteristically painless
 - Fistulas—seen in up to 50% of patients with colonic disease.
 - May present prior to other manifestations.
 - UC:
 - No perianal involvement
- RLQ pain/mass often mistaken for appendicitis.
- Severe toxicity/abdominal pain—must exclude toxic megacolon.
- Extraintestinal:

- Eye:
 - Uveitis
 - Episcleritis
 - Keratitis
- Oral:
 - Aphthous stomatitis
- Dermatologic:
 - Erythema nodosum
 - Pyoderma gangrenosum

ESSENTIAL WORKUP

- May present as initial onset of disease or exacerbation of existing disease.
- Maintain high index of suspicion because of subtle presentation of Crohn's disease.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Nothing diagnostic
- CBC:
 - Anemia secondary to chronic or acute blood loss
- Electrolytes, BUN/creatinine, glucose
- Stool exam:
 - Occult blood
 - *Clostridium difficile*
 - Fecal leukocytes may be present.
 - O & P and culture to rule out infectious cause of enteritis
- ESR is always elevated.
- Newer, investigational, serologic tests may have use as adjunctive diagnostic aids, screening tests, or predictors in therapy.

Imaging

- Lifetime radiation dose is cumulative and IBD patients have repeated exposure; consider MRI when available.
- Upright chest and abdominal radiographs for:
 - Toxic megacolon (>6 cm dilation)
 - Obstruction
 - Air in wall of colon (may indicate impending perforation)
 - Perforation—subdiaphragmatic air or free air outlining liver or gall bladder
- CT abdomen/MRI:
 - Distinguish abscess from localized inflammatory mass in Crohn's.
- Colonoscopy with biopsy can confirm diagnosis of UC or Crohn's:
 - Can be withheld with severe symptoms owing to perforation risk.
- Contrast imaging of small bowel, especially terminal ileum, may confirm diagnosis

of Crohn's.

- MRI can be useful in Crohn's perianal disease and avoids ionizing radiation.

DIFFERENTIAL DIAGNOSIS

- Infectious enteritis
- Pseudomembranous colitis (*C. difficile*)
- Appendicitis
- Diverticulitis
- Diverticulosis
- Functional bowel disease
- Lymphoma involving bowel
- Ischemic colitis
- Gonococcal or chlamydial proctitis
- HIV
- Colon cancer
- Vasculitis
- Amyloidosis



TREATMENT

PRE HOSPITAL

Vital sign stabilization as per BLS

INITIAL STABILIZATION/THERAPY

- IV 0.9% NS volume replacement if dehydrated
- Transfusion if significant blood loss

ED TREATMENT/PROCEDURES

- Nasogastric (NG) suction if obstruction or toxic dilation suspected
- Broad-spectrum antibiotics for fulminant UC or suspected perforation
- Consider steroid replacement if stress doses are required for those recently on oral steroids.
- Surgical evaluation indications:
 - Free perforation
 - Intestinal obstruction
 - Massive, unresponsive hemorrhage
 - Toxic dilation:
 - Not an absolute indication for surgery
 - Intensive medical management with small bowel decompression and close radiographic monitoring and surgical consultation
- Walled-off perforation with abscess:
 - Usually not an indication for emergent surgery
 - Careful observation for peritonitis

- **Medical therapy:**
 - Treatment is usually not initiated unless diagnosis is already established.
 - Refill or restart medications in patient with known disease.
 - ED-prescribed medical regimen should be individualized, and consultation with gastroenterologist strongly recommended:
 - Aminosalicylate (sulfasalazine/mesalamine) in mild to moderate case.
 - Antidiarrheal agent (diphenoxylate) is used—but withhold if severe disease or suspect toxic megacolon.
 - Steroid (prednisone, budesonide or hydrocortisone enema, ACTH) is used for moderate to severe disease.
 - Antibiotics (metronidazole and/or ciprofloxacin) aid in treatment of Crohn's with colon/perineal involvement.
 - Immunosuppressive agents (azathioprine, methotrexate) are used in severe disease.
 - Monoclonal antibodies neutralize cytokine tumor necrosis factor (TNF)- α and inhibit binding to TNF- α receptors (infliximab [Remicade]). Used as parenteral therapy in disease unresponsive to other modalities. Not an ED drug, but be aware of potential severe adverse reactions, infusion reactions, autoimmune diseases, and infections.

Pediatric Considerations

If nonaccidental trauma is suspected, prompt referral to appropriate child protective agencies is required along with medical treatment.

MEDICATION

- Ciprofloxacin: 500 mg (peds: 10–20 mg/kg q12) PO q12h
- Hydrocortisone enema: 60 mg per rectum
- Mesalamine enemas: 1–4 g retention enema—retain overnight. Adult.
- Mesalamine suppository: 500 mg per rectum BID. Adult.
- Mesalamine tablets:
 - Asacol 800 mg PO TID
 - Pentasa 1,000 mg PO QID
- Methylprednisolone: 125–250 mg IV load (peds: 2 mg/kg IV load, maintenance as adult), then 0.5–1 mg/kg/dose q6h for 5 days
- Metronidazole: 250–500 mg (peds: 30 mg/kg/24h) PO TID
- Prednisone: 40–60 mg (peds: 1–2 mg/kg) PO daily
- Sulfasalazine (Azulfidine): 500 mg (peds: 30 mg/kg) PO QID



FOLLOW-UP

DISPOSITION

Admission Criteria

- Surgical indication:
 - Massive, unresponsive hemorrhage
 - Perforation
 - Toxic dilation
 - Obstruction
- Severe flare-up:
 - Electrolyte imbalance
 - Severe dehydration
 - Severe pain
 - High fever
 - Significant bleeding

Discharge Criteria

- Initial presentation of diarrhea, mild pain, without toxicity, with close follow-up
- Mild to moderate exacerbation of known disease without obstruction, severe bleeding, severe pain, dehydration, with close follow-up, on renewed therapy or with addition of steroid

Issues for Referral

Extraintestinal manifestations

- Ocular
- Dermatologic

FOLLOW-UP RECOMMENDATIONS

Gastroenterologist or primary care as managing physician with surgical consultation as indicated

PEARLS AND PITFALLS

- With severe flare, rule out toxic megacolon.
- Consider Crohn's in children with growth/puberty delay.
- Consider Crohn's with perianal disease.
- Rule out *C. difficile* with flares; the incidence of *C. difficile* complicating IBD is increasing.
- Avoid antidiarrheals/spasmodic in severe UC.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Abdominal Pain
- Diarrhea



CODES

ICD9

- 555.9 Regional enteritis of unspecified site
- 556.9 Ulcerative colitis, unspecified
- 558.9 Other and unspecified noninfectious gastroenteritis and colitis

ICD10

- K50.90 Crohn's disease, unspecified, without complications
- K51.90 Ulcerative colitis, unspecified, without complications
- K52.9 Noninfective gastroenteritis and colitis, unspecified

INFLUENZA

Philip Shayne • Carli Blomquist



BASICS

DESCRIPTION

- Acute, usually self-limited, viral infection
- Transmission: By dispersion in small-particle aerosols created by sneezing, coughing, and talking
- Virus is deposited on respiratory tract epithelium and absorbed.
- Incubation period: 1–4 days (avg. 2)
- Mean duration in adults: 4 days
- Seasonal outbreaks most common in February.
- 2009 novel H1N1 pandemic peaked in fall and early winter of that year. Children and pregnant women had particularly high complication rates.
- Complications:
 - Primary influenza viral pneumonia
 - Secondary bacterial pneumonia
 - Exacerbations of COPD
 - Otitis and sinusitis in children
 - Reactive airway disease
 - Rare complications: Myositis, myocarditis, pericarditis, Guillain–Barré syndrome, and aseptic meningitis
 - ARDS and multisystem organ failure
- Key features:
 - Seasonal epidemics are spread by high attack rates in immunologically naive children.
 - Intermittent unpredictable pandemics
 - Mortality results largely from pulmonary complications.

Pediatric Considerations

- Children exhibit more lower respiratory involvement (croup, bronchitis, bronchiolitis, pneumonitis) and higher temperatures than adults.
- Children were particularly susceptible to complications of novel H1N1 influenza virus.
- Myalgias in the calf muscle
- Febrile convulsions occur in ~ 10% of children < 5 yr of age with influenza infection.
- Reye syndrome:
 - Influenza may be a predisposing factor.
 - Rare and severe complication associated with salicylate use (children taking

- aspirin chronically are recommended to get vaccinated)
- Acute liver and brain injury

ETIOLOGY

- Caused usually by 1 of 2 influenza types, A or B, the latter usually less severe.
- Influenza A subtypes are classified by hemagglutinin antigens H1, H2, or H3 and less importantly by the neuraminidase subtype.
- Vaccine targets the subtype antigen, which is also the target of natural immunity.
- Annual epidemics are seasonal:
 - Caused by *antigenic drift*—new variants from minor changes in surface protein
 - Duration of epidemic < 6 wk
- Pandemics:
 - Unpredictable
 - Caused by *antigenic shift*—major changes in virus structure
- Waterfowl reservoir of influenza virus
- Avian flu has proven difficult to transmit to humans and between humans, but infection is often very severe.
- The 3 most common strains in 2012 (most vaccinations cover) were influenza B viruses, influenza A (H1N1), and influenza A (H3N2)



DIAGNOSIS

- Complicated by similar acute infections caused by other respiratory viruses
- CDC defines influenza-like illness (ILI) as cough or sore throat in a patient with fever > 100°F and no alternative diagnosis.

SIGNS AND SYMPTOMS

- Local status of the epidemic (see CDC weekly status update <http://www.cdc.gov/flu/weekly/>) is by far the most important predictor of influenza in a patient with ILI.
- Despite poor discriminating properties of specific symptoms, a rise in ILI cases accurately predicts onset of the seasonal influenza epidemic.

History

- No single finding on history has much predictive power. Influenza can be asymptomatic or fatal.
- Fever and cough together is somewhat specific for influenza but insensitive.
- Specificity of findings depends on prevalence of other circulating viruses. E.g., RSV epidemics are also accompanied by high frequency of fever.

Physical-Exam

- Fever: Degree of fever is correlated with likelihood of influenza in randomized

trials of persons with ILI.

- There is no consistent relationship between physical findings and influenza positivity across multiple studies, but there are very few studies of ED patients.
- Many patients have evidence of reactive airway disease with bronchoconstriction.

Geriatric Considerations

Elderly may present with high fever, lassitude, and confusion without pulmonary complications.

ESSENTIAL WORKUP

Clinical diagnosis based on the signs and symptoms of influenza during the winter months in the setting of a known outbreak

DIAGNOSIS TESTS & NTERPRETATION

Lab

- CBC (optional):
 - _ WBC: Normal to mildly decreased
- Pulse oximetry/arterial blood gas for significant pulmonary symptoms

Imaging

CXR for prominent lower respiratory signs or symptoms:

- Normal (50–90%)
- Bilateral interstitial infiltration

Diagnostic Procedures/Surgery

- Culture of nasopharyngeal swab or aspirate is more sensitive than pharynx.
- Yield declines rapidly with duration of symptoms. Infrequently positive after day 2.
- Rapid influenza diagnostic tests and direct fluorescent antibody tests are inexpensive, rapid, and specific but often of very low sensitivity. Some are able to discriminate between A and B, but not subtypes of A.
- Polymerase chain reaction (PCR) tests have short turnaround time, are both sensitive and specific, and can discriminate H1 from H2 antigens; a combination of H1 negative and H2 negative is very specific for 2009 H1N1.
- Viral culture: Turnaround time too long for ED use, although OK for local surveillance.

DIFFERENTIAL DIAGNOSIS

- Other respiratory viruses
- Bronchitis
- Atypical pneumonia
- Epstein–Barr infection (infectious mononucleosis)

- Anthrax is very rare and much more likely to include dyspnea and nausea.



TREATMENT

PRE HOSPITAL

Vaccination and respiratory hygiene for EMS personnel during outbreaks

INITIAL STABILIZATION/THERAPY

Aggressive fluid resuscitation, supplemental oxygen, and positive-pressure ventilation as clinical circumstances dictate

ED TREATMENT/PROCEDURES

- Supportive and symptomatic:
 - Antipyretics (acetaminophen or NSAIDs)—avoid aspirin
 - Cough suppressants (rarely useful)
 - Rehydration
- Antivirals are effective if given within 48 hr of symptom onset:
 - Antiviral resistance patterns vary each season; confirm at CDC update page.
 - The neuraminidase inhibitors (NI) zanamivir and oseltamivir are generally active against types A and B.
 - The adamantanes amantadine and rimantadine are only effective against influenza A, but not current strains (currently not recommended for use due to resistance).
 - Antivirals reduce symptom duration by less than 1 day. Indirect evidence of benefit in severe disease.
 - Costly, except for amantadine
 - Recommended for:
 - Patient with severe illness
 - Immunocompromised patients
 - Patients at high risk for complications
- PREVENTION:
 - Inactivated, polyvalent influenza vaccine recommended annually for:
 - Adults > 50 yr
 - Residents of nursing homes and long-term care facilities
 - Children of age 6–4 yr
 - Children of age 6 mo–18 yr on chronic aspirin therapy
 - High-risk individuals (asthma, COPD, cardiovascular disease, immunocompromised, diabetics)
 - Health care workers
 - Morbidly obese (BMI > 40)
 - American Indians/Alaska natives
 - Caretakers of children < 6 mo old

- Women who will be pregnant during influenza season
- Attenuated-live intranasal vaccine (FluMist) is currently approved for healthy people of age 2–49 yr.
- Contraindicated for:
 - Pregnant women
 - Close contacts and health care workers for severely immunocompromised patients
 - Children < 5 yr with h/o recurrent wheezing
 - Children/adolescents receiving aspirin
 - Severe allergy to chicken eggs
 - People with comorbidities placing them in high risk for influenza complications
- Chemoprophylaxis in the following settings:
 - Postexposure prophylaxis for exposed family members, especially high risk
 - Short-term prophylaxis during outbreak of influenza A in high-risk patients who did not receive vaccine
 - In conjunction with vaccine in high-risk patients (including those with HIV infections) expected to respond poorly to vaccine
 - In lieu of vaccine when vaccine is contraindicated in high-risk individuals
 - In individuals providing care for high-risk persons
 - Extended duration, season-long prophylaxis of health care workers is effective but consumes large quantity of stockpiled drug.
 - Exceptions:
 - *Could interfere with live-virus vaccine*
 - Should not be started for at least 2 wk after inoculation
- Patients with evidence of bronchoconstriction and reduced breath sounds may benefit from bronchodilators such as albuterol.

Pregnancy Considerations

- Inactivated vaccine is recommended for women expected to be pregnant during influenza season.
- Live-attenuated virus is contraindicated in pregnancy.

MEDICATION

- Oseltamivir: 75 mg PO BID for 3–4 days:
 - Postexposure prophylaxis: 75 mg PO daily for 7–10 days
- Zanamivir: 10 mg nasal insufflation (2 inhalations) q12h for 3–5 days
- Amantadine: 200 mg PO initially, then 100 mg PO BID for 3–5 days (halve dose if age > 65 yr)
- Rimantadine: 200 mg PO initially, then 100 mg PO BID for 3–5 days
- Albuterol 2.5 mg in 3 mL by nebulizer or metered-dose inhaler with spacer

DISPOSITION***Admission Criteria***

- Hypoxia (pneumonia or reactive airway disease)
- Severe dehydration
- Alteration in mental status

Discharge Criteria

Most patients will have a short, self-limited course provided they are able to tolerate fluids and antipyretics.

Issues for Referral

Consultation with infectious disease specialist when uncertain of local disease status, diagnostic uncertainty, local antiviral resistance patterns

FOLLOW-UP RECOMMENDATIONS

Call back for PCR test result.

PEARLS AND PITFALLS

- Become familiar with online CDC weekly update since flu changes each season.
- In most patients, neither testing nor antiviral treatment is necessary.
- In patients with respiratory distress or hypoxia, consider concurrent reactive airway disease.
- ED policy: Institute respiratory hygiene:
 - Etiquette posters and alcohol hand soap available

ADDITIONAL READING

- Call SA, Vollenweider MA, Hornung CA, et al. Does this patient have influenza? *JAMA*. 2005;293:987–997.
- CDC 2012 flu update: <http://www.cdc.gov/flu/professionals/index.htm>
- Harper SA, Bradley JS, Englund JA, et al. Seasonal influenza in adults and children—diagnosis, treatment, chemoprophylaxis, and institutional outbreak management: Clinical practice guidelines of the Infectious Diseases Society of America. *Clin Infect Dis*. 2009;48:1003–1032.
- Prevention and control of influenza: Recommendations of the Advisory Committee on Immunization Practices (ACIP), 2008. *MMWR Recomm Rep*. 2008;57(RR-7):1–60.
- Writing Committee of the WHO Consultation on Clinical Aspects of Pandemic (H1N1) 2009 Influenza, Bautista E, Chotpitayasunondh T, et al. Clinical aspects on

pandemic 2009 influenza A (H1N1) virus infection. *N Engl J Med.* 2010;362:1708–1719.



See Also (Topic, Algorithm, Electronic Media Element)

- Anthrax
- Asthma, Pediatric and Adult
- Pneumonia, Pediatric and Adult



CODES

ICD9

- 487.1 Influenza with other respiratory manifestations
- 488.02 Influenza due to identified avian influenza virus with other respiratory manifestations
- 488.82 Influenza due to identified novel influenza A virus with other respiratory manifestations

ICD10

- J09.X2 Flu due to ident novel influenza A virus w oth resp manifest
- J10.1 Flu due to oth ident influenza virus w oth resp manifest
- J11.1 Influenza due to unidentified influenza virus with other respiratory manifestations

INTRACEREBRAL HEMORRHAGE

Atul Gupta • Rebecca Smith-Coggins

BASICS

DESCRIPTION

Hemorrhage into brain parenchyma:

- Compression of brain tissues
- Secondary injury results from:
 - Cerebral edema
 - Increased intracranial pressure (ICP)
 - Potential of brain herniation

ETIOLOGY

Intracerebral hemorrhage can occur spontaneously or from trauma:

- Uncontrolled or acute HTN (most common)
- Vascular malformations:
 - Arteriovenous malformation
 - Venous angiomas
 - Ruptured cerebral aneurysms
- Neoplasm (particularly melanoma and glioma)
- Anticoagulant therapy (warfarin, heparin)
- Thrombolytic agents
- Illicit drugs (cocaine, amphetamines)
- Bleeding disorders (hemophilia)
- Cerebral amyloid angiopathy
- Traumatic hemorrhage secondary to blunt or penetrating injury

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Severe headache, typically sudden in onset
- Seizure
- Evidence of head injury
- Neck stiffness
- Vomiting
- Anticoagulation therapy
- Altered level of consciousness (may be comatose):
 - Altered mental status may occur as late as 24–48 hr after head injury

Physical-Exam

- HTN
- Nuchal rigidity
- Altered mental status
- Variable neurologic deficits depending on site of intracerebral hemorrhage:
 - Putamen hemorrhage (35%):
 - Contralateral hemiparesis
 - Contralateral hemisensory loss
 - Occasional dysphagia
 - Occasional neglect
 - Lobar hemorrhage (30%):
 - Variable signs depending on involved area
 - Cerebellar hemorrhage (15%):
 - Vomiting
 - Ataxia
 - Nystagmus
 - Thalamic hemorrhage (10%):
 - Similar to putamen, but may also have eye movement abnormalities
 - Caudate hemorrhage (5%):
 - Confusion
 - Memory loss
 - Hemiparesis
 - Gaze paresis
 - Pontine hemorrhage (5%):
 - Quadriplegia
 - Pinpoint pupils
 - Ataxia
 - Sensorimotor loss

ESSENTIAL WORKUP

- Manage airway if indicated
- Immediate noncontrast head CT:
 - Acute hemorrhage appears as high-density lesion

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC
- Coagulation studies (PT/PTT, INR, platelets)
- Electrolytes; BUN, creatinine
- Pregnancy test in women of childbearing age
- EKG
- Consider toxicology screen

Imaging

- CT as above
- MRI may be useful but currently not as available or rapid as CT

Diagnostic Procedures/Surgery

- CT angiography:
 - Gaining increasing acceptance as a diagnostic tool in acute setting
 - Up to 15% of patients may show an underlying vascular etiology on CTA, potentially changing acute management
 - Contrast extravasation (spot sign) may represent ongoing bleeding
 - Highest risk of hematoma expansion with poor outcome and mortality

DIFFERENTIAL DIAGNOSIS

- Seizure:
 - Todd paralysis
- CNS infection
- CNS mass
- Electrolyte or acid–base abnormality
- Intoxication
- Wernicke encephalopathy
- Migraine headache
- Transient ischemic attack
- Nonhemorrhagic acute cerebrovascular accident
- Air embolism
- Differential diagnosis once bleed is seen on CT:
 - Spontaneous hemorrhage:
 - Hypertensive hemorrhage
 - Arteriovenous malformation
 - Neoplasm
 - Traumatic hemorrhage:
 - Subarachnoid hemorrhage
 - Subdural hematoma
 - Epidural hematoma

Pediatric Considerations

Additional differential diagnoses include:

- Moyamoya disease
- Acute infantile hemiplegia



TREATMENT

PRE HOSPITAL

- C-spine precautions if head or neck injury is suspected
- Elevation of head with C-spine control
- Initial pre-hospital responder must ascertain neurologic defect to be able to note progression of symptoms

INITIAL STABILIZATION/THERAPY

- Manage airway and resuscitate as needed:
 - _ Patients with depressed level of consciousness should be intubated immediately for controlled ventilation
- Early neurosurgical consultation

ED TREATMENT/PROCEDURES

- Prompt neurosurgery and/or neurology consultation
- BP management:
 - _ Must use caution in BP control because acute lowering of BP to normal in setting of increased ICP could reduce cerebral perfusion to ischemic levels
 - _ Use labetalol, nicardipine, esmolol, enalapril to lower diastolic BP initially by 10%
 - _ Normotensive levels should be achieved over 12–24 hr
 - _ May use nitroprusside, nitroglycerin, or hydralazine as an alternative
- Treatment of elevated ICP:
 - _ Controlled ventilation to PaCO₂ of 35 Torr
 - _ Fluid restriction; elevate head of bed 30°
 - _ Mannitol—osmotic diuresis
 - _ Use furosemide as an alternative
- Correct coagulopathies:
 - _ Consider fresh frozen plasma (FFP), platelets, prothrombin complex concentrates, vitamin K
- Consider anticonvulsants
 - _ Phenytoin, fosphenytoin

MEDICATION

- Esmolol: 0.5–1 mg/kg initial bolus IV, followed by 50–150 µg/kg/min infusion
- Enalapril: 1.25–5 mg q6h (risk of precipitous BP lowering, test dose 0.625 mg)
- FFP: 10–20 mL/kg IV
- Fosphenytoin: 15–20 mg/kg phenytoin equivalents (PE) at rate of 100–150 mg/min IV/IM
- Furosemide: 20–40 mg (peds: 0.5–1 mg/kg/dose) IV; may repeat as necessary
- Hydralazine: 10–40 mg (peds: 0.1–0.2 mg/kg/dose; max. 20 mg/dose) IV; may repeat as necessary
- Labetalol: 20 mg (peds: 0.3–1 mg/kg/dose; max. 20 mg/dose) IV; may give additional 40–80 mg IV q10min to max. 300 mg
- Mannitol: 1 g/kg IV

- Nicardipine: 5–15 mg/h infusion
- Nitroprusside: Start 0.25–10 µg/kg/min IV (max. 10 µg/kg/min); titrate to effect
- Phenytoin: 15–20 mg/kg/dose (peds: 15 mg/kg) at rate of < 40–50 mg/min
- Platelet: 1–2 U IV in consultation with neurosurgery
- Prothrombin complex concentrates: 500–1,000 IU IV
- Vitamin K: 5–10mg IV over 30 min



FOLLOW-UP

DISPOSITION

Admission Criteria

- To OR if surgical intervention is indicated
- To ICU if intubated, altered level of consciousness, or on IV infusion for BP control
- Admit to neurologic observation unit if normal neurologic exam without evidence of progression of bleed and hemodynamically stable

Discharge Criteria

All patients with intracerebral hemorrhage should be admitted

Issues for Referral

Rehabilitation is a key aspect of recovery

FOLLOW-UP RECOMMENDATIONS

- Treating HTN in the nonacute setting is the most important step to reduce the risk of intracerebral hemorrhage
- Discontinuation of smoking, alcohol use, and cocaine use prevents recurrence of intracerebral hemorrhage

PEARLS AND PITFALLS

- Brain imaging is a crucial part of emergent evaluation of patients with headache, HTN, and/or altered level of consciousness
- Cautious BP control because acute lowering of BP to normal in setting of ICP could reduce cerebral perfusion to ischemic levels
- Consider delayed intracranial bleed in patients on anticoagulation with head trauma

ADDITIONAL READING

- Broderick J, Connolly S, Feldmann E, et al. Guidelines for the management of spontaneous intracerebral hemorrhage in adults: 2007 update: A guideline from the American Heart Association/American Stroke Association Stroke Council, High

Blood Pressure Research Council, and the Quality of Care and Outcomes in Research Interdisciplinary Working Group. *Stroke*. 2007;38:2001–2023.

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- Naval NS, Nyquist PA, Carhuapoma JR. Management of spontaneous intracerebral hemorrhage. *Neurol Clin*. 2008;26:373–384.
- Nishijima DK, Offerman SR, Ballard DW, et al. Immediate and delayed traumatic intracranial hemorrhage in patients with head trauma and preinjury warfarin or clopidogrel use. *Ann Emerg Med*. 2012;59:460–468.
- Nishijima DK, Offerman SR, Ballard DW, et al. Risk of traumatic intracranial hemorrhage in patients with head injury and preinjury warfarin or clopidogrel use. *Acad Emerg Med*. 2013;20:140–145.



See Also (Topic, Algorithm, Electronic Media Element)

- Headache
- Hypertensive Emergencies
- Seizure



CODES

ICD9

- 431 Intracerebral hemorrhage
- 853.00 Other and unspecified intracranial hemorrhage following injury without mention of open intracranial wound, unspecified state of consciousness

ICD10

- I61.9 Nontraumatic intracerebral hemorrhage, unspecified
- S06.360A Traum hemor cereb, w/o loss of consciousness, init

INTUSSUSCEPTION

Roger M. Barkin

BASICS

DESCRIPTION

- The proximal bowel invaginates into the distal bowel, producing infarction and gangrene of the inner bowel:
 - >80% involve the ileocecal region.
- Often occurs with a pathologic lead point in children >2 yr:
 - Hypertrophied lymphoid patches may be present in infants.
 - Children >2 yr: 1/3 of patients have pathologic lead point.
 - Children >6 yr: Lymphoma is the most common lead point.
 - Adults usually have a pathologic lead point.
- The most common cause of intestinal obstruction within the 1st 2 yr of life
- Epidemiology in US:
 - Most frequently between 5 and 9 mo of age
 - Incidence is 2.4 cases per 1,000 live births.
 - Male > female predominance of 2:1
 - Mortality <1%
- Morbidity increases with delayed diagnosis.

ALERT

Patients, particularly those in the pediatric age group, with a picture of potential intestinal obstruction, especially with hematest-positive stool or altered mental status, need to have intussusception considered.

ETIOLOGY

- Most cases (85%) have no apparent underlying pathology.
- Predisposing conditions that create a lead point for invagination, esp. in older children and adults:
 - Masses/tumors:
 - Lymphoma
 - Lipoma
 - Polyp
 - Hypertrophied lymphoid patches
 - Meckel diverticulum
 - Infection:
 - Adenovirus or rotavirus infection
 - Parasites
 - Foreign body
 - Henoch–Schönlein purpura

- Celiac disease and cystic fibrosis (small intestine intussusception)



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Classic triad (present in < 50% of patients):
 - Abdominal pain
 - Vomiting, often bilious
 - Stools have blood and mucus (“currant jelly” stools)
- Recurrent painful episodes accompanied by pallor and drawing up of the legs; intermittent fits of sudden intense pain with screaming and flexion of legs:
 - Occur in 5–20 min intervals
- Mental status changes:
 - Irritability
 - Lethargy or listlessness; child can be limp or have a rag doll appearance.
 - May precede abdominal findings.
- Stool variable:
 - Heme-positive (occult), bloody, or “currant jelly”
- Preceding illness several days or weeks prior to the onset of abdominal pain:
 - Diarrhea
 - Viral syndrome
 - Henoch–Schönlein purpura
- Recurrent intussusception occurs in < 10% of patients.

Physical-Exam

- Fever
- Abdomen distended and swollen:
 - A “sausage” mass may be palpated in the right upper quadrant.
 - May have absent cecum in right iliac fossa.
 - Peristaltic wave may be present.
 - Rectal exam may reveal bloody stool and palpable mass.
- Dependent on the time from onset to diagnosis; perforation with peritonitis and sepsis may be present.

ESSENTIAL WORKUP

- The diagnosis is suggested by the history and is proven radiographically.
- A heme-positive stool may aid in the diagnosis, particularly in the presence of lethargy or listlessness.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC
- Serum electrolytes, BUN
- Type and cross-match

Imaging

- Abdominal radiograph:
 - Abnormal in 35–40% of patients
 - Decreased bowel gas and fecal material in the right colon
 - Abdominal mass
 - Apex of intussusceptum outlined by gas
 - Small bowel distention and air–fluid levels secondary to mechanical obstruction
 - May aid in excluding intestinal perforation.
- Enema:
 - Often both diagnostic and therapeutic. Reoccurrences do happen.
 - 74% successful if intussusception present ≤ 24 hr
 - 32% effective when present > 24 hr
 - The more distal the intussusception, the lower is the ability to reduce it radiographically.
 - Recurrent disease (up to 10%) has similar success to initial episode.
 - Complications include bowel perforation, reduction of necrotic bowel, incomplete reduction with delay in surgery, and overlooking pathologic lead point.
 - Hypovolemic shock reported following reduction secondary to endotoxins and cytokines.
 - Barium:
 - Traditional standard for diagnosis and treatment
 - Characteristic coiled-spring appearance
 - Air:
 - Fluoroscopic guidance
 - Avoids peritoneal contamination if perforation
 - Increasingly used for diagnosis and treatment
 - Contraindications:
 - Peritonitis
 - Perforation
 - Unstable patients secondary to sepsis or shock
- US is highly accurate and may be useful as a screening technique; operator dependent:
 - Typical appearance is a “donut” or “bull’s eye” structure, with hyperechoic core surrounded by hypoechoic rim of homogeneous thickness.

Diagnostic Procedures/Surgery

If enema is unsuccessful in reducing, surgery is required on an emergent basis.

DIFFERENTIAL DIAGNOSIS

- Infection
- Acute gastroenteritis
- Appendicitis
- Inflammatory bowel disease
- Infectious mononucleosis
- Pneumonia
- Pharyngitis/group A streptococcal
- Pyelonephritis
- Colic
- Intestinal obstruction/peritonitis
- Strangulated hernia
- Malrotation/volvulus
- Hirschsprung disease
- Trauma
- Intestinal vascular/hemorrhagic disorder
- Anal fissure/hemorrhoids
- Ulcer disease
- Vascular malformations
- Henoch–Schönlein purpura
- Polyp
- Protein-sensitive enterocolitis
- Diabetes mellitus
- Coagulopathy



TREATMENT

PRE HOSPITAL

- IV access
- IV bolus of 20 mL/kg of 0.9% NS or lactated Ringer (LR) if evidence of hypovolemia, abdominal distention, peritonitis, sepsis
- Diagnosis rarely confirmed in pre-hospital setting

INITIAL STABILIZATION/THERAPY

- IV access and initiation of 0.9% NS or LR at 20 mL/kg bolus
- Nasogastric tube

ED TREATMENT/PROCEDURES

- Stabilize patient hemodynamically.
- Surgical consultation

- Abdominal radiograph film series
- Interventional radiography for reduction if no contraindications:
 - Enemas are 75–80% successful at reduction, reflecting duration of condition.
 - Recurrences may also be reduced radiographically.
- Antibiotics:
 - Initiate if evidence of peritonitis, perforation, or sepsis.
 - Ampicillin, clindamycin, and gentamicin
 - Ampicillin/sulbactam
- Laparotomy:
 - Indications:
 - Enema is unsuccessful.
 - Enema is contraindicated.
 - Pathologic lead point
 - Multiple recurrences
 - Procedure:
 - Gentle milking of the intussusceptum
 - Resection of any nonviable bowel as well as any lead points that are identified

MEDICATION

First Line

- Ampicillin: 100–200 mg/kg/d q4h IV
- Clindamycin: 30–40 mg/kg/d q6h IV
- Gentamicin: 5–7.5 mg/kg/d q8h IV
- Ampicillin/sulbactam 100–200 mg/kg/d q6h IV



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients undergoing successful enema reduction should be observed for complications or recurrence.
- Patients undergoing surgery

Discharge Criteria

- May be considered after a *very prolonged* period of observation following successful enema reduction:
 - Stable patient with normal mental status
 - Symptomatic relief of abdominal pain during the postreduction period
 - Parents have appropriate understanding to watch for potential

reoccurrence, even after prolonged period observation

Issues for Referral

Surgeon should be aware of patients with potential diagnosis of intussusception.

PEARLS AND PITFALLS

Infants with intermittent abdominal pain, impaired mental status, and blood in stools should generally have intussusception considered.

ADDITIONAL READING

- Bajaj L, Roback MG. Postreduction management of intussusception in a children's hospital emergency department. *Pediatrics*. 2003;112:1302–1307.
- Hryhorczuk AL, Strouse PJ. Validation of US as a first-line diagnostic test for assessment of pediatric ileocolic intussusception. *Pediatr Radiol*. 2009;39:1075–1079.
- Kleizen KJ, Hunck A, Wijnen MH, et al. Neurological symptoms in children with intussusception. *Acta Paediatr*. 2009;98:1822–1824.
- Saverino BP, Lava C, Lowe LH, et al. Radiographic findings in the diagnosis of pediatric ileocolic intussusception: Comparison to a control population. *Pediatr Emerg Care*. 2010;26:281–284.
- Willetts IE, Kite P, Barclay GR, et al. Endotoxin, cytokines and lipid peroxides in children with intussusception. *Br J Surg*. 2001;88:878–883.



CODES

ICD9

560.0 Intussusception

ICD10

K56.1 Intussusception

IRITIS

Jessica Freedman

BASICS

DESCRIPTION

- Inflammation of anterior uveal tract
- Iritis and anterior uveitis are synonymous.
- Uveitis secondary to trauma is also called traumatic iritis.

ETIOLOGY

- Most cases are idiopathic, but may be traumatic or associated with numerous infectious and noninfectious systemic diseases.
- May be acute or chronic.
- Noninfectious systemic diseases include the following:
 - _ Ankylosing spondylitis
 - _ Reiter syndrome
 - _ Sarcoidosis
 - _ Behçet disease
 - _ Inflammatory bowel disease
 - _ Juvenile rheumatoid arthritis
 - _ Kawasaki syndrome
 - _ Interstitial nephritis
 - _ IgA nephropathy
 - _ Drug reactions
 - _ Sjögren syndrome
 - _ Psoriatic arthritis
- Infectious conditions include the following:
 - _ Viral:
 - Rubella
 - Measles
 - Adenovirus
 - Herpes simplex virus
 - Herpes zoster virus
 - HIV
 - Mumps
 - Varicella
 - Cytomegalovirus
 - West Nile virus
 - _ Bacterial:
 - Tuberculosis

- Syphilis
- Pertussis
- Brucellosis
- Lyme disease
- Chlamydia
- Rickettsia
- Gonorrhea
- Leprosy
- Fungal:
- Malignancies include the following:
 - Leukemia
 - Lymphoma
 - Multiple sclerosis
 - Malignant melanoma
- Other causes include the following:
 - Cocaine use
 - Exposure to pesticides
 - Corneal foreign body
 - Blunt trauma

DIAGNOSIS

SIGNS AND SYMPTOMS

- Acute presentation:
 - Ocular pain, red eye
 - Photophobia (consensual)
 - Lacrimation
 - Decreased visual acuity (usually mild)
 - Cells and flare in anterior chamber; hypopyon
 - Posterior synechiae (adhesions of iris to lens)
 - Miosis
 - Low intraocular pressure (occasionally may be high)
 - Injection of perilimbal vessels (ciliary flush)
- Chronic presentation:
 - Recurrent episodes
 - Few or no acute symptoms

ESSENTIAL WORKUP

- History and review of systems:
 - Up to 50% may be associated with systemic disease.
- Slit-lamp exam:
 - Inflammatory cells (leukocytes) or “flare” in the anterior chamber are diagnostic.

- Flare is a homogeneous fog secondary to protein leakage into aqueous humor.
- Use short, wide beam to best appreciate cells and flare.
- Cellular deposits with more severe inflammation
- Intraocular pressure measurement
- If topical anesthesia relieves pain, probably not iritis.

DIAGNOSIS TESTS & NTERPRETATION

- None usually indicated
- Tailored outpatient workup if history, signs, and symptoms point strongly to a certain cause (with referral to ophthalmology, rheumatology, or internal medicine)

Lab

- TB:
 - Purified protein derivative (PPD)
- Sarcoidosis:
 - PPD
- Ankylosing spondylitis:
 - ESR
 - HLA-B27
- Inflammatory bowel disease:
 - HLA-B27
- Reiter syndrome:
 - HLA-B27
 - Cultures of conjunctiva and urethra
- Psoriatic arthritis:
 - HLA-B27
- Lyme disease:
 - Immunoassays
- Juvenile rheumatoid arthritis:
 - Antinuclear antibody
 - Rheumatoid factor
- Sarcoidosis:
 - ACE
 - Serum lysozyme level
- STI:
 - Rapid plasma reagin or VDRL test
 - Fluorescent treponemal antibody absorption test
 - Appropriate cultures

Imaging

- Ankylosing spondylitis:
 - Sacroiliac spine radiograph

- Sarcoidosis:
 - CXR
- TB:
 - CXR

Diagnostic Procedures/Surgery

US biomicroscopy can be used to help to diagnose pathologies.

DIFFERENTIAL DIAGNOSIS

- Acute angle-closure glaucoma
- Conjunctivitis
- Corneal abrasion
- Corneal foreign body
- Episcleritis
- Intraocular foreign body
- Keratitis
- Posterior segment tumor



TREATMENT

INITIAL STABILIZATION/THERAPY

- Goal:
 - Reduce inflammation and prevent complications
- Cycloplegic agent (short-acting):
 - Decreases pain, photophobia
 - Prevents development of posterior synechiae

ED TREATMENT/PROCEDURES

- Cycloplegia
- Topical steroids if indicated:
 - Use with caution, in consultation with ophthalmologist.
 - May cause significant complications (i.e., progression of herpes simplex virus keratitis)
- Treat secondary glaucoma.
- Supportive measures:
 - Warm compresses
 - Dark glasses
 - Analgesia
- Identification of cause:
 - Initiate appropriate management.
- Ankylosing spondylitis:
 - Systemic anti-inflammatory agents

- Physical therapy
- Inflammatory bowel disease:
 - Systemic steroids
 - Sulfadiazine
 - Vitamin A
- Reiter syndrome:
 - Treat urethritis (and sexual contacts).
- Behçet disease:
 - Systemic steroids or immunosuppressive agents
- Infectious causes:
 - Appropriate management of underlying infection

MEDICATION

- Cycloplegic:
 - Cyclopentolate 1–2% for mild to moderate inflammation: 1 drop TID (lasts up to 24 hr)
 - Homatropine 2% or 5% for moderate inflammation: 1 drop TID (lasts up to 3 days)
 - Atropine 1% for moderate to severe inflammation (should only be used in consultation with ophthalmologist): 1 drop TID (lasts 7–14 days)
- Topical steroid (should only be used in consultation with ophthalmologist):
 - Prednisolone acetate 1%: 1 drop q1–6h, depending on severity
- Analgesic:
 - Tylenol or tylenol with codeine

Pediatric Considerations

- Cycloplegics not recommended in children <6 yr:
 - May cause systemic anticholinergic toxicity with blurred vision, flushing, tachycardia, hypotension, and hallucinations.



FOLLOW-UP

DISPOSITION

Admission Criteria

Not indicated unless significant systemic illness

Issues for Referral

- Iritis:
 - Refer to ophthalmologist within 24 hr for follow-up care and possible steroid therapy.
- Inflammatory bowel disease:

- Gastroenterology consult
- Reiter syndrome:
 - Rheumatology consult
- Psoriatic arthritis:
 - Rheumatology consult
- Juvenile rheumatoid arthritis:
 - Rheumatology consult

PEARLS AND PITFALLS

- If topical anesthesia relieves pain, probably not iritis.
- Must be differentiated from other, vision-endangering forms of eye pain:
 - Keratitis
 - Herpes simplex conjunctivitis
 - Bacterial conjunctivitis
 - Acute angle-closure glaucoma
 - Traumatic globe rupture

ADDITIONAL READING

- Bertolini J, Pelucio M. The red eye. *Emerg Med Clin North Am.* 1995;13:561–579.
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- Weinberg RS. Uveitis. *Ophthalmol Clin North Am.* 1999;12:71–79.



See Also (Topic, Algorithm, Electronic Media Element)

- Conjunctivitis
- Red Eye



CODES

ICD9

- 364.00 Acute and subacute iridocyclitis, unspecified
- 364.3 Unspecified iridocyclitis
- 364.10 Chronic iridocyclitis, unspecified

ICD10

- H20.00 Unspecified acute and subacute iridocyclitis
- H20.9 Unspecified iridocyclitis
- H20.10 Chronic iridocyclitis, unspecified eye

IRON POISONING

Sean M. Bryant



BASICS

DESCRIPTION

- Peak concentrations are 2–4 hr postingestion
- Serum concentrations not reliable if obtained > 4–6 hr after ingestion:
 - Enteric coated or sustained release—warrants serial levels
- Postabsorption: Iron redistributes into tissues, and fall in serum iron occurs as free iron shifts intracellularly resulting in cellular injury
- Injury patterns:
 - Corrosive injury to intestinal mucosa may result in profound fluid loss (shock), hemorrhage, and perforation
 - Liver receives largest load of iron because of portal venous circulation—(hemorrhagic periportal necrosis)
- Free iron:
 - Concentrates in mitochondria, disrupting oxidative phosphorylation; catalyzes lipid peroxidation and free radical formation, resulting in cell death; increases anaerobic metabolism and acidosis
 - Causes myocardial depression, venodilation, and cerebral edema
- Hydration of ferric form liberates 3 protons, resulting in acidemia

ETIOLOGY

Elemental iron ingestion:

- Nontoxic < 20 mg/kg
- Moderate to severe > 40 mg/kg
- Lethality possible > 60 mg/kg
- Elemental iron equivalents:
 - Ferrous sulfate, 20% (325 mg = 65 mg Fe)
 - Ferrous gluconate, 12%
 - Ferrous fumarate, 33%
- Prenatal vitamins vary from 60–90 mg elemental iron per tablet
- Children's vitamins may contain 5–18 mg elemental iron per tablet

Pediatric Considerations

- Historically notorious for the highest mortality rate among pediatric accidental exposures (adult iron products)
- Children's chewable iron products have been shown to be safe



DIAGNOSIS

SIGNS AND SYMPTOMS

- Classically divided into 5 stages:
 - **Stage 1: GI** (0.5–6 hr):
 - Abdominal pain
 - Vomiting
 - Diarrhea
 - Hematemesis
 - Hematochezia
 - **Stage 2: Latent/quiescent** (6–24 hr):
 - Resolution of GI symptoms
 - Deceptive phase (ongoing injury?)
 - Possible hypotension and acidosis
 - **Stage 3: Shock and organ failure** (6–72 hr):
 - Hypoperfusion
 - Metabolic acidosis
 - Coma
 - Coagulopathy
 - **Stage 4: Hepatic failure** (2–3 days):
 - Coagulopathy
 - Hypoglycemia
 - Jaundice
 - Elevated LFTs (transaminases) and bilirubin
 - **Stage 5: Obstruction** (2–4 wk):
 - Gastric outlet and small bowel obstruction
 - Abdominal pain, vomiting
- Patient may present in or skip any of the 5 stages
- If onset of stage 1 does not occur within 6 hr, likely not significant ingestion

ESSENTIAL WORKUP

Acute iron poisoning is a clinical diagnosis, regardless of lab results

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Serum iron levels (mg/dL):
 - Peak absorption 2–6 hr
 - 4 hr is most common time for peak
 - Delayed peak with enteric coated/sustained release
- Electrolytes, BUN/creatinine, glucose:
 - Anion gap metabolic acidosis
 - Hyperglycemia early
 - Hypoglycemia late
- Arterial blood gas:

- Metabolic acidosis
- CBC:
 - Anemia with significant hemorrhage
 - Leukocytosis
- Liver function
- Coagulation profile
- Lactate
- Type and screen if hemorrhage
- Total iron-binding capacity is not useful and not recommended

Imaging

Abdominal radiograph check for:

- Tablets (children's chewables rarely visible)
- Absence of pill fragment interpretation:
 - Patient did not ingest iron
 - Iron was in solution or has already dissolved
 - Patient ingested pediatric multivitamin product
 - Absence of radiopacities does not rule out significant or lethal ingestion
- Perforation

DIFFERENTIAL DIAGNOSIS

- Sepsis
- Acetaminophen toxicity
- Toxic ingestions causing anion gap acidosis:
 - Salicylate
 - Cyanide
 - Methanol
 - Ethylene glycol
- Mushrooms
- Heavy metals
- Theophylline toxicity
- GI bleed from other causes (alcoholic liver disease)



TREATMENT

PRE HOSPITAL

Collect prescription bottles/medications for identification in the ED

INITIAL STABILIZATION/THERAPY

- ABCs:
 - Intubate if profoundly unstable
 - Venous access and fluids for hypotension

- Cardiac monitor and pulse oximetry
- Naloxone, thiamine, dextrose (or Accu-Chek) as indicated for altered mental status

ED TREATMENT/PROCEDURES

- Decontamination:
 - Poorly adsorbed by activated charcoal
 - Gastric lavage has not been shown to change outcome
 - NaHCO₃, phospho soda, and oral deferoxamine are not recommended
 - If pill fragments are visualized on x-ray, or history of significant ingestion:
 - Consider whole bowel irrigation (with NG GoLytely: Peds: 10–15 mL/kg/h; adult: 1–2 L/h) while monitoring progression with radiograph (KUB).
 - Caution with GI bleed
 - Endoscopy or gastrotomy can remove bezoar formation after massive ingestions (> 240 mg/kg)
- Chelation with deferoxamine (DFO):
 - DFO is a highly specific chelator of parenteral iron
 - IV infusion results in more constant DFO levels and is route of choice:
 - Administer as soon as possible (< 24 hr)
 - Administration techniques:
 - Increase IV infusion rate to 15 mg/kg/h over 20 min, monitoring for hypotension
 - Decrease infusion rate if hypotension
 - Infusion rates as high as 45 mg/kg/h have been tolerated
 - Disregard manufacturer's recommendation of max. daily doses of 6 g in serious iron exposures
 - IM DFO challenge test is not advocated
 - Interpret serum levels cautiously:
 - Time since ingestion must be considered: Treatment may be indicated in patient who presents late, after distribution stage (> 8 hr postingestion), with serum iron level < 350 mg/dL
 - If serum iron levels are not readily available, base treatment decisions on clinical status
 - Length of infusion (controversial):
 - DFO–iron complex causes urine to turn *vin rose* color; this suggests continuing infusion until urine returns to normal
 - Resolution of signs and symptoms of significant toxicity is criterion for discontinuing DFO
 - Prolonged DFO therapy > 24–48 hr may precipitate adult respiratory distress syndrome
 - In severe cases with continued signs and symptoms, infusion may be continued cautiously at lower dose
 - Controversies:

- Safety of DFO infusions given for > 24 hr
- Maximal infusion rates and total amount
- Serum iron concentration warranting treatment
- Endpoint of treatment (best endpoint is resolution of poisoning, i.e., acidemia)
- Role of extracorporeal elimination
- Contact regional poison center for moderate to severe iron exposures
 - (1-800-222-1222)

MEDICATION

- Dextrose: D₅₀W 1 amp (50 mL or 25 g; peds: D₂₅W 2–4 mL/kg) IV
- Naloxone (Narcan): 2 mg (peds: 0.1 mg/kg) IV or IM initial dose
- Thiamine (vitamin B₁): 100 mg (peds: 50 mg) IV or IM



FOLLOW-UP

DISPOSITION

Admission Criteria

- GI symptoms or dehydration
- Altered mental status
- Hypotension, lethargy, metabolic acidosis, or shock
- Serum iron > 500 mg/dL
- Serum iron > 350 mg/dL *and* pills seen on KUB
- Rising serum iron levels
- Patients treated with deferoxamine
- ICU admission for coma, shock, metabolic acidosis, or iron levels > 1,000 mg/dL

Discharge Criteria

- Asymptomatic with negative radiograph
- Minimal to no symptoms after 6-hr observation
- Mild GI symptoms that have resolved without evidence of metabolic acidosis and serum iron < 350 mg/dL

Issues for Referral

Contact regional poison center for mild to moderate toxicity

FOLLOW-UP RECOMMENDATIONS

- Follow-up after discharge may be indicated in patients who are at risk of developing gastric outlet obstruction
- Psychiatric referral for patients with intentional overdose

PEARLS AND PITFALLS

- DFO may be indicated in patients who present late, after distribution stage (> 8 hr postingestion), or with serum iron level < 350 mg/dL and signs of intracellular poisoning (e.g., anion gap metabolic acidosis)
- Resolution of GI symptoms does not indicate that there is no ongoing toxicity that may progress over time

ADDITIONAL READING

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- Chang TP, Rangan C. Iron poisoning: A literature-based review of epidemiology, diagnosis, and management. *Pediatr Emerg Care*. 2011;27:978–985.
- Tenenbein M. Unit-dose packaging of iron supplements and reduction of iron poisoning in young children. *Arch Pediatr Adolesc Med*. 2005;159:557–560.



CODES

ICD9

964.0 Poisoning by iron and its compounds

ICD10

- T45.4X1A Poisoning by iron and its compounds, accidental, init
- T45.4X4A Poisoning by iron and its compounds, undetermined, init

IRRITABLE BOWEL SYNDROME

Scott A. Miller



BASICS

DESCRIPTION

- Syndrome of abdominal pain or discomfort associated with altered bowel habits and no other pathology explaining symptoms
- Prevalence estimated to be 10–20%

ETIOLOGY

- Uncertain pathophysiology, but many possibilities
- Altered GI motility:
 - _ Increased gut sensitivity (visceral hyperalgesia):
 - Exaggerated response to normal GI physiology
- Mucosal inflammation:
 - _ Postinfectious:
 - After bacterial enteritis, 10% have persistent IBS symptoms
- Mucosal lymphocyte infiltration
- Altered microflora in small bowel or feces
- Food sensitivity is a possibility but not proven
- Psychosocial dysfunction:
 - _ More anxiety, somatoform disorders, and history of abuse in patients who seek care
 - _ No evidence of increased psychiatric illness in those who do not seek care



DIAGNOSIS

SIGNS AND SYMPTOMS

- Abdominal pain or discomfort:
 - _ Relief with defecation
- Altered stool frequency
- Altered stool consistency
- Bloating or distention
- Passage of mucus
- Feeling of incomplete emptying

ALERT

- Consider further diagnostic workup if any of the following “alarm” features are present:
 - _ Onset > 50

- Nocturnal symptoms
- Unintentional weight loss
- Iron-deficiency anemia
- Hematochezia
- Family history of colorectal cancer, inflammatory bowel disease, or celiac sprue

History

- Rome III diagnostic criteria: Recurrent abdominal pain or discomfort 3 days/mo in the last 3 mo associated with ≥ 2 of:
 - Improvement with defecation
 - Onset associated with a change in frequency of stool
 - Onset associated with a change in form (appearance) of stool
- Other symptoms consistent with IBS:
 - Abdominal distention or bloating
 - Passage of mucus in stools
 - Altered stool passage (straining, urgency, or feeling of incomplete evacuation)
 - Postprandial upper abdominal discomfort
 - Symptoms of gastroesophageal reflux
 - Flatulence
- Female < male, 1.5–2:1 overall, higher in those who seek care

Physical-Exam

- Usually well appearing with normal physical
- May have tender sigmoid or palpable sigmoid cord

ESSENTIAL WORKUP

Clinical diagnosis: Careful history crucial

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Typically no abnormalities found
- Labs to be considered (to exclude other pathology), but not required:
 - CBC:
 - Should not have leukocytosis or anemia
 - Normal ESR and CRP useful in excluding inflammatory conditions
 - Serum chemistry, thyroid studies unlikely to be useful
 - Stool for ova and parasites:
 - Most useful for diarrhea workup
 - Consider outpatient serum test for celiac

Imaging

Only necessary if excluding emergent pathology

Diagnostic Procedures/Surgery

Colonoscopy/flexible sigmoidoscopy for select patients (outpatient)

DIFFERENTIAL DIAGNOSIS

- Celiac disease
- Inflammatory bowel disease:
 - Ulcerative colitis/proctitis
 - Crohn's disease
- Infectious enteritis
- Small-intestinal bacterial overgrowth
- Lactose intolerance
- Colorectal cancer
- Diverticular disease
- Biliary disease
- Diabetic gastroparesis
- Pancreatitis
- Thyroid malfunction
- Obstruction
- Peptic ulcer disease
- Acute intermittent porphyria



TREATMENT

PRE HOSPITAL

No specific treatment required

INITIAL STABILIZATION/THERAPY

- Symptomatic treatment
- Pain control
- Administer fluids if dehydrated

ED TREATMENT/PROCEDURES

- Empathetic approach and therapeutic physician–patient relationship is most important.
- Exercise:
 - Improves gastric emptying and constipation
- Diet:
 - Many believe symptoms have a food trigger, but not yet proven.
 - Exclusion diets starting with gluten or lactose can be empirically considered.

- Avoid beans, cabbage, uncooked broccoli, other flatulent foods if symptomatic.
- Constipation symptoms:
 - High-fiber diet, fiber supplements
- Abdominal pain:
 - Antispasmodics like hyoscyamine and dicyclomine may be helpful short-term
- Probiotics:
 - Bifidobacteria appear more effective than lactobacilli
- Antidepressants:
 - TCAs and possibly SSRIs appear to be effective at relieving global IBS symptoms and reducing abdominal pain.
- Psychological therapies appear effective.

MEDICATION

First Line

- Dicyclomine: 10–20 mg PO q6h
- Hyoscyamine: 0.125–0.25 mg PO or sublingual not to exceed 12 tab/day

Second Line

- Amitriptyline: 25 mg PO at bedtime (or another TCA)
- Fluoxetine: 20 mg PO daily (or another SSRI)
- Bifidobacteria probiotic



FOLLOW-UP

DISPOSITION

Admission Criteria

Uncertain diagnosis with suspicion of an emergent abdominal condition

Discharge Criteria

Almost all patients can be managed as outpatients.

Issues for Referral

Some may benefit from GI or psychiatric referral.

FOLLOW-UP RECOMMENDATIONS

Most important is follow-up with primary care physician to foster a therapeutic physician–patient relationship.

PEARLS AND PITFALLS

- Beware of other emergent pathology.
- IBS is common, so it is likely the underlying cause of many abdominal workups done in the ED.

ADDITIONAL READING

- American College of Gastroenterology Task Force on Irritable Bowel Syndrome, Brandt LJ, Chey WD, et al. An evidence-based position statement on the management of irritable bowel syndrome. *Am J Gastroenterol*. 2009;104 (Suppl 1):S1–S35.
- Whelan K, Quigley EM. Probiotics in the management of irritable bowel syndrome and inflammatory bowel disease. *Curr Opin Gastroenterol*. 2013;29(2):184–189.
- Vidlock EJ, Chang, L. Irritable bowel syndrome: Current approach to symptoms, evaluation, and treatment. *Gastroenterol Clin North Am*. 2007;36(3):665–685, x.



See Also (Topic, Algorithm, Electronic Media Element)

- Constipation
- Diarrhea
- Gastroenteritis
- Inflammatory Bowel Disease



CODES

ICD9

- 306.4 Gastrointestinal malfunction arising from mental factors
- 564.1 Irritable bowel syndrome

ICD10

- F45.8 Other somatoform disorders
- K58.0 Irritable bowel syndrome with diarrhea
- K58.9 Irritable bowel syndrome without diarrhea

IRRITABLE INFANT

David H. Rubin



BASICS

DESCRIPTION

- Most children have some period of the day when they are most irritable, usually toward the evening:
 - Normal infant crying ranges from 1–4 hr by 6 wk of age.
 - During the 1st 6 mo of life, 1 mo olds have the highest prevalence of crying
- Irritability is based on a comparison with the child's normal behavior pattern
- Colic is the most common cause of inconsolable crying in infants, occurring in as many as 25% of healthy children:
 - Episodes of paroxysmal screaming accompanied by drawing up knees and oftentimes passage of flatus
 - Usually begins at 2–3 wk and may continue through 12 wk
 - Diagnosis of exclusion

ETIOLOGY

- Bites: Spider/insect bite
- Burn
- Cardiac (supraventricular tachycardia, congestive heart failure, aberrant left coronary artery, coarctation of the aorta, endocarditis, myocarditis)
- Child abuse
- Corneal abrasion/foreign body (eyelash) in eye
- Diaper pin
- Diphtheria, pertussis, and tetanus (DPT) and other vaccine reactions
- Endocrine/metabolic (inborn errors of metabolism, metabolic acidosis, hypernatremia, hypoglycemia, hypocalcemia, hyperthyroid—direct or by transplacental passage of maternal thyroid stimulating immunoglobulins)
- Foreign body, fracture, tourniquet (hair around digit or penis)
- GI (gastroenteritis, colic, gastroesophageal reflux, esophagitis, volvulus, malrotation constipation, cow's milk protein intolerance, anal fissure, intussusception, appendicitis)
- Genitourinary (incarcerated hernia, testicular torsion, genital tourniquets, urinary retention)
- Iron deficiency/anemia
- Medications/toxins: Aspirin, antihistamines, atropine, adrenergics, home remedies, new prescription, mercury)
- Meningitis
- Minor acute infections (upper respiratory infection, otitis media, thrush,

gingivostomatitis)

- Neurologic (increased intracranial pressure: Mass, hydrocephalus, intracranial hemorrhage, hematoma—subdural, epidural, skull fracture)
- Osteomyelitis
- Parental anxiety
- Pneumonia
- Sickle cell crisis
- Splinter
- Teething
- Trauma
- UTI
- Vascular

DIAGNOSIS

SIGNS AND SYMPTOMS

- Vital signs
- Chief complaint
- Chronology of events

History

Obtain complete history (including neonatal history) and information regarding routine feeding, crying.

Physical-Exam

- Assess vital signs including rectal temperature and pulse oximetry.
- Measure and plot for percentiles: Height, weight, and head circumference.
- Perform a thorough physical exam with infant completely undressed.

ESSENTIAL WORKUP

This is usually directed by a comprehensive history and physical exam. Specific studies may be obtained.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC, urinalysis, chemistries, and cultures as indicated by history and physical exam
- Stat blood glucose at bedside if indicated.
- Stool hemoccult test if GI signs or symptoms

Imaging

- Chest radiograph to exclude cardiopulmonary disease
- Skeletal survey, if indicated
- CT scan of the head, chest, etc. usually directed by history and physical exam
- Contrast radiograph studies such as barium enema for specific indications

Diagnostic Procedures/Surgery

- Fluorescein eye exam
- ECG

DIFFERENTIAL DIAGNOSIS

See etiology above. It is essential to distinguish benign, self-limited conditions from those that might be life threatening.



TREATMENT

PRE HOSPITAL

As determined by history, physical exam, and lab studies

INITIAL STABILIZATION/THERAPY

- Manage underlying conditions; stabilize airway, breathing, and circulation (ABCs).
- Immediate removal of hair tourniquets and/or splinters

ED TREATMENT/PROCEDURES

- Initial evaluation of the child focusing on parent–child interaction and then on potential underlying conditions
- Colic responds to soothing, rhythmic activities, avoiding stimulants (coffee, cola), minimizing daytime sleep:
 - Soy or hydrolyzed casein formula may be transiently beneficial.
 - Parents must reduce stress
 - No proven pharmacologic therapy
 - Probiotics may be useful
- Support, empathy, close follow-up
- Prolonged observation of the child is usually appropriate.

MEDICATION

Dependent on the underlying condition

First Line

Dependent on the underlying condition

Second Line

Dependent on the underlying condition



FOLLOW-UP

DISPOSITION

Admission Criteria

- Life-threatening underlying condition
- Significant parental stress secondary to crying infant

Discharge Criteria

- No serious condition
- Functional and supportive family
- Excellent follow-up is essential; parents must feel that their observations and concerns are not being ignored. Close follow-up and ongoing observation are mandatory to reevaluate the child and provide support to the family.

Issues for Referral

Determined by specific specialty related issues

FOLLOW-UP RECOMMENDATIONS

Long-term follow-up strongly recommended

PEARLS AND PITFALLS

- Address life-threatening/serious causes of irritability first:
 - Cardiovascular: Supraventricular tachycardia, congestive heart failure, endocarditis/myocarditis
 - Neurologic: Subdural/epidural, meningitis, intracranial hemorrhage, increased intracranial pressure, skull fracture
 - GI: Volvulus, intussusception, appendicitis, peritonitis
 - Metabolic: Metabolic acidosis, electrolyte disturbances
 - Genitourinary: UTI, torsion of testis, incarcerated hernia
 - Pulmonary: Foreign body, pneumothorax, pneumonia
 - Dermatologic: Strangulated digit
 - Toxicologic: Toxic ingestion, immunization reaction
 - Trauma
 - Ophthalmologic: Corneal abrasion, glaucoma
 - Other: Child abuse, transplacental passage of maternal medications that may cause irritability
- Detailed history and complete physical exam in the noncritically ill child is crucial before obtaining any lab or radiologic studies

ADDITIONAL READING

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CODES

ICD9

- 780.91 Fussy infant (baby)
- 780.92 Excessive crying of infant (baby)
- 789.7 Colic

ICD10

- R10.83 Colic
- R68.11 Excessive crying of infant (baby)
- R68.12 Fussy infant (baby)

IRRITANT GAS EXPOSURE

Patrick M. Whiteley • Sean M. Bryant



BASICS

DESCRIPTION

- An irritant is any noncorrosive substance that on immediate, prolonged, or repeated contact with respiratory mucosa will induce a local inflammatory reaction.
- Respiratory irritants are inhaled as gases, fumes, particles, or liquid aerosols.
- Inhaled irritants:
 - Pulmonary toxicity is determined primarily by their water solubility.
- Inhalation accidents frequently involve a mixture of irritant gases as well as chemical asphyxiants:
 - Carbon monoxide
 - Hydrogen cyanide
 - Hydrogen sulfide
 - Oxides of nitrogen
- Risk factors include exposure to potential irritants:
 - Occupational
 - Leisure
 - Intentional
 - Accidental
- Pathophysiology:
 - Cellular injury through interaction with respiratory mucosal water with subsequent formation of acids, alkalis, and free radicals

ETIOLOGY

- Settings:
 - Industrial: Chemical manufacturing, mining, plastics, and petroleum industries
 - Home: Improper use or storage of cleaning chemicals
 - Fires: Combustion yields toxic gases.
 - Civil Disturbance: Riot control agents.
- **Immediate onset** of upper airway inflammation with highly water-soluble irritant gases or with aerodynamic diameter > 5 mm:
 - Ammonia (fertilizers, refrigerants, dyes, plastics, synthetic fibers, cleaning agents):
 - Immediate symptoms range from mild edema and erythema to full-thickness burns and airway obstruction.
 - Sulfur dioxide (fumigants used on produce, bleaching, tanning, brewing,

- wine making, combustion of coal, and smelting of sulfide-containing ores):
 - Combines with water, forming sulfuric acid.
- Hydrogen chloride (formed during combustion of chlorinated hydrocarbons such as polyvinyl chloride):
 - Combines with water, forming hydrochloric acid.
- Chloramine (generated when ammonia and bleach are mixed):
 - When exposed to moist surfaces, releases hypochlorous acid.
- Acrolein (production of plastics, pharmaceuticals, synthetic fibers; formed during combustion of petroleum products, cellulose, wood, paper):
 - May cause protein damage via free radical production and sulfhydryl binding.
- Formaldehyde (production of plywood, particle board, insulation; combustion product of gas stoves and heaters):
 - Combines with water to form sulfuric acid and formic acid.
- Hydrogen fluoride (combustion of fluorinated hydrocarbons):
 - Depletes calcium stores, resulting in cell death.
- Riot control agents (Capsaicin [OC], Chlorobenzylidenemalononitrile [CS], and Chloroacetophenone [CN]):
 - Lacrimation agents which cause temporary ocular discomfort.
- **Latent period** of minutes to hours before onset of symptoms with irritant gases of intermediate water solubility or aerodynamic diameter of 1–5 mm:
 - Chlorine (product of chlorinated chemicals; bleaching agent):
 - Upper and lower airway damage after reacting with water to form hydrochloric and hypochlorous acids
- **Delayed onset** of symptoms up to 24 hr after inhalation with irritant gases of poor water solubility or aerodynamic diameter < 1 mm (with little or no warning of exposure):
 - Oxides of nitrogen produced:
 - In manufacture of dyes and fertilizers
 - By electric arc welding and gas blowing
 - By fermentation of nitrogen-rich silage (silo-filler's disease)
 - In combustion of nitrocellulose and polyamides
 - Phosgene/carbonyl chloride (arc welding and pesticide production: Combustion of chlorinated hydrocarbons and solvents)
 - Ozone (produced during arc welding)
 - Cadmium oxide (oxyacetylene welding and electroplating)



DIAGNOSIS

SIGNS AND SYMPTOMS

- Dependent on water solubility
- Highly water-soluble gases:

- Eye, nose, throat burning
- Shortness of breath
- Wheezing
- Cough
- Hoarseness
- Stridor
- Obstruction
- Intermediate water solubility:
 - Upper and lower tract involvement
 - Mucosal irritation
 - Bronchospasm
 - Dyspnea
 - Wheezing
 - Cough
 - Rales
 - Possible delayed pulmonary edema
- Other:
 - Dermal irritation
 - Headache
 - Nausea
 - Vomiting
 - Confusion
 - Seizures
 - Syncope

History

- Known exposures
- Type of chemical/industry
- Rapidity of symptom
- Material safety data sheet from exposure site
- Water solubility of agent

Physical-Exam

- HEENT:
 - Conjunctival injection
 - Lacrimation
 - Chemosis
- Respiratory
- Stridor
 - Voice changes
 - Dyspnea
 - Wheezing

- Cough
- GI:
 - Vomiting
- Dermatologic:
 - Skin erythema/irritation
 - Erythematous rash
- Neurologic:
 - Confusion
 - Seizure activity

ESSENTIAL WORKUP

History of exposure to irritant gases in addition to noted symptoms confirm diagnosis.

DIAGNOSIS TESTS & INTERPRETATION

ECG in the following patients:

- Elderly
- Cardiac history
- Evidence of significant pulmonary symptoms

Lab

- Arterial blood gas to assess:
 - Oxygenation
 - Ventilation status
 - pH
 - Pulse oximetry is unreliable.
- Carbon monoxide level:
 - If smoke inhalation with concomitant irritant gas inhalation (see “Carbon Monoxide Poisoning”)
- Methemoglobin level:
 - If oxides of nitrogen are suspected
- Serum calcium level:
 - If hydrogen fluoride is suspected
- Lactate:
 - Elevation may indicate cellular poisoning from carbon monoxide or cyanide.
- Pregnancy test in all females of childbearing age
- Rapid dextrose
- Cardiac enzyme levels if acute coronary syndrome suspected

Imaging

CXR:

- Frequently normal on initial presentation
- May take up to 24 hr to reveal pulmonary edema or evidence of diffuse injury.

Diagnostic Procedures/Surgery

- Spirometry:
 - Assess evidence suggesting airway narrowing and bronchoconstriction.
- Direct laryngoscopy:
 - Assess evidence of upper airway edema.
- Corneal fluorescein:
 - Assess evidence of corneal burns/injury.

DIFFERENTIAL DIAGNOSIS

- Asthma exacerbation
- Allergic stimuli (pollen)
- Physical stimuli (cold air)
- Bronchitis
- Pneumonia
- Occupational asthma
- Hypersensitivity pneumonitis
- Congestive heart failure



TREATMENT

PRE HOSPITAL

Rescuer's goal is to prevent self-contamination with use of protective clothing or equipment (self-contained breathing apparatus).

INITIAL STABILIZATION/THERAPY

- ABCs:
 - 100% oxygen through a tight-fitting, nonbreathing face mask
 - Early intubation may be necessary to protect airway from edema.
 - Mechanical ventilation
 - Continuous positive airway pressure or positive end-expiratory pressure may enhance oxygenation.
- Decontaminate by removing clothes and irrigating skin and ocular tissues.

ED TREATMENT/PROCEDURES

- Inhaled nebulized β_2 -adrenergic agonists (albuterol) for bronchoconstriction
- Inhaled/IV/PO corticosteroids: Beclomethasone, methylprednisolone, prednisone:
 - Controversial
 - No controlled trials that document benefit of acute corticosteroids after irritant gas inhalation.
- Nebulized sodium bicarbonate (3.75% solution) after chlorine gas exposure:
 - Reported to improve oxygenation in several case reports/series.
- Nebulized calcium gluconate after acute hydrogen fluoride inhalation:

- Reported, but with no proven benefit
- Hydroxocobalamin or cyanide antidote kit if hydrogen cyanide is suspected (see “Cyanide Poisoning”)
- Oxygen or hyperbaric oxygen therapy if carbon monoxide poisoning documented

MEDICATION

- Albuterol: 0.5 mL (peds: 0.03 mL or 0.15 mg/kg/dose) of 0.5% solution diluted in NS to 3 mL aerosolized
- Beclomethasone MDI: 1–2 sprays (40–80 µ/spray) BID
- Calcium gluconate: Nebulized (2.5–3% solution) prepared by adding 0.15 g of calcium gluconate to 6 mL of NS
- Methylprednisolone: 80–125 mg (peds: 1–2 mg/kg) IV
- Prednisone: 40–80 mg (peds: 1–2 mg/kg) PO
- Sodium bicarbonate: Nebulized (3 mL of 8.4% sodium bicarbonate mixed with 2 mL of NS to prepare 5 mL of 5% solution)



FOLLOW-UP

DISPOSITION

Admission Criteria

- ICU admission:
 - Intubated patients
 - Significant respiratory insufficiency or potential upper airway obstruction
- Persistently symptomatic with bronchospasm or oxygen requirement
- Exposure to irritant gases that affect peripheral airways:
 - Delayed pulmonary edema and respiratory failure may occur.
- Conservative treatment for children, pregnant women, elderly patients, or those with pre-existing chronic obstructive pulmonary or coronary disease

Discharge Criteria

- Mild exposures that respond well to supportive care and have no oxygen requirement or bronchospasm after 4–6 hr of observation
- Follow-up chest radiograph during observation and prior to discharge, especially if any symptoms are present or clinically worsening

Issues for Referral

Intensive care for patients with early evidence of acute lung injury

FOLLOW-UP RECOMMENDATIONS

- Occupational medicine referral for work-related exposures.
- Pulmonary follow-up for repeated symptomatic exposures.

PEARLS AND PITFALLS

- Beware of delayed onset of low-solubility agents. These exposures may require 23-hr observation for delayed respiratory symptoms.
- Avoid exposure of agent to first responders, with appropriate decontamination.

ADDITIONAL READING

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- Taylor AJ. Respiratory irritants encountered at work. *Thorax*. 1996;51:541–545.
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- Weiss SM, Lakshminarayan S. Acute inhalation injury. *Clin Chest Med*. 1994;15:103–116.



See Also (Topic, Algorithm, Electronic Media Element)

- Carbon Monoxide Poisoning
- Cyanide Poisoning



CODES

ICD9

- 986 Toxic effect of carbon monoxide
- 987.7 Toxic effect of hydrocyanic acid gas
- 987.9 Toxic effect of unspecified gas, fume, or vapor

ICD10

- T57.3X1A Toxic effect of hydrogen cyanide, accidental (unintentional), initial encounter
- T58.91XA Toxic effect of carb monx from unsp source, acc, init
- T59.91XA Toxic effect of unsp gases, fumes and vapors, acc, init

ISONIAZID POISONING

Sean M. Bryant



BASICS

DESCRIPTION

- Complexes with and inactivates pyridoxal-5 phosphate, the active form of pyridoxine (vitamin B₆)
- Inhibits pyridoxine phosphokinase, hindering the conversion of pyridoxine to its active form
- Yields a net decrease in γ -aminobutyric acid (GABA) production:
 - Depressed GABA causes cerebral excitability and seizures
- Inhibits lactate dehydrogenase, decreasing the conversion of lactate to pyruvate:
 - Contributes to the profound anion gap metabolic acidosis
- Chronic toxicity:
 - Interferes with synthesis of nicotinic acid (niacin)
 - May cause syndrome indistinguishable from pellagra after months of therapy (niacin deficiency)
- Some actions similar to the monoamine oxidase inhibitors:
 - Reports of a tyramine-like reaction to isoniazid (INH)
 - Rare cases of mania, diaphoresis, depression, obsessive–compulsive disorder, and psychosis
- Pharmacokinetics:
 - Rapidly absorbed, peak levels within 1–2 hr
 - Volume of distribution is 0.6 L/kg and protein binding is low (10%)
 - Renally excreted within 24 hr after acetylation in the liver
 - Half-life is < 1 hr in fast acetylators and 2–4 hr in slow-acetylating individuals

ETIOLOGY

- High-risk groups include:
 - Immigrants
 - Homeless
 - HIV infected
 - Alcoholics
 - Lower socioeconomic status populations
- Slow acetylators (60% of African Americans and Whites compared to 20% of Asians) are more prone to chronic effects/toxicity
- LD50 estimated at 80–150 mg/kg
- Ingestions < 1.5 g lead to mild toxicity, and those of 10 g or more often result in fatality

SIGNS AND SYMPTOMS

- Acute toxicity:
 - Neurologic:
 - Altered mental status
 - Seizures refractory to standard therapy
 - Agitation
 - Coma
 - Dizziness
 - Ataxia
 - Hyper-reflexia
 - Slurred speech
 - Hallucinations
 - Psychosis
 - GI:
 - Nausea
 - Vomiting
 - Renal:
 - Anuria
 - Oliguria
 - Cardiovascular:
 - Hypotension
 - Tachycardia
 - Shock
 - Cyanosis
 - Metabolic:
 - Profound anion gap metabolic acidosis (elevated lactate)
 - Hyperthermia
- Chronic toxicity:
 - Neurologic:
 - Peripheral neuropathy
 - Optic neuritis, optic atrophy
 - Psychosis
 - Insomnia
 - Vertigo
 - Pellagra
- GI hepatitis:
 - Liver failure, hepatitis
 - Nausea, vomiting, constipation
 - Anorexia

Without specific history of ingestion, initiate general workup for:

- Altered mental status
- Seizures
- Metabolic acidosis

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Arterial blood gas:
 - Profound metabolic acidosis
- Electrolytes, BUN/creatinine, glucose:
 - Elevated anion gap acidosis
 - Hyperglycemia
- CBC:
 - Acute toxicity:
 - Leukocytosis
 - Eosinophilia
 - Chronic toxicity:
 - Agranulocytosis
 - Eosinophilia
 - Hemolysis
 - Anemia

Imaging

- CXR:
 - Evidence of tuberculosis increases suspicion for ingestion/toxicity.
 - Evaluate for aspiration pneumonia.
- CT/lumbar puncture if indicated and questionable history

DIFFERENTIAL DIAGNOSIS

- Toxins:
 - Tricyclic antidepressants
 - Salicylates (aspirin)
 - Theophylline
 - Methanol/ethylene glycol
 - Lithium
 - Carbon monoxide
 - Cocaine/cyanide
 - Agents that cause metabolic acidosis
- CNS:
 - Cerebrovascular accident
 - Intracranial hemorrhage/mass/trauma/abscess
- Hypoglycemia

- Uremia
- Thyrotoxicosis



TREATMENT

PRE HOSPITAL

Collect prescription bottles/medications for identification in the ED

INITIAL STABILIZATION/THERAPY

- ABCs:
 - Supplemental oxygen
 - Intubate if necessary for airway protection
 - Secure IV access
 - Cardiac monitor
 - 0.9% NS access
- Naloxone, thiamine, D50W (Accu-Chek) if altered mental status

ED TREATMENT/PROCEDURES

- Vitamin B₆ (pyridoxine):
 - Specific antidotal treatment for INH toxicity
 - Goal: 1 g of pyridoxine for each gram of INH ingested (1 g q2–3min)
 - 5 g for unknown amount ingested
 - May repeat in 20 min for refractory seizures or persistent coma
 - If insufficient quantity of pyridoxine available, contact other hospital pharmacies and the regional poison control center to obtain more
 - If no parenteral pyridoxine available, crush tablets and give as a slurry via NG tube
- Seizure control:
 - Pyridoxine restores deficiency in GABA
 - Benzodiazepines are synergistic with pyridoxine
 - Phenytoin has no role
- Gastric decontamination after stabilization:
 - Consider gastric lavage only in life-threatening ingestions presenting within 1 hr with a protected airway (being aware of potential seizure activity and obtundation)
 - Activated charcoal (AC) dosed at 10:1 ratio (AC:drug)
- Hemodialysis:
 - Persistent symptoms despite adequate therapy
 - Renal insufficiency in symptomatic patients
- Sodium bicarbonate:
 - Acidosis usually resolves spontaneously after elimination of seizures

MEDICATION

- Dextrose: D50W 1 amp (50 mL or 25 g) (peds: D25W 2–4 mL/kg) IV
- Diazepam (benzodiazepine): 5–10 mg (peds: 0.2–0.5 mg/kg) IV
- Lorazepam (benzodiazepine): 2–6 mg (peds: 0.03–0.05 mg/kg) IV
- Naloxone (Narcan): 2 mg (peds: 0.1 mg/kg) IV/IM initial dose
- Pyridoxine (vitamin B₆): 1 g IV for each gram of INH ingested (see above)
- Thiamine (vitamin B₁): 100 mg (peds: 50 mg) IV/IM



FOLLOW-UP

DISPOSITION

Admission Criteria

- ICU admission for refractory seizures, severe acidosis, coma, altered mental status
- Uncontrolled nausea/vomiting, unclear history of ingestion, or suicidal
- Consult regional poison center:
 - (1-800-222-1222)

Discharge Criteria

- Symptoms are usually observed within 45 min of an acute overdose but may be delayed for ≥ 2 hr
- Discharge if asymptomatic after 6 hr

FOLLOW-UP RECOMMENDATIONS

Psychiatric referral for intentional overdoses or suicidal patients

PEARLS AND PITFALLS

- Inadequate appreciation and management of INH poisoning:
 - Refractory seizures to standard treatments is a fundamental clue to INH poisoning
 - Severe acidemia with elevated lactate in altered patients with seizures
- Never paralyze a seizing patient without the use of continuous EEG monitoring
- Goal of pyridoxine therapy is gram for gram of INH
- If pyridoxine adequately treats seizures, may give more if patient remains comatose

ADDITIONAL READING

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- Osterhoudt KC, Henretig FM. A 16-year-old with recalcitrant seizures. *Pediatr Emerg*

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- Tajender V, Saluja J. INH induced status epilepticus: Response to pyridoxine. *Indian J Chest Dis Allied Sci.* 2006;48:205–206.



See Also (Topic, Algorithm, Electronic Media Element)

Seizures



CODES

ICD9

961.8 Poisoning by other antimycobacterial drugs

ICD10

- T37.1X1A Poisoning by antimycobac drugs, accidental, init
- T37.1X4A Poisoning by antimycobacterial drugs, undetermined, init

ISOPROPANOL POISONING

Paul Kolecki



BASICS

DESCRIPTION

- CNS depressant effect of isopropanol is 2 to 3 times as potent as that of ethanol.
- Many products that contain isopropanol also contain methanol, ethylene glycol, and ethanol.
- Rapidly absorbed following oral ingestion
- Ketogenic, but does not cause significant acidosis
- Metabolized by alcohol dehydrogenase to acetone (a CNS depressant):
 - Concomitant ethanol ingestion doubles half-life of isopropanol but not that of acetone.
 - Acetone eliminated by lung and kidney
- Half-life:
 - Isopropanol: 3–16 hr
 - Acetone: 7.5–26 hr

ETIOLOGY

- Isopropanol (isopropyl alcohol): Clear, colorless, volatile liquid with faint odor of acetone and bitter taste
- Available as 70% rubbing alcohol solution:
 - May contain blue dye that was added to inhibit its abuse (“blue heaven”)
- Found in:
 - Various toiletries
 - Disinfectants
 - Window-cleaning solutions
 - Paint remover
 - Solvents
 - Jewelry cleaners
 - Detergents
 - Antifreeze
 - Hand sanitizers
- Typical adult patient: Chronic alcoholic who has been on drinking binge and recently depleted his or her ethanol supply
- Dermal and rectal administration can cause systemic toxicity.



DIAGNOSIS

SIGNS AND SYMPTOMS

- Usually occur within 30–60 min of ingestion
- Neurologic:
 - Lethargy
 - Weakness
 - Headache
 - Inebriation
 - Vertigo
 - Ataxia
 - Apnea
 - Coma
 - Initial excitation phase seen with ethanol ingestion is absent.
- GI:
 - Nausea/vomiting
 - Abdominal pain
 - Gastritis
 - Hematemesis
- Cardiovascular:
 - Hypotension
 - Tachycardia
 - Myocardial depression
 - Peripheral vascular dilation
- Pulmonary:
 - Respiratory depression
 - Hemorrhagic tracheobronchitis
- Dermatologic:
 - Skin irritation
 - Burns
- Ocular:
 - Irritation
 - Lacrimation

Pediatric Considerations

- Accidental ingestions common in <6-yr olds.
- Rubbing alcohol sponge baths may cause inhalational toxicity.

ESSENTIAL WORKUP

- History of ingestion
- Odor of isopropanol or acetone on patient's breath

DIAGNOSIS TESTS & NTERPRETATION

Lab

- Electrolytes, BUN, creatinine (Cr), glucose:

- Hypoglycemia occurs.
- Does *not* produce significant acidosis unless accompanied by end-organ hypoperfusion.
- Acetone can produce false elevation of serum Cr:
 - o When acetone level > 40 mg/dL, Cr values rise at ~ 1 mg Cr/100 mg/dL acetone.
 - o Cr returns to baseline following acetone metabolism.
- CBC:
 - Decreased hematocrit with significant hemorrhagic gastritis
- Arterial blood gas:
 - Acidosis rare unless owing to hypoperfusion or coingestant.
- Urinalysis:
 - Ketones present.
- Serum ketones are present.
- Isopropanol level:
 - Coma with level > 150 mg/dL
- Serum osmolality:
 - Osmolar gap: Difference between measured and calculated osmolality
 - Calculated osmolality = $2 \text{ Na}^+ \text{ BUN}/2.8 + \text{glucose}/18 + \text{ethanol}/4.6$.
 - Osmolar gap is present if measured minus calculated osmolality is > 10.
 - Gap increases by 1 mOsm/kg for each 5.9 mg/dL of isopropanol and 5.5 mg/dL of acetone.

Imaging

- CXR: For aspiration pneumonia with altered mental status and vomiting
- CT head: Concomitant head injury occurs.

DIFFERENTIAL DIAGNOSIS

- For CNS depression and elevated osmolar gap includes:
 - Ethanol
 - Ethylene glycol
 - Methanol
 - Glycerol
 - Mannitol

Pediatric Considerations

Prone to hypoglycemia following exposure



TREATMENT

PRE HOSPITAL

Search for and transport all bottles and medications that may have been ingested by the

patient when an overdose is suspected.

INITIAL STABILIZATION/THERAPY

- ABCs:
 - Maintain airway and assist in ventilation if necessary.
- Hypotension:
 - Treat initially with 0.9% NS IV fluid bolus.
 - Initiate dopamine or norepinephrine infusion if hypotension persists.
 - Packed RBCs with significant hemorrhagic gastritis
- Place NG tube and irrigate for patients with hematemesis.
- Naloxone, thiamine, dextrose (or Accu-Chek) if altered mental status

ED TREATMENT/PROCEDURES

- Primarily supportive therapy—no specific antidote
- Irrigate skin/eyes for dermal or ocular exposure.
- Consider activated charcoal:
 - For coingestants
 - Large doses can absorb significant amounts of isopropanol.
- Do **not** treat with ethanol infusion or 4-methylpyrazole.
- Hemodialysis:
 - Effectively removes isopropanol and acetone.
 - Most managed with supportive care alone.
 - Indications:
 - Hemodynamic instability despite fluid replacement and use of pressors
 - Levels > 400 mg/dL (associated with severe hypotension and prolonged coma)

MEDICATION

- Activated charcoal slurry: 1–2 g/kg up to 90 g PO
- Dextrose: D50W 1 amp: 50 mL or 25 g (peds: D25W 2–4 mL/kg) IV
- Dopamine: 2–20 mg/kg/min IV
- Naloxone (Narcan): 2 mg (peds: 0.1 mg/kg) IV or IM initial dose
- Thiamine (vitamin B₁): 100 mg (peds: 50 mg) IV or IM



FOLLOW-UP

DISPOSITION

Admission Criteria

Moderate to severe isopropanol toxicity (altered mental status, hypotension)

Discharge Criteria

- Observe asymptomatic patients following ingestion for 2–4 hr before discharge.
- Mild intoxication that resolves over 4–6 hr

Issues for Referral

GI referral for endoscopy for patients with recurrent hematemesis.

FOLLOW-UP RECOMMENDATIONS

Alcohol detox or psychiatry referral for patients with intentional ingestion

PEARLS AND PITFALLS

- Supportive care is the primary treatment.
- Do **not** treat with ethanol infusion or 4-methylpyrazole.

ADDITIONAL READING

- Emadi A, Coberly L. Intoxication of a hospitalized patient with an isopropanol-based hand sanitizer. *N Engl J Med*. 2007;356:530–531.
- Kraut JA, Kurtz I. Toxic alcohol ingestions: Clinical features, diagnosis, and management. *Clin J Am Soc Nephrol*. 2008;3:208–225.
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See Also (Topic, Algorithm, Electronic Media Element)

- Alcohol Poisoning
- Ethylene Glycol Poisoning
- Methanol Poisoning



CODES

ICD9

- 976.0 Poisoning by local anti-infectives and anti-inflammatory drugs
- 980.2 Toxic effect of isopropyl alcohol
- 982.8 Toxic effect of other nonpetroleum-based solvents

ICD10

- T51.2X1A Toxic effect of 2-Propanol, accidental (unintentional), initial encounter
- T52.8X1A Toxic effect of organic solvents, accidental, init

JAUNDICE

Andrew K. Chang • Albert Izzo

BASICS

DESCRIPTION

Yellow pigmentation of tissues and body fluids due to hyperbilirubinemia, usually present at levels of >2.5 mg/dL

ETIOLOGY

- Unconjugated (indirect) hyperbilirubinemia: Unconjugated bilirubin is the direct breakdown product of heme, is water insoluble, and is measured as indirect bilirubin:
 - Hemolytic:
 - Excessive production of unconjugated bilirubin
 - Hepatic:
 - Decreased hepatobiliary excretion of bilirubin by:
 - Defective uptake (drugs, Crigler–Najjar syndrome)
 - Defective conjugation (Gilbert syndrome drugs)
 - Defective excretion of bilirubin by the liver cell (drugs, Dubin–Johnson syndrome)
- Conjugated (direct) hyperbilirubinemia:
 - Conjugated bilirubin is water soluble and measured as direct bilirubin.
 - In conjugated hyperbilirubinemia, bilirubin is returned to the bloodstream after conjugation in the liver instead of draining into the bile ducts.
 - Hepatocellular dysfunction:
 - Hepatitis
 - Cirrhosis
 - Tumor invasion
 - Toxic injury
 - Intrahepatic (nonobstructive) cholestasis
 - Extrahepatic (obstructive) cholestasis

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Cholestasis:
 - Pruritus
 - Pale stools

- Dark urine
- Malignancy:
 - Anorexia
 - Weight loss
 - Malaise
- Abdominal pain

Physical-Exam

- Icterus of sclera and tongue base (levels > 2.5 mg/dL)
- Right upper quadrant tenderness:
 - Courvoisier rule:
 - Painless jaundice and a palpable, nontender gallbladder represent malignant common duct obstruction.
- Stigmata of cirrhosis:
 - Abdominal collateral circulation including caput medusae, hepatosplenomegaly, or hepatic atrophy
 - Ascites
 - Spider telangiectasia
 - Palmar erythema
 - Dupuytren contractures
 - Asterixis
 - Encephalopathy
 - Gynecomastia
- Palpable gallbladder
- Hepatomegaly
- Splenomegaly
- Abdominal mass
- Evidence of cachexia
- Excoriations (primary biliary cirrhosis, obstruction)
- Kayser–Fleischer rings:
 - Wilson disease

ESSENTIAL WORKUP

- History and physical exam, together with routine lab tests, will suggest the diagnosis in ~ 80% of patients with jaundice.
- Bilirubin level—severity may suggest cause:
 - Malignancy causes highest levels (10–30 mg/dL).
 - Choledocholithiasis rarely exceeds 15 mg/dL.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Urine dipstick is 74% sensitive for bilirubin.

- Alkaline phosphatase:
 - If no bone disease and not pregnant, then elevation suggests impaired biliary tract function.
 - 2X normal: Hepatitis and cirrhosis
 - 3X normal: Extrahepatic biliary obstruction (i.e., choledocholithiasis) and intrahepatic cholestasis (i.e., drug-induced and biliary cirrhosis)
- Aminotransferases—provide evidence of hepatocellular damage:
 - Alanine aminotransferase (ALT, SGPT): Primarily in the liver
 - Aspartate aminotransferase (AST, SGOT): Liver, heart, kidney, muscle, and brain
- γ -Glutamyl transpeptidase—throughout hepatobiliary system, pancreas, heart, kidneys, and lungs:
 - May be the most sensitive indicator of biliary tract disease.
 - Confirms hepatic origin of an elevated alkaline phosphatase.
- 5'-Nucleotidase—widespread tissue distribution:
 - Confirms hepatic origin of an elevated alkaline phosphatase level.
- Albumin: Decreased with severe liver disease
- PT: Elevation is an important prognostic indicator in patients with acute hepatitis.

Imaging

- US: Most effective initial imaging technique:
 - >90% effective in identifying cholelithiasis
 - Ductal dilation is a reliable indicator of extrahepatic obstruction:
 - A dilated common bile duct (CBD) and gallbladder suggest distal obstruction, whereas dilation of the intrahepatic ducts (without CBD dilation) suggests proximal obstruction.
- Tumors of the liver and head of pancreas are usually well visualized.
- Distinguishes solid liver tumors from cystic structures.
- Plain radiographs:
 - May show evidence of hepatic and splenic enlargement or biliary calcifications
- Hepatic nuclear scan (hepatobiliary iminodiacetic acid scan):
 - Accurate method of diagnosing acute cholecystitis or cystic duct obstruction
 - Time consuming (usually several hours)
- CT:
 - Superior to US in detecting pancreatic and intra-abdominal tumors.
 - Can help differentiate fluid-containing structures.

Diagnostic Procedures/Surgery

Endoscopic retrograde cholangiopancreatography (ERCP):

- Diagnostic:
 - Stones are seen as filling defects within bile duct lumen.

- Malignancies are seen as strictures.
- Therapeutic:
 - Extraction of CBD stones and insertion of stents to bypass malignant obstructions
 - Biopsy under direct vision

DIFFERENTIAL DIAGNOSIS

- Prehepatic:
 - Hemolysis (sickle cell, other hemoglobinopathies)
 - Ineffective erythropoiesis
 - Drugs
 - Gilbert syndrome: Usually benign inherited form of unconjugated hyperbilirubinemia
 - Crigler–Najjar syndrome
 - Prolonged fasting
- Hepatocellular:
 - Hepatitis (infectious, alcoholic, autoimmune, toxin, drug induced)
 - Cirrhosis
 - Postischemic
 - Hemochromatosis
- Intrahepatic cholestasis:
 - Idiopathic cholestasis of pregnancy
 - Drugs
 - Dubin–Johnson syndrome
 - Rotor syndrome
 - Benign recurrent cholelastia
 - Familial syndromes
 - Sepsis
 - Postoperative jaundice
 - Lymphoma
- Extrahepatic obstruction:
 - Common duct stone
 - Biliary stricture
 - Bacterial cholangitis
 - Sclerosing cholangitis
 - Carcinoma (ampulla, gallbladder, pancreas), cholangiosarcoma
 - Pancreatitis, pancreatic pseudocyst
 - Hemobilia
 - Duodenal diverticula
 - Ascariasis
 - Postlaparoscopic cholecystectomy complications
 - Congenital biliary atresia
 - Congenital choledochal cyst

Pediatric Considerations

Intrahepatic cholestasis:

- Cardiovascular (congenital heart disease, congestive heart failure, shock, asphyxia)
- Metabolic or genetic (α_1 -antitrypsin deficiency, trisomy 18 and 21, cystic fibrosis, Gaucher disease, Niemann–Pick disease, glycogen storage disease type IV)
- Infectious (bacterial sepsis, cytomegalovirus, enterovirus, herpes simplex virus, rubella, syphilis, TB, varicella, viral hepatitis)
- Hematologic (severe isoimmune hemolytic disease)



TREATMENT

INITIAL STABILIZATION/THERAPY

- Isotonic IV fluid therapy if dehydrated
- Toxic-appearing patients:
 - Supplemental oxygen, cardiac monitoring
 - Nasogastric suction and bladder catheterization

ED TREATMENT/PROCEDURES

- For bacterial cholangitis/sepsis, obtain blood cultures and administer parenteral antibiotics:
 - Ampicillin, gentamicin, and metronidazole *or*
 - Ticarcillin, or piperacillin, and metronidazole *or*
 - Cefoxitin and tobramycin
- Obstructive extrahepatic jaundice:
 - Surgical consult
- Choledocholithiasis:
 - ERCP papillotomy, balloon or basket retrieval, or open surgery
- Obstructive intrahepatic or nonobstructive jaundice:
 - Medical management:
 - Withdraw causative drug, ethanol
 - Interferon for chronic hepatitis B and C
 - Penicillamine and phlebotomy for Wilson disease and hemochromatosis
 - Corticosteroids for chronic hepatitis of autoimmune origin

Pediatric Considerations

- Exchange transfusion:
 - Emergent treatment of markedly elevated bilirubin (> 20 mg/dL in full-term infants) and for correction of anemia caused by isoimmune hemolytic disease
- Phototherapy—for neonatal jaundice when bilirubin = 17 mg/dL:

- Measure bilirubin once to twice daily and stop when bilirubin has been reduced by about 4–5 mg/dL.
- Phenobarbital: In sepsis and drug-induced causes; decreases conjugated bilirubin.
- Metalloporphyrins: Investigational inhibitors of heme oxygenase

MEDICATION

- Ampicillin: 2 g IV q6h (peds: 25 mg/kg IV q6–8h)
- Cefoxitin: 2 g IV q6h (peds: 40–160 mg/kg/d div. q6–12h)
- Gentamicin: 5–2 mg/kg IV q8h
- Metronidazole: 7.5 mg/kg IV q6h (peds: Same)
- Piperacillin/tazobactam: 3.375 g IV q6h (peds: 300 mg/kg/d div. q6h [> 2 mo of age])
- Ticarcillin/clavulanate: 3.1 g IV q6h (peds: 75–100 mg/kg/d div. q6h)
- Tobramycin: 1 mg/kg IV q6h (peds: Same)



FOLLOW-UP

DISPOSITION

Admission Criteria

- Bacterial cholangitis
- Intractable pain
- Intractable emesis
- Associated pancreatitis
- Elevated PT

Discharge Criteria

- No evidence of infection (evaluate as outpatient)
- Tolerating liquids

ADDITIONAL READING

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- Roche SP, Kobos R. Jaundice in the adult patient. *Am Fam Physician.* 2004;69:299–304.
- Udell JA, Wang CS, Tinmouth J, et al. Does this patient with liver disease have cirrhosis? *JAMA.* 2012;307(8):832–842.
- Wang Q, Gurusamy KS, Lin H, et al. Preoperative biliary drainage for obstructive jaundice. *Cochrane Database Syst Rev.* 2008;16(3):CD005444.



CODES

ICD9

- 277.4 Disorders of bilirubin excretion
- 774.6 Unspecified fetal and neonatal jaundice
- 782.4 Jaundice, unspecified, not of newborn

ICD10

- E80.6 Other disorders of bilirubin metabolism
- P59.9 Neonatal jaundice, unspecified
- R17 Unspecified jaundice

KAWASAKI DISEASE

Adam Z. Barkin

BASICS

DESCRIPTION

- Acute inflammatory process involving multiple organs
- Leading cause of childhood-acquired heart disease in developed countries
- Vasculitis is most severe in medium-sized arteries
- Acute cardiac sequelae:
 - Coronary artery aneurysm:
 - Often lead to stenosis after healing
 - Giant aneurysm:
 - May rupture
 - Myocarditis
 - Pericarditis
- Stages:
 - Acute (lasts 1–2 wk):
 - Fever
 - Oral mucosal erythema
 - Conjunctival injection
 - Erythema and edema of hands and feet
 - Cervical adenopathy
 - Aseptic meningitis
 - Hepatic dysfunction
 - Diarrhea
 - Myocarditis
 - Pericardial effusion (20–40%)
 - No aneurysms by ECHO
 - Subacute (when fever, rash, and lymphadenopathy resolve until about 4 wk):
 - Anorexia
 - Irritability
 - Desquamation of hands and feet
 - Thrombocytosis
 - Coronary artery aneurysms (20% if untreated)
 - Risk for sudden death is highest
 - Convalescent phase (about 6–8 wk):
 - Clinical signs are absent
 - ESR normalizes
 - Epidemiology:

- 80% of cases occur in children < 4 yr old; peak at 1–2 yr; rare in infants < 3 mo old
- Adult cases have been reported
- Asians are at highest risk
- Males > females 1.5:1
- Genetics:
 - _ Possible genetic predisposition
- Risks for nonresponse to standard therapy (10–15%):
 - _ Elevated band count
 - _ Low albumin level
 - _ Abnormal initial ECHO
- Risks for development of coronary artery aneurysms:
 - _ Extremes of age
 - _ Male gender
 - _ Prolonged fever
 - _ Persistent fever after treatment
 - _ Delay in diagnosis
 - _ Increased WBC and/or band count
 - _ Low hematocrit
 - _ Significant increase in CRP and/or ESR

ETIOLOGY

- Unknown—believed to be infectious based on manifestations of disease, epidemics, and increased numbers of cases in winter and early spring
- Current theory:
 - _ Activation of immune system in response to infection
 - _ Genetically susceptible host
 - _ May explain why certain ethnicities have higher incidence of disease
 - More prominent in Asian countries



DIAGNOSIS

- Classic diagnostic criteria:
 - _ Fever for 5 days + 4 of the 5 following criteria:
 - Bilateral conjunctival injection
 - Changes in oral mucosa
 - Polymorphous erythematous rash
 - Changes in hands or feet—edema, erythema, desquamation
 - Cervical lymphadenopathy > 1.5 cm (least common)
- Atypical cases can be seen without meeting diagnostic criteria
 - _ Fever for > 5 days + 2 or 3 clinical criteria with ESR > 40 and CRP > 3
 - _ If > 3 of the below can diagnose incomplete Kawasaki disease
 - Albumin < 3

- Anemia for age
- ALT elevation
- WBC > 15,000
- Urine > 10 WBC per high power field
- Platelets > 450,000 after 7 days
- Thrombocytosis
 - Changes in hands or feet—edema, erythema, desquamation
 - Cervical lymphadenopathy > 1.5 cm (least common)

SIGNS AND SYMPTOMS

History

- Temperature > 38.5°C (often spiking) for at least 5 days:
 - Begins abruptly and may last > 2 wk
- Cardiac:
 - Shortness of breath
 - Chest pain
- HEENT:
 - Eyes:
 - Conjunctivitis
 - Photophobia
 - Mouth:
 - Erythema
 - Dry and fissured lips
- Skin rash
- Musculoskeletal:
 - Arthralgia, arthritis
- Neurologic:
 - Extreme irritability
- GI:
 - Diarrhea
 - Vomiting
 - Abdominal pain

Physical-Exam

- Cardiac:
 - Evidence of congestive heart failure
 - Evidence of pericarditis
 - Rub
 - Evidence of valvular disease
 - Murmur
- HEENT:
 - Eyes:

- Bilateral conjunctival injection without exudates
- Bulbar conjunctiva is more frequently involved than palpebral conjunctiva
- Usually within 2 days of onset of fever and lasting 1–2 wk
- Photophobia, uveitis, iritis
- Mouth:
 - Erythema, dry and fissured lips, strawberry tongue, pharyngeal erythema
- Lymph:
 - Cervical lymphadenopathy (node diameter >1.5 cm)
- Neurologic:
 - Irritability
 - Meningismus
- Skin:
 - Rash, primarily on the trunk
 - May be maculopapular, scarlatiniform, or erythema multiforme–like; erythroderma
 - Changes in the hands or feet—erythema, edema (acute phase); unwilling to bear weight
 - Desquamation (subacute phase) of the tips of fingers and toes 2–3 wk after onset of illness
- Genitourinary:
 - Urethritis
 - Meatitis
- GI:
 - Hydrops of the gallbladder

ESSENTIAL WORKUP

Must think of the diagnosis in a febrile child with rash

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC:
 - WBC—normally elevated with shift to left in acute phase
 - Normocytic anemia
 - Leukopenia and thrombocytopenia are rare
 - Suspect viral infection
 - Thrombocytopenia is a risk factor for development of coronary artery disease
- Urinalysis:
 - Sterile pyuria
 - Proteinuria

- ESR elevated from 1st wk until 4–6 wk
- Increased C-reactive protein
- CSF pleocytosis
- Cultures: Negative blood, urine, CSF, throat
- Increased transaminases and bilirubin

Imaging

- ECHO to evaluate for coronary artery aneurysm:
 - Acute phase (baseline)
 - 2–3 wk
 - 6–8 wk
- CXR

Diagnostic Procedures/Surgery

- ECG if concern about MI or pericarditis
- Slit-lamp exam—uveitis

DIFFERENTIAL DIAGNOSIS

- Viral infections:
 - Adenovirus
 - Enterovirus
 - Measles
 - Epstein–Barr virus
 - Rubella
 - Rubeola
 - Influenza
- Bacterial infection:
 - Scarlet fever (responds rapidly to penicillin)
 - Staphylococcal scalded-skin syndrome
 - Rickettsial disease, including Rocky Mountain Spotted Fever and leptospirosis
 - Cervical adenitis
- Immune-mediated:
 - Stevens–Johnson syndrome
 - Erythema multiforme
 - Serum sickness
 - Connective tissue disease (i.e., Lupus)
 - Other forms of vasculitis



TREATMENT

PRE HOSPITAL

- ABCs
- Oxygen

INITIAL STABILIZATION/THERAPY

ABCs with focus on cardiovascular system

ED TREATMENT/PROCEDURES

- Initiate IV gammaglobulin (IVIG) and aspirin therapy:
 - Do not generally need to monitor salicylate levels because of decreased absorption and increased clearance
- Treatment within the 1st 10 days of illness reduces cardiac sequelae from range of 20–25% to range of 2–4%.
- Cardiology consultation
- Treatment of MIs as in adults

MEDICATION

First Line

- IVIG: 2 g/kg IV over 10–12 hr; retreatment may be required for persistent (> 48–72 hr) or recrudescent fever:
 - Requires close cardiac monitoring
 - Should be started within the 1st 10 days of illness
 - 3–4% failure rate after 2 doses
- Aspirin: 80–100 mg/kg/d PO q6h until about day 14 when fever has resolved; then 3–5 mg/kg/d PO daily for 6–8 wk. Do not exceed 4g/24 h (peds: Do not exceed 120 mg/kg/24 h):
 - Anti-inflammatory
 - Antiplatelet
 - Potentiates the action of IVIG
 - Reduces the occurrence of aneurysms when given with IVIG
 - Alternative dosing at 30 mg/kg/d during acute and subacute phases

Second Line

If no response to 2nd dose of IVIG

- Corticosteroids:
 - Methylprednisolone 30 mg/kg over 3 hr
 - May improve outcome in conjunction with IVIG

May also consider:

- Infliximab
- Cyclosporine A
- Methotrexate



FOLLOW-UP

DISPOSITION

Admission Criteria

- Admit all patients who fulfill diagnostic criteria for Kawasaki disease
- Admit toxic-appearing patients who do not yet meet the criteria for Kawasaki disease

Discharge Criteria

- Nontoxic children who do not fulfill diagnostic criteria
- Close follow-up is required

Issues for Referral

Cardiology consultation for all patients

PEARLS AND PITFALLS

- Prompt diagnosis and therapy can prevent coronary aneurysms in 95%
- Aspirin and IVIG are mainstays of therapy
- Must consider the diagnosis in febrile children presenting to the ED for multiple visits
- Restrict steroids to children with 2 IVIG failures

ADDITIONAL READING

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- Newburger JW, Sleeper LA, McCrindle BW, et al. Randomized trial of pulsed corticosteroid therapy for primary treatment of Kawasaki disease. *N Engl J Med*. 2007;356:663–675.
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- Scuccimarri R. Kawasaki disease. *Pediatr Clin N Am*. 2012;59:425–445
- Tacke CE, Burgner D, Kuipers IM, et al. Management of acute and refractory



See Also (Topic, Algorithm, Electronic Media Element)

Myocardial Infarction



CODES

ICD9

- 429.0 Myocarditis, unspecified
- 446.1 Acute febrile mucocutaneous lymph node syndrome [MCLS]
- 447.6 Arteritis, unspecified

ICD10

- I51.4 Myocarditis, unspecified
- I77.6 Arteritis, unspecified
- M30.3 Mucocutaneous lymph node syndrome [Kawasaki]

KNEE DISLOCATION

Kelly Anne Foley



BASICS

DESCRIPTION

- Defined by the position of the tibia in relation to the distal femur:
 - _ Anterior dislocation:
 - Most common dislocation, accounts for 60%
 - Hyperextension of the knee
 - Rupture of the posterior capsule at 30°
 - Rupture of the posterior cruciate ligament (PCL) and popliteal artery (PA) occurs at 50°
 - _ Posterior dislocation:
 - Direct blow to the anterior tibia with the knee flexed at 90°, “dashboard injury”
 - Anterior cruciate ligament (ACL) is usually spared.
 - _ Medial dislocation:
 - Varus stress causing tear to the ACL, PCL, and lateral collateral ligament (LCL)
 - _ Lateral dislocation:
 - Valgus stress causing tear to the ACL, PCL, and medial collateral ligament (MCL)
- Associated injuries:
 - _ PA injury:
 - Occurs in 35% of dislocations.
 - Anterior dislocations place traction on PA and cause contusion or intimal injury, which may result in delayed thrombosis.
 - Posterior dislocations cause direct intimal fracture and transection of the artery with immediate thrombosis.
 - _ Peroneal nerve injury:
 - Less common than PA injury
 - If present, must rule out concomitant arterial insult
 - Medial dislocation causes injury by traction of the nerve.
 - Rotary injuries have a high incidence of traction and transection.

ETIOLOGY

High-energy injuries such as motor vehicle crashes, auto–pedestrian accidents, and athletic injuries (football most common)



DIAGNOSIS

SIGNS AND SYMPTOMS

- Grossly deformed knee
- Grossly unstable knee in AP plane or on varus/valgus stress
- Lack of distal pulse:
 - PA injury is primary concern.
- Signs of distal ischemia:
 - Pallor, paresthesia, pain, paralysis

History

Mechanism of injury with high level of suspicion

Physical-Exam

- Distal pulses
- Distal nerve function:
 - Hypesthesia of 1st web space, inability to dorsiflex foot
- Ligamentous laxity

ESSENTIAL WORKUP

- History of mechanism of injury
- Complete and careful physical exam:
 - Pulses—palpation, Doppler, ankle–brachial index (ABI), and cap refill
 - Neurologic—sensation to 1st web space and great toe, movement of toes, dorsiflexion of foot
- AP and lateral knee radiographs
- Documented repeat exam if any closed reduction is attempted

DIAGNOSIS TESTS & INTERPRETATION

Imaging

- AP/lateral radiograph of knee:
 - Essential to rule out concomitant fractures
- MRI within 1 wk of injury to define ligamentous injury

Diagnostic Procedures/Surgery

- ABI—likelihood of significant arterial injury requiring surgery low if ≥ 0.9
- Peripheral vascular ultrasonography
- Arteriogram should be considered:
 - High suspicion of PA injury
 - Poor pulses or distal perfusion after reduction
 - Peroneal nerve injury
 - Ischemic symptoms despite normal pulses

DIFFERENTIAL DIAGNOSIS

- Tibial plateau fracture
- Supracondylar femoral fracture
- Ligamentous/tendonous avulsion fracture



TREATMENT

PRE HOSPITAL

- Management of ABCs
- Documentation of pulses and motor response essential
- Splint knee in slight flexion to prevent PA traction or compression.

INITIAL STABILIZATION/THERAPY

- ABCs especially when motor vehicle crash or auto-pedestrian accident
- Fluid resuscitation; hypotension may alter distal pulses and perfusion.
- Closed reduction immediately for any limb ischemia
- Early surgical consult in an open injury or high suspicion of arterial injury

ED TREATMENT/PROCEDURES

- Closed reduction by longitudinal traction and lifting femur into normal alignment without placing pressure on popliteal fossa
- Posterior leg splint/knee immobilizer with knee in 15–20° of flexion
- Repeat neurovascular exam after manipulation and at frequent intervals.
- IV analgesia for patient comfort
- Surgical consult (orthopedic and vascular): Open injury, PA injury, or unable to reduce

MEDICATION

First Line

- Narcotic analgesia IV
- Avoid PO meds, as surgery may be necessary.



FOLLOW-UP

DISPOSITION

Admission Criteria

All patients require admission for observation of limb perfusion and PA repair if necessary.

Discharge Criteria

All patients should be admitted.

Issues for Referral

Eventual repair of ligamentous injuries:

- Usually at 3 wk
- Arthroscopic surgery is contraindicated for 2 wk after injury to prevent compartment syndrome.

FOLLOW-UP RECOMMENDATIONS

- Orthopedics for ligamentous repair
- Vascular for PA injury

PEARLS AND PITFALLS

- Failure to revascularize PA within 6–8 hr: Amputation rate approaches 90%.
- Peroneal nerve injury:
 - Poor prognosis for recovery
- Delayed compartment syndrome may occur.

ADDITIONAL READING

- Kelleher HB, Mandavia D. Dislocation, knee. *eMedicine* [serial online]. 2011. Available at www.emedicine.medscape.com/article/823589-overview
- Mills WJ, Barei DP, McNair P. The value of ankle-brachial index for diagnosing arterial injury after knee dislocation: A prospective study. *J Trauma*. 2004;56(6):1261–1265.
- Nicandri GT, Chamberlain AM, Wahl CJ. Practical management of knee dislocations: A selective angiography protocol to detect limb-threatening vascular injuries. *Clin J Sport Med*. 2009;19(2):125–129.
- Seroyer ST, Musahl V, Harner CD. Management of the acute knee dislocation: The Pittsburgh experience. *Injury*. 2008;97(7):710–718.



CODES

ICD9

- 836.50 Dislocation of knee, unspecified, closed
- 836.51 Anterior dislocation of tibia, proximal end, closed
- 836.52 Posterior dislocation of tibia, proximal end, closed

ICD10

- S83.106A Unspecified dislocation of unspecified knee, init encntr
- S83.116A Anterior disloc of proximal end of tibia, unsp knee, init
- S83.126A Posterior disloc of proximal end of tibia, unsp knee, init

KNEE INJURIES: ACL, PCL, MCL, MENISCUS

Ilona A. Barash

BASICS

DESCRIPTION

- Cruciate ligament injuries:
 - Anterior cruciate ligament (ACL):
 - From the posteromedial aspect of the lateral femoral condyle to the intraspinus area on the tibia
 - Prevents excessive anterior translation of the tibia, internal rotation of the tibia on the femur, or hyperextension of the knee.
 - Posterior cruciate ligament (PCL):
 - Twice as strong and twice as thick as the normal ACL, less commonly injured
 - From anterolateral aspect of medial femoral condyle to the posterior tibia
- Meniscal tears:
 - Medial meniscus injury most common
 - More firmly attached to the joint capsule and less mobile than lateral meniscus
 - Tears are the result of tensile or compressive forces between the femoral and tibial condyles
 - Extension of meniscal tear may result in a free segment that may become displaced into the joint, resulting in a true locked joint.
- Medial collateral ligament:
 - From the posterior aspect of medial femoral condyle to the tibia, distal to joint
 - Often accompanied by other injury:
 - Hyperextension with external rotation (ACL/PCL injured 1st)
 - Anterior stress (ACL injured 1st)

EPIDEMIOLOGY

Incidence and Prevalence Estimates

- ACL:
 - Most commonly injured knee ligament
 - 200,000 ACL injuries annually in US
 - 2/3 of all ACL injuries are noncontact
 - Female gender: 3× greater risk
- Associated injuries:

- ~50% ACL injuries are associated with meniscal tears
- ACL injuries commonly have chondral and subchondral injuries
- Meniscus:
 - Medial meniscus injury 10 × more common than lateral
 - True locked joint in only 30%

ETIOLOGY

- Cruciate ligament injuries:
 - ACL: Often deceleration with flexion and rotation, or hyperextension
 - Usually sports-related, especially skiing and football
 - Plant-and-pivot or stop-and-jump mechanism
 - PCL:
 - “Dashboard injury”: Flexed knee with posteriorly directed force to the anterior proximal tibia (motor vehicle crash or direct trauma)
 - Fall on flexed knee
- Meniscus Injury:
 - Sudden rotary motion of knee associated with squatting, pivoting, turning, and bending
 - Common in sports with low stance positions (wrestling/football) or kneeling position (carpet installers, plumbers)
- Medial collateral ligament injuries:
 - Direct trauma to lateral knee
 - Most common: Valgus stress with external rotation on flexed knee:
 - From catching a ski tip
 - Side tackle (football)

Pediatric Considerations

- The ACL is the most frequently injured knee ligament in children.
- Isolated MCL injury infrequent before growth plate closure (< 14-yr old)

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Cruciate ligament injuries:
 - Feeling knee “give way,” pop, tearing sensation
 - Most patients report immediate knee dysfunction, but some may ambulate despite complete ACL rupture because of stability from supporting structures.
 - Large, almost immediate effusion -- patients report significant swelling
- Medial collateral ligament:

- Tearing sensation and immediate pain in medial aspect of knee
- Medial pain and tenderness may be more pronounced with partial tears than with complete tears.
- Medial meniscus injury:
 - Patient may recall the knee “giving way”
 - Inability to fully extend knee is common
 - Effusion is found in 50% and usually occurs over 6–12 hr.
 - Pain is often intermittent and localized to the joint line
 - Unlike ligamentous injury, patients often report completion of activities at time of injury
 - Degenerative meniscal tears tend to have a more insidious, atraumatic presentation, with mild swelling, vague joint line pain, and sometimes with mechanical symptoms. Often associated with osteoarthritis.

Physical-Exam

- Ability to bear weight reduced with all injuries
- Palpate for pain on:
 - bony prominences for fracture
 - growth plates in children
 - medial and lateral joint line (meniscus and collateral ligament injury)
- Range of motion:
 - Locking: May occur with ACL (interposition of torn cruciate), meniscus injury, loose body (arthritis)
 - Pseudolocking may be present from pain, effusion, or spasm
- Effusion:
 - Immediate (within 2–3 hr) usually indicates a significant intra-articular injury including ACL
 - About 70% of acute knee hemarthroses are caused by ACL injury, but lack of effusion does not rule out ACL injury
 - MCL, meniscus, PCL injuries have more delayed effusion (12–24 hr)
 - Warmth, erythema: Consider infection
- Neurovascular exam:
 - Distal pulses
 - 1st dorsal web-space sensation (deep peroneal nerve)
 - Ankle/toe dorsiflexion
- Stress testing: *Always compare the injured to the uninjured side* (asymmetry is more reliable than absolute degree of laxity):
 - Pain and spasm can limit the utility of all stress testing in the acute phase
 - *Lachman test* is most reliable for ACL:
 - Knee flexed 20°, patient supine with thigh supported and hip slightly externally rotated. Quickly bring the tibia forward on the femur, 1 hand holding proximal tibia, the other stabilizing the femur just above the patella, evaluating for quality of the endpoint and degree of

- anterior translation of the tibia
- Pain with motion = partial tear or disruption
- Quantification of degree of movement less important than simply positive or negative interpretation of test
- *Pivot shift test*: More specific for ACL injury but unreliable without anesthesia and painful acutely. Not recommended routinely in the ED.
- *Anterior/posterior drawer sign*:
 - Knee flexed 90°, patient supine, hip flexed 45°, foot neutral and stabilized (sit on foot)
 - Observe for posterior sag of tibia, positive with PCL injury
 - Posterior drawer (PCL): Movement of tibia back with application of posterior pressure
 - Anterior drawer (ACL): Movement of tibia forward with anterior distraction force
- Quadriceps active test (PCL):
 - Patient supine, knee flexed at 90°, hip flexed at 45°
 - Patient attempts to extend knee against examiner's counterforce
 - Positive if the tibia translates anteriorly during quad activation
- Varus/valgus stress testing: Evaluate in extension and 20° flexion for MCL and LCL laxity
- Meniscus: Wait until acute pain is controlled:
 - *McMurray*: While palpating joint lines, extend the knee while internally and then externally rotating. Pain and click is positive.
 - *Apley*: With patient prone, flex knee to 90°, provide axial load and internally/externally rotate lower leg. Pain is positive.

Pediatric Considerations

- Children and adolescents show more laxity on exam than adults
- Examine hip and obtain radiograph if any concern for hip pathology (especially slipped capital femoral epiphysis)
- Have a high suspicion for epiphyseal growth plate injuries

DIAGNOSIS TESTS & INTERPRETATION

Lab

- If cause of knee effusion not clearly traumatic, synovial aspirate can be sent for cell count, Gram stain, culture, crystals
- Arthrocentesis is usually not indicated after trauma except to relieve symptoms from tense effusion

Imaging

- Ottawa knee rules (adults): Plain films required for patients with any of 5

findings:

- Age \geq 55
- Isolated tenderness of patella
- Tenderness at head of fibula
- Inability to flex 90°
- Inability to bear weight both immediately and in ED (4 steps)
- Standard radiography:
 - Obtain on all suspected ACL injuries due to high risk of fractures
 - Important in children to evaluate for tibial spine and growth plate fractures
 - Views: AP, lateral, oblique, notch
 - Special attention to avulsion fractures of the medial/lateral tibial spine and lateral tibial plateau, which can be seen with ACL/PCL injuries and may be more likely to be treated operatively
 - Fat–fluid level for fracture.
- MRI is around 95% sensitive for ACL tears and other intra-articular disorders (menisci, PCL, osteonecrosis, osteochondral lesions, occult fractures) and even more specific, but it is rarely indicated emergently.
- Arteriograms to evaluate vascular integrity for suspected dislocations
- US useful to diagnose cysts and popliteal artery aneurysms

Pediatric Considerations

Ottawa knee rules do not apply to children.

ESSENTIAL WORKUP

- Neurovascular evaluation
- Exclusion of fractures and infection
- Evaluate for multidirectional instability
- Valgus/varus stress at 20° of flexion
- Extensor mechanism function
- Lachman test for ACL injury

DIFFERENTIAL DIAGNOSIS

- Growth plate injury
- Tibial plateau bony injury, other fracture
- Transient knee dislocation
- Transient patellar dislocation
- Hip injury causing referred pain
- Nontraumatic causes of knee effusion and pain including septic joint, gout, osteoarthritis, rheumatoid arthritis



TREATMENT

PRE-HOSPITAL STABILIZATION AND INITIAL THERAPY

- ABC's, ATLS
- Immobilize knee
- Document neurovascular function
- Apply ice, elevate, analgesia

ED TREATMENT/PROCEDURES

- Reduce locked knee from meniscus injury within 1st 24 hr after injury:
 - With patient seated, hang extremity off edge of exam table at 90°: This with analgesia alone may reduce locked joint.
 - Assist with applying gentle traction and rotation of tibia
- Arthrocentesis may afford relief with large effusions and assist in reducing locked joint:
 - Follow with compressive dressing
- Treatment (if no fracture):
 - Rest, Ice, Compression, Elevation
 - Weight Bearing as Tolerated, crutches for comfort if needed
 - May provide knee immobilization for protection, but encourage motion out of brace as much as possible, especially if follow-up may be delayed

MEDICATION

- Pain control: NSAIDs preferred over opioids
- Ibuprofen: 400–600 mg (peds: 5–10 mg/kg) PO QID.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Isolated ACL, PCL, meniscus, or collateral ligament injury rarely requires emergent hospitalization
- Low threshold to admit possible knee dislocations for monitoring
- Fractures often need ORIF to limit post-traumatic arthritis

Discharge Criteria

Most patients can be managed as outpatients with appropriate referral.

Issues for Referral

- Re-exam is recommended at 48 hr if ED exam is inconclusive or if history suggests more significant injury than initial exam demonstrates (i.e., severe symptoms, hearing “pop”).
- Orthopedic referral within 1–2 wk if significant ligamentous injury is present.

- Surgical repair of all lesions may be considered for patients wishing to return to sports or active lifestyles.

PEARLS AND PITFALLS

- Do a careful neurovascular exam, and always examine 1 joint above and below the pain for associated injury or referred pain
- Have a high index of suspicion for a reduced total knee dislocation if patient has multidirectional knee instability or injuries to multiple ligaments
- Do not miss: Knee dislocation, fractures, septic joint, referred pain from hip, neurovascular injury

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- Meuffels DE, Poldervaart MD, Diercks RL, et al. Guideline on anterior cruciate ligament injury: A multidisciplinary review by the Dutch Orthopaedic Association. *Acta Orthop*. 2012;83(4):379–869.
- Noyes FR. *Noyes' Knee Disorders: Surgery, Rehabilitation, Clinical Outcomes*. Philadelphia, PA: Saunders-Elsevier; 2010.
- Ryzewicz M, Peterson B, Siparsky PN, et al. The diagnosis of meniscus tears: The role of MRI and clinical examination. *Clin Orthop Relat Res*. 2007;455:123–133.



CODES

ICD9

- 836.0 Tear of medial cartilage or meniscus of knee, current
- 844.1 Sprain of medial collateral ligament of knee
- 844.2 Sprain of cruciate ligament of knee

ICD10

- S83.419A Sprain of medial collateral ligament of unsp knee, init
- S83.519A Sprain of anterior cruciate ligament of unsp knee, init
- S83.529A Sprain of posterior cruciate ligament of unsp knee, init

LABOR

Jonathan B. Walker • James S. Walker



BASICS

Labor denotes the sequence of physiologic occurrences that result in a fetus being transported from the uterus through the birth canal.

DESCRIPTION

- Labor brings about changes in the cervix to allow passage of fetus through birth canal
- Synchronous, coordinated contractions of the uterus
- Contractions progress in magnitude, duration, and frequency to produce dilation of the cervix and ultimate delivery
- Labor is divided into 3 stages:
 - Stage 1 (cervical stage): From onset of uterine contractions to full dilation of cervix
 - Stage 1 is further divided into latent and active phases:
 - In the *latent phase*, uterine contraction with little change in cervical dilation or effacement; contractions are mild, short (< 45 sec), and irregular
 - This is followed by the *active phase*, which begins around time of cervical dilation of 3–4 cm; contractions are strong, regular (every 2–3 min), and last longer (> 45 sec)
 - Stage 2: From onset of complete cervical dilation to time of delivery of infant
 - Stage 3: From time of delivery of baby to time of placental delivery
- Total duration of labor varies with each woman
- Generally, lengths of 1st and 2nd stages of labor are significantly longer for nulliparous woman:
 - Nulliparous: Mean length for 1st stage of labor is 14.4 hr and for 2nd stage of labor is 1 hr
 - Parous: Mean length of 1st stage of labor is 7.7 hr and for 2nd stage of labor is 0.2 hr
- Length of 2nd stage of labor is greatly influenced by “3 Ps”:
 - Passenger (infant size and presentation)
 - Passageway (size of bony pelvis and soft tissues)
 - Powers (uterine contractions)
- Problems with any of these 3 Ps can cause abnormal progression of labor:
 - Fetal malposition, uterine dysfunction, cephalopelvic disproportion
- False labor (Braxton Hicks contractions):
 - Irregular, nonsynchronous contractions of uterus several weeks to days

before onset of true labor, and do not cause cervical dilation

ETIOLOGY

- Premature labor occurs in 8–10% of pregnancies.
- 30–40% of premature labor is caused by uterine, cervical, or urinary tract infections
- Premature rupture of membranes is defined as rupture of amniotic/chorionic membranes at least 2 hr before onset of labor in patient before 37 wk gestation:
 - This occurs in only 3% of pregnancies but accounts for 30–40% of all premature births



DIAGNOSIS

SIGNS AND SYMPTOMS

- Symptoms of labor:
 - Intermittent low abdominal pain with or without low back pain
 - Occurring regularly at least every 5 min
 - Lasting 30–60 sec
- Preterm labor is of sufficient frequency and intensity to bring about changes in dilation or effacement of cervix before 37 wk
- Labor is not associated with vaginal bleeding:
 - Patients with 3rd-trimester abdominal pain or vaginal bleeding should raise suspicion of placenta previa or placental abruption
- Sudden release of clear fluid from vagina or feeling of constant perineal wetness can represent rupture of membranes:
 - This is not always associated with labor but often leads to onset of labor

History

- Gestational age
- Prenatal care
- Previous pregnancies:
 - Complications
 - C-section
- Recent infections

Physical-Exam

- Assess fundal height:
 - Centimeters from pubic bone to top of uterus
 - Correlates with number of weeks after 2nd trimester
 - Can help determine gestational age if unknown
- Sterile pelvic exam to assess cervical dilation and effacement

Do not perform a pelvic exam if vaginal bleeding is present.

ESSENTIAL WORKUP

- Patients presenting in possible labor should have *immediate sterile pelvic exam* to assess dilation, effacement of cervix, and possibility of imminent delivery.
- Bimanual pelvic exam should NOT be done in 3rd-trimester patient with vaginal bleeding until US can be done to assess for placenta previa or placental abruption.
- Patients with suspected rupture of membranes should have sterile speculum exam with visual exam of cervix and collection of fluid from vaginal area
- Suggestive of rupture of membranes:
 - _ Presence of *ferning* when fluid is allowed to dry on a slide
 - _ Presence of *pooling* of fluid in vagina
 - _ *Change of color of litmus paper* from yellow to blue
- Patients with preterm labor and cervical changes should have urinalysis with culture and cervical cultures
- Fetal monitoring should be initiated

DIAGNOSIS TESTS & INTERPRETATION

Lab

- If patient is in labor, CBC, type, and screen should be sent.
- Urinalysis for proteinuria
- In patients with no prenatal care, obtain Rh factor and antibody screen.
- Cervical cultures and urine culture in patients with preterm labor

Imaging

- Not generally needed
- 3rd-trimester patients with abdominal pain and vaginal bleeding should have emergent US to evaluate for placenta previa or abruption.

DIFFERENTIAL DIAGNOSIS

- Braxton Hicks contractions (false labor) are irregular uterine contractions without associated cervical changes:
 - _ Contractions can be every 10–20 min
- Round uterine ligament pain, musculoskeletal back pain
- Other common causes of abdominal pain, such as appendicitis, ovarian cyst, diverticulitis, nephrolithiasis, UTI



TREATMENT

PRE HOSPITAL

- Emergency medical services personnel should place patients in labor on oxygen

- and in left lateral recumbent position to maximize delivery of oxygen to uterus
- Maternal transport of high-risk obstetric patients before delivery results in improved outcomes instead of transfer of neonate after delivery
- Air transport of high-risk obstetric patients has been shown to be beneficial and cost effective
- Patients in labor who are transported by aircraft should have high-flow oxygen available in the event of cabin decompression at high altitudes

INITIAL STABILIZATION/THERAPY

If delivery is imminent (presenting part visible), prepare for immediate vaginal delivery in ED (see “Delivery, uncomplicated”)

ED TREATMENT/PROCEDURES

- Unless delivery is imminent, patient should be sent directly to the labor and delivery (L&D) unit
- If transport to L&D will be delayed, or if transfer to another facility is necessary, these steps should be taken:
 - Consider IV antibiotics for unknown group B Streptococcus status
 - IV hydration with 1 L NS or 5% dextrose in lactated Ringer over 30–60 min
 - Maternal monitoring
 - Fetal monitoring
 - If labor needs to be arrested (premature fetus), begin a tocolytic such as β -agonist terbutaline or magnesium sulfate:
 - Magnesium toxicity is suggested by loss of deep tendon reflexes
 - High doses of magnesium can cause cardiac dysrhythmias and respiratory depression.

MEDICATION

- Magnesium sulfate: 4–6 g IV over 30 min, followed by 2–6 g/hr
- Terbutaline: 0.25 mg SC; may repeat same dose in 30 min

ALERT

Consider antibiotic prophylaxis for patients with history of cardiac lesions.



FOLLOW-UP

DISPOSITION

Admission Criteria

- All patients in labor who are not at risk for imminent delivery should be admitted to L&D
- Preterm patients in labor demand immediate obstetric consultation and should be admitted to L&D for further treatment

Discharge Criteria

Patients with false labor may be discharged only after obstetric consultation, confirmation of fetal well-being, and close follow-up is arranged:

- False labor may progress to true labor

PEARLS AND PITFALLS

- If vaginal bleeding is present, must rule out placental abruption or previa
- Do not perform a digital exam if bleeding is present
- Pelvic exam must be sterile in a patient in labor
- False labor may progress to true labor

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See Also (Topic, Algorithm, Electronic Media Element)

- Delivery, Uncomplicated
- Placental Abruption
- Placenta Previa



CODES

ICD9

- V22.0 Supervision of normal first pregnancy
- V22.1 Supervision of other normal pregnancy
- V23.9 Supervision of unspecified high-risk pregnancy

ICD10

- Z34.00 Encntr for suprvsn of normal first pregnancy, unsp trimester
- Z34.80 Encounter for suprvsn of normal pregnancy, unsp trimester
- Z34.90 Encntr for suprvsn of normal pregnancy, unsp, unsp trimester

LABYRINTHITIS

Amira M. Bass • Charles V. Pollack, Jr.



BASICS

DESCRIPTION

- Inflammatory disorder of the inner ear
- Inflammation decreases afferent firing from the labyrinth
 - CNS interprets the decreased signal as head rotation away from the diseased labyrinth
 - The imbalance in firing from the labyrinth results in spontaneous nystagmus with fast phase away from the pathologic side
- Form of unilateral vestibular dysfunction that typically cause balance disorders and vertigo, and may be associated with hearing loss and tinnitus
- Peak onset 30–60 yr old
- Associated with upper respiratory tract infection in 50% of patients
- Symptoms predominantly with head movement but can persist at rest
- Recovery phase gradual over weeks to months

ETIOLOGY

- 3 most common causes of peripheral vertigo include, benign paroxysmal positional vertigo (BPPV), Ménière disease, and labyrinthitis
- Labyrinthitis:
 - Serous: Viral or bacterial
 - Suppurative: Bacterial
 - Autoimmune: Wegener or polyarteritis nodosa
 - Vascular ischemia
 - Head injury or ear trauma
 - Medications:
 - Aminoglycosides, loop diuretics, antiepileptics (phenytoin)
 - Allergies
 - Chronic
- BPPV:
 - Dislodgement of otoconia debris:
 - Idiopathic: 49%
 - Post-traumatic: 18%
 - Sequela of labyrinthitis: 15%
 - Sequela of ischemic insult

Pediatric Considerations

- Suppurative and serous labyrinthitis:

- Usually secondary to acute otitis media, mastoiditis, or meningitis
- BPPV:
 - Onset 1–5 yr of age
 - Symptoms: Abrupt onset of crying, nystagmus, diaphoresis, emesis, ataxia
 - Recurrences for up to 3 yr
 - Migraine–BPPV complex is the most common etiology of pediatric vertigo
- Ménière disease:
 - Rare before 10 yr of age

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Vertigo:
 - Peripheral vertigo
 - Sudden onset
 - Associated with movement, head position
 - Sensation of room spinning or off balance
- Nausea and vomiting
- Episodes of hearing impairment:
 - Unilateral or bilateral
 - Mild or profound
 - Tinnitus (consider Ménière disease)
- Otorrhea (consider otitis media, tympanic membrane [TM] perforation)
- Otalgia (consider otitis media, mastoiditis, cholesteatoma)
- Associated with recent infections or sick contacts
- Predisposing factors include ear surgery, diabetes mellitus, stroke, migraine, and trauma
- Head/cervical spine trauma is a direct causal agent, as it dislodges inner ear particles
- Associated with family history of hearing loss or ear diseases

Physical-Exam

- Complete head and neck exam
- Inspect external ear (erythema, swelling, evidence of surgery), ear canal (otorrhea, vesicles), and TM and middle ear (perforation, cholesteatoma, middle ear effusion, or otitis media)
- Mastoid tenderness (mastoiditis)
- Ocular exam, including range of movements, pupillary response, and fundoscopy, to assess for papilledema
- Nystagmus:

- Augmented by head movement or rapid head shaking
- Positional
- Horizontal, frequently with rotational component
- Direction is constant
- Attenuates with fixation
- Fatigable
- Complete neurologic and cardiac exams:
 - Assess for other causes of symptoms
 - Cranial nerves, Romberg test, tandem gait, cerebellar function
 - Orthostatic vitals, carotid and vertebral bruits
- May be associated with facial weakness or asymmetry (consider stroke or Ramsay Hunt syndrome), neck pain or stiffness (consider meningitis), and visual changes (consider central cause of vertigo)
- Caloric testing:
 - Irrigate external ear canal with cold water for 20 sec (1st inspect to confirm absence of TM perforation).
 - Normal response causes horizontal nystagmus with the fast phase away from the irrigated ear
 - Labyrinthitis produces partial or complete loss of response
- Dix–Hallpike maneuver:
 - Tests for evidence of BPPV

ESSENTIAL WORKUP

- Careful neurologic exam to exclude central cause of vertigo
- Exclude underlying infections:
 - Acute otitis media, meningitis, mastoiditis, Ramsay Hunt syndrome (herpetic lesions on the TM)
- Orthostatics
- Auditory evaluation

DIAGNOSIS TESTS & INTERPRETATION

- Indicated only if evaluating patients for central vertigo or more unusual etiologies of peripheral vertigo
- Chemistry panel and electrolytes if significant or refractory nausea and vomiting
- Lumbar puncture if clinical suspicion for meningitis or subarachnoid hemorrhage

Lab

- Finger-stick glucose
- Syphilis screening
- Rheumatoid factor
- Chemistry panel and electrolytes

Imaging

- **Indications:**
 - Findings suggestive of central vertigo:
 - Acute or gradual onset
 - Not positional but may be exacerbated by head movements
 - Pure direction—vertical, horizontal, or torsional
 - Direction may change
 - Nonfatigable
 - High cardiovascular risk factors
 - Focal neurologic findings
- **Head CT:**
 - Fine cuts through the cerebellum
- **MRI and MRA:**
 - Evaluate the posterior fossa, the 8th cranial nerve, and the vertebrobasilar circulation
 - Imaging study of choice in patients suspected of central vertigo

ALERT

Consider brain imaging in patients >45 yr, children, and patients with cardiovascular risk factors.

Diagnostic Procedures/Surgery

- **Electronystagmography:** May help in diagnosing difficult cases
- **Infrared nystagmography:** Torsional eye movement can be demonstrated directly

DIFFERENTIAL DIAGNOSIS

- **Peripheral vertigo:**
 - Otitis media
 - Vestibular neuritis
 - Acoustic neuroma
 - Autoimmune inner ear disease
 - BPPV
 - Cholesteatoma
 - Ménière disease (associated tinnitus, “fullness,” or hearing loss)
 - Ootosyphilis
 - Ototoxic drugs (loop diuretics, aminoglycosides, streptomycin, salicylates, ethanol)
 - Herpes zoster (Ramsey Hunt syndrome)
 - Perforated TM
 - Perilymphatic fistula (symptoms accentuated with Valsalva)
 - Post-traumatic vestibular concussion
 - Suppurative labyrinthitis (toxic appearance)
 - Temporal bone fracture
- **Central vertigo—**often presents with symptoms indistinguishable from peripheral

vertigo because the labyrinth has a monosynaptic connection to the brainstem:

- Brainstem ischemia
 - Cerebellar hemorrhage
 - Inferior cerebellar ischemia
 - CNS lesions (tumors)
 - Chiari malformation
 - Multiple sclerosis (paresthesia, optic neuritis)
 - Partial seizures of temporal lobe
 - Vestibular–masseter syndrome (associated masseter muscle weakness)
 - Vestibular migraine (30% have vertigo independent of headaches)
 - Wallenberg syndrome (associated Horner's syndrome, crossed sensory signs)
- Cardiac arrhythmia (presyncopal symptoms)
 - Hypoglycemia (gradual onset, not positional)
 - Hypotension (exacerbated with standing)
 - Cervicogenic disease (onset with rotational neck movement)
 - Hypothyroidism
 - Alcohol or drug induced



TREATMENT

PRE HOSPITAL

- Cardiac monitor for arrhythmia
- Finger-stick glucose to exclude hypoglycemia
- Acute stroke assessment
- Antiemetics for nausea and vomiting
- IV fluids for dehydration
- Fall precautions

INITIAL STABILIZATION/THERAPY

- Bed rest and hydration
- Fall precautions

ED TREATMENT/PROCEDURES

- Medications are minimally beneficial for BPPV
- Avoid chronic use (up to 48 hr) to encourage development of vestibular compensation
- Medications for symptomatic relief:
 - Vestibular suppressants: Diazepam, meclizine, scopolamine
 - Antiemetics: Ondansetron, prochlorperazine, promethazine
 - Corticosteroids: Poor evidence for efficacy
- Debris repositioning is primary therapy for BPPV. Effective relief in 50–80% of patients:

- Epley maneuver
- Vestibular enhancement exercises
- Surgery for failed medical and physical therapy:
 - Posterior canal plugging to occlude canal
 - Nerve section

MEDICATION

- Diazepam (benzodiazepine): 2–10 mg IV; 5–10 mg (0.1–0.3 mg/kg/24 h) PO q6–12h
- Dimenhydrinate: 5 mg/kg/24 h PO, IM, IV, or PR
- Meclizine (antihistamine): 25 mg (50 mg/24 h for patient > 12 yr) PO q6h
- Lorazepam: 0.5–2 mg IV, IM, or PO q6h (peds: 0.05 mg/kg IV/PO q4–8h)
- Ondansetron: 4–8 mg IV, IM, or PO q8h (peds: 1 mo–12 yr and < 40 kg: 0.1 mg/kg IV; > 12 yr and > 40 kg: 4 mg IV)
- Prochlorperazine: 5–10 mg (peds: 0.3 mg/kg/24 h IM or PO for patient > 2 yr old) IV, IM, or PO q6–8h
- Promethazine: 12.5–25 mg (peds: 1.5–2 mg/kg/24 h) IV or PO q4–6h
- Scopolamine (anticholinergic, not approved in pediatrics): 0.4 mg PO q4–6h; 1.5-mg transdermal patch q3d

Pediatric Considerations

Bacterial labyrinthitis:

- Antibiotics IV
- Surgical debridement

Pregnancy Considerations

- Class D medication: Diazepam, lorazepam
- Class C medication: Prochlorperazine
- Class B medication: Famciclovir
- Class B medication: Corticosteroids

First Line

- Meclizine
- Ondansetron for nausea/vomiting

Second Line

- Diazepam or lorazepam
- Prochlorperazine or promethazine (beware dystonic or dysphoric reactions)



FOLLOW-UP

DISPOSITION

Admission Criteria

- Symptoms concerning for an acute stroke or central etiology of vertigo
- Intractable nausea and vomiting
- Severe dehydration
- Unsteady gait

Discharge Criteria

- Tolerate oral fluids
- Steady gait
- Normal neurologic exam
- Avoid driving, heights, and operating dangerous equipment
- Fall precautions
- Arrange neurology or otolaryngology follow-up

Issues for Referral

- Recurrent symptoms
- Concern for cholesteatoma
- Possible severe underlying conditions such as vertebrobasilar ischemia or brainstem tumor will need consultation from neurologist or neurosurgeon

FOLLOW-UP RECOMMENDATIONS

- Vestibular rehabilitation for patients with persistent vestibular symptoms and chronic vertigo due to peripheral vestibular etiology
- Auditory brainstem response test is indicated in younger children.
- Surgical therapy in the form of labyrinthectomy/posterior canal occlusion/vestibular nerve section, etc., can be considered in cases of refractory vertigo and unsuccessful canalith repositioning procedure.

PEARLS AND PITFALLS

- Counsel patients regarding occupation, fall risk, and driving
- Failure to diagnose life-threatening conditions like meningitis, cerebrovascular ischemia, or brain tumors
- Take caution while performing physical maneuvers for BPPV, as violent hyperextension at cervical spine can cause vertebral artery dissection

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See Also (Topic, Algorithm, Electronic Media Element)

- Dizziness
- Vertigo
- Ménière Disease
- Otitis Media
- Mastoiditis



CODES

ICD9

- 386.30 Labyrinthitis, unspecified
- 386.31 Serous labyrinthitis
- 386.35 Viral labyrinthitis

ICD10

- H83.01 Labyrinthitis, right ear
- H83.02 Labyrinthitis, left ear
- H83.09 Labyrinthitis, unspecified ear

LACERATION MANAGEMENT

Gordon S. Chew

BASICS

DESCRIPTION

- A laceration is a disruption in skin integrity most often resulting from trauma.
- May be single or multiple layered

ETIOLOGY

Multiple causes

DIAGNOSIS

SIGNS AND SYMPTOMS

Lacerations may be accompanied by:

- Bleeding
- Tissue foreign bodies
- Hematoma
- Pain or numbness
- Loss of motor function
- Diminished pulses, delayed capillary refill

History

- Mechanism and circumstances of injury
- Time of injury
- History of foreign body (glass, splinter, teeth)
- Tetanus immunization
- Comorbid condition or medications that may impede wound healing

Physical-Exam

- Evaluate nerve and motor function.
- Document associated neurovascular injury.
- Assess presence of devitalized tissue, debris from foreign materials, bone or joint violation, tendon injury:
 - Avoid digital exploration if the object is believed to be sharp.

ESSENTIAL WORKUP

- Consider repair in OR if unable to be performed safely within the ED, especially for children requiring deep sedation.
- Consider surgical consultation for complex lacerations, especially involving eyes

and face.

Pediatric Considerations

Assess for possible nonaccidental trauma.

DIAGNOSIS TESTS & INTERPRETATION

Imaging

- Evaluation for possible foreign bodies
- Plain radiography:
 - Soft-tissue views may aid in visualization.
 - Objects with the same density as soft tissue may not be seen (wood, plants).
- US
- CT scan
- MRI with metal precautions

DIFFERENTIAL DIAGNOSIS

- Skin avulsion
- Contusion
- Abrasion



TREATMENT

PRE HOSPITAL

- Obtain hemostasis, or control of bleeding with direct pressure.
- Straighten any flaps of skin whose blood supply may be strangulated.
- Apply splint if needed.
- Universal precautions

INITIAL STABILIZATION/THERAPY

- Airway, breathing, and circulation management (ABCs)
- Control hemostasis.
- Remove rings or jewelry if needed. Swollen fingers with rings can become ischemic.

ED TREATMENT/PROCEDURES

- Time of onset:
 - Lacerations may be closed primarily ≤ 8 hr old in areas of poorer circulation.
 - Lacerations may be closed ≤ 12 hr old in areas of normal circulation.
 - On face, lacerations may be closed ≤ 24 hr if clean and well irrigated.
 - If not closed, wound may heal by secondary intention or by delayed primary closure (DPC) in 3–5 days.

- Analgesia and conscious sedation:
 - Adequate analgesia is crucial for good wound management.
 - Conscious sedation may be required (see “Conscious Sedation”).
- Local anesthetics:
 - Topical:
 - TAC (tetracaine, adrenaline, cocaine)
 - EMLA (eutectic mixture, lidocaine, prilocaine)
 - LET (lidocaine, epinephrine, tetracaine)
 - Local/regional:
 - Lidocaine, bupivacaine
 - Epinephrine will cause vasoconstriction and improve duration of action of anesthetic.
 - Avoid epinephrine in the penis, digits, toes, ears, eyelids, tip of nose, skin flaps (necrosis), and severely contaminated wounds (impairs defense).
 - For patient comfort, inject slowly with small-gauge needle; buffer every 9 mL of 1% lidocaine with 1 mL 8.4% sodium bicarbonate.
 - Consider a 1% diphenhydramine solution in the lidocaine-allergic patient.
- Exploration and removal of foreign body:
 - Indications for removal of a foreign body include:
 - Potential or actual injury to tendons, nerves, vasculature
 - Toxic substance or reactive agent
 - Continued pain
- Irrigation and debridement:
 - Surrounding intact skin may be cleaned with an antiseptic solution (povidone-iodine, chlorhexidine):
 - Do not use antiseptic solution within the wound itself because it may impair healing.
 - Scrub with a fine-pore sponge only if significant contamination or particulate matter.
 - Irrigation with ≥ 200 mL of normal saline (NS):
 - Optimal pressure (5–8 psi) generated with 30-mL syringe through 18–20G needle
 - Try to avoid shaving hair. Clip if necessary:
 - Increased skin infection rate after shaving
 - Never shave or clip eyebrow as it may not grow back with a normal appearance
 - Debride devitalized and contaminated tissue.
- Wound repair:
 - Universal precautions
 - Wounds that cannot be cleaned adequately should heal by secondary intention or DPC.

- Reapproximate all anatomic borders carefully (e.g., skin–vermilion border of lip).
- Consider tissue adhesive for wounds with clean borders, low tension.
- Single-layered closure:
 - Simple interrupted sutures:
 - Avoid in lacerations under tension.
 - Horizontal mattress sutures (running or interrupted):
 - Edematous finger and hand wounds
 - Ideal in skin flaps where edges at risk for necrosis
 - Vertical mattress:
 - For wounds under greater tension
 - 1 stitch that provides both deep and skin closure
 - Half-buried horizontal mattress sutures:
 - Ideal for closing the vertex of a v- or y-shaped laceration where ischemia is a concern
- Multiple-layered closure:
 - Closes deep tissue dead space
 - Lessens tension at the epidermal level, improves cosmetic result
 - Buried interrupted absorbable suture, simple or running nonabsorbable sutures for epidermis
- Dressing:
 - Dress wound with antibiotic ointment and nonadherent semiporous dressing.
 - Inform patient about scarring and risk for infection, use of sunscreen.
 - Apply splint if needed.
- Antimicrobial agents:
 - Uncomplicated lacerations do not need prophylactic antibiotics.
 - If antibiotics are used, initiate before wound manipulation or as early as possible.
 - Lacerations with high likelihood of infection:
 - Animal, human bites, especially to hand (see “Hand Infection”)
 - Contaminated with dirt, bodily fluids, feces
 - Tetanus immunization

MEDICATION

- See “Conscious Sedation.”
- Tetanus (Tdap/Td for adolescents–adults, DTap for peds): 0.5 mL IM
- Local anesthetics:
 - Topical, applied directly to wound with cotton, gauze:
 - EMLA (eutectic mixture, 5% lidocaine, and prilocaine): Apply for 60 min. Note: each g of EMLA contains 2.5 g of lidocaine, do not exceed 3 mg/kg lidocaine
 - TAC (0.5% tetracaine, 1:2,000 adrenaline, 11.8% cocaine): Apply for

- 20–30 min. Apply from 2–5 mL to wound
 - LET (4% lidocaine, 1:1,000 epinephrine, 0.5% tetracaine): Apply for 20–30 min. Apply 1–3 mL. Do not exceed 3 mg/kg lidocaine.
- Injected:
 - Bupivacaine (max.: 2 mg/kg; duration 3–10 hr)
 - Lidocaine (max.: 4.5 mg/kg; duration 1.5–3.5 hr)
- Suture materials:
 - Absorbable:
 - For use in mucous membranes and buried muscle/fascial layer closures
 - Natural: Dissolve < 1 wk, poor tensile strength, local inflammation: Plain gut, chromic gut, fast-absorbing gut (for certain facial lacerations where cosmesis is important)
 - Synthetic braided: Tensile strength diminishing over 1 mo, mild inflammation: Polyglycolic acid (Dexon), polyglactin 910 (Vicryl)
 - Synthetic monofilament: Tensile strength 70% at 1 mo: Polydioxanone (PDS), polyglyconate (Maxon)
 - Nonabsorbable:
 - Greatest tensile strength
 - Monofilament: Nylon (Ethilon, Dermalon), polypropylene (Prolene), polybutester (Novafil) can stretch with wound edema, polyethylene (stainless steel)
 - Multifilament: Cotton, Dacron, silk (local inflammation)
 - Needle types:
 - Cutting (cuticular and plastic) types are most often used in outpatient wound repair.
- Staples:
 - For linear lacerations of scalp, torso, extremities
 - Avoid in hands, face, and areas requiring CT or MRI.
- Adhesive tapes (Steri-Strips):
 - For lacerations that are clean, small, and under minimal tension
 - Avoid in wounds that have potential to become very swollen.
 - Pretreat wound edges with tincture of benzoin to improve adhesion.
- Tissue adhesives:
 - Good cosmetic results have been achieved in simple lacerations with low skin tension.
 - An alternative to sutures/staples, especially in children



FOLLOW-UP

DISPOSITION

Admission Criteria

- Few lacerations by themselves necessitate admission unless they require significant debridement or ongoing IV antibiotics, or are complicated by extensive wound care issues or comorbid processes (head injury, abdominal trauma).
- It is unsafe for a child to return home when nonaccidental trauma is suspected.

Discharge Criteria

- Wounds at risk for infection or poor healing require a wound check within 48 hr.
- Time of suture removal dependent on location and peripheral perfusion:
 - _ Scalp: 7–10 days
 - _ Face: 3–5 days
 - _ Oral: 7 days
 - _ Neck: 4–6 days
 - _ Abdomen, back, chest, hands, feet: 7–10 days
 - _ Upper extremity: 7–10 days
 - _ Lower extremity: 10–14 days
 - _ Overlying joints: 10–14 days

Issues for Referral

- Lacerations of the eye where tear duct injury is suspected require immediate referral.
- Complicated lacerations (tendon involvement) may require further repair in the outpatient surgical office.
- Be sure to discuss temporary skin closure and splinting with your surgical consultant.
- Specific follow-up should be arranged prior to patient discharge.

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See Also (Topic, Algorithm, Electronic Media Element)

Hand Infection



CODES

ICD9

- 879.8 Open wound(s) (multiple) of unspecified site(s), without mention of complication
- 882.0 Open wound of hand except finger(s) alone, without mention of complication
- 883.0 Open wound of finger(s), without mention of complication

ICD10

- S61.219A Laceration w/o fb of unsp finger w/o damage to nail, init
- S61.419A Laceration without foreign body of unsp hand, init encntr
- T14.8 Other injury of unspecified body region

LARYNGITIS

Yi-Mei Chng

BASICS

DESCRIPTION

- Inflammation of the mucosa of the larynx
- The most common cause is viral upper respiratory infection
- Peaks parallel epidemics of individual viruses
- Most common during late fall, winter, early spring

ETIOLOGY

- Viral upper respiratory infections most common in acute laryngitis:
 - Influenza A and B
 - Parainfluenza types 1 and 2
 - Adenovirus
 - Coronavirus
 - Coxsackievirus
 - Respiratory syncytial virus
 - Measles
 - Rhinovirus
- Bacterial infections much less common:
 - β -Hemolytic streptococcus
 - *Streptococcus pneumoniae*
 - *Haemophilus influenzae* (HiB)
 - *Moraxella catarrhalis*
 - *Bordetella pertussis*
 - Diphtheria
 - Tuberculosis
 - Syphilis
 - Leprosy
- Laryngopharyngeal reflux (LPR) from gastroesophageal reflux disease (GERD) (especially in adults)
- Fungal infections (often associated with inhaled steroid use or immunocompromise)
- Allergic
- Voice abuse or misuse
- Inhalation or ingestion of caustic substances or other irritants
- Autoimmune (rheumatoid arthritis, relapsing polychondritis, Wegener granulomatosis, or sarcoidosis)
- Trauma

- Idiopathic

Pediatric Considerations

- Acute spasmodic laryngitis (spasmodic croup)
- More likely to be infectious.
 - Consider HiB, diphtheria, etc., if not immunized
- Consider foreign body



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Hoarseness
- Abnormal-sounding voice
- Throat swelling
- Throat tickling
- Feeling of throat rawness
- Constant urge to clear the throat
- Cough
- Fever
- Malaise
- Dysphagia

Physical-Exam

- Regional lymphadenopathy
- Stridor in infants
- Hoarse voice
- Pharyngeal erythema, exudates, and/or edema
- Asymmetrical breath sounds in case of foreign body

ESSENTIAL WORKUP

- Acute laryngitis:
 - In most cases, the history and inspection of the throat suffice to distinguish between infectious and noninfectious laryngitis:
 - Infectious laryngitis usually lasts about 7–10 days.
 - Have increased suspicion for epiglottitis in persons who have not had HiB vaccine
- Chronic laryngitis (> 3 wk):
 - The workup should be directed toward chronic infections, GERD, neurologic disorders, and tumors
 - Visualization of the larynx should be performed but may not need to be

done in the ED

- The patient should be referred to an ear–nose–throat specialist for further workup

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Blood tests are not generally indicated:
 - An elevated WBC count is not a reliable way to distinguish between bacterial and viral illness
- Throat culture:
 - Indicated when exam suggests a bacterial infection such as significant exudate in the throat or on the vocal folds

Imaging

Soft-tissue neck films:

- Rarely indicated because fiberoptic laryngoscopy provides a more comprehensive assessment
- Mostly used if epiglottitis or foreign body suspected, though high-risk patients should not be sent to radiology

Diagnostic Procedures/Surgery

Fiberoptic laryngoscopy:

- Red, inflamed vocal cords, with rounded edges
- Occasionally hemorrhage or exudates
- Endolaryngeal pus is more common in bacterial laryngitis than viral
- Demonstration of laryngeal pseudomembrane to distinguish diphtheria from other infectious forms of laryngitis

DIFFERENTIAL DIAGNOSIS

- Asthma
- Epiglottitis
- Esophageal reflux
- Vocal nodules
- Laryngeal or thyroid malignancy
- Croup/laryngotracheobronchitis
- Foreign-body inhalation or other trauma



TREATMENT

PRE HOSPITAL

Supportive care and ambulance transport are not generally indicated

ALERT

- Stridor can mean obstruction of the laryngeal or tracheal parts of the airway, particularly in children
- An otolaryngologist should evaluate laryngitis after trauma to the neck
- Beware of esophageal injuries in laryngitis associated with caustic ingestions
- If there are signs of respiratory distress, epiglottitis should be suspected:
 - Transport sitting up
 - Provide supplemental oxygen
 - Intubation may be difficult or impossible and should only be attempted in patients in extremis

INITIAL STABILIZATION/THERAPY

Stabilization is only required if the patient shows signs of respiratory distress:

- The patient should be managed for epiglottitis
- Supplemental oxygen via a nonrebreather mask
- Orotracheal intubation when time permits in the OR
- The neck should be prepped and the equipment ready for a surgical airway

ED TREATMENT/PROCEDURES

- Antibiotics are not 1st-line therapy in adults with acute laryngitis:
 - In a systematic review of randomized controlled trials investigating the use of antibiotics vs. placebo, antibiotics offered no objective improvement in symptoms over placebo
- Vocal rest (avoid whispering, as it promotes hyperfunctioning of the larynx):
 - If patient must speak, use a soft sighing voice
- Humidified air
- Increase fluid intake
- Analgesics
- Smoking cessation
- Symptoms usually resolve in 7–10 days, if viral cause
- Use of inhaled steroids for laryngitis is controversial and not part of current best practices.

MEDICATION

Depends on cause of laryngitis.

- Mucolytics like guaifenesin if related to upper respiratory infection or allergy
- Acetaminophen or NSAIDs for symptomatic relief if associated with viral syndrome
- Proton pump inhibitors for GERD-related laryngitis:
 - Esomeprazole magnesium: 20–40 mg (peds: 10 mg for patients 1–11 yr) PO daily
 - Omeprazole: 20 mg PO BID
- Diflucan for candidal laryngitis
- If caused by croup: Dexamethasone (0.6 mg/kg) PO or IM × 1

- Antihistamines can dry out the vocal cords, make it harder to clear secretions and exudate
- Cochrane Review found no benefit in using antibiotics to treat acute laryngitis
 - Antibiotics may be considered in high-risk patients or in cases where a positive Gram stain and culture has been obtained



FOLLOW-UP

DISPOSITION

Admission Criteria

- Tuberculous laryngitis:
 - Highly contagious requiring isolation
- Signs of epiglottitis, respiratory distress, neck trauma, or anaphylaxis
- Respiratory compromise

Discharge Criteria

Most patients with uncomplicated laryngitis can be discharged if they have no difficulty breathing and are able to keep adequately hydrated.

Issues for Referral

Refer patients with chronic laryngitis to otolaryngologist. Patients with >3 wk of laryngitis without obvious benign cause should be evaluated with laryngoscopy to rule out more serious conditions such as carcinoma.

FOLLOW-UP RECOMMENDATIONS

- With otolaryngology if not improved in 2–3 wk
- With primary care or gastroenterology if symptoms of GERD

PEARLS AND PITFALLS

- Most acute laryngitis is of viral origin
 - Antibiotics likely with no benefit
- Consider life-threatening causes of altered phonation such as epiglottitis
- Laryngitis not associated with upper respiratory infection may be related to GERD
- Patients with chronic or nonresolving laryngitis should follow up with otolaryngologist

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Croup
- Epiglottitis



CODES

ICD9

- 464.00 Acute laryngitis without mention of obstruction
- 464.01 Acute laryngitis with obstruction
- 476.0 Chronic laryngitis

ICD10

- J04.0 Acute laryngitis
- J05.0 Acute obstructive laryngitis [croup]
- J37.0 Chronic laryngitis

LARYNX FRACTURE

David Della-Giustina • Katja Goldflam



BASICS

DESCRIPTION

- Direct transfer of severe forces to the larynx
- Simple mucosal tears to fractured and comminuted cartilage:
 - Epiglottis, thyroid, arytenoid, cricoid, corniculate, and cuneiform cartilages

ETIOLOGY

- Rare: 1/5,000–1/42,000 ED visits
- <1% of all blunt trauma
- Directly related mortality is 2–15%
- Blunt or penetrating trauma to the anterior neck associated with motor vehicle or motorcycle crash, assault, or recreational activities.
- Typical mechanism is hyperextension of neck with a direct blow to the exposed anterior neck.
- “Clothesline” injury is a classic mechanism (victim struck in neck by cord, wire, or branch hung across path of travel).
- Iatrogenic injuries from intubation are becoming more common with an aging population.

Pediatric Considerations

Bicycle handlebars:

- Extended neck hits the bar, compressing structures between the bar and vertebral column.



DIAGNOSIS

SIGNS AND SYMPTOMS

- May be delayed for hours
- Blunt trauma recognition is most challenging
- Blood, cervical collar, or polytrauma may distract from subtle findings
- Neck tenderness
- Bruising or abrasions over the anterior neck
- Hoarseness or voice changes
- Hemoptysis
- Dysphonia
- Stridor
- SC or mediastinal emphysema

- Dyspnea
- Pneumothorax
- Loss of normal cartilaginous landmarks of neck
- Difficulty with mechanical ventilation

ESSENTIAL WORKUP

- Endoscopic evaluation should take precedence over radiography, as mucosal edema may contribute to airway compromise more than skeletal injury.
- Cervical spine imaging:
 - Plain radiographs are not very helpful and should not supplant cervical CT scan
- CXR:
 - Identify pneumothorax, SC emphysema, and pneumomediastinum
- CT scan (with IV contrast) of cervical spine with fine cuts of larynx:
 - Contrast may identify vascular injuries
 - Recommended unless the patient is going directly to surgery
 - Useful even in cases of apparently less severe symptoms and minor abnormalities on indirect laryngoscopy
- Pulse oximetry

ALERT

MRI has not gained acceptance:

- Length of time
- Physical demands on injured patient
- Less helpful for skeletal structures

DIAGNOSIS TESTS & INTERPRETATION

Lab

Arterial blood gas potentially useful if the patient is having respiratory difficulty:

- Identifies hypoxia, hypercarbia

Diagnostic Procedures/Surgery

- Fiberoptic laryngoscopy:
 - Visualization of injuries involving the airway, vocal cords, ideally with a nasopharyngoscope
- Angiography:
 - Penetrating injuries
 - Only when concern exists for possible vascular injuries
- CT angiogram offers advantages to conventional angiography:
 - Readily accessible and less invasive
 - Can be rapidly performed
 - Few complications

- Provides useful information on cervical soft tissues, aerodigestive tract, spinal canal, and spinal cord
- Fiberoptic bronchoscopy and esophagoscopy
- Surgery:
 - As indicated by severity of injury
 - Emergent surgical repair if necessary

DIFFERENTIAL DIAGNOSIS

Associated injuries:

- Intracranial injuries (13%)
- Open neck injuries (9%)
- Cervical spine injuries (8%)
- Esophageal injuries (3%)
- Carotid artery injury
- Phrenic nerve injury
- Hypoxic cerebral injury
- Airway edema
- Aspiration pneumonitis
- Air embolism

Pediatric Considerations

- The pediatric larynx is located higher in the neck and is more cartilaginous and mobile than in adults; thus, pediatric patients are more resistant to laryngeal fractures.
- Loosely attached submucosal tissue allows for greater soft-tissue trauma, massive edema, and hematoma formation:
 - With smaller airway diameter, airway compromise can occur rapidly.
- Symptoms can vary from neck tenderness or hoarseness to respiratory distress and stridor.
- CT imaging may not add much to the physical and fiberoptic exam of the child as fractures of the poorly mineralized larynx may not be visualized.



TREATMENT

PRE HOSPITAL

- Cautions:
 - Aggressive airway management may be necessary: Oxygen, suctioning
 - Cervical spine immobilization
 - Injury may be overlooked if patient is intubated pre-hospital for other injuries owing to loss of subjective complaints.
- Controversies:
 - Elective intubation is not advocated.

INITIAL STABILIZATION/THERAPY

Airway management is of primary concern:

- Severe injuries may require operative management.
- Early intubation to preclude progressive respiratory compromise.
- Formal tracheostomy under local anesthesia may be required rather than endotracheal intubation when more severe neck injury is present.
- *Avoid* repeated orotracheal intubation attempts:
 - Proceed to surgical airway.
- Cricothyrotomy for severe maxillofacial injuries and injuries cephalad to cricothyroid cartilage.
- *Avoid* cricothyrotomy if hematoma present over the cricothyroid membrane or there is evidence of cricotracheal disruption.
- Emergent tracheostomy may be the only option to secure an airway.

Pediatric Considerations

- Elective intubation is not recommended.
- Mandatory flexible fiberoptic laryngoscopy
- CT scan if management course is in doubt

ED TREATMENT/PROCEDURES

- Supplemental humidified oxygen
- Elevate head of bed to decrease cerebral and neck soft tissue edema
- Maintain NPO status
- Voice rest as much as possible
- Obtain IV access
- Consult otolaryngologist for surgical evaluation
- Positive end-expiratory pressure and volume-controlled ventilation for severe pulmonary injury associated with acute respiratory distress syndrome or aspiration pneumonitis

MEDICATION

- For laryngeal injury with SC emphysema:
 - Assume that the mucosa of the upper airway has communicated with the deep tissue of the neck:
 - Ampicillin/sulbactam: 1.5–3 g IV (peds: 50 mg/kg IV) q6h
 - Clindamycin: 600–900 mg IV q8h (peds: 25–40 mg/kg/24h IV)
 - Histamine-2 blockers to prevent irritation to mucosal injuries (e.g., ranitidine 150–300 mg IV; peds: 2–4 mg/kg/d div. q6h IV) or proton-pump inhibitors (e.g., pantoprazole 40 mg IV, no pediatric dosing)
- For laryngeal edema, steroids may be indicated:
 - Not routinely used, but may be used for massive edema.
 - Methylprednisolone 250 mg IV q4h (faster acting)
 - Dexamethasone 8–10 mg IV q8h (peds: 0.15–0.6 mg/kg/dose IV)

Pediatric Considerations

If stridor present, consider nebulized racemic epinephrine: 2.25% 0.25–0.5 mL in 2.5 mL NS.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients with true laryngeal injuries must be admitted to a monitored setting for observation and airway management; prepare for emergent surgical repair of laryngeal defect.
- Patients with suspected laryngeal injury or highly suspicious mechanism must be admitted to a monitored setting for observation and serial flexible fiberoptic laryngoscopic exams.

Pediatric Considerations

Mandatory admission is recommended in all patients for oximetry, oxygen, and serial fiberoptic laryngoscopic exams.

Discharge Criteria

Patients without evidence of serious laryngeal injury or airway edema or compromise after an appropriate period of observation in the ED (usually 6 hr):

- Patients can appear deceptively normal for several hours after injury; if there is any doubt, admit to a monitored setting.

ADDITIONAL READING

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CODES

ICD9

- 807.5 Closed fracture of larynx and trachea
- 807.6 Open fracture of larynx and trachea

ICD10

- S12.8XXA Fracture of other parts of neck, initial encounter
- S12.8XXD Fracture of other parts of neck, subsequent encounter
- S12.8XXS Fracture of other parts of neck, sequela

LEAD POISONING

Vinodinee L. Dissanayake

BASICS

DESCRIPTION

- Lead has multiple mechanisms of toxicity:
 - Binds sulfhydryl groups and affects multiple enzymatic processes
 - Resembles Ca^{2+} thereby interfering with Ca^{2+} -dependent processes, such as cell signaling
 - May have mutagenic potential and play a role in human carcinogenesis
- Distribution:
 - Up to 99% of lead is bound to erythrocytes after initial absorption.
 - Ultimately redistributed into bone:
 - 95% of total body lead in adults
 - 70% of total body lead in children
 - High lead levels in the serum compromise the blood–brain barrier and result in lead entry into the CNS and neurotoxicity.
- Often coexists with iron deficiency; this allows for increased lead absorption in the gut.
- Impairs heme synthesis, leading to elevated free erythrocyte protoporphyrin (FEP); these complex with zinc, resulting in elevated zinc protoporphyrin (ZPP).
- Levels correlate poorly with symptoms:
 - Associated with drops in intelligence quotient (IQ) and increase in violent behavior

ETIOLOGY

- Acute toxicity:
 - Most often due to inhalation of an environmental source or ingestion of substance containing lead
 - Pottery glaze
 - Certain folk remedies
 - Cosmetics
 - Jewelry
 - Weights
 - Home-distilled alcoholic beverages
 - Lead dust from ammunition and primer
- Chronic toxicity:
 - Occupational exposures (usually via inhalation):
 - Battery manufacturing/recycling
 - Bridge painting

- Construction workers
- De-leading
- Electronic waste recycling
- Firing range instructors
- Mining and smelting
- Pottery workers
- Welders
- Home exposures (pediatric poisoning):
 - Lead-based paint inhalation/ingestion from toys and walls
 - Contaminated water from old pipes
 - Lead dust from the clothing of a parent exposed at work
 - Imported foods
 - Folk medicines

DIAGNOSIS

SIGNS AND SYMPTOMS

- Neurologic:
 - Seizures (may be prolonged and refractory)
 - Encephalopathy
 - Learning disabilities
 - Psychiatric disturbances
 - Cerebral edema
 - Peripheral motor neuropathy (wrist drop), classic but rare finding in chronic toxicity
- GI:
 - Colicky abdominal pain (lead colic)
 - Ileus
 - Nausea/vomiting
 - Lead lines on gingival line (Burton lines) appear as bluish tint (indication of lifetime burden, not acute exposure).
 - Hepatitis/pancreatitis
- Cardiovascular:
 - HTN (generally secondary to renal failure)
 - Myocarditis and conduction defects
- Renal:
 - Chronic renal insufficiency with long-term exposure
- Hematologic:
 - Anemia (due to interference with globin chain synthesis)
 - Increases RBC fragility, so decreased RBC life span
- Musculoskeletal:
 - Lead lines from increased Ca^{2+} deposition at epiphyses (do not consist of

- lead itself)
- Decreased bone strength and growth

ESSENTIAL WORKUP

Blood lead level (BLL)

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Whole-BLL:
 - There is no normal BLL
 - In pediatric cases, educational interventions begin at BLL ≥ 10 $\mu\text{g}/\text{dL}$
 - In pediatric cases, chelation therapy is instituted at BLL ≥ 45 $\mu\text{g}/\text{dL}$
 - In adults, chelation therapy is usually considered at BLL ≥ 70 $\mu\text{g}/\text{dL}$
 - 100 $\mu\text{g}/\text{dL}$ may present with severe encephalopathy; cognitive effects increase with rising levels
 - Expect that BLL may rise after treatment is completed due to redistribution
- CBC:
 - For presence of anemia
 - RBC indices and iron studies
- Electrolytes, BUN, creatinine, glucose:
 - For renal insufficiency
- Transaminases, liver function tests prior to chelation administration
- FEP or ZPP

Imaging

- Plain abdominal radiographs to look for radiopaque foreign body
- Long-bone series to look for lead lines (specifically in children)
- Cranial CT and other studies as indicated by patient's condition

DIFFERENTIAL DIAGNOSIS

- Acute toxicity:
 - Acute appendicitis/colitis/gastroenteritis
 - Celiac disease
 - Cholera
 - Distributive shock
 - Encephalopathy
 - Toxic ingestions
 - *Amanita* mushroom poisoning
 - Cyclic antidepressants or other seizure-inducing toxins
 - Organophosphates
- Chronic toxicity:
 - Addison disease

- Guillain–Barré syndrome or other neuropathy
- Vitamin deficiency (B3, B6, or B12)
- Wernicke–Korsakoff syndrome



TREATMENT

PRE HOSPITAL

- Support airway/breathing and circulation
- Cardiac monitoring
- Seizure management

ALERT

- If possible to do so safely, bring containers in suspected overdose or poisoning.
- Decontaminate skin for obvious dermal exposures.

INITIAL STABILIZATION/THERAPY

- ABCs:
 - Cardiac monitor
 - Isotonic crystalloids as needed for hypotension; vasopressors for refractory hypotension
- Naloxone, thiamine, and dextrose (D50W) as indicated for altered mental status
- Cardiovascular:
 - Isotonic crystalloids to support BP
 - Vasopressors for refractory hypotension (rare)
- Neurologic:
 - Treat seizures with benzodiazepines.
 - Assist ventilation for respiratory failure due to neuromuscular weakness.
- Renal:
 - Hemodialysis for renal failure
- Alimentary:
 - Dextrose, enteral, or parenteral feeding may be beneficial

ED TREATMENT/PROCEDURES

- Decontamination:
 - If opacities are seen on upright abdominal film, institute whole-bowel irrigation at 1–2 L/hr of polyethylene glycol until abdominal films are clear
 - Activated charcoal is not effective.
- Evaluate need for chelation therapy:
 - BLL
 - Acuity of exposure
 - Clinical symptoms
 - Consultation with a medical toxicologist or poison center

Adult Considerations

- Most likely exposures are via inhalation and caused by occupational exposure or ethnic products
- Adults with encephalopathy or those with BLL: > 100 mg/dL may need chelation
 - Begin with dimercaprol (BAL) and continue for 5 days
 - Start edetate calcium disodium (CaNa₂ EDTA) after 2nd dose of BAL
- Asymptomatic patients with BLL of 70–100 µg/dL may be treated with an oral chelating agent, succimer (DMSA)
- Chelation is not indicated for asymptomatic adults with BLL < 70 µg/dL

Pediatric Considerations

- Currently, BLL ≥ 10 µg/dL require investigative and educational interventions:
 - Investigation into the cause of the exposure and repeat monitoring must occur
 - Parental education should be initiated
- BLL ≥ 45 µg/dL:
 - Chelation therapy is initiated
 - Asymptomatic children are treated with DMSA
 - Symptomatic children or those with BLL ≥ 70 µg/dL are treated with BAL and CaNa₂ EDTA
 - Consult with medical toxicologist/poison center when chelation therapy is considered

Pregnancy Considerations

- Much controversy about fetal lead toxicity
- Consult maternal–fetal medicine and medical toxicologist/poison center in pregnant patients with elevated BLL.

MEDICATION

- Chelating agents:
 - Dimercaprol (BAL), 3 mg/kg deep IM q4h for 3–5 days if mild to moderate symptoms; 4 mg/kg IM q4h for 5 days for severe symptoms (seizure, encephalopathy):
 - Caution: Contraindicated in patients with peanut allergies
 - Edetate calcium disodium (CaNa₂ EDTA), 50 mg/kg/d as continuous IV infusion (adults and peds) or 1 g/m²/d as continuous IV infusion
 - Treat for 5 days and start 4 hr after BAL
 - Succimer (DMSA):
 - Adults: 10 mg/kg PO q8h for 5 days, then q12h for 14 days
 - Peds: 350 mg/m² q8h for 5 days, then q12h for 14 days
- Dextrose 50%: 25 g (50 mL; peds: 0.5 g/kg D25W) IV for hypoglycemia

- Diazepam: 5–10 mg (peds: 0.1 mg/kg) IV for seizure control
- Lorazepam: 2–4 mg IV or IM
- Naloxone: 0.4–2 mg (peds: 0.1 mg/kg) IV
- Thiamine: 100 mg (peds: 1 mg/kg) IM or IV



FOLLOW-UP

DISPOSITION

Admission Criteria

- Symptomatic lead intoxication
- Children at high risk for re-exposure in their current environment
- Children with difficulty tolerating DMSA
- Pregnant patients with elevated lead levels—consult obstetrics and toxicology.

Discharge Criteria

- Asymptomatic patients not requiring IV chelation therapy
- Chronically exposed patients who do not require admission should be referred for outpatient evaluation
- Ensure home environment is safe for patient prior to discharge
- Ensure pediatric patients tolerate oral chelation therapy prior to discharge

FOLLOW-UP RECOMMENDATIONS

Follow up with medical toxicologist or primary care physician.

PEARLS AND PITFALLS

- Heel sticks may result in falsely elevated BLL; repeat positive blood tests for confirmation
- Secure social worker support to ensure safe home environment prior to discharge
- Inquire and test siblings or family members in a patient with lead toxicity
- Do not give BAL if patient has peanut allergy

ADDITIONAL READING

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A special thanks goes to Dr. Harry C. Karydes, who contributed to the previous edition.



CODES

ICD9

- 984.0 Toxic effect of inorganic lead compounds
- 984.1 Toxic effect of organic lead compounds
- 984.9 Toxic effect of unspecified lead compound

ICD10

- T56.0X1A Toxic effect of lead and its compounds, accidental, init
- T56.0X4A Toxic effect of lead and its compounds, undetermined, init

LEGG–CALVÉ–PERTHES DISEASE

Sarah V. Espinoza



BASICS

DESCRIPTION

- Idiopathic avascular necrosis of the femoral head in children
- Genetics:
 - Increased frequency with factor V Leiden and anticardiolipin antibodies

Pediatric Considerations

Exclusively a pediatric disease

ETIOLOGY

- Successive vascular occlusions causing characteristic findings
- Growing evidence implicating hypercoagulable states
- May be multifactorial
- Risk factors include tobacco smoke, wood smoke, low birth weight, birth length < 50 cm
- Progression through 4 stages of disease:
 - Initial stage: Dense femoral head causing intermittent synovitis
 - Fragmentation: Femoral head becomes soft and deforms causing loss of motion
 - Healing: New bone grows into femoral head
 - Residual: Healed femoral head with some deformity
- More common in boys: Male > female, 4:1
- More common among Caucasians
- Most commonly occurs between ages of 3 and 7:
 - Range 18 mo–18 yr
- Bilateral in 10–15% of cases
- Associated with short stature, deprived populations, delayed & disproportionate growth



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Frequently insidious onset
- Limp often presenting complaint
- Pain:

- Aching in hip, groin, anteromedial thigh, or anteromedial knee
- May be mild
- Aggravated by activity, relieved by rest
- Muscle spasm common complaint early in course of disease

Physical-Exam

- Tenderness over anterior aspect of hip joint
- Joint stiffness:
 - Limitation of internal rotation seen earliest
 - Limited abduction
 - Contractures of adductors
- Muscle atrophy and shortening of leg on affected side are late findings
- Otherwise well appearing and afebrile
- May be asymptomatic

ESSENTIAL WORKUP

- Radiographs of hip most important study for diagnosis in ED
- Consider and exclude septic arthritis (usually an acute febrile illness)

DIAGNOSIS TESTS & INTERPRETATION

Lab

- No specific lab studies diagnostic of Legg–Calvé–Perthes (LCP)
- CBC, C-reactive protein (CRP), or ESR, if septic arthritis a concern

Imaging

- Characteristic imaging findings combined with consistent history and physical exam establish diagnosis
- Plain radiographs, MRI, and nuclear scintigraphy (bone scan) are the main diagnostic modalities used
- Hip radiographs:
 - AP & Frog lateral view of affected hip
 - Image both hips to detect contralateral disease
 - Assess stage, extent, and severity
 - Can be normal during 1st 3–6 mo
 - Usually abnormal at time of presentation
 - 5 stages seen in sequence:
 - Cessation of growth at the capital femoral epiphysis; smaller femoral head epiphysis and widening of articular space on affected side
 - Subchondral fracture
 - Resorption of bone
 - Reossification of new bone
 - Healed stage

- CT:
 - Shows precise information on anatomic relationship between femoral head and acetabulum
 - May have role in operative planning, staging
- MRI:
 - Sensitive in the diagnosis of LCP and provides good anatomic images
 - Detects abnormalities earlier than plain radiographs
 - Used to assess the extent of femoral head infarction
 - Variety of findings depending on imaging protocol used
- Bone scan:
 - Precedes x-ray changes by an average of 3 mo
 - Evaluates patterns associated with revascularization and recanalization
- US:
 - Shows effusion in the hip but is not specific for LCP
 - Evaluate for thickening of the synovial membrane
 - Evaluate deformity and containment of femoral head
- Arthrography:
 - Used to evaluate method of treatment

Diagnostic Procedures/Surgery

Arthrocentesis of hip definitive test to exclude septic arthritis if significant concern for this; may need orthopedic consultation

DIFFERENTIAL DIAGNOSIS

- Unilateral involvement:
 - Transient (a.k.a. toxic) synovitis
 - Septic arthritis
 - Osteomyelitis
 - Sickle cell anemia
 - Juvenile rheumatoid arthritis
 - Rheumatic fever
 - Trauma:
 - Femoral neck fracture
 - Hip dislocation
 - Slipped capital femoral epiphysis
 - Tuberculosis
 - Tumor
- Bilateral involvement:
 - Hypothyroidism
 - Epiphyseal dysplasia
 - Gaucher disease



TREATMENT

PRE HOSPITAL

Clinical course is subacute; less likely to present via ambulance

INITIAL STABILIZATION/THERAPY

Not a life-threatening condition; clinical instability mandates identification of alternative diagnosis

ED TREATMENT/PROCEDURES

- Main ED intervention is pain control
- Restrict from vigorous activity
- May need crutches if weight bearing painful

MEDICATION

First Line

Ibuprofen: 10 mg/kg/dose PO q6–8h PRN pain

Second Line

Diazepam: 0.1–0.2 mg/kg/dose (max. 5 mg) PO q6–8h PRN muscle spasm



FOLLOW-UP

DISPOSITION

Admission Criteria

Need for admission rare, indicated for:

- Severe pain or muscle spasm not controlled by PO medications
- Social considerations; bedrest/care at home not possible

Discharge Criteria

- Adequate pain control with PO medications
- Orthopedic follow-up arranged in 1–2 wk

Issues for Referral

- Age of onset shown to affect outcome; onset younger than 6 with better outcome, if older than 8, shown to have poorer outcome
- For more severe disease, a range of treatment options exists, including conservative treatment, orthotics, traction, and surgical osteotomy, determined by consulting orthopedist
- Degenerative arthritis and possible need of a replacement is the main long-term

complication

FOLLOW-UP RECOMMENDATIONS

Orthopedic consultation to determine further management; may be outpatient

PEARLS AND PITFALLS

Abrupt onset, presence of fever, unstable patient, or toxic appearance suggest diagnosis other than LCP.

ADDITIONAL READING

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CODES

ICD9

732.1 Juvenile osteochondrosis of hip and pelvis

ICD10

- M91.10 Juvenile osteochondrosis of head of femur, unspecified leg
- M91.11 Juvenile osteochondrosis of head of femur, right leg
- M91.12 Juvenile osteochondrosis of head of femur, left leg

LEUKEMIA

Linda Mueller

BASICS

DESCRIPTION

- Neoplasms of WBCs that have undergone a malignant transformation
- Hyperleukocytosis:
 - Occurs with WBC $> 100,000/\text{mm}^3$
 - Leads to occlusions of small vessels primarily in brain or lungs
 - Present with confusion, stupor, or shortness of breath

Chronic Myelogenous Leukemia

- Overproduction of granulocytic WBCs (neutrophils)
- Neutrophil function preserved
- Thrombocytosis
- Basophilia
- Philadelphia chromosome present in bone marrow of $> 95\%$

Chronic Lymphocytic Leukemia

- Most common leukemia in adults
- Overproduction of monoclonal lymphocytes
- Cells accumulate in lymph nodes, bone marrow, liver, spleen
- Particularly prone to herpes virus infections

Acute Leukemias

- Proliferation of undifferentiated immature cells:
 - Acute myelogenous leukemia (AML)—immature myeloid cells
 - Acute lymphocytic leukemia (ALL)—immature lymphoid cells (blasts)
- Rapidly fatal

ETIOLOGY

- Cause unknown
- Familial clustering in chronic lymphocytic leukemia (CLL)
- Increased incidence of AML, ALL, and chronic myelogenous leukemia (CML) with ionizing radiation

Pediatric Considerations

- Usually have ALL:
 - Most common pediatric cancer
- 60–80% remission in those who are standard risk

- Better overall prognosis, except if < 1 yr of age
- May develop leukostasis at lower levels
- Allopurinol dose is 3 mg/kg.
- Ceftazidime dose is 50 mg/kg.

Pregnancy Considerations

- 90% of leukemias are AML or ALL.
- Myeloid leukemias are more common.
- CLL is very rare in pregnancy.
- Chemotherapeutics may cause birth defects and/or preterm labor.
- Same prognosis as nonpregnant; do not delay therapy.
- Transfuse earlier than nonpregnant; keep hemoglobin > 9.8 mg/dL.

Geriatric Considerations

More likely to present with CLL and CML

DIAGNOSIS

SIGNS AND SYMPTOMS

Chronic Myelogenous Leukemia

- Asymptomatic
- Fatigue
- Weight loss
- Left upper quadrant pain, tenderness
- Abdominal fullness
- Splenomegaly (most common)
- Later stage:
 - Headaches
 - Bone pain
 - Arthralgias
 - Fever
 - Leukotactic symptoms:
 - Dyspnea
 - Drowsiness
 - Confusion

Chronic Lymphocytic Leukemia

- Asymptomatic
- Fatigue
- Lethargy
- Weight loss

- Lymphadenopathy
- Splenomegaly
- Hepatomegaly

Acute Myelogenous Leukemia

- Fever
- Fatigue
- Pallor
- Headache
- Angina
- Congestive heart failure, dyspnea on exertion
- Bone pain
- Granulocytic sarcoma (isolated mass of leukemic blasts)
- Easy bleeding (thrombocytopenia):
 - Petechiae
 - Ecchymosis
 - Epistaxis
 - Hemorrhage
- Infections (granulocytopenic)
- Organ involvement with advanced ALL:
 - Lymphadenopathy
 - Hepatomegaly
 - Splenomegaly
 - Leukemic meningitis:
 - Headache
 - Nausea
 - Seizures

History

- Radiation exposure
- Exposure to alkylating agents
- Recent viral infection, particularly Epstein–Barr

Physical-Exam

- Signs of bleeding (petechiae, purpura)
- Hepatomegaly and splenomegaly
- Presence of chloromas (AML blast tumors)
- Sausage-like hemorrhagic retinal veins are pathognomic for hyperviscosity.

ESSENTIAL WORKUP

- CBC/platelets:
 - CML:

- WBC range, 10,000–1 million/mm³
- Neutrophils predominate.
- Thrombocytosis in 50%
- CLL:
 - Absolute lymphocytosis >5,000
 - WBC range, 40,000–150,000/mm³
- Acute leukemia (AML/ALL):
 - Anemia
 - Thrombocytopenia
 - Elevation/depression of WBCs

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Electrolytes, BUN, creatinine, glucose, calcium
- Uric acid level:
 - Frequently elevated, especially in ALL
- Lactate dehydrogenase:
 - Increased in acute leukemias
- Coagulation profile:
 - PT/PTT, fibrinogen, fibrin-split products
 - If disseminated, suspect intravascular coagulation.
- Blood/urine cultures if fever
- Arterial blood gases/pulse oximetry for shortness of breath

Imaging

CXR for infectious workup

Diagnostic Procedures/Surgery

- Bone marrow biopsy:
 - Required to make diagnosis
 - CML—hypercellular with myeloid hyperplasia
 - CLL—lymphocytosis (30–100%)
 - Acute leukemia—hypercellular with blast cells, which replace normal marrow
- Leukocyte alkaline phosphatase test:
 - Decreased in neutrophils in CML
- Ph1 chromosome present in CML

DIFFERENTIAL DIAGNOSIS

- CML:
 - Lymphoma

- Myeloproliferative syndromes
- Systemic lupus erythematosus
- Infection—bacterial, fungal, mycobacterial
- CLL:
 - Pertussis
 - Infectious lymphocytosis
 - Cytomegalovirus
 - Epstein–Barr virus/mononucleosis
 - Hepatitis
 - Rubella
- Acute leukemia:
 - Aplastic anemia
 - Leukemoid reactions to infections



TREATMENT

INITIAL STABILIZATION/THERAPY

- 100% oxygen for hypoxia/shortness of breath
- IV access with 0.9% NS
- Initiate platelet transfusion for severe bleeding from thrombocytopenia.
- Begin broad-spectrum antibiotics for fever and granulocytopenia.
- Treat disseminated intravascular coagulation (see “Disseminated Intravascular Coagulation”).

ED TREATMENT/PROCEDURES

- Treat leukostasis:
 - Rehydrate with 500-mL bolus (20 mL/kg) IV 0.9% NS
 - Administer acetazolamide to alkalinize urine.
 - Initiate allopurinol.
 - Arrange for leukapheresis.
 - Whole-brain radiation or dexamethasone for CNS effects
 - Administer hydroxyurea for CML: 20–30 mg/kg single dose daily
- Transfuse packed RBCs for symptomatic anemia:
 - May require irradiated, filtered, and HLA-type–specific blood
- Post-ED treatment:
 - CLL:
 - Chemotherapy
 - Prednisone for immune-mediated thrombocytopenia
 - Radiation to localized nodular masses/enlarged spleen
 - CML:
 - Interferon therapy
 - Chemotherapy

- Bone marrow transplantation
- ALL:
 - Chemotherapy
 - CNS prophylaxis with intrathecal–methotrexate/cranial radiation
 - Bone marrow transplantation
- AML:
 - Chemotherapy
 - Bone marrow transplantation

MEDICATION

First Line

- Aggressive IVF, start with normal saline, then alkalinize
- Packed RBC and platelets as needed

Second Line

- Ceftazidime if febrile
- Allopurinol or rasburicase and diuretics if at risk for tumor lysis



FOLLOW-UP

DISPOSITION

Admission Criteria

- Newly diagnosed leukemia with:
 - Symptomatic anemia
 - WBC > 30,000
 - Thrombocytopenia
- ICU admission for unstable patients with disseminated intravascular coagulation, blast crisis, or bleeding

Discharge Criteria

Asymptomatic patients without significant lab abnormalities

Issues for Referral

Hematology for any patient presenting with new leukemia

PEARLS AND PITFALLS

- Monitor for tumor lysis and secondary hyperkalemia.
- Hyperleukocytosis may present as respiratory failure or hemorrhage.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Hyperviscosity Syndrome



CODES

ICD9

- 204.10 Chronic lymphoid leukemia, without mention of having achieved remission
- 205.10 Chronic myeloid leukemia, without mention of having achieved remission
- 208.90 Unspecified leukemia, without mention of having achieved remission

ICD10

- C91.10 Chronic lymphocytic leuk of B-cell type not achieve remis
- C92.10 Chronic myeloid leukemia, BCR/ABL-positive, not having achieved remission
- C95.90 Leukemia, unspecified not having achieved remission

LEUKOCYTOSIS

Sierra Beck • Steven M. Lindsey



BASICS

DESCRIPTION

Definition:

- Any elevation of total number of white blood cells (WBCs) beyond expected value
- Normal range for total WBCs ($/\text{mm}^3$):
 - Adults: 4,500–11,000
 - Children: WBC count decreases with age:
 - Infant, 1 wk old: 5,000–21,000
 - Toddler, 1 yr old: 6,000–17,500
 - Child, 4 yr old: 5,500–15,500
 - Pregnancy:
 - 1st trimester: 5,000–14,000
 - 2nd trimester: 5,000–15,000
 - 3rd trimester: 5,000–17,000
- Normal ranges shift upward with:
 - Exercise
 - Female gender
 - Smoking
 - Daytime hours
- Given wide range of normal values, numbers must be interpreted in clinical context
- Specific subsets
- Neutrophil predominance (neutrophilia):
 - Absolute neutrophil count $>7,500/\text{mm}^3$
 - Half of circulating neutrophils are adherent to blood vessel walls. They can be rapidly released (demarginate) in response to acute stressors. This can double the WBC count.
 - An additional pool of mature neutrophils, immature metamyelocytes, and band neutrophils are stored in the bone marrow. These can be released increasing the neutrophil count typically during inflammation or infection. Release of immature forms results in a “left shift.”
- Lymphocyte predominance (lymphocytosis)
 - Absolute lymphocyte count $>4,000/\text{mm}^3$
 - Stored in the spleen, lymph nodes, thymus, and bone marrow. They are typically released in response to foreign antigens or viral infections
- Hyperleukocytosis (WBC $>100,000/\text{mm}^3$):

- Seen primarily in hematologic malignancies
- Associated with leukostasis which can lead to cerebral infarction, pulmonary insufficiency, death

EPIDEMIOLOGY

- CBC most common test ordered from the emergency department
- Leukocytosis is one of the most commonly found lab abnormalities.
- Elevated WBC count can be found in 17% of ED patients in whom a CBC is checked (Callaham)

ETIOLOGY

- Neutrophil predominance:
 - Demargination/stress reaction:
 - Stress
 - Exercise
 - Surgery
 - Seizures
 - Trauma
 - Hypoxia
 - Pain
 - Vomiting
 - Inflammation:
 - Rheumatoid arthritis
 - Gout
 - Inflammatory bowel disease
 - Infection, generally bacterial
 - Lab error
 - Labor
 - Leukemoid reaction (TB, Hodgkin, sepsis, metastatic CA)
 - Medications:
 - β -Agonist (epinephrine, cocaine, parenteral albuterol)
 - Corticosteroids
 - Lithium
 - Granulocyte colony stimulating factor
 - Metabolic disorders:
 - DKA
 - Thyrotoxicosis
 - Uremia
 - Malignancy, nonhematogenous
 - Myeloproliferative disorders:
 - Chronic myeloid leukemia
 - Polycythemia vera
 - Pregnancy

- Rapid RBC turnover:
 - o Hemorrhage
 - o Hemolysis
- Tissue necrosis:
 - o Cancer
 - o Burns
 - o Infarction
- Lymphocyte predominance:
 - Infection, generally viral, early stages:
 - o Mononucleosis
 - o VZV
 - o CMV
 - o Viral hepatitis
 - Bacterial infection, specifically:
 - o Pertussis
 - o TB
 - o Syphilis
 - o Rickettsia
 - o Babesia
 - o Bartonella
 - Hypothyroidism
 - Immunologic responses:
 - o Immunization
 - o Autoimmune diseases
 - o Graft rejection
 - Lymphoproliferative disease:
 - o Acute lymphocytic leukemia
 - o Chronic lymphocytic leukemia
 - o Non-Hodgkin lymphoma
 - Splenectomy



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Depends upon presenting complaint
- Symptoms suggestive of infection:
 - Cough
 - Fever
 - Rash
 - GI symptoms

- Symptoms suggestive of long-term inflammation:
 - Joint pain
 - Rash
- Symptoms suggestive of malignancy:
 - Weight loss
 - Fatigue
 - Night sweats

Physical-Exam

- Focal signs of infection:
 - Cellulitis/abscess
 - Otitis
 - Pharyngitis
 - Pneumonia
- Signs of malignancy:
 - Hepatosplenomegaly
 - Lymphadenopathy
 - Pallor
 - Bleeding
- Signs of chronic inflammatory conditions:
 - Joint pain and swelling
 - Rash

DIAGNOSIS TESTS & INTERPRETATION

- Interpretation of leukocytosis:
 - Elevated WBC counts are highly nonspecific and rarely change management. They have equal chances of appropriately and inappropriately influencing care
 - Duration of leukocytosis:
 - Hours to days: More likely to be acute event (infection, acute leukemia)
 - Months to years: Chronic inflammatory states or hematologic malignancies (rheumatoid arthritis, solid organ tumors, chronic leukemias, lymphomas)
- Cell count and differential:
 - If obtained be sure to evaluate absolute cell counts, percentile counts will be spuriously elevated if other cell lines are low
 - Look for the presence of a “left shift” (immature cells in circulation). Normal ratio is 1 band cell for every 10 neutrophils in circulation. This may indicate acute infection, or malignancy. Demargination should not cause a left shift.
 - Differential rarely provides additional helpful information and cannot reliably distinguish between bacterial and viral infections.

- Manual differential or peripheral blood smear:
 - Can be ordered if concern for lab error. Nucleated RBCs, or clumped platelets may cause spurious results in automated tests.
- RBC and platelet counts:
 - Low counts may suggest malignancy or bone marrow infiltration
- Pediatrics:
 - Evaluation of young febrile children (< 36 mo):
 - WBC >15,000 is associated with a high risk of serious bacterial infection and in the appropriate clinical context should prompt clinicians to consider antibiotics, blood cultures, and possible admission.
 - Providers should not be reassured by only moderately elevated WBC counts 15–25,000
 - Conversely, the presence of a significantly elevated WBC count >25,000 does not signify more significant illness
 - Crying shown to elevate WBC count 113%

ESSENTIAL WORKUP

- Dependent upon clinical scenario
- Cell count differential with absolute cell counts may be helpful if the etiology is not apparent based on history and physical exam alone, or if hematologic malignancy is considered
- If hematologic malignancy is suspected patients will require peripheral blood smear and bone marrow biopsy following admission

DIFFERENTIAL DIAGNOSIS

See etiology. Narrow diagnosis based on corresponding clinical presentation.



TREATMENT

- Based on underlying disease process.
- Leukostasis secondary to the extremely high WBC counts of malignancy may require acute management with:
 - IV hydration
 - Transfusion
 - Allopurinol
 - Hydroxyurea
 - Hematology consult for leukapheresis



FOLLOW-UP

DISPOSITION

Dependent upon clinical scenario. Avoid making disposition decisions based solely on

the WBC count.

PEARLS AND PITFALLS

- Be aware that the decision making of health care providers is significantly influenced by the presence of a leukocytosis
- Increased admission rate
- Increased number of tests and cost
- Wide variety of conditions can cause a leukocytosis, including normal variants
- Poor sensitivity and specificity for predicting severity of illness
- Extremely high WBC counts typically in the setting of hematologic malignancy can be associated with leukostasis which can be life threatening and require emergent therapy

ADDITIONAL READING

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- Shah SS. Clinical significance of extreme leukocytosis in the emergency department evaluation of young febrile children. *Acad Emerg Med.* 2003;10(5):443–444.



CODES

ICD9

- 288.8 Other specified disease of white blood cells
- 288.60 Leukocytosis, unspecified
- 288.61 Lymphocytosis (symptomatic)

ICD10

- D72.820 Lymphocytosis (symptomatic)
- D72.828 Other elevated white blood cell count
- D72.829 Elevated white blood cell count, unspecified

LIGHTNING INJURIES

Tarlan Hedayati • Sheila T. Wan

BASICS

DESCRIPTION

- Lightning is a discharge of energy that occurs cloud to cloud (90%) or cloud to ground (10%).
- Exposure to lightning:
 - Brief duration (1–100 msec)
 - Typically occurs during outdoor activity
 - Highest incidence in summer months, between 3 and 6 pm
 - Fatality rate of 8–10%

ETIOLOGY

- Mechanism of injury—electrical:
 - Direct strike (5%)
 - Contact potential (15–25%):
 - Current passes through an object the victim is touching.
 - Side splash (20–30%):
 - Current jumps from nearby object to the victim.
 - Earth potential rise/ground current (40–50%):
 - Current moves through the ground surface and may injure multiple victims.
 - Current moves through hard-wired telephone lines, metallic pipes, or a structure's electrical equipment, causing lightning injury to victims indoors.
 - Upward streamer (10–15%):
 - Negatively charged lightning strikes from a cloud and induces positive current from the ground to rise and meet it to complete the lightning channel.
- Mechanism of injury—trauma:
 - Barotraumas
 - Blunt trauma:
 - Muscle contractions can throw the victim and/or cause a fall.
 - Thermal burn

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Consider lightning strike in unwitnessed falls, cardiac arrests, or unexplained coma in an outdoor setting.
- Conscious patients may report:
 - Muscle aches and pains
 - Chest pain
 - Shortness of breath
 - Extremity pain or discoloration
 - Burns
 - Neurologic deficits including:
 - Paresthesia
 - Dysesthesias
 - Weakness or paralysis
 - Visual disturbance or blindness
 - Headache
 - Confusion or amnesia
 - Hearing loss or deafness
 - Dizziness

Physical-Exam

- HEENT:
 - Blunt head trauma
 - Ruptured tympanic membrane with ossicular disruption (up to 50%)
 - Ophthalmic injuries:
 - Cataracts
 - Corneal lesions
 - Intraocular hemorrhages
 - Retinal detachment
- Neck:
 - Cervical spine injury
- Cardiopulmonary injuries:
 - Primary cardiac arrest:
 - Cardiac asystole:
 - Due to direct current injury
 - May resolve spontaneously as the heart's intrinsic automaticity resumes.
 - Hypertension: Transient
 - Pulmonary contusion or hemorrhage
 - Respiratory arrest:
 - Caused by paralysis of medullary respiratory center
 - May persist longer than primary cardiac arrest and lead to hypoxia-induced secondary cardiac arrest and/or brain injury
- Extremities:

- Fractures/dislocations
- Muscle tears, contusions
- Compartment syndromes
- Mottled or cold:
 - Caused by autonomic vasomotor instability
 - Usually resolves spontaneously in a few hours
- Skin:
 - Burns:
 - May evolve over several hours after injury
 - Discrete entrance and exit wounds are uncommon.
 - Superficial in nature; deep burns uncommon
 - Direct thermal injury is uncommon due to the brevity of electrical currents.
 - Thermal burns can arise from evaporation of water on skin, ignited clothing, and heated metal objects (buckles, jewelry).
 - Feathering pattern of fernlike “burns” are pathognomonic of lightning injuries and resolve within 24 hr.
- Neurologic injuries:
 - Confusion, cognitive or memory defects
 - Altered level of consciousness (>70% of cases)
 - Flaccid motor paralysis
 - Seizures
 - Cerebrovascular accident
 - Fixed dilated pupils due to either serious head injury or autonomic dysfunction
- Shock:
 - Neurogenic (spinal injury)
 - Hypovolemic (trauma)

ESSENTIAL WORKUP

Confirmatory history from bystanders or rescuers of the circumstances of the injury

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC
- Urinalysis for myoglobin (rare)
- Electrolytes for acidosis
- BUN, creatinine for renal function
- Troponin, creatine kinase, and cardiac enzymes for muscle/cardiac damage

Imaging

- CXR:

- Pulmonary edema
- Pulmonary contusion/hemorrhage
- Rib fractures
- Cervical spine radiograph
- Head CT for altered mental status or significant head trauma
- Relevant imaging for specific injuries

Diagnostic Procedures/Surgery

EKG:

- Prolonged QT (most common)
- Nonspecific ST changes
- Premature ventricular contractions
- Atrial fibrillation
- Ventricular tachycardia
- Acute MI (rare)

DIFFERENTIAL DIAGNOSIS

Other causes of coma, cardiac dysrhythmias, or trauma:

- Hypoglycemia
- Intoxication
- Drug overdose
- Cardiovascular disease
- Cerebrovascular accident
- Seizure
- Syncope



TREATMENT

PRE HOSPITAL

- Field triage should rapidly focus on providing ventilatory support to unconscious victims or those in cardiopulmonary arrest:
 - Prevents primary cardiac arrest from degenerating into hypoxia-induced secondary cardiac arrest
 - Conscious victims are at lower risk for imminent demise.
- Spine immobilization for:
 - Cardiopulmonary arrest (suspected trauma)
 - Significant mechanical trauma
 - Suspected loss of consciousness at any time
- Cover superficial burns with sterile saline dressings.
- Immobilize injured extremities.
- Rapid extrication to decrease risk for repeat lightning strikes

INITIAL STABILIZATION/THERAPY

- ABCs
- Standard advanced cardiac life support measures for cardiac arrest
- Diligent primary and secondary survey for traumatic injuries and other causes of collapse/injury:
 - Maintain cervical spine precautions until cleared.
- Treat altered mental status with glucose, naloxone, or thiamine as indicated.
- Hypotension requires volume expansion, blood products, and/or pressor agents.

ED TREATMENT/PROCEDURES

- IV access
- Cardiac monitor and pulse oximetry
- Clean and dress burns.
- Tetanus prophylaxis
- Treat myoglobinuria if present:
 - Diuretics, such as furosemide or mannitol
 - Alkalinize urine to a pH of 7.45 with IV sodium bicarbonate
- Volume expansion:
 - Do not follow burn treatment formulas because lightning burns are rarely the cause of fluid loss.
 - Occult deep burn injury is rare when compared with other types of electrical current injury.
 - Titrate volume administration to urine output.
 - Fluid loading may be dangerous if patient has concomitant head injury.
- Compartment syndrome:
 - Must be distinguished from vasospasm, autonomic dysfunction, and paralysis, which are usually self-limited phenomena.
 - Fasciotomy will rarely be necessary.
- NSAIDs and high-dose steroids have been proposed to reduce long-term neurologic and corneal damage.

MEDICATION

- Furosemide: 1 mg/kg IV slow bolus q6h
- Mannitol: 0.5 mg/kg IV, repeat PRN
- Sodium bicarbonate: 1 amp IV push (peds: 1 mEq/kg) followed by 2–3 amps/L D5W IV fluid



FOLLOW-UP

DISPOSITION

Admission Criteria

- Postcardiac arrest patients
- History of change in mental status/altered level of consciousness
- History of chest pain, dysrhythmias, or ECG changes:
 - May not resolve spontaneously
 - 24–48 hr observation period to identify potentially unstable cases
 - Myoglobinuria
 - Acidosis
 - Extremity injury with or at risk for compartment syndrome

Discharge Criteria

Asymptomatic patients with no injuries

FOLLOW-UP RECOMMENDATIONS

- Close follow-up with subspecialists may be required due to the risk for delayed sequelae:
 - Neurology:
 - Memory deficit
 - Attention deficit
 - Aphasia
 - Sleep disturbance
 - Prolonged paresthesia and dysesthesias
 - Ophthalmology
 - ENT
- Psychology/psychiatry:
 - Anxiety
 - Depression
 - Personality changes
 - Post-traumatic stress disorder

PEARLS AND PITFALLS

- Do not follow burn treatment formulas for lightning burns and injuries.
- Be diligent in the primary and secondary survey so as not to miss occult injuries.
- Have a low threshold to admit and monitor patients with cardiopulmonary complaints, as unstable dysrhythmias may occur 24–48 hr post injury.

ADDITIONAL READING

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- Price T, Cooper MA. Electrical and lightning injuries. In: Marx JA, Hockenberger RS, Walls RM, et al., eds. *Rosen’s Emergency Medicine.* 6th ed. Philadelphia, PA: Mosby; 2006.



See Also (Topic, Algorithm, Electronic Media Element)

Electrical Injury



CODES

ICD9

- 949.0 Burn of unspecified site, unspecified degree
- 994.0 Effects of lightning
- 994.8 Electrocution and nonfatal effects of electric current

ICD10

- T30.0 Burn of unspecified body region, unspecified degree
- T75.00XA Unspecified effects of lightning, initial encounter
- T75.09XA Other effects of lightning, initial encounter

LITHIUM POISONING

Sean M. Bryant



BASICS

DESCRIPTION

- GI absorption is rapid:
 - Regular release: Peak serum levels 2–4 hr
 - Sustained release: Peak serum levels 4–12 hr
- Half-life 24 hr
- Slow distribution (at least 6 hr)
- Volume of distribution 0.6–0.9 L/kg
- Elimination:
 - *Not* metabolized
 - Renal excretion (unchanged)
 - Reabsorbed in the *proximal* tubules by sodium transport mechanism
 - Elimination half-life (therapeutic) is 20–24 hr and prolonged in chronic users
- Therapeutic and toxic indices:
 - Therapeutic and toxic effects occur *only* when lithium is intracellular
 - Narrow toxic-to-therapeutic ratio
 - Therapeutic level 0.6–1.2 mEq/L (postdistribution)
 - Because of small size, renal handling is similar to sodium, potassium, and magnesium
- Risk factors:
 - Acute conditions increasing risk of toxicity:
 - Dehydration (larger percent reabsorbed)
 - Overdose
 - Chronic conditions:
 - Hypertension
 - Diabetes mellitus
 - Renal failure
 - Congestive heart failure
 - Advanced age
 - Dose change
 - Drug interactions
 - Lithium therapy
 - Low-salt diet
 - The following may result in increased serum lithium levels due to decreased renal clearance or exacerbated effects:
 - NSAIDs

- Thiazide diuretics
- ACE inhibitors
- Phenytoin
- Tricyclic antidepressants
- Phenothiazines

ETIOLOGY

- Acute or chronic conditions affecting lithium clearance
- Overdose

DIAGNOSIS

SIGNS AND SYMPTOMS

- Acute toxicity:
 - _ Less common/serious than chronic toxicity
 - _ Neurologic (mild):
 - Weakness
 - Fine tremor
 - Lightheadedness
 - _ Neurologic (moderate):
 - Ataxia
 - Slurred speech
 - Blurred vision
 - Tinnitus
 - Weakness
 - Coarse tremor
 - Fasciculations
 - Hyper-reflexia
 - Apathy
 - _ Neurologic (severe):
 - Confusion
 - Coma
 - Seizure
 - Clonus
 - Extrapyrarnidal symptoms
 - _ GI:
 - Very common
 - Nausea/vomiting
 - Diarrhea
 - Abdominal pain
 - _ Cardiac:
 - Prolonged QT, ST depression
 - T-wave flattening *most common* ECG abnormality

- U-waves
- Serious dysrhythmias (rare)
- Chronic toxicity:
 - _ Neurologic:
 - Most common
 - Same symptoms as acute
 - Severe toxicity includes parkinsonism, psychosis, and memory deficits
 - _ Renal:
 - Nephrogenic diabetes insipidus
 - Interstitial nephritis
 - Distal tubular acidosis
 - Direct cellular damage
 - _ Dermatologic:
 - Dermatitis
 - Ulcers
 - Localized edema
 - _ Endocrine:
 - Hypothyroidism
 - _ Hematologic:
 - Leukocytosis
 - Aplastic anemia

History

- Time of last dose ingested
- Ingestion history:
 - _ Acute (1-time overdose)
 - _ Chronic (scheduled dosing)
 - _ Acute on chronic (overdose in patients who regularly take lithium)

Physical-Exam

Perform complete neurologic exam

ESSENTIAL WORKUP

- Lithium level: Goal = postdistribution:
 - _ Because of prolonged distribution, repeat every 2 hr to ensure trend
- Stratify patient into 1 of 3 categories of toxicity to interpret level and predict toxicity: Acute, acute on chronic, chronic:
 - _ Acute toxicity:
 - Intentional overdose in patient not previously taking lithium
 - Poor correlation between lithium level and symptoms because intracellular distribution has not yet occurred
 - Toxic levels may appear in asymptomatic patients
 - Lithium level > 4 mEq/L may result in toxic sequelae because of

slowed clearance

- Acute on chronic toxicity:
 - Intentional or accidental overdose in patient on lithium therapy
 - Lithium level > 3 mEq/L usually associated with symptoms
- Chronic toxicity:
 - Patients on lithium therapy who progressively develop toxicity secondary to factors other than acute ingestion
 - Stronger correlation between lithium level and symptoms
 - Lithium level > 1.5 mEq/L may correlate with toxicity

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Electrolytes, BUN, creatinine, and glucose levels to determine electrolyte disturbances/renal function
- Aspirin and/or acetaminophen levels as indicated by history
- Urinalysis:
 - Specific gravity

DIFFERENTIAL DIAGNOSIS

- Consider lithium toxicity with altered mental status and fasciculations
- Endocrine:
 - Hypoglycemia
- Toxicologic:
 - Cholinergic substances
 - Heavy-metal poisoning
 - Neuroleptic overdose
 - Black widow/scorpion envenomation
 - Strychnine poisoning



TREATMENT

PRE HOSPITAL

- Transport all appropriate pill bottles to the hospital
- IV access, oxygen, and cardiac monitoring

INITIAL STABILIZATION/THERAPY

- ABCs
- Secure IV access with 0.9% NS
- Cardiac monitor
- Naloxone, thiamine, dextrose (or Accu-Chek) if altered mental status
- Benzodiazepines for seizures

ADDITIONAL TREATMENT

General Measures

- Correct electrolyte abnormalities
- Maintain well-hydrated state
- Continuous cardiac monitoring
- Observe for neurologic changes
- Prevent absorption:
 - Consider gastric lavage only if patient presents within 1 hr of acute life-threatening ingestion and has protected airway
 - Activated charcoal:
 - Lithium is not adsorbed by charcoal
 - Administer 1 dose of activated charcoal if possible coingestants
 - Whole-bowel irrigation:
 - Polyethylene glycol (PEG) solution (GoLytely)
 - Sustained-release lithium products
 - Flushes lithium through gut
 - Administer (2 L/hr per nasogastric tube) until rectal effluent is clear
 - Contraindications include bowel obstruction or perforation, ileus or hypotension, and unprotected airway in obtunded or seizing patient
- Enhance elimination:
 - IV fluids:
 - Rapidly correct any pre-existing fluid deficit with 0.9% NS at 150–300 mL/hr (or 2× maintenance)
 - Saline hydration improves glomerular filtration and decreases proximal tubule reabsorption of lithium
 - Maintain urine output, 1–2 mL/kg/hr
 - Limited value once glomerular filtration rate maximized
 - Sodium bicarbonate offers no additional advantage
 - Loop, thiazide, and osmotic diuretics not recommended:
 - Dehydration may result in worsening toxicity
 - No direct effect on renal reabsorption because lithium is reabsorbed in proximal tubules
 - Kayexalate (sodium polystyrene sulfonate):
 - Animal and human studies indicate some efficacy
 - Complications may include hypokalemia, hyperkalemia, fluid overload, and dysrhythmias
 - Dialysis:
 - Peritoneal dialysis is not recommended
 - Hemodialysis may be recommended for augmenting elimination (see below)
- Hemodialysis is recommended for severe cases or acute ingestions with high levels

indicating imminent toxicity:

- Controversial indications (validated criteria yet to be established):
 - o Severe and progressive neurologic abnormalities
 - o Renal insufficiency
 - o Altered mental status (e.g., placidly tolerating a rectal tube for GI effects would be considered substantial obtundation)
 - o Ventricular dysrhythmia/cardiogenic shock
 - o History of congestive heart failure or pulmonary edema
 - o Acute ingestions with levels $> 4\text{--}5$ mEq/L
 - o Chronic ingestions with levels $> 2.5\text{--}3$ mEq/L
- Endpoint is lithium level < 1 mEq/L
- Repeat lithium level 6 hr after dialysis checking for evidence of redistribution
- May need to repeat dialysis due to rebound effect (redistribution of intracellular lithium)
- May reduce the potential for developing permanent neurologic sequelae with chronic toxicity

MEDICATION

- Dextrose: D50 1 amp: 25 g (peds: D25W 4 mL/kg) IV
- Diazepam: 5 mg (peds: 0.2–0.4 mg/kg) IV q5min until seizures controlled
- Naloxone: 2 mg (peds: 0.1 mg/kg) IV or via endotracheal tube
- PEG solution: 2 L/hr (peds: 2 mL/kg/h) via nasogastric tube
- Thiamine: 100 mg IV



FOLLOW-UP

DISPOSITION

Admission Criteria

- Symptomatic
- Requiring hemodialysis
- Lithium level unchanged, increased, or > 2 mEq/L despite ED intervention
- Moderate to severe symptoms with chronic levels > 4 mEq/L warrant admission to ICU
- Intentional ingestion

Discharge Criteria

Decreasing lithium levels every 2–4 hr in *asymptomatic* patient *and* serum lithium level < 2 mEq/L (nonsuicidal patients)

Issues for Referral

Intentional overdose:

- Psychiatry consultation

FOLLOW-UP RECOMMENDATIONS

Psychiatry follow-up to ensure correct dosing regimen in those with chronic poisoning

PEARLS AND PITFALLS

- Erroneously interpreting a predistribution lithium concentration as “toxic” in patients without symptoms or history of overdose
- Aggressive hydration in patients with pulmonary edema, renal insufficiency, or mental status changes

ADDITIONAL READING

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- Waring WS. Management of lithium toxicity. *Toxicol Rev.* 2006;25:221–230.



CODES

ICD9

985.8 Toxic effect of other specified metals

ICD10

- T56.891A Toxic effect of other metals, accidental (unintentional), initial encounter
- T56.892A Toxic effect of other metals, intentional self-harm, initial encounter
- T56.894A Toxic effect of other metals, undetermined, init encntr

LUDWIG ANGINA

Paul Blackburn

BASICS

DESCRIPTION

-
- In a couple of places (concerning airway and oxygenation) I put one of the references in parentheses to direct the inquisitive to an article in *Annals of Emergency Medicine* that many will not have heard about. This is different than what the format usually directs, and these can very easily be cut.
- Also, following GlideScope and EZ-IO being mentioned, I followed the format seen in other publications of putting the company name and address in parentheses following first mention of the devices in the text. These can obviously be omitted
- Named for German physician Wilhelm Friedrich von Ludwig, who 1st described this in 1836 as a rapidly progressive, gangrenous cellulitis and edema of soft tissues of the neck, floor of the mouth
- Gangrene is serosanguineous infiltration with little or no frank pus or primary abscesses
 - _ Contiguous spread may encircle the airway or involve the mediastinum
 - _ Emergent interventions rarely include surgical or aspiration techniques
- Most deaths are due to airway compromise, occlusion, and resultant asphyxia
 - _ Mortality exceeded 50% in preantibiotic era, currently <8%

ETIOLOGY

- Odontogenic in 90% of adult cases, usually from 2nd, 3rd mandibular molars
- Less commonly: Mandibular fractures, oral lacerations, contiguous infections, errant drug injections, tongue piercings
- Polymicrobial: *β-hemolytic strep* commonly associated with anaerobes such as peptostreptococcus, pigmented bacteroides
 - _ Microbiologic analyses may guide therapy

Pediatric Considerations

- Frequently no clear etiology or site of origin
- Ideally, a destination facility will have specialty expertise available (surgery and subspecialties, anesthesia) and be properly equipped to provide emergent intervention

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Nonspecific constitutional symptoms: Fever, malaise, anxiety
- Pain: Tongue, throat, jaw, chest, neck stiffness
- Dysphagia, dysphonia
- Dentition, dental care suboptimal

Physical-Exam

- Febrile, toxic, tripod “sniffing” posture
- Stridor, “hot potato” voice
- HEENT:
 - Tongue progressively displaced upward in both posterior, anterior directions at unpredictable rate
 - Airway increasingly compromised
 - Drooling, salivary incontinence
 - Trismus impedes diagnosis and complicates treatment measures
- Physical exam findings beyond those of the head and neck area are often noncontributory or unrelated

ESSENTIAL WORKUP

- The diagnosis is usually clinically evident
 - No study or procedure needed to confirm the diagnosis
- Loss of airway patency can be unexpected, precipitous, and calamitous
- Securing airway patency and initiating treatment take precedent over workup considerations

DIAGNOSIS TESTS & INTERPRETATION

Lab

No test will establish the diagnosis; assess severity or direct therapy

Imaging

Contrast-enhanced CT:

- CT of the neck with IV contrast enhancement is the study of choice:
 - Standard cross-sectional imaging extends from skull base to aortic arch
 - Best for evaluating the mediastinum, deep space infection location and extent, degree of airway involvement.
 - Findings include streaky or “dirty” fat in areas of inflammation; adenopathy (submandibular, submental, anterior and posterior cervical chains); perhaps pus or gas formation
 - Potential limitations: Patient must remain supine for the study duration. Scanning location often away from optimal resuscitation, intervention capability.

Plain Radiographs:

- **Soft tissue lateral neck x-ray** may demonstrate altered anatomy, especially in the upper airway
- **Chest x-ray** of little utility, including detecting presence and extent of mediastinal involvement
- **Panorex** may detect odontogenic or mandibular pathology, but of no use imaging soft tissue

Contrast-enhanced MRI:

- Information obtained is the same, of no greater value than contrast-enhanced CT:
 - Potential limitations: patient must remain supine, motionless for the study duration. Scanning location often away from optimal resuscitation, intervention capability.

Ultrasound:

- Detects gas in tissues, abscesses, reactive lymphadenopathy
- May locate, outline the airway amongst edematous, distorted tissues of the anterior neck
- A guide for abscess or fluid aspiration

Diagnostic Procedures/Surgery

No surgery or invasive procedure will establish the diagnosis, assess severity, or direct therapy

DIFFERENTIAL DIAGNOSIS

- Infectious: Cellulitis, epiglottitis, tracheitis, peritonsillar abscess
- Traumatic: Penetrating injury, sublingual hematoma from fracture, soft tissue injury
- Angioneurotic edema
- Neoplasia



TREATMENT

PRE HOSPITAL

- Transport in position of comfort
 - Allow adult tripod “sniffing” position, to suction themselves
 - Allow pediatric transport, simple interventions (blow-by O₂, nebulizer treatments) on mother’s lap
- Maximize oxygenation:
 - FIO₂ of 100%
 - Consider concurrent O₂ delivery systems, such as facemask and nasal cannula
- Jet insufflation: An infrequently used temporizing rescue device for oxygenation
 - Potential limitations: Few experienced with device assembly or use.

- Newer rescue devices easier to place and use.

Pediatric Considerations

- Minimize patient upset, agitation
 - Allow transport, simple interventions (blow-by O₂, nebulizer treatments) in parent's embrace.
 - Question the necessity for any interventions: IV access, blood draws, O₂ mask, monitor leads.
 - Transport to facility best able to care for this complex patient if possible.

INITIAL STABILIZATION/THERAPY

Airway Measures

- Maximize oxygenation
- Maintain in position of comfort
- Gather supplies/personnel for back-up airway techniques
- See “Airway Management” below.

Vascular Access

- Vascular access: Provides rapid, titratable, predictable medication delivery
 - Intraosseus (IO) access useful with poor peripheral access, resuscitations, pediatric access, adverse prehospital conditions
 - Commercially available device provides IO access rapidly, effectively
- 2nd access recommended: Rescue backup, concurrent polypharmacy.

ED TREATMENT/PROCEDURES

- Immediate priorities are to secure the airway and to institute medical treatment. Diminishing consensus on need for acute surgical intervention other than airway related.
- Infrequently see treatable abscess formation, fluid collections on initial presentation.

Airway management:

- Rescue airway devices may be difficult to place, altered effectiveness due to anatomy distortion, trismus, excessive secretions
- Avoid blind intubation techniques to reduce laryngospasm, iatrogenic injury, bleeding, further tissue distortion
- Equipment considerations:
 - Smaller ET tubes
 - Prelubricate with gel or viscous lidocaine
 - Use stylet or bougie for tube support
 - Bend distal tube into “hockey stick” shape
- Rapid-sequence intubation (RSI) agents may cause abrupt loss of muscle tone, airway architecture, or precipitate airway compromise

- Concern for impending respiratory failure increases with stridor, voice change, trismus, tripod posture, sialorrhea

Definitive management:

- Traditional surgical gold standard: Tracheostomy using local anesthesia:
 - Potential difficulties: Surgeon, specialist availability, facility capabilities not uniform
- Traditional nonsurgical gold standard intubation using fiberoptic guidance:
 - Potential difficulties: Fiberoptic scopes expensive, fragile, require specific cleaning regimens. Short scopes often lack suction or irrigation ports, visualization easily impaired. Their use is not intuitive to the infrequent operator
- **Best management option “double setup”**
 - Patient in an operating theater equipped, prepared to establish surgical airway
 - Nonsurgical intervention attempted
 - Immediate surgical intervention if unsuccessful or clinical deterioration
- **Intubation:** Anticipate distorted anatomy:
 - Sitting, awake a preferred option
 - Sequential topical applications

MEDICATION

- **IV administration:** Preferred route of administration as previously outlined
- IO considerations:
 - Lidocaine flush reduces infusion pain
 - Flow rates same as IV for routine fluids, medication administration
 - Avoid hyperosmolar agents, potential marrow injury
- **Antibiotics:** Empiric use of broad-spectrum antibiotics justifiable, for use until return of culture and antibiogram results, which should direct further therapy:
 - Ampicillin/sulbactam: 1.5–3 g IM/IV q6h (peds: 300 mg/kg/d div. q6 if <1yr, <40 kg; 1.5–3 g IV q6h if >1 yr, >40 kg); max. 12 g/d
 - Cefoxitin: 1–2 g IV q6–8h (peds: 80–160 mg/kg/d div. q4–6h); max. 12g/d
 - Clindamycin: 600–900 mg IM/IV q8h (peds: 15–25 mg/kg/d div. q6–8h)
 - Piperacillin/tazobactam: 3.375 g IV q6h (peds: If >9 mo, <40 kg; 300 mg/kg/d IV div. q8h)
 - Ticarcillin/clavulanate: 3.1 g IV q4–6h (peds: If >3 mo, <60 kg; 200–300 mg/kg/d div. q4–6h)
- **Analgesia:** Pain control should be a primary concern
- **Antiemetics:** Proactive, prophylactic use for medication-related or condition-induced symptoms
- **Steroids:** Recommend empiric use of longer acting steroids to reduce:
 - swelling
 - inflammation
 - systemic stress dose replenishment

- **Hyperbaric oxygen:** Consider if mediastinitis or necrotizing fasciitis



FOLLOW-UP

DISPOSITION

Admission Criteria

- All are admitted:
 - Airway encroachment and obstruction can be progressive and unpredictable
- ICU or closely monitored setting due to unpredictable progression of symptoms

Issues for Referral

- This is a clinical diagnosis with unpredictable progression:
 - Early specialty consultation is necessary for possible assistance with airway management or drainage
- Early transfer to higher level of care if the illness acuity exceeds the clinician's level of expertise or if the facility is not adequately equipped for such management

Pregnancy Considerations

- Mother is susceptible to all aspects and complications as nongravid patients
- Focus: Airway management, oxygenation, treatment of sepsis if present

Geriatric Considerations

Chronic comorbid conditions, chronic medications, less physiologic reserve can all complicate the presentation and treatment

COMPLICATIONS

- Asphyxia
- Spread into thoracic cavity:
 - Empyema
 - Mediastinitis
 - Lung abscess
- Pericarditis
- Internal jugular vein thrombosis
- Carotid artery erosion and/or infection
- Sepsis/bacteremia
- Subphrenic abscess

PEARLS AND PITFALLS

Pearls:

- Prepare to manage airway immediately

- Consult appropriate medical specialists as soon as possible, whether for transfer to a higher level of care, or to the operating suite for “double setup” management
- Video laryngoscopy is intuitive and easy to use, provides rapid, safe, high probability intubation success

Pitfalls:

- Failure to appreciate the progressive nature, unpredictable rate, extent of advancement
- Diagnostic testing and/or imaging should not delay definitive airway management or other therapy

ADDITIONAL READING

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- Ludwig BJ, Foster BR, Saito N, et al. Diagnostic imaging in nontraumatic pediatric head and neck emergencies. *Radiographics*. 2010;30(3):781–799.
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- Tobias JD, Ross AK. Intraosseous infusions: A review for the anesthesiologist with a focus on pediatric use. *Anesth Analg*. 2010;110:391–401
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- Wolfe MM, Davis JW, Parks SN. Is surgical airway necessary for airway management in deep neck infections and Ludwig angina? *J Crit Care*. 2011;26:11–14.



CODES

ICD9

528.3 Cellulitis and abscess of oral soft tissues

ICD10

K12.2 Cellulitis and abscess of mouth

LUNATE DISLOCATION

Emi M. Latham



BASICS

DESCRIPTION

- Dislocation of the lunate relative to the radius and distal row of metacarpals, most are volar but can be dorsal.
- Usually from high-energy hyperextension with ulnar deviation of the wrist.

ETIOLOGY

- Implies disruption of all 4 perilunate ligaments and radiocarpal ligament (Mayfield classification, stage IV)
- In volar dislocations, median nerve injury occurs in the carpal tunnel.
- Associated fractures of the radial styloid, scaphoid, capitate, and triquetrum are common and, if present, should raise suspicion of an occult perilunate ligamentous injury.



DIAGNOSIS

SIGNS AND SYMPTOMS

Frequently missed injury.

History

- Often from fall or motor vehicle accident.
- Pain and tenderness in the wrist.

Physical-Exam

- Mass or swelling in the wrist, either dorsally or volarly, depending on direction of dislocation.
- Gross deformity can be masked by swelling.
- May display signs of median nerve injury.

ESSENTIAL WORKUP

- Clinical exam is frequently not diagnostic.
- Assess skin integrity and neurovascular status, including 2-point discrimination.
- Radiographs as outlined below.

DIAGNOSIS TESTS & INTERPRETATION

Imaging

- Radiographic imaging to include 3 views of the wrist.
- Lateral view most useful:
 - Disruption of the normal imaginary longitudinal line through the centers of the radius, lunate, and capitate indicates dislocation or subluxation.
 - In volar dislocations, the lunate is frequently tilted with the opening of the “cup” toward the palm (spilled teacup sign)
- Posteroanterior (PA) view:
 - The dislocated lunate has a triangular (as opposed to the usual quadrangular) appearance.
 - Disruption of a smooth and continuous arc formed by the radiocarpal row suggests lunate dislocation.

Pediatric Considerations

Radiograph can be difficult to interpret unless full ossification is present.

Geriatric Considerations

Other fractures are common.

DIFFERENTIAL DIAGNOSIS

- Lunate fracture.
- Perilunate dislocation.
- Scapholunate dissociation.
- Scaphoid fracture.



TREATMENT

PRE HOSPITAL

- Dress open wounds.
- Immobilize in neutral position.

INITIAL STABILIZATION/THERAPY

Immobilize in position of comfort with a volar or “sugar tongs” splint.

ED TREATMENT/PROCEDURES

- Identify multiple trauma or other injuries.
- Contact a hand surgeon for immediate reduction and possible operative intervention.
- Closed reduction can be difficult or unstable.
- Open reduction and internal fixation are frequently required.

Pediatric Considerations

Although serious injury is unusual, children with wrist pain should be splinted and

referred for ongoing evaluation of possible occult fractures.

MEDICATION

First Line

Analgesics:

- **Morphine:**
 - Pediatrics: 0.05–0.20 mg/kg IV up to 1.5 mg. Use preservative-free formulation q4h
 - Adults: 4–8 mg IV
- **Acetaminophen with hydrocodone:**
 - Pediatrics > 12 yo: 2.5–10 mg hydrocodone every 4–6 h to max 60 mg/24 h or 4 g acetaminophen/24 h.
 - Adults: 5–10 mg hydrocodone ever 4–6 h as needed not to exceed 60 mg/24 h or 4 g acetaminophen/24 h
- **Acetaminophen with codeine (adults):**
 - Pediatrics: 0.5–1 mg/kg/dose based on codeine content PO q4–6h; do not exceed 5 doses of 10–15 mg/kg/24 h of acetaminophen
 - Adults: 30–60 mg/dose PO q4–6h; do not exceed 4 g/24 h of acetaminophen
- **Hydrocodone and acetaminophen:**
 - Pediatrics < 12 yr old: 0.1–0.2 mg/kg based on hydrocodone content PO q4–6h; do not exceed 5 doses of 10–15 mg/kg/24 h of acetaminophen
 - Pediatrics > 12 yr old: 750 mg apap PO q4h, not to exceed 10 mg hydrocodone per dose
 - Adults: Do not exceed 4 g/24h of acetaminophen PO q4–6h

Second Line

NSAIDs:

- **Ibuprofen:**
 - Pediatrics: 5–10 mg/kg q6–8h, max. dose 40 mg/kg/d PO div. TID/QID
 - Adults: 600 mg PO q6h
- **Naproxen:**
 - Pediatrics > 2 yr old: 2.5 mg/kg/d PO BID (not to exceed 10 mg/kg/d)
 - Adults: 250–500 mg PO BID
- **Acetaminophen with codeine (pediatrics):**
 - Patients may metabolize codeine at variable speeds: poor metabolizers which may lead to under-response, or "ultra-fast" metabolizers which can lead to high levels of morphine, hence undesirable side effects such as apnea and death.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Admission is often necessary for definitive care.
- Open fracture, presence of multiple trauma, or other more serious injuries mandates admission.

Discharge Criteria

Patients with closed dislocations or fractures that have been adequately reduced and immobilized in the ED may be discharged with orthopedic follow-up.

FOLLOW-UP RECOMMENDATIONS

- For those reduced and discharged with splint, follow-up with orthopedics.
- No return to play until fully healed.

PEARLS AND PITFALLS

- Failure to diagnose wrist dislocations.
- Missed median nerve injury.
- Avascular necrosis of the lunate (Kienböck disease)
- Degenerative joint disease.

ADDITIONAL READING

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- Mayfield JK, Johnson RP, Kilcoyne RK. Carpal dislocations: Pathomechanics and progressive perilunar instability. *J Hand Surg Am.* 1980;5(3):226–241.
- Perron AD, Brady WJ, Keats TE, et al. Orthopedic pitfalls in the ED: Lunate and perilunate injuries. *Am J Emerg Med.* 2001;19(2):157–162.



CODES

ICD9

- 833.02 Closed dislocation of radiocarpal (joint)
- 833.09 Closed dislocation of wrist, other

ICD10

- S63.024A Dislocation of radiocarpal joint of right wrist, initial encounter
- S63.026A Dislocation of radiocarpal joint of unspecified wrist, initial encounter

- S63.096A Other dislocation of unspecified wrist and hand, initial encounter

LYME DISEASE

Moses S. Lee



BASICS

DESCRIPTION

- Most common tick-borne illness in North America
- Endemic in Northeast, Upper Midwest, and northwestern California

ETIOLOGY

- Peak April–November; 80–90% in summer months
- Spirochete *Borrelia burgdorferi* introduced by Ixodes tick:
 - *Ixodes dammini* (deer tick) most common
- < 50% of patients recall tick bite.
- Pathogenesis—combination of:
 - Organism-induced local inflammation
 - Cytokine release
 - Autoimmunity
- No person-to-person transmission
- *Borrelia miyamotoi*, a spirochete related to *B. burgdorferi*, has recently been described as causing disease similar to Lyme disease.



DIAGNOSIS

SIGNS AND SYMPTOMS

Stage I (early):

- Onset few days to a month after tick bite (arthropod transmission)
- 30–50% of patients recall tick bite.
- Erythema chronicum migrans (ECM):
 - Pathognomonic finding:
 - Bull's-eye rash
 - Maculopapular, irregular expanding annular lesion:
 - Single or multiple
 - Central clearing with red outer border
 - Diameter > 5 cm
- Regional adenopathy
- Low-grade intermittent fever
- Headache
- Myalgia
- Arthralgias
- Fatigue

- Malaise

Stage II (secondary, disseminated):

- Days to weeks after tick bite
- Intermittent and fluctuating symptoms with eventual disappearance
- Triad of aseptic meningitis, cranial neuritis, and radiculoneuritis:
 - Facial (Bell) palsy most common cranial neuritis
 - May present without rash
 - Prognosis generally good
- Cardiac:
 - Tachycardia
 - Bradycardia
 - Atrioventricular block
 - Myopericarditis

Stage III (tertiary, late):

- Onset > 1 yr after disease onset
- Acrodermatitis chronica atrophicans:
 - Extensor surfaces of extremities, especially lower leg
 - Initial edematous infiltration evolving to atrophic lesions
 - Resembles scleroderma
- Arthritis:
 - Brief arthritis attacks
 - Monoarthritis
 - Oligoarthritis
 - Occasionally migratory
 - Most common joints (descending order):
 - Knee
 - Shoulder
 - Elbow

Other:

- GI:
 - Hepatitis
 - Right upper quadrant pain
- Ocular:
 - Keratitis
 - Uveitis
 - Iritis
 - Optic neuritis
- Jarisch–Herxheimer reaction:
 - Worsening of symptoms a few hours after treatment initiated
 - More common in patients with multiple ECM lesions
- Babesiosis occurs simultaneously in endemic areas.

Persistent Lyme disease:

- Articular and neurologic symptoms despite treatment:

– Chronic axonal polyneuropathy or encephalopathy

Recurrent Lyme disease:

- Relapse despite treatment
- 2nd episodes less severe

Pediatric Considerations

- More likely than adults to be febrile
- Only 50% of children with arthralgias have history of ECM.
- Facial palsy is accompanied by aseptic meningitis in 1/3.
- Asymptomatic cardiac involvement with abnormal ECGs
- Appropriately treated children have excellent prognosis for unimpaired cognitive functioning.
- Untreated children may have keratitis

Pregnancy Considerations

No clear evidence that Lyme disease during pregnancy causes harm to fetus

History

- History of tick bite in endemic areas
- Flu-like illness in the summer

Physical-Exam

- Rash
- Joint, cardiac, and neurologic findings in later organ involvement

ESSENTIAL WORKUP

- Clinical diagnosis:
 - Presence of ECM obviates serologic tests.
- Careful search for tick
- Lumbar puncture when meningeal signs
- Arthrocentesis for acute arthritis
- ECG

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC:
 - Leukocytosis
 - Anemia
 - Thrombocytopenia
- ESR:
 - > 30 mm/hr

- Most common lab abnormality
- Electrolytes, BUN, creatinine, glucose
- Liver function tests:
 - Elevated liver enzymes (γ -glutamyl transferase most common)
- Culture:
 - Low yield
 - Not indicated
- CSF:
 - Pleocytosis
 - Elevated protein
 - Obtain CSF spirochete antibodies.
- Special tests:
 - Serology:
 - Obtain ELISA, immunofluorescence assay, and western blot when disease is suggested without ECM lesion.
 - Antibodies may persist for months to years.
 - Positive serology or previous Lyme disease does not ensure protective immunity.
 - Polymerase chain reaction assay:
 - Highly specific and sensitive
 - Not available for routine use
 - Joint fluid:
 - Cryoglobulin increased 5-fold compared with serum
 - Joint films may show soft tissue, cartilaginous, osseous changes.

DIFFERENTIAL DIAGNOSIS

- Other tick-borne illnesses:
 - Deer tick usually larger (1 cm) than Ixodid ticks (1–2 mm)
 - Rocky Mountain spotted fever
 - Tularemia
 - Relapsing fever
 - Colorado tick fever
 - Tick-bite paralysis
- Rheumatic fever:
 - Rash of erythema marginatum
 - Temporomandibular joint arthritis more common than in Lyme disease
 - Valvular involvement rather than heart block
 - Chorea may be isolated finding.
- Viral meningitis
 - Syphilis
 - Septic arthritis
 - Parvovirus B19 infection—polyarticular arthritis
 - Infectious endocarditis

- Juvenile rheumatoid arthritis
- Reiter syndrome
- Brown recluse spider bite
- Fibromyalgia
- Chronic fatigue syndrome



TREATMENT

INITIAL STABILIZATION/THERAPY

- 20 mL/kg of 0.9% NS IV fluid bolus for dehydration
- IV access for neurologic and cardiac involvement
- Cardiac monitoring
- Temporary pacemaker for heart block

ED TREATMENT/PROCEDURES

- Remove tick:
 - Disinfect site.
 - With blunt instrument, grasp tick close to skin and pull upward with gentle pressure.
- Medications:
 - Aspirin as adjunctive therapy for cardiac involvement
 - NSAIDs for arthritis or arthralgias
- Vaccine (Lymerix) for prevention of disease:
 - A recombinant surface protein
 - For persons in high/moderate risk areas
 - For travelers to endemic areas
 - 3 doses (0–1 mo–2 mo)
- Stage I:
 - Amoxicillin, doxycycline (for those ≥ 8 yr of age), or cefuroxime (21 days)
 - Azithromycin (14–21 days)
 - Parenteral therapy in pregnant patients
- Stage II:
 - Oral therapy for isolated Bell palsy and mild involvement:
 - Amoxicillin with probenecid (30 days) or doxycycline (avoid if pregnant or ≥ 8 yr old; 10–21 days)
 - Parenteral therapy for more severe involvement (meningitis, carditis, severe arthritis):
 - Ceftriaxone, cefotaxime (14–21 days), or penicillin G (14–28 days)
- Stage III:
 - Parenteral therapy:
 - Penicillin G, cefotaxime (14–21 days), or ceftriaxone (14–28 days)

MEDICATION

First Line

- Amoxicillin: 500 mg (peds: 50 mg/kg/24 h) PO TID for those <8 yr of age or unable to tolerate doxycycline.
- Aspirin: 80–100 mg/kg/d (peds: 50–100 mg/kg/d in 6 div. doses) PO; do not exceed 4 g/24 h (peds: Do not exceed 120 mg/kg/24 h or 4 g/24 h)
- Doxycycline: 100 mg PO BID for 14–21 days for children ≥8 yr and adults (except if pregnant)
- Ceftriaxone: 2 g (peds: 100 mg/kg/24 h) IV daily (1st line for late-term disease)

Second Line

- Azithromycin: 500 mg PO daily
- Cefuroxime axetil, 500 mg BID (all ages)
- Cefotaxime: 2 g (peds: 100–150 mg/kg/24 h) IV q8h
- Penicillin G: 20–24 million U IV q4–6h
- Probenecid: 500 mg PO TID



FOLLOW-UP

DISPOSITION

Admission Criteria

- Meningoencephalitis
- Telemetry/ICU admission for carditis

Discharge Criteria

Patients treated with oral therapy

PEARLS AND PITFALLS

- Duration of treatment for later organ involvement will be ≥ 30 days.
- Be aware of coinfections with *Anaplasmosis* and *Babesiosis*.

ADDITIONAL READING

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- Marques AR. Lyme disease: A review. *Curr Allergy Asthma Rep*. 2010;10:13–20.

- Steere AC, Coburn J, Glickstein L. The emergence of Lyme disease. *J Clin Invest.* 2004;113(8):1093–1101.



CODES

ICD9

- 088.81 Lyme Disease
- 320.7 Meningitis in other bacterial diseases classified elsewhere
- 711.80 Arthropathy associated with other infectious and parasitic diseases, site unspecified

ICD10

- A69.20 Lyme disease, unspecified
- A69.21 Meningitis due to Lyme disease
- A69.23 Arthritis due to Lyme disease

LYMPHADENITIS

John Mahoney • Dolores Gonthier

BASICS

DESCRIPTION

- Lymph nodes may be swollen and tender as part of the systemic response to infection:
 - Become engorged with lymphocytes and macrophages
 - May be primarily infected
 - Infection in distal extremity may result in painful tender adenopathy proximally
- Acute suppurative lymphadenitis may occur after pharyngeal or skin infection

ETIOLOGY

- Most frequently caused by bacterial infection
- Most common organisms in pyogenic lymphadenitis:
 - *Staphylococcus aureus*—including resistant strains such as community-associated methicillin-resistant *S. aureus* (CA-MRSA):
 - CA-MRSA risk factors include prior MRSA infection, household contact of CA-MRSA patient, military personnel, incarcerated persons, athletes in contact sports, IV drug users, men who have sex with men
 - Different antibiotic susceptibility than nosocomial MRSA
 - CA-MRSA now sufficiently prevalent to warrant coverage in empiric treatment
 - Suspect CA-MRSA in unresponsive infections
 - Group A β -hemolytic *Streptococcus*
- Cervical lymphadenitis:
 - Usually pharyngeal or periodontal process
 - Streptococcus and anaerobes
- Axillary lymphadenitis:
 - *Streptococcus pyogenes* (group A β -hemolytic *Streptococcus*)
- Nosocomial MRSA:
 - Risk factors: Recent hospital or long-term care admission, surgery, injection drug use, vascular catheter, dialysis, recent antibiotic use, unresponsive infection
 - Resistant to most antibiotics (see “Treatment”)

Pediatric Considerations

- Acute unilateral cervical suppurative lymphadenitis:
 - Most common at age <6 yr

- Group A *Streptococcus*, *S. aureus*, and anaerobes are most common causes



DIAGNOSIS

SIGNS AND SYMPTOMS

- Painful swelling, inflammation/infection of lymph nodes
- Commonly presents simultaneously with acute cellulitis or abscess if pyogenic cause
- Axillary lymphadenitis:
 - Fever, axillary pain, and acute lymphedema of arms and chest, without features of cellulitis or lymphangitis; ipsilateral pleural effusion may be present

History

- Occupation
- Exposure to pets
- Sexual behavior
- Drug use
- Travel history
- Associated symptoms:
 - Sore throat
 - Cough
 - Fever
 - Night sweats
 - Fatigue
 - Weight loss
 - Pain in nodes
- Duration of lymphadenopathy

Physical-Exam

- Extent of lymphadenopathy (localized or generalized)
- Size of nodes:
 - Abnormal size by site:
 - General: > 1 cm
 - Epitrochlear: > 0.5 cm
 - Inguinal: > 1.5 cm
- Presence or absence of nodal tenderness
- Signs of inflammation over node
- Skin lesions
- Splenomegaly
- Enlargement of supraclavicular or scalene nodes is always abnormal

ESSENTIAL WORKUP

- Acute regional lymphadenitis is clinical diagnosis, often part of larger syndrome (cellulitis)
- History and physical exam to reveal infectious source

DIAGNOSIS TESTS & NTERPRETATION

Lab

- WBC is not essential:
 - Possible leukocytosis with left shift or normal
- CBC, Epstein–Barr virus (EBV), cytomegalovirus (CMV), HIV, and other serologies based on clinical findings

Imaging

US or CT in patients who do not improve or progress to suppuration

Diagnostic Procedures/Surgery

Consider percutaneous needle aspiration or surgical drainage in patients who do not improve or progress to suppuration

DIFFERENTIAL DIAGNOSIS

- Common infections:
 - Adenovirus
 - Scarlet fever
 - Cat scratch disease
 - Fungal
 - Herpes zoster
- Unusual infections:
 - Sporotrichosis (rose thorns)
 - Diphtheria
 - West Nile fever
 - Plague
 - Anthrax
 - Typhoid
 - Rubella
- Venereal infections:
 - Syphilis
 - Genital herpes
 - Chancroid
 - Lymphogranuloma venereum
- Other systematic infections causing generalized lymphadenitis:
 - HIV
 - Infectious mononucleosis (EBV or CMV)

- Toxoplasmosis
- Tuberculosis
- Infectious hepatitis
- Dengue
- Drug reaction:
 - Phenytoin
 - Allopurinol
- Silicone implants
- Malignancy
- Rheumatologic disorders
- Systemic lupus erythematosus
- Sarcoidosis
- Amyloidosis
- Serum sickness

Pediatric Considerations

- Kawasaki disease
- PFAPA syndrome (periodic fever, aphthous stomatitis, pharyngitis, cervical adenitis)



TREATMENT

INITIAL STABILIZATION/THERAPY

Ensure airway, breathing, and circulation management and hemodynamic stability

ED TREATMENT/PROCEDURES

- General principles:
 - Antibiotics based on involved primary organ/suspected pathogen (see also “Cellulitis”)
 - Consider local prevalence of MRSA and other resistant pathogens in addition to usual causes
 - Usual outpatient treatment: 7–10 days
 - Elevation
 - Application of moist heat
 - Analgesics
- Drainage of abscesses if present:
 - Obtain culture if drainage performed, especially to help identify resistant pathogens
- Skin origin:
 - Outpatient:
 - Oral cephalexin plus trimethoprim/sulfamethoxazole (TMP/SMX) (to cover CA-MRSA)

- Alternatives to cephalexin: Oral dicloxacillin, macrolide, or levofloxacin
- Alternatives to TMP/SMX: Clindamycin or doxycycline
- Inpatient:
 - IV nafcillin or equivalent, plus IV vancomycin (to cover CA-MRSA)
- Pharyngeal or periodontal origin:
 - Outpatient:
 - Oral penicillin VK
 - Alternatives: Oral clindamycin or amoxicillin/clavulanate
 - Inpatient:
 - IV penicillin G (aqueous) and IV metronidazole
 - Alternatives: IV ampicillin/sulbactam or IV clindamycin
- Axillary lymphadenitis:
 - Outpatient:
 - Oral penicillin VK
 - Alternatives: Oral macrolide or amoxicillin/clavulanate
 - Inpatient:
 - IV penicillin G (aqueous)
 - Alternatives: IV ampicillin/sulbactam
- Acute unilateral cervical suppurative lymphadenitis:
 - Outpatient:
 - Oral penicillin VK
 - Alternatives: Oral clindamycin or amoxicillin/clavulanate
- MRSA:
 - Nosocomial MRSA:
 - IV vancomycin or PO or IV linezolid
 - CA-MRSA:
 - PO: TMP/SMX, clindamycin or doxycycline
 - IV: Vancomycin or clindamycin

MEDICATION

- Amoxicillin/clavulanate: 500–875 mg (peds: 45 mg/kg/24 h) PO BID or 250–500 mg (peds: 40 mg/kg/24 h) PO TID
- Ampicillin/sulbactam: 1.5–3 g (peds: 100–300 mg/kg/24 h up to 40 kg; > 40 kg, give adult dose) IV q6h
- Cephalexin: 500 mg (peds: 50–100 mg/kg/24 h) PO QID
- Clindamycin: 450–900 mg (peds: 20–40 mg/kg/24 h) PO or IV q6h
- Dicloxacillin: 125–500 mg (peds: 12.5–25 mg/kg/24 h) PO q6h
- Doxycycline: 100 mg PO BID for adults
- Erythromycin base: (adult) 250–500 mg PO QID
- Linezolid: 600 mg PO or IV q12h (peds: 30 mg/kg/d divided q8h)
- Metronidazole: (adult) 15 mg/kg IV once, followed by 7.5 mg/kg IV q6h
- Nafcillin: 1–2 g IV q4h (peds: 50–100 mg/kg/24 h divided q6h); max. 12 g/24 h

- Penicillin VK: 250–500 mg (peds: 25–50 mg/kg/24 h) PO q6h
- Penicillin G (aqueous): 4 mIU (peds: 100,000–400,000 U/kg/24 h) IV q4h
- Rifampin: 600 mg PO BID for adults
- TMP/SMX: 2 DS tabs PO q12h (peds: 6–10 mg/kg/24 h TMP divided q12h)
- Vancomycin: 1 g IV q12h (peds: 10 mg/kg IV q6h, dosing adjustments required age <5 yr); check serum levels



FOLLOW-UP

DISPOSITION

Admission Criteria

- Toxic appearing
- History of immune suppression
- Concurrent chronic medical illnesses
- Unable to take oral medications
- Unreliable patients

Discharge Criteria

- Mild infection in a nontoxic-appearing patient
- Able to take oral antibiotics
- No history of immune suppression or concurrent medical problems
- Has adequate follow-up within 24–48 hr

Issues for Referral

- If not found in context of acute infection and not quick to resolve with course of antibiotics, evaluate for more serious underlying causes (e.g., malignancy)
- Lymph node biopsy may be helpful in the following circumstances:
 - Clinical findings indicate likely malignancy
 - Lymph node size > 1 cm
 - Supraclavicular location

FOLLOW-UP RECOMMENDATIONS

- Follow-up within 24–48 hr for response to treatment
- If symptoms worsen—including new or worsening lymphangitis, new or increasing area of redness over the node, worsening fever—patient should be instructed to return sooner

PEARLS AND PITFALLS

- Staph species are the most common cause of acute regional lymphadenitis due to pyogenic bacteria

- Empiric antibiotic coverage must extend to include CA-MRSA, in addition to coverage for other staph species and strep

ADDITIONAL READING

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- Thomas KT, Feder HM Jr, Lawton AR, et al. Periodic fever syndrome in children. *JPediatr.* 1999;135:15–21.



See Also (Topic, Algorithm, Electronic Media Element)

- Cellulitis
- Lymphangitis
- MRSA



CODES

ICD9

- 289.1 Chronic lymphadenitis
- 289.3 Lymphadenitis, unspecified, except mesenteric
- 683 Acute lymphadenitis

ICD10

- I88.9 Nonspecific lymphadenitis, unspecified
- L04.0 Acute lymphadenitis of face, head and neck
- L04.2 Acute lymphadenitis of upper limb

LYMPHANGITIS

John Mahoney



BASICS

DESCRIPTION

- Lymphangitis is the infection of lymphatics that drain a focus of inflammation
- Histologically, lymphatic vessels are dilated and filled with lymphocytes and histiocytes:
 - Inflammation frequently extends into perilymphatic tissues and may lead to cellulitis or abscess formation

ETIOLOGY

- Acute lymphangitis:
 - Likely caused by bacterial infection
 - Most commonly group A β -hemolytic *Streptococcus*
 - Less commonly due to other strep groups, and occasionally *Staphylococcus aureus*, including resistant strains such as community-associated methicillin-resistant *S. aureus* (CA-MRSA):
 - CA-MRSA risk factors: Prior MRSA infection, household contact of CA-MRSA patient, military personnel, incarcerated persons, athletes in contact sports, IV drug users, men who have sex with men
 - Different antibiotic susceptibility than nosocomial MRSA
 - CA-MRSA now sufficiently prevalent to warrant empiric treatment
 - Suspect CA-MRSA in unresponsive infections or if multiple or recurrent abscesses
 - Other organisms:
 - *Pasteurella multocida* (cat or dog bite)
 - *Spirillum minus* (rat-bite fever)
 - *Wuchereria bancrofti* (filariasis): Consider in immigrants from Africa, Southeast Asia/Pacific, and tropical South America with lower-extremity involvement
- Chronic lymphangitis:
 - Usually caused by mycotic, mycobacterial, and filarial infections
 - *Sporothrix schenckii* (most common cause of chronic lymphangitis in US):
 - Inoculation occurs while gardening or farming (rose thorn)
 - Organism is present on some plants and in sphagnum moss
 - Multiple SC nodules appear along course of lymphatic vessels
 - Typical antibiotics and local treatment fail to cure lesion
 - *Mycobacterium marinum*:
 - Atypical Mycobacterium

- Grows optimally at 25–32°C in fish tanks and swimming pools
- May produce a chronic nodular, single wart-like or ulcerative lesion at site of abrasion
- Additional lesions may appear in distribution similar to sporotrichosis
- *Nocardia brasiliensis*
- *Mycobacterium kansasii*
- *W. bancrofti*

DIAGNOSIS

SIGNS AND SYMPTOMS

- Acute lymphangitis:
 - Warm, tender erythematous streaks develop and extend proximally from the source of infection
 - Regional lymph nodes often become enlarged and tender (lymphadenitis).
 - Peripheral edema of involved extremity
 - Systemic manifestations:
 - Fever
 - Rigors
 - Tachycardia
 - Headache
- Chronic (nodular) lymphangitis:
 - Erythematous nodule, chancreiform ulcer, or wart-like lesion develops in SC tissue at inoculation site
 - Often presents without pain or evidence of systemic infection
 - Multiple lesions possible along lymphatic chain

History

History and physical exam directed at discovering source of infection

Physical-Exam

- Fever
- Erythematous streaks from source of infection proceeding toward regional lymph nodes

ESSENTIAL WORKUP

Lymphangitis is a clinical diagnosis

DIAGNOSIS TESTS & NTERPRETATION

Lab

- WBC is unnecessary but often elevated

- Gram stain and culture of initial lesion to focus antimicrobial selection and reveal resistant pathogens (MRSA):
 - Aspirate point of maximal inflammation or punch biopsy
 - Essential if treatment failure
- If sporotrichosis or *M. marinum* infection is suspected, diagnosis should be confirmed by culture of organism from wound
- Blood culture may reveal organism

Imaging

- Imaging is not commonly performed
- Plain radiographs may reveal abscess formation, SC gas, or foreign bodies if these are suspected
- Extremity vascular imaging (doppler US) can help rule out deep venous thrombosis

DIFFERENTIAL DIAGNOSIS

- Thrombophlebitis; deep venous and superficial:
 - Differentiation from lymphangitis:
 - Absence of initial traumatic or infectious focus
 - No regional lymphadenopathy
- IV line infiltration
- Smallpox vaccination, normal variant of usual reaction to vaccination
- Phytophotodermatitis:
 - Linear inflammatory reaction, mimics lymphangitis
 - Lime rind, lime juice, and certain plants can act as photosensitizing agents



TREATMENT

INITIAL STABILIZATION/THERAPY

If patient is septic, manage airway and resuscitate as indicated

ED TREATMENT/PROCEDURES

- Antimicrobial therapy should be initiated with first dose in ED
- General principles:
 - Consider local prevalence of MRSA and other resistant pathogens in addition to usual causes
 - Usual outpatient treatment: 7–10 days
 - Elevation
 - Application of moist heat
- Acute lymphangitis, empiric coverage:
 - Outpatient:
 - Oral cephalexin plus trimethoprim/sulfamethoxazole (TMP/SMX) (to cover CA-MRSA)

- Alternatives to cephalexin: Oral dicloxacillin, macrolide, or levofloxacin
- Alternatives to TMP/SMX: Clindamycin or doxycycline
- Inpatient: IV nafcillin or equivalent
- Lymphangitis after dog or cat bite: IV ampicillin/sulbactam
- MRSA:
 - _ Nosocomial MRSA: IV vancomycin or PO or IV linezolid
 - _ CA-MRSA:
 - PO: TMP/SMX, clindamycin, or doxycycline
 - IV: Vancomycin or clindamycin
- Sporotrichosis:
 - _ Itraconazole or saturated solution of potassium iodide (SSKI)
- *M. marinum*:
 - _ Localized granulomas are usually excised
 - _ Antimicrobial therapy is usually reserved for more severe infections:
 - Limited data on what combination of agents should be used
 - Rifampin and ethambutol may be best choice

MEDICATION

- Ampicillin/sulbactam: 1.5–3 g (peds: 100–300 mg/kg/24 h up to 40 kg; > 40 kg, give adult dose) IV q6h
- Cephalexin: 500 mg (peds: 50–100 mg/kg/24 h) PO QID
- Clindamycin: 450–900 mg (peds: 20–40 mg/kg/24h) PO or IV q6h
- Dicloxacillin: 125–500 mg (peds: 12.5–25 mg/kg/24h) PO q6h
- Doxycycline: 100 mg PO BID for adults
- Erythromycin base: (Adult) 250–500 mg PO QID
- Itraconazole (adult): 200 mg PO daily, continue until 2–4 wk after all lesions resolve (usually 3–6 mo); peds: Not approved for use
- Levofloxacin: (Adult only) 500–750 mg PO or IV daily
- Linezolid: 600 mg PO or IV q12h (peds: 30 mg/kg/24 h div. q8h)
- Nafcillin: 1–2 g IV q4h (peds: 50–100 mg/kg/24 h div. q6h); max. 12 g/24 h
- Rifampin: 600 mg PO BID for adults
- TMP/SMX: 2 DS tabs PO q12h (peds: 6–10 mg/kg/24 h TMP div. q12h)
- Vancomycin: 1 g IV q12h (peds: 10 mg/kg IV q6h, dosing adjustments required for age <5 yr); check serum levels



FOLLOW-UP

DISPOSITION

Admission Criteria

- Toxic appearing

- History of immune suppression
- Concurrent chronic medical illnesses
- Unable to take oral medications
- Unreliable patients

Discharge Criteria

- Mild infection in a nontoxic-appearing patient
- Able to take oral antibiotics
- No history of immune suppression or concurrent medical problems
- Adequate follow-up within 24–48 hr

FOLLOW-UP RECOMMENDATIONS

- Follow-up within 24–48 hr
- Sooner if worsening symptoms, including worsening fever or other systemic symptoms
- Outline the border of erythema before discharge to aid in assessing response to therapy

PEARLS AND PITFALLS

Empiric antibiotic coverage must extend to include CA-MRSA, in addition to coverage for other staph species and strep.

ADDITIONAL READING

- Pasternack MS, Swartz MN. Lymphadenitis and lymphangitis. In: Mandell GL, Bennett JE, Dolin R, eds. *Mandell, Douglas and Bennett's Principles and Practice of Infectious Diseases*. 7th ed. New York, NY: Elsevier/Churchill Livingstone; 2010:1323–1334.
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- Smego RA, Castiglia M, Asperilla MO. Lymphocutaneous syndrome: A review of nonsporothrix causes. *Medicine (Baltimore)*. 1999;78:38–63.



See Also (Topic, Algorithm, Electronic Media Element)

- Cellulitis
- Lymphadenitis
- MRSA



CODES

ICD9

- 041.12 Methicillin resistant Staphylococcus aureus in conditions classified elsewhere and of unspecified site
- 457.2 Lymphangitis
- 682.9 Cellulitis and abscess of unspecified sites

ICD10

- A49.02 Methicillin resis staph infection, unsp site
- I89.1 Lymphangitis
- L03.91 Acute lymphangitis, unspecified

LYMPHOGRANULOMA VENEREUM

Joel Kravitz

BASICS

DESCRIPTION

- Sexually transmitted disease
- Primary stage:
 - Painless papule, pustule, or ulcer
- Secondary stage:
 - Spread to regional lymph nodes
 - Fluctuant inguinal lymphadenopathy (buboes)
 - Lymphadenopathy may be unilateral or bilateral
- Responsive to antibacterial therapy
- Tertiary stage:
 - If untreated, significant tissue damage and destruction may result
- Endemic in Southeast Asia, Latin America, parts of Africa, and the Caribbean
- Increasing incidence among men who have sex with men
- Also known as:
 - Struma
 - Tropical bubo
 - Nicolas–Favre–Durand disease

ETIOLOGY

Chlamydia trachomatis serotypes L1, L2, and L3

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Primary genital lesions:
 - Incubation: 3–30 days after sexual exposure to *C. trachomatis*
 - Painless genital chancre lasts 2–3 days (rarely, a papule or vesicle)
 - Often transient and not noticed
 - May present as proctitis
- Secondary stage:
 - Systemic symptoms:
 - Fever and malaise
 - Myalgias
 - Lymphadenopathy; usually inguinal:

- May ulcerate and drain pus
- Proctitis:
 - Rectal bleeding
 - Tenesmus
 - Constipation
- Tertiary stage:
 - Symptoms mimic inflammatory bowel disease or proctocolitis
 - Elephantiasis
 - Strictures

Physical-Exam

- Primary stage:
 - Painless papule, pustule, or ulcer
 - Usually anogenital region
- Secondary stage:
 - Tender inguinal adenopathy:
 - Occurs 1–3 wk after initial inoculation
 - Adenopathy is unilateral in 2/3 of cases
 - Bubo (large inguinal lymph nodes) form in inguinal and femoral chains
 - Groove sign: Scarred or coalescent buboes above and below inguinal ligament give a linear depression parallel to the inguinal ligament (seen in 30%)
 - Anal-receptive patients may develop hemorrhagic proctocolitis
 - Perirectal lymphatic inflammation causes fistulae and strictures
- Tertiary disease (invasive if untreated):
 - Chronic proctocolitis:
 - Abdominal pain
 - Rectal bleeding
 - Genital strictures
 - Perineal and perianal fistulae
 - Elephantiasis of the ipsilateral leg

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Standard *Chlamydia* DNA probes *do not* test for lymphogranuloma venereum (LGV) strain
- False-positive VDRL in 20%
- Serologic testing and culture are the standard
- Complement fixation titers > 1:64 are consistent with LGV infection

Diagnostic Procedures/Surgery

Bubo aspiration—specific but expensive and impractical

DIFFERENTIAL DIAGNOSIS

- Genital herpes (ulcers usually not seen in LGV)
- Syphilis—nodes nontender, longer incubation
- Chancroid—multiple ulcers, no systemic symptoms
- Granuloma inguinale—lesions painless and bleed easily



TREATMENT

PRE HOSPITAL

No pre-hospital issues

INITIAL STABILIZATION/THERAPY

No field or ED stabilization required

ED TREATMENT/PROCEDURES

If large, buboes may need to be aspirated or drained to avoid or minimize scarring

MEDICATION

First Line

Doxycycline: 100 mg PO BID for 3 wk

Second Line

- Azithromycin: 1,000 mg PO weekly for 3 wk
- Erythromycin: 500 mg PO QID for 3 wk

Pregnancy Considerations

Erythromycin is the recommended regimen in pregnancy and during lactation



FOLLOW-UP

DISPOSITION

Admission Criteria

Hospitalization is rarely needed (i.e., severe systemic symptoms)

Discharge Criteria

Immunocompetent patient without systemic involvement

Issues for Referral

- Outpatient follow-up is required to confirm diagnosis and cure
- Rectal infection may require retreatment

FOLLOW-UP RECOMMENDATIONS

- Ensure that sexual partners are tested and treated
- Sexual contacts within 60 days should be tested and treated with antichlamydial therapy

PEARLS AND PITFALLS

- Diagnosis is based on clinical suspicion, epidemiologic patterns, and exclusion of other etiologies
- Consider this diagnosis in men who have sex with men
- Treat to avoid tertiary disease which is not responsive to antibiotic therapy alone
- Treatment course is at least 3 wk of antibiotics

ADDITIONAL READING

- Centers for Disease Control and Prevention: 2002 guidelines for treatment of sexually transmitted diseases. Available at: <http://www.cdc.gov/std/treatment/2010/STD-Treatment-2010-RR5912.pdf#page=28>.
- McLean CA, Stoner BP, Workowski KA. Treatment of lymphogranuloma venereum. *Clin Infect Dis*. 2007;44:S147–S152.
- White JA. Manifestations and management of lymphogranuloma venereum. *Curr Opin Infect Dis*. 2009;22:57–66.
- White J, Ison C. Lymphogranuloma venereum: What does the clinician need to know? *Clin Med*. 2008;8:327–330.



CODES

ICD9

099.1 Lymphogranuloma venereum

ICD10

A55 Chlamydial lymphogranuloma (venereum)

MALARIA

Jordan Moskoff

BASICS

DESCRIPTION

- Protozoan infection transmitted through the *Anopheles* mosquito
- Incubation period 8–16 days
- Periodicity of the disease is due to the life cycle of the protozoan:
 - Exoerythrocytic phase: Immature sporozoites migrate to liver, where they rapidly multiply into mature parasites (merozoites).
 - Erythrocytic phase: Mature parasites are released into circulation and invade RBCs.
 - Replication within RBCs followed 48–72 hr later by RBC lysis and release of merozoites into circulation, repeating cycle
 - Fever corresponds to RBC lysis.
- *Plasmodium falciparum*:
 - Cause of most cases and almost all deaths
 - Usually presents as an acute, overwhelming infection
 - Able to infect red cells of all ages:
 - Results in greater degree of hemolysis and anemia
 - Causes widespread capillary obstruction:
 - Results in end-organ hypoxia and dysfunction
 - More moderate infection in people who are on or who have recently stopped prophylaxis with an agent to which the *P. falciparum* is resistant
 - Post-traumatic immunosuppression may cause relapse of malaria in patients who have lived in endemic areas.
- *Plasmodium vivax* and *Plasmodium ovale*:
 - May present with an acute febrile illness
 - Dormant liver stages (hypnozoites) that may cause relapse 6–11 mo after initial infection
- *Plasmodium malariae*:
 - May persist in the bloodstream at low levels up to 30 yr

ETIOLOGY

- Transmission usually occurs from the bite of infected female *Anopheles* mosquito.
- North American transmission possible:
 - *Anopheles* mosquitoes on east and west coasts of US.
 - Transmission may also occur through infected blood products and shared needles.

Pediatric Considerations

- Sickle cell trait protective
- Cerebral malaria more common in children
- In highly endemic areas with minimal lab capability, all children presenting with febrile illness may be treated.

Pregnancy Considerations

Pregnant patients, especially primigravida, at higher risk

DIAGNOSIS

SIGNS AND SYMPTOMS

- **Timing:**
 - _ P. falciparum—exhibits within 8 wk of return
 - _ P. vivax—delayed several months
 - _ Most symptomatic within 1 yr
- **General:**
 - _ Malaise
 - _ Chills
 - _ Fever—usually $> 38^{\circ}\text{C}$
 - _ Classic malaria paroxysm:
 - 15 min to 1 hr of chills
 - Followed by 2–6 hr of nondiaphoretic fever $\leq 39\text{--}42^{\circ}\text{C}$
 - Profuse diaphoresis followed by defervescence
 - Pattern every 48 hr (P. vivax and P. ovale) or every 72 hr (P. falciparum)
 - Fever pattern may be varied; rare to have classical fever.
 - _ Orthostatic hypotension
 - _ Myalgias/arthralgias
 - _ Hematology
 - _ Hemolysis:
 - Blackwater fever; named from the dark color of the urine partially due to hemolysis in overwhelming P. falciparum infections
 - _ Jaundice
 - _ Splenomegaly:
 - More common in chronic infections
 - May cause splenic rupture
- **CNS—cerebral malaria:**
 - _ Headache
 - _ Focal neurologic findings
 - _ Mental status changes
 - _ Coma

- Seizures
- GI:
 - Emesis
 - Diarrhea
 - Abdominal pain
- Pulmonary:
 - Shortness of breath
 - Rales
 - Pulmonary edema
- Severe malaria:
 - One or more of the following:
 - >20% mortality even with optimal management
 - Prostration; unable to sit up by oneself
 - Impaired consciousness
 - Respiratory distress or pulmonary edema
 - Seizure
 - Circulatory collapse
 - Abnormal bleeding
 - Jaundice
 - Hemoglobinuria
 - Severe anemia

ESSENTIAL WORKUP

Oil emersion light microscopy of a thick-smear Giemsa stain:

- Demonstrates intraerythrocytic malaria parasites
- Cannot exclude diagnosis without three negative smears in 48 hr
- Only high degrees of parasitemia will be evident on a standard CBC smear.

DIAGNOSIS TESTS & NTERPRETATION

Lab

- CBC:
 - Anemia—25%
 - Thrombocytopenia—70% have < 150
 - Leukocytopenia
- Electrolytes, BUN, creatinine, glucose:
 - Renal failure
 - Hypoglycemia (rare)
 - Lactic acidosis
 - Hyponatremia
- Urinalysis
- Liver function tests:
 - Increased in 25%

- Increased bilirubin and lactate dehydrogenase are the signs of hemolysis

Imaging

Chest radiograph—for pulmonary edema

Diagnostic Procedures/Surgery

- Immunofluorescence assay, enzyme-linked immunosorbent assay, or DNA probes:
 - Differentiates the type of Plasmodium present
 - 5–7% will have mixed infections.
- Lumbar puncture/ CSF analysis:
 - Performed to distinguish cerebral malaria from meningitis
 - CSF lactate/protein elevated with malaria
 - CSF pleocytosis/hypoglycemia absent with malaria

DIFFERENTIAL DIAGNOSIS

- Meningitis
- Encephalitis
- Stroke
- Acute renal failure
- Acute hemolytic anemia
- Sepsis
- Hepatitis
- Viral diarrheal illness
- Hypoglycemic coma
- Heat stroke



TREATMENT

INITIAL STABILIZATION/THERAPY

- ABCs
- 0.9% NS fluid bolus for hypotension
- Immediate cooling if temperature $>40^{\circ}\text{C}$
- Acetaminophen
- Mist/cool-air fans
- Naloxone, D₅₀W (or Accu-Chek), and thiamine if altered mental status

ED TREATMENT/PROCEDURES

- Dependent on considering this diagnosis and identifying the type of malaria present and geographic area of acquisition
- Assume drug resistant until proven otherwise.
- To counter resistance Artemisinin combinations of antimalarials are recommended 1st line.

- Artemisinin-based combination therapy – choice is based on geographic region, check WHO database
 - Artemether + Lumefantrine
 - Artesunate + Amodiaquine
 - Artesunate + Mefloquine
 - Artesunate + Sulfadoxine–Pyrimethamine
- Severe falciparum—IV treatment:
 - Artesunate can be given IV or IM
 - Artemisinin can be given rectally
- Supportive therapy for complications
- Chemoprophylaxis: Must be based on region of travel, check WHO database
 - Malarone
 - Daily medication
 - Very well tolerated
 - Safe in children > 5 kg – pediatric dosing
 - Unsafe in pregnancy
 - 250/100 mg PO daily
 - Begin 1–2 days prior to entering malaria area and continue for 7 days after leaving area
 - Chloroquine:
 - Drug of choice for travelers who want weekly medication
 - Safe in pregnancy
 - 300 mg PO weekly
 - Begin 2 wk prior to departure and continue for 4 wk after return
 - Mefloquine:
 - Weekly medication
 - Safe in pregnancy; do not use with certain psychiatric conditions
 - 250 mg PO weekly
 - Begin 2 wk before departure and continue for 4 wk after return
 - Doxycycline:
 - Daily medication
 - Least expensive
 - Unsafe in pregnancy
 - Unsafe in children < 8 y/o
 - Risk with sun exposure
 - 100 mg PO daily
 - Begin 1 day prior to entering area and continue for 4 wk after return
 - Primaquine:
 - Daily medication
 - Cannot use in G6PD deficiency
 - Unsafe in pregnancy
 - 30 mg PO every day
 - Begin 1 day prior to entering area and continue 1 wk after return

- Vaccine is not available, but several are in field trials.

MEDICATION

- Acetaminophen: 500 mg (peds: 10–15 mg/kg) PO q4–6h; do not exceed 5 doses/24 h; max. 4 g/24 h
- Artemether (20 mg)–lumefantrine (120 mg): 6 dose regimen PO BID × 3 days
- Artesunate (50 mg) + Amodiaquine (153 mg): 3 dose regimen PO QD × 3 days
- Artesunate (50 mg) + Sulfadoxine
- Pyrimethamine (500/25): 3 dose regimen 1 tabs of Artesunate PO QD × 3 and 1 tab
- Sulfadoxine–Pyrimethamine PO QD × 1 day
- Artesunate (50 mg) + Mefloquine (250 mg): 3 dose regimen 1 tab of Artesunate PO QD × 3 days and Mefloquine PO split over 2–3 days.
- Dextrose: D₅₀W 1 amp—50 mL or 25 g (peds: D₂₅W 2–4 mL/kg) IV
- Naloxone (Narcan): 2 mg (peds: 0.1 mg/kg) IV or IM initial dose
- Thiamine (vitamin B₁): 100 mg (peds: 50 mg) IV or IM



FOLLOW-UP

DISPOSITION

Admission Criteria

- ICU admission for severe *P. falciparum* infection
- Suspected acute *P. falciparum* infection
- Severe dehydration
- Inability to tolerate oral solution/medication
- > 3% of RBC containing parasites

Discharge Criteria

- Non-*P. falciparum* infection
- Able to tolerate oral medications

PEARLS AND PITFALLS

Consider in patients with appropriate exposure/epidemiology and in exposed patients with fever and consistent signs and symptoms.

ADDITIONAL READING

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- Centers for Disease Control and Prevention. Malaria hotline: 770-488-7788.
- Centers for Disease Control and Prevention. Traveler's Health. Available at www.cdc.gov/travel/contentYellowBook.aspx.
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- Garner P, Gelband H, Graves P, et al. Systemic reviews in malaria: Global policies need global reviews. *Infect Dis Clin North Am*. 2009;23:387–404.
- WHO. Guidelines for the Treatment of Malaria. 2006; 266 p.



CODES

ICD9

- 084.0 Falciparum malaria [malignant tertian]
- 084.1 Vivax malaria [benign tertian]
- 084.6 Malaria, unspecified

ICD10

- B50.9 Plasmodium falciparum malaria, unspecified
- B51.9 Plasmodium vivax malaria without complication
- B54 Unspecified malaria

MALLORY-WEISS SYNDROME

Galeta C. Clayton

BASICS

DESCRIPTION

- Partial-thickness intraluminal longitudinal mucosal tear of distal esophagus or proximal stomach
- Sudden increase in intra-abdominal and/or transgastric pressure causes:
 - Mild to moderate submucosal arterial and/or venous bleeding:
 - May be related to underlying pathology
 - “Mushrooming” of stomach into esophagus during retching has been observed endoscopically.
 - Responsible for ~5% of all cases of upper GI bleeding

ETIOLOGY

- Associated with:
 - Forceful coughing, laughing, or retching
 - Lifting
 - Straining
 - Blunt abdominal trauma
 - Seizures
 - Childbirth
 - Cardiopulmonary resuscitation
- Risk factors:
 - Alcoholics:
 - Especially after recent binge
 - Patients with hiatal hernia
 - Hyperemesis gravidarum
- Greater bleeding associated with:
 - Portal hypertension
 - Esophageal varices
 - Coagulopathy

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Multiple bouts of nonbloody vomiting and/or retching followed by hematemesis:
 - Most bleeding is small and resolves spontaneously.

- Massive life-threatening hemorrhage can occur.
- Epigastric pain
- Back pain
- Dehydration:
 - Dizzy, light-headed; syncope

Physical-Exam

- Hematemesis
- Melena
- Postural hypotension
- Shock

ESSENTIAL WORKUP

- CBC
- Rectal exam for occult blood

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Prothrombin time (PT), partial thromboplastin time (PTT), INR
- Electrolytes, BUN, creatinine, glucose, LFTs
- Amylase/lipase if abdominal pain
- Type and cross-match:
 - At least 4 U of packed red blood cells (PRBCs) if bleeding is severe
- ECG if elderly or with cardiac history

Imaging

- Upright chest radiograph for free air from esophageal or gastric perforation
- Upper endoscopy (esophagogastroscopy):
 - Procedure of choice to locate, identify, and treat source of bleeding

DIFFERENTIAL DIAGNOSIS

- Nasopharyngeal bleeding
- Hemoptysis
- Esophageal rupture (Boerhaave syndrome)
- Esophagitis
- Gastritis
- Gastroenteritis
- Duodenitis
- Ulcer disease
- Varices
- Carcinoma
- Vascular-enteric fistula

- Hemangioma



TREATMENT

PRE HOSPITAL

- Airway control:
 - 100% oxygen or intubate if unresponsive or airway patency in jeopardy
- If hemodynamically unstable or massive hemorrhage:
 - Initiate 2 large-bore IV catheters.
 - 1 L bolus (peds: 20 mL/kg) lactated Ringer (LR) solution or 0.9% normal saline (NS)
 - Trendelenburg position

INITIAL STABILIZATION/THERAPY

- ABCs:
 - IV access with at least 1 large-bore catheter; more if unstable
 - Central catheter placement if unstable for more efficient delivery of fluids and monitoring of central venous pressure
 - IV fluids of either 0.9% NS (or LR) at 250 mL/h if stable; wide open if hemodynamically unstable
 - Dopamine for persistent hypotension unresponsive to aggressive fluid resuscitation
- Large-bore Ewald tube placement with evidence of large amount of bleeding:
 - Safe
 - Will not aggravate Mallory–Weiss tear
 - Lavage blood from stomach with water while patient is on side in Trendelenburg position.
- Nasogastric (NG) tube placement to check for active bleeding
- Transfuse O-negative red blood cells immediately if hypotensive and not responsive to 2 L of crystalloid.
- Most bleeding stops spontaneously with conservative therapy.

ED TREATMENT/PROCEDURES

- NPO
- Transfuse PRBCs if unstable or lowering hematocrit with continued hemorrhage.
- Place Foley catheter to monitor urine output.
- Monitor fluid status closely.
- With continuing hemorrhage, arrange for immediate endoscopy:
 - Control bleeding endoscopically via:
 - Electrocoagulation
 - Injection therapy (epinephrine)
 - Band ligation

- Hemoclips
- Application of blood-clotting agents
 - Esophageal balloon tamponade
 - Arterial embolization
- Intravenous vasopressin in massive bleeding and unavailable endoscopy
- In persistent/unresponsive hemorrhage, angiographic infusion of vasopressin
- Surgery—last but definitive treatment modality using techniques to oversew bleeding site or perform gastrectomy
- Failure of above may require gastric arterial embolization in patients of poor surgical risk.
- Antiemetics for nausea/vomiting
- Proton pump inhibitors or H₂ blockers for gastric acid suppression.
- Avoid Sengstaken-Blakemore tubes (especially in presence of hiatal hernia).

MEDICATION

- Dopamine: 2–20 µ/kg/min IV piggyback (IVPB)
- Ondansetron 4 mg IV
- Pantoprazole 20–40 mg IV
- Vasopressin: 0.1–0.5 IU/min IVPB titrating up to 0.9 IU/min as necessary



FOLLOW-UP

DISPOSITION

Admission Criteria

- ICU admission for:
 - Continued or massive hemorrhage
 - Hemodynamic instability
 - Extreme age
 - Poor underlying medical condition
 - Complications
- General floor admission for
 - Stable patients with minimal bleed on presentation that has since cleared
 - Patients with risk factors for rebleeding (portal HTN, coagulopathy)

Discharge Criteria

- History of minimal bleed that has stopped
- Hemodynamically stable
- Normal/stable hematocrit
- Negative or trace heme-positive stool
- Negative or trace gastric aspirate

Issues for Referral

Consult GI in ED if significant upper GI bleeding or if you suspect that requires urgent endoscopy.

FOLLOW-UP RECOMMENDATIONS

GI follow-up for outpatient endoscopy if clinically stable for discharge.

PEARLS AND PITFALLS

- Place 2 large-bore IVs for patients with upper GI bleed.
- For massive GI bleed, initiate blood transfusion early.
- Contact GI early for emergent endoscopy with significant bleeding.
- Active bleeding at the time of initial endoscopy and a low initial hematocrit is associated with a complicated clinical course.
- Rebleeding usually occurs within 24 hr, and is most common in patients with coagulopathies.

ADDITIONAL READING

- Fujisawa N, Inamori M, Sekino Y, et al. Risk factors for mortality in patients with Mallory-Weiss syndrome. *Hepatogastroenterology*. 2011;58:417–420.
- Kim JW, Kim HS, Byun JW, et al. Predictive factors of recurrent bleeding in Mallory-Weiss syndrome. *Korean J Gastroenterol*. 2005;46(6):447–454.
- Takhar SS. Upper gastrointestinal bleeding. In: Wolfson AB, Hendey GW, Ling LJ, et al., eds. *Clinical Practice of Emergency Medicine*. 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2010:548–550.
- Wu JC, Chan FK. Esophageal bleeding disorders. *Curr Opin Gastroenterol*. 2004;20:386–390.



See Also (Topic, Algorithm, Electronic Media Element)

Gastrointestinal Bleeding



CODES

ICD9

530.7 Gastroesophageal laceration-hemorrhage syndrome

ICD10

K22.6 Gastro-esophageal laceration-hemorrhage syndrome

MALROTATION

Moon O. Lee



BASICS

DESCRIPTION

- Incomplete rotation and fixation of intestine during embryogenesis during transition from extracolonic position during week 10 of gestation
- Risk factor:
 - Heterotaxia syndromes
- Associated conditions:
 - Gastrointestinal anomalies:
 - Duodenal stenosis, atresia, web
 - Meckel diverticulum
 - Intussusception
 - Gastroesophageal reflux
 - Omphalocele or gastroschisis
 - Congenital diaphragmatic hernia
 - Abdominal wall defect
 - Hirschsprung disease
 - Metabolic acidosis
 - Congenital cardiac anomalies; present in 27% of patients with malrotation; increases morbidity to 61%

ETIOLOGY

- Duodenojejunal junction remains right of midline
- Cecum remains in the upper left abdomen with abnormal mesenteric attachments
- Volvulus is complication of malrotation when small bowel rotates around superior mesenteric artery and vein resulting in vascular compromise to midgut
- Abnormal anatomy predisposes to obstruction and other conditions
- Usually found in combination with other congenital anomalies (70%): Cardiac, esophageal, urinary, anal
- Epidemiology:
 - 1 in 500 live births
 - High mortality in infants: Up to 24%
 - Necrotic bowel at surgery increases mortality by 25×.
 - Incidence:
 - In neonates, male-to-female ratio 2:1
 - 75% diagnosed newborn period
 - 90% diagnosed by age 1 yr of life
 - Can present during adulthood

SIGNS AND SYMPTOMS

- Neonates:
 - Bilious emesis
 - Abdominal distention
 - Bloody stools
 - Constipation/obstipation
 - Difficulty feeding
 - Poor weight gain
- > 1 yr: Abdominal pain followed by bilious emesis
- Older children and adolescents:
 - Chronic vomiting
 - Intermittent colicky abdominal pain
 - Diarrhea
 - Hematemesis
 - Constipation
 - May not exhibit abnormal physical findings at time of presentation (50–75%)
- Adults: Symptoms vague and nonspecific
- General:
 - Dehydration, acidosis
 - Peritonitis
 - Ischemic bowel
 - Sepsis, shock

History

- Vomiting in infant is the most common sign, but may or may not be bilious
- Signs of small bowel obstruction in early infancy
- Bilious vomiting associated with abdominal pain
- In older children and adults, the most common symptom is abdominal pain
- Other pertinent history—acute or chronic abdominal pain, poor feeding, lethargy, malabsorption, chronic diarrhea

Physical-Exam

- Abdominal exam may show distension from obstruction
- Blood in the stool indicates bowel ischemia
- Evaluate for congenital anomalies

ESSENTIAL WORKUP

Diagnosis is suggested by history and physical exam findings and is delineated by contrast radiography.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC
- Venous blood gas
- Electrolytes, BUN, creatinine, glucose
- Urinalysis/urine culture
- Type and screen
- Prothrombin time, partial thromboplastin time, international normalized ratio
- Lactate

Imaging

- Plain abdominal radiographs:
 - _ Diagnostic in < 30%
 - _ Volvulus likely if accompanied by:
 - Duodenal obstruction
 - Gastric distention with paucity of intraluminal gas distal to volvulus in complete volvulus
 - Generalized distention of small-bowel loops
 - “Double-bubble sign” can be seen on upright film from partial duodenal obstruction causing distension of stomach and duodenum
- Upper GI contrast studies:
 - _ 95% sensitive and 86% accurate
 - _ Findings:
 - Absence of ligament of Treitz or on the right side of the abdomen with misplaced duodenum
 - Dilation of proximal duodenum with termination in conical or beak shape
 - Spiral or corkscrew appearance of duodenum with volvulus
 - Proximal jejunum on right side of abdomen (although readily displaced in neonates)
 - Thickening of small-bowel folds
- Contrast enema:
 - _ Can be useful to determine position of cecum in equivocal cases
 - _ Evaluates position of cecum in midline of upper abdomen or to left of midline
 - _ > 20% false-negative results
- Ultrasound:
 - _ US can be very sensitive in experienced hands
 - _ US shows abnormal relationship between superior mesenteric artery and vein in malrotation
 - _ “Whirlpool” sign on Doppler US of superior mesenteric artery and vein

twisting around the base of mesenteric pedicle seen in volvulus

– Normal ultrasound does not exclude malrotation

- CT:

– Little benefit in infants and children

– More likely to be used for diagnosis in adults

DIFFERENTIAL DIAGNOSIS

- Early life:

– Hirschsprung disease

– Necrotizing enterocolitis

– Intussusception

- Children with acute abdominal pain and peritoneal signs:

– Appendicitis

– Intussusception

– Overwhelming sepsis

- Older children and adults with vague abdominal pain:

– Irritable bowel syndrome

– Peptic ulcer disease

– Biliary and pancreatic disease

– Psychiatric disorders



TREATMENT

ALERT

Midgut volvulus may result in need for rapid volume and electrolyte replacement/resuscitation to correct severe hypovolemia and metabolic acidosis.

PRE HOSPITAL

Rapid transport to ED

INITIAL STABILIZATION/THERAPY

- ABCs
- NS (0.9%) IV fluid bolus (20 mL/kg) for shock, sepsis, or dehydration
- Consider nasogastric tube
- 2 IVs and/or CV catheter
- Initiate broad-spectrum antibiotics for signs of sepsis or peritonitis

ED TREATMENT/PROCEDURES

- Emergent surgical correction
- May require transfer to facility with pediatric surgical expertise when associated with midgut volvulus for:
 - Detorsion of volvulus
 - Restoration of intestinal perfusion

- Resection of obviously necrotic areas
- Replacement of long segments with questionable vascular integrity back into abdominal cavity for return evaluation and possible celiotomy in 36 hr
- Diet:
 - NPO

MEDICATION

- Broad-spectrum antibiotics prior to surgery
- Correct fluid and electrolyte abnormalities
- Vasopressors



FOLLOW-UP

DISPOSITION

Admission Criteria

- Acute abdomen
- Surgical intervention
- Significant dehydration
- Acidosis
- Sepsis
- Shock

Discharge Criteria

Stable, asymptomatic, incidental finding without associated condition, although patients are usually admitted

- Pediatric surgical evaluation prior to discharge

Issues for Referral

Diagnostic evaluation often requires tertiary care pediatric hospital with pediatric surgical and pediatric radiologic expertise.

FOLLOW-UP RECOMMENDATIONS

As dictated by pediatric surgical service

PEARLS AND PITFALLS

- Early recognition of child with acute abdomen
- Prompt treatment of acidosis and shock
- Prompt referral to appropriate facility

ADDITIONAL READING

- Applegate KE. Evidence-based diagnosis of malrotation and volvulus. *Pediatr Radiol*. 2009;39:S161–S163.
- Fleisher GR, Ludwig S, eds. *Textbook of Pediatric Emergency Medicine*. 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2010.
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CODES

ICD9

751.4 Anomalies of intestinal fixation

ICD10

Q43.3 Congenital malformations of intestinal fixation

MANDIBULAR FRACTURES

David W. Munter

BASICS

DESCRIPTION

- Typically due to a direct force
- The most common area fractured is the angle, followed by the condyle, molar, and mental regions.
- Because of its thickness, the mandibular symphysis is rarely fractured.
- Multiple fractures are seen in > 50% of cases owing to the ring-like structure of the mandible.
- Bilateral mandibular fractures most commonly result from motor vehicle accidents (MVAs).
- Open fractures are common, including lacerations of the gum overlying a fracture.

ETIOLOGY

- The mandible is the 3rd most common facial fracture following nasal and zygomatic fractures.
- MVAs, personal violence, contact sports, or industrial accidents
- Patients are often intoxicated and unable to give a clear history of events.
- Facial and head lacerations and facial fractures are the most commonly associated injuries.

Pediatric Considerations

- Mandibular fractures are uncommon in children < 6 yr of age; when they do occur, they are often greenstick fractures and can be managed with soft diet alone.
- Inform parents that because any fracture of the mandible may damage permanent teeth, follow-up with a specialty consultant is advisable.
- Refer pediatric patients to a specialist with experience in children due to issues with growth plates and permanent teeth.

DIAGNOSIS

SIGNS AND SYMPTOMS

- Mandibular pain
- Facial asymmetry, deformity, and dysphagia
- Malocclusion, decreased range of motion of the temporomandibular joint (TMJ), trismus, or a grating sound conducted to the ear
- Gum laceration, subungual or gum hematoma

History

- Mechanism of injury
- Malocclusion, dental pain, associated injuries

Physical-Exam

- Inspect maxillofacial area for deformity, including ecchymosis or swelling.
- Malocclusion, trismus, or facial asymmetry
- Loose, fractured, or missing teeth; gross malalignment of teeth; separation of tooth interspaces, bleeding at the base of teeth; gum lacerations between teeth; and ecchymosis or hematoma of the floor of the mouth
- Step-off, bony disruption, or point tenderness with palpation along the entire length of the mandible
- Protrusion or lateral excursion of the jaw
- Interference with normal mandibular function, including decreased range of motion or deviation of the mandible with opening:
 - The examiner should be able to insert three fingers between the mandible and the maxilla.
 - Inability of the patient to hold a tongue depressor laterally between the teeth when pulled by the examiner, or attempted to be broken by twisting (positive tongue blade test)
- Paresthesia of the lower lip or gums indicates secondary damage to the inferior alveolar nerve.
- Inability to note motion of the mandibular condyles when palpated through the external ear canals suggests mandible fracture.
- Tenderness of the condyle at the TMJ

ESSENTIAL WORKUP

- Diagnosis of mandibular fractures requires radiographs – mandibular series or panorex.
- Panorex superior for evaluation of all of the mandible except condyles
- Low index for obtaining facial bone CT if associated injuries are suspected

DIAGNOSIS TESTS & INTERPRETATION

Lab

Only indicated if immediate operative intervention is indicated, or for evaluation of other injuries

Imaging

- Plain films or dental panoramic views should be obtained.
- Plain films including an anteroposterior (AP), bilateral obliques, and Towne view should be obtained:
 - Mandibular views are best for evaluating the condyles and neck of mandible

(most common site of fracture).

- Dental panoramic view may be obtained:
 - Panorex best evaluates the symphysis and body (less common fracture site).
- If condylar fracture is still suspected and not noted on initial radiographs, obtain CT of the condyles in the coronal plane.
- Missing teeth that cannot be found mandate a chest radiograph to rule out aspiration.
- Obtain cervical spine films if the neck cannot be cleared clinically
- Obtain facial bone CT if other injuries of the face suspected.

DIFFERENTIAL DIAGNOSIS

- Contusions
- Dislocation of the mandible:
 - If a single condyle is dislocated, the jaw will deviate away from the side of the dislocation.
 - If fractured, the jaw will deviate toward the fractured side.
- Isolated dental trauma



TREATMENT

PRE HOSPITAL

- Cautions:
 - Protect the airway.
 - Protect the cervical spine.
 - Preserve any avulsed teeth.

INITIAL STABILIZATION/THERAPY

- 20–40% of patients with mandibular fractures have associated injuries:
 - Treatment is directed toward immediate, potentially lethal injuries such as airway obstruction, aspiration, major hemorrhage, cervical spine injury, and intracranial injury.
- Airway must be protected.
- Cervical spine precautions
- If oral intubation cannot be performed, nasotracheal intubation should be performed unless associated facial injuries are present, in which case cricothyrotomy may be indicated.

ED TREATMENT/PROCEDURES

- With the exception of condylar fractures, many mandibular fractures are associated with mucosal, gingival, or tooth socket disruption and should be considered open fractures:
 - Antibiotics such as penicillin, clindamycin, amoxicillin, amoxicillin/clavulanate or azithromycin to cover intraoral anaerobic

pathogens

- Tetanus prophylaxis for open fractures
- Analgesia such as acetaminophen, ibuprofen, or narcotic medications
- Definitive care usually consists of reduction and fixation by wiring upper and lower teeth in occlusion for 4–6 wk or by ORIF:
 - Linear, nondisplaced, or greenstick fractures may be treated with soft diet without wiring.
- If mandible dislocation is present, while the jaw is open apply bilateral downward pressure on the occlusal surface of the posterior lower teeth while grasping the mandible:
 - The goal is to free the condyle from its anterior position to the eminence.
 - Reduction is facilitated by muscle relaxants (diazepam or midazolam) or anesthetic injection of mastication muscles.
 - A bite block should be used, or the examiner's fingers should be wrapped in gauze to prevent injury.

MEDICATION

- Acetaminophen: 500 mg (peds: 10–15 mg/kg, do not exceed 5 doses/24h) PO q4–6h, do not exceed 4 g/24h
- Amoxicillin/clavulanate: 500/125–875/125 mg PO BID (peds: 40 mg/kg/d of amoxicillin PO BID)
- Amoxicillin: 500 mg PO TID (peds: 40 mg/kg PO div. TID)
- Azithromycin: 500 mg PO day 1 followed by 250 mg day 2–4 (peds: 10 mg/kg day 1 followed by 5 mg/kg day 2–4)
- Clindamycin: 150–450 mg PO QID (peds: 10–20 mg/kg/24h)
- Diazepam: 5–10 mg (peds: 0.1–0.2 mg/kg) IV
- Ibuprofen: 600–800 mg (peds: 20–40 mg/kg/24h) PO TID–QID
- Midazolam: 2–5 mg (peds: 0.02–0.05 mg/kg/dose, max. dose 0.4 mg/kg total and not > 10 mg) IV over 2–3 min
- Penicillin VK: 250–500 mg (peds: 25–50 mg/kg/24h) PO QID



FOLLOW-UP

DISPOSITION

Admission Criteria

- Significant displacement or associated dental trauma—open fractures require urgent specialty consultation for possible admission.
- The severity of associated trauma may indicate admission.
- Any patient with the potential for airway compromise should be admitted.
- An unreliable patient with nondisplaced fractures should be admitted for definitive fixation.

- In the pediatric population, if the mechanism of injury is not appropriate to the injuries seen, pediatric or child protective services consultation should be obtained.

Discharge Criteria

Patients with nondisplaced, closed fractures may be discharged on analgesics and a soft diet.

FOLLOW-UP RECOMMENDATIONS

Oral or maxillofacial surgeon within 2–3 days for uncomplicated fractures

PEARLS AND PITFALLS

- The most sensitive sign of a mandibular fracture is malocclusion.
- Failure to recognize that a gum laceration overlying a mandibular fracture represents an open fracture which requires antibiotics.
- Missing mandibular condyle fractures when only a panorex film is obtained – if there is condyle tenderness or malocclusion, obtain plain films or CT.
- Missing teeth must be accounted for, if not found, obtain a chest x-ray to rule out aspiration.
- A nonfractured mandible should be able to hold a tongue blade between the molars tightly enough to break it off. There should be no pain in attempting to rotate the tongue blade between the molars.

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See Also (Topic, Algorithm, Electronic Media Element)

- Dental Trauma
- Facial Fractures



CODES

ICD9

- 802.20 Closed fracture of mandible, unspecified site
- 802.21 Closed fracture of mandible, condylar process
- 802.25 Closed fracture of mandible, angle of jaw

ICD10

- S02.61XA Fracture of condylar process of mandible, init for clos fx
- S02.65XA Fracture of angle of mandible, init for clos fx
- S02.609A Fracture of mandible, unsp, init encntr for closed fracture

MARINE ENVENOMATION

Armando Marquez, Jr. • Timothy B. Erickson

BASICS

DESCRIPTION

Marine envenomation refers to poisoning caused by sting or bite from a vertebrate or invertebrate marine species.

ETIOLOGY

- Sponges:
 - Contain sharp spicules with irritants that cause pruritic dermatitis
- Coelenterates (Cnidaria jellyfish):
 - Contain stinging cells known as nematocysts on their tentacles
 - Fluid-filled cysts eject sharp, hollow thread-tube on contact.
 - Thread-tube penetrates skin and envenomates the victim.
 - Box jellyfish can kill within minutes
- Starfish:
 - Sharp, rigid spines are coated with slimy venom.
- Sea urchins:
 - Hollow, sharp spines filled with various toxins
- Sea cucumbers:
 - Hollow tentacles secrete holothurin, a liquid toxin.
- Cone shells:
 - Venom injected through dart-like, detachable tooth.
 - Active peptides interfere with neuromuscular transmission.
 - Presents with puncture wounds similar to wasp stings.
- Stingrays:
 - Most common cause of human marine envenomations.
 - Tapered spines attached to tail inject venom into victim.
- Scorpion fish:
 - Lionfish usually mild; stonefish can be life threatening.
 - Sharp spines along dorsum and pelvis of fish
 - Often stepped on inadvertently
 - Neurotoxic venom
- Catfish:
 - Dorsal and pectoral spines contain venom glands.
- Sea snakes:
 - Hollow fangs with associated venom glands
 - Highly neurotoxic venom blocks neuromuscular transmission.

SIGNS AND SYMPTOMS

- Sponges:
 - Itching and burning a few hours after contact
 - Local joint swelling and soft tissue edema
 - Fever
 - Malaise
 - Dizziness
 - Nausea
 - Muscle cramps
 - In severe cases, desquamation in 10 days to 2 mo
- Coelenterates (Cnidaria jellyfish):
 - Mild envenomation:
 - Immediate stinging sensation
 - Pruritus
 - Paresthesia, burning sensation
 - Throbbing
 - Blistering/local edema/wheal formation
 - Moderate/severe:
 - Neurologic: Ataxia, paralysis, delirium, seizures
 - Cardiovascular: Anaphylaxis, hemolysis, hypotension, dysrhythmias
 - Respiratory: Bronchospasm, laryngeal edema, pulmonary edema, respiratory failure
 - Musculoskeletal: Muscle cramps or spasm, arthralgias
 - Gastrointestinal: Nausea, vomiting, diarrhea, dysphagia, hypersalivation/thirst
 - Ophthalmologic: Conjunctivitis, corneal ulcers, elevated intraocular pressure
- Echinodermata:
 - Starfish:
 - Immediate pain
 - Bleeding
 - Mild edema
 - Paresthesias, nausea, vomiting if severe
 - Sea urchins:
 - Intense pain and severe local muscle aches
 - Nausea, vomiting
 - Paresthesias, hypotension, or respiratory distress with multiple stings
 - Sea cucumbers:
 - Mild contact dermatitis
 - Corneal and conjunctival involvement: Severe reactions can lead to

blindness.

- Mollusks:
 - Cone shells:
 - Puncture wounds similar to wasp stings
 - Sharp burning and stinging
 - Paresthesias indicate severe envenomation.
 - Can evolve into muscular paralysis and respiratory failure, dysphagia, syncope, disseminated intravascular coagulation
- Stingrays:
 - Puncture wounds or jagged lacerations
 - Local, intense pain, edema, bleeding; necrosis if severe
 - Nausea, vomiting, diarrhea
 - Diaphoresis
 - Headache
 - Tachycardia
 - Seizures
 - Paralysis
 - Hypotension
 - Dysrhythmias
- Scorpion fish:
 - Intense local pain for 6–12 hr
 - Erythema may progress to cellulitis.
 - Headache
 - Nausea, vomiting, diarrhea
 - Pallor
 - Delirium
 - Seizures
 - Fever
 - Hypertension
- Catfish:
 - Local pain, ischemic appearance progressing to erythema
 - Swelling, bleeding, and edema
 - Local muscle spasms
 - Diaphoresis
 - Neuropathy, fasciculations, weakness, syncope
- Sea snakes:
 - Bite initially causes very little pain.
 - Pin-like pairs of fang marks
 - Onset from 5 min to 6 hr
 - Muscle pain, lower extremity paralysis, arthralgias
 - Trismus, blurred vision, dysphagia, drowsiness
 - Severe signs include:
 - Ascending paralysis

- Aspiration
- Coma
- Renal and liver failure
- _ If untreated, 25% mortality

History

- Time of envenomation
- Body part envenomated
- Activity when envenomated (scuba diving, swimming, surfing, fishing, boating, pet care)
- Type of water (salt water, fresh water, aquarium)
- Geographic location (resort, international, remote, local, aquarium, zoo, pet store)
- Onset of symptoms, pain
- Mental status changes
- Near drowning

Physical-Exam

- Vital signs
- Airway
- Mental status
- Cardiopulmonary exam
- Dermatologic exam, foreign bodies, cellulitis, blistering

ESSENTIAL WORKUP

- Careful history, repeated evaluation of wound sites
- Assessment of ABCs

DIAGNOSIS TESTS & NTERPRETATION

Lab

- CBC
- Electrolytes, BUN, creatinine, and glucose levels
- LFT
- Urinalysis
- Arterial blood gases if severe symptoms

Imaging

Soft tissue radiographs to detect foreign body

DIFFERENTIAL DIAGNOSIS

- Allergic reaction
- Cellulitis
- Gastroenteritis

- Aspiration pneumonia
- Near drowning



TREATMENT

PRE HOSPITAL

- Remove victim from water source.
- Control airway, breathing.
- Control hemorrhage.
- Detoxify venom with proper wound irrigation as discussed below.

INITIAL STABILIZATION/THERAPY

- Airway, breathing, and circulation management (ABCs)
- Establish IV access with 0.9% normal saline (NS).

ED TREATMENT/PROCEDURES

- General:
 - _ Prepare for anaphylactic reactions (epinephrine/steroids).
 - _ Prepare for intubation if needed.
 - _ Diphenhydramine for itch, burn, hives
 - _ Tetanus prophylaxis
 - _ Corticosteroids for severe local reactions
 - _ Narcotic analgesia for severe pain
 - _ Antibiotic prophylaxis for the following:
 - Large lacerations or burns
 - Deep puncture wounds
 - Grossly contaminated wounds
 - Elderly or chronically ill
 - _ Antibiotic choices:
 - Trimethoprim/sulfamethoxazole (TMP-SMX; Bactrim)
 - Tetracycline
 - Ciprofloxacin
 - 3rd-generation cephalosporin
- Sponges:
 - _ Gently dry skin and remove spicule:
 - Adhesive tape may aid in removal.
 - _ 5% acetic acid (vinegar) (or 40–70% isopropyl alcohol) soaks QID for 10–30 min
- Coelenterates (Cnidaria jellyfish):
 - _ Rinse wound with salt water or seawater:
 - Hypotonic (fresh or tap water solutions), trigger more nematocysts
 - _ Do not rub skin to avoid release of nematocysts.

- Inactivate toxin with 30-min soak of 5% acetic acid (vinegar)
- Remove remaining nematocysts with razor, clam shell.
- Apply topical anesthetics once nematocysts are removed.
- Sea Safe jellyfish sunblock products are available.
- Box-jellyfish sting envenomation (Australia) emergent cases:
 - Administer *Chironex* antivenin: 1 amp (20,000 U) IV diluted 1:5 with crystalloid.
- Corticosteroids for severe reactions
- Starfish:
 - Immerse in nonscalding hot water for pain relief.
 - Irrigate and explore all puncture wounds.
 - Prophylactic antibiotics for significant wounds
- Sea urchins:
 - Immerse in nonscalding hot water for pain relief.
 - Remove any remaining spines.
 - Prophylactic antibiotics for significant wounds.
- Sea cucumbers:
 - Immerse in nonscalding hot water for pain relief.
 - 5% acetic acid soaks
 - Ocular involvement:
 - Proparacaine for pain
 - Copious irrigation with NS
 - Careful slit-lamp exam
- Cone shells:
 - Hot water immersion for pain relief
 - Be prepared for cardiac or respiratory support.
- Stingrays:
 - Copious irrigation with removal of any visible spines
 - Local suction is controversial.
 - Hot water soaks for pain relief
 - Narcotics for pain control
 - High incidence of bacterial infection:
 - Administer prophylactic antibiotics for significant wounds.
- Scorpion fish:
 - Hot water soaks for pain relief and venom inactivation
 - Copious irrigation, removal of any visible spines
 - Local lidocaine or regional block for severe pain
 - Surgical exploration for deep penetration/foreign bodies
 - Stonefish antivenin for severe envenomations:
 - One 2-mL amp diluted in 50-mL saline IV slow
 - May cause serum sickness
- Catfish:
 - Hot water soaks for pain relief and venom inactivation

- Copious irrigation, removal of any visible spines
- Consider local lidocaine, regional block, or narcotics for severe pain.
- Surgical exploration for deep penetration, foreign bodies
- Leave puncture wounds open to heal.
- Consider prophylactic antibiotics for hand, foot, or deep wounds.
- Sea snakes:
 - Immobilize bitten extremity.
 - Apply pressure bandage for venous occlusion (pre-hospital).
 - Keep victim warm and still.
 - Polyvalent sea snake antivenin reduces mortality to 3%:
- May require 3–10 amps (1000 U each)
- Prepare early for assisted ventilation.

MEDICATION

- Cefixime: 400 mg (peds: 8 mg/kg/24h) PO daily
- Ciprofloxacin: 500 mg PO BID
- Epinephrine: 0.3–0.5 mL SC 1:1,000 (peds: 0.01 mL/kg)
- Tetracycline: 500 mg PO QID (caution with photosensitivity)
- TMP-SMX (Bactrim DS): 1 tab [peds: 5 mg liquid (40/200/5 mL)/10 kg per dose] PO BID (caution with photosensitivity)



FOLLOW-UP

DISPOSITION

Admission Criteria

Significant signs of systemic involvement or need for antivenom administration

Discharge Criteria

No signs of systemic illness after 8 hr of observation

Issues for Referral

Zoos, aquariums for available supplies of antivenom; poison control centers: 800-222-1222

PEARLS AND PITFALLS

- Most toxins are detoxified with either temperature change (hot water) or pH alteration (more acidic).
- Specific antivenoms for box jellyfish, stone fish, and sea snake envenomations are available but in limited supply; acquire early in treatment course.

ADDITIONAL READING

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CODES

ICD9

- 692.89 Contact dermatitis and other eczema due to other specified agents
- 989.5 Toxic effect of venom

ICD10

- T63.511A Toxic effect of contact with stingray, accidental (unintentional), initial encounter
- T63.621A Toxic effect of contact with other jellyfish, accidental (unintentional), initial encounter
- T63.691A Toxic effect of contact with other venomous marine animals, accidental (unintentional), initial encounter

MASTITIS

Hao Wang • Marco Coppola



BASICS

DESCRIPTION

- Infection of the breast causing pain, swelling, and erythema
- Most commonly in women who are breast-feeding
- Often with systemic symptoms also:
 - Malaise
 - Fever
- Incidence may be as high as 33% in lactating woman
- Onset typically 2–3 wk to months postpartum
- 75–95% occur before infant is 3 mo old
 - Rare during 1st postpartum week
- More common in advanced maternal age and patients with diabetes
- Complications:
 - Recurrence
 - Abscess
 - Sepsis
 - Necrotizing fasciitis
 - Fistula
 - Scarring
 - Breast hypoplasia

Pediatric Considerations

Can occur in full-term infants < 2 mo of age

ETIOLOGY

- *Staphylococcus aureus* most common
- Less common causes:
 - Coagulase-negative *Staphylococcus*
 - *Streptococcus* spp.
 - *Escherichia coli*
 - *Haemophilus influenzae*
 - *Candida albicans*
- Risk factors:
 - Cleft lip or palate
 - Cracked nipples
 - Infant attachment issues
 - Local milk stasis

- Nipple piercing
- Poor maternal nutrition
- Previous mastitis
- Primiparity
- Restriction from a tight bra
- Sore nipples
- Short frenulum in infant
- Use of a manual breast pump
- Yeast infection

DIAGNOSIS

SIGNS AND SYMPTOMS

- Fever and chills
 - Temperature usually $> 38.3^{\circ}\text{C}$ (101°F)
- General malaise
- Tachycardia
- Breast pain, induration, erythema, warmth; usually unilateral
- Onset typically 2–3 wk to months postpartum while breast-feeding
- Rare during 1st postpartum week

History

- Flu-like symptoms
- Fever, malaise, and myalgia
- Breast redness, swelling
- Breast pain
- Decreased milk outflow

Physical-Exam

- Breast is:
 - Warm
 - Tender
 - Indurated
 - Erythematous – often in a wedge-shaped pattern
- Usually unilateral breast involvement
- Purulent nipple discharge can occur
- Axillary lymph nodes may be enlarged

ESSENTIAL WORKUP

Physical exam with special attention to detecting abscess:

- Abscess is frequently difficult to detect, but is more common in periareolar area
- Purulent nipple discharge with palpation

Pediatric Considerations

- In neonates:
 - Consider the presence of abscess formation and systemic symptoms of infection (e.g., lethargy, poor feeding, fever)
 - Sepsis workup may be needed if neonates are febrile and ill appearing
 - A complete blood count (CBC) with differential and blood culture need to be considered before the initiation of antibiotics

DIAGNOSIS TESTS & INTERPRETATION

Lab

Breast milk culture is usually not required

Imaging

- Consider breast US if abscess is suspected
- Mammography is not indicated acutely

DIFFERENTIAL DIAGNOSIS

- Breast engorgement:
 - Transient fever $< 39^{\circ}\text{C}$ of 4–16 hr duration
 - Appearing 48–72 hr postpartum
 - Bilateral nonerythematous engorgement
- Carcinoma (inflammatory)
- Cyst, tumor
- Abscess formation



TREATMENT

PRE HOSPITAL

Generally no pre-hospital treatment needed

INITIAL STABILIZATION/THERAPY

No specific stabilization

ED TREATMENT/PROCEDURES

- Continue breast-feeding:
 - Child and mother are colonized with the same organisms
 - Milk from a breast with mastitis may be protective
 - If an infant does not like the taste of milk from a breast with mastitis, then encourage the mother to pump and discard
- Massage
- Hot/cold therapy
- Improve breast-feeding technique:

- May need a lactation consultant
- Maintain good maternal hydration.
- If mild symptoms and early in disease, antibiotics may not be necessary.
- Oral antibiotics for 7–14 days:
 - β -Lactamase-resistant penicillin (e.g., dicloxacillin)
 - 1st-generation cephalosporin (e.g., cefalexin)
 - Clindamycin or trimethoprim/sulfamethoxazole (TMP/SMX) or erythromycin if penicillin allergic
- Surgical consultation if evidence of abscess
- If considering methicillin-resistant *S. aureus* (MRSA), treat according to local susceptibility patterns:
 - Clindamycin
 - TMP/SMX
 - Vancomycin

ALERT

Vertical transmission of HIV (mother to infant) may be increased in mothers with mastitis.

MEDICATION

- Amoxicillin/clavulanate: 875 mg PO q12h
- Cephalexin: 500 mg PO q6h for 10 days
- Clindamycin: 300 mg PO q6h for 10 days
- Dicloxacillin: 500 mg PO q6h for 10 days (1st-line treatment)
- Erythromycin: 500 mg PO q6h for 10 days
- Mupirocin 2% ointment TID
- TMP/SMX: 160/800 mg PO q12h:
 - Avoid in compromised infants and healthy infants <2 mo old
- If MRSA positive: Vancomycin 1 g IV q12h

First Line

Dicloxacillin

Second Line

- Amoxicillin/clavulanate
- Cephalexin
- Erythromycin
- TMP/SMX



FOLLOW-UP

DISPOSITION

Admission Criteria

- Incision and drainage under general anesthesia may be necessary and require admission
- Immunocompromised or evidence of septicemia
- Patients with diabetes may account for 1/3 of mastitis cases
- Neonatal mastitis generally requires admission

Discharge Criteria

- Most patients may be managed in outpatient setting
- Most symptoms resolve within 48 hr of therapy
- In simple mastitis, breast-feeding may be continued, including using affected breast:
 - Gently massage to enhance drainage
 - Counsel that this will not harm baby
- Breast support, warm compresses, and analgesia for comfort
- In frank abscess, discontinue breast-feeding until purulent discharge resolves
- Follow-up should be arranged to exclude diagnosis of inflammatory carcinoma

FOLLOW-UP RECOMMENDATIONS

- Patients should follow up with primary care physician
- Lactation consultant may be helpful

PEARLS AND PITFALLS

- Most cases respond to lactation and warm compresses without antibiotics
- Cessation of breast-feeding will lead to increased milk stasis and increased risk for abscess formation
- One of the most common complications of mastitis is cessation of breast-feeding

ADDITIONAL READING

- Dixon JM, Khan LR. Treatment of breast infection. *BMJ*. 2011;342:d396.
- Jahanfar S, Ng CJ, Teng CL. Antibiotics for mastitis in breastfeeding women. *Cochrane Database Syst Rev*. 2009;(1):CD005458.
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See Also (Topic, Algorithm, Electronic Media Element)

- Abscess
- Cellulitis
- Community-acquired MRSA



CODES

ICD9

- 611.0 Inflammatory disease of breast
- 675.24 Nonpurulent mastitis associated with childbirth, postpartum condition or complication
- 778.7 Breast engorgement in newborn

ICD10

- N61 Inflammatory disorders of breast
- O91.23 Nonpurulent mastitis associated with lactation
- P83.4 Breast engorgement of newborn

MASTOIDITIS

Jonathan Fisher • Colby Redfield



BASICS

DESCRIPTION

- Inflammation of the mastoid air cells of the temporal bone, generally caused by direct extension of acute purulent otitis media
- Middle ear and mastoid air cells are contiguous via the aditus to mastoid antrum
- Fluid accumulation from closure of channel due to otitis media creates opportunity for infection
- Manifestation ranges from clinically insignificant inflammation of mastoid air cells to infection and destruction of the bone
- Acute mastoiditis:
 - Occurs to some degree in all cases of otitis media
 - Early signs and symptoms are those of acute otitis media
 - Usually secondary to contamination with infectious material trapped in the mastoid by inflammatory obstruction of the channel between middle ear and mastoid air cells
- Acute mastoiditis with periostitis:
 - As infection progresses, periosteum of the mastoid bone is involved, causing periostitis
 - Subperiosteal abscess may be present
- Acute mastoid ostitis (also called coalescent mastoiditis):
 - Progression of the infection within the mastoid air cells leads to destruction of the mastoid trabeculae, causing coalescence of bony trabeculae
 - Mastoid empyema or a draining fistula may be present
 - May progress to severe head and neck complications if untreated
- Masked mastoiditis:
 - Mastoid infection, which lingers after an acute otitis media has been treated
 - May progress to acute or coalescent mastoiditis
- Chronic mastoiditis:
 - Infection lasting > 3 mo
- Mastoiditis can be a complication of a primary disorder:
 - Leukemia
 - Mononucleosis
 - Sarcoma of the temporal bone
 - HIV
 - Kawasaki disease
- Mastoiditis used to be more common prior to the use of antibiotics for acute otitis media

- More common in young children and infants

ETIOLOGY

- Organisms in acute mastoiditis are similar to those in acute otitis media, but differ in frequency:
 - *Streptococcus pneumoniae*
 - Group A streptococcus
 - *Staphylococcus aureus*
 - *Haemophilus influenzae*
- Gram-negative enteric bacteria most common with chronic mastoiditis:
 - *Pseudomonas aeruginosa*
 - *Escherichia coli*
 - *Proteus mirabilis*
 - *Bacteroides* species
- Other less common causes:
 - *Mycobacterium tuberculosis*
 - *Aspergillus* species in immunocompromised states

Pediatric Considerations

- More frequently seen in the pediatric population due to strong association with otitis media
- *S. pneumoniae* is the most common cause in children

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Ear pain
- Otorrhea
- Mild to severe hearing loss
- Fever
- Headache
- History of irritability in a child
- History of recurrent otitis media

Physical-Exam

- Tenderness, edema, and erythema over the mastoid
- Lateral and inferior displacement of the auricle
- Loss of the postauricular crease
- Swelling of the posterior and superior ear canal wall
- Tympanic membrane abnormalities consistent with severe otitis media

- Purulent fluid drainage from the auditory canal
- Bulging tympanic membrane

ESSENTIAL WORKUP

Mastoiditis is a clinical diagnosis

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC:
 - Leukocytosis
- Cultures of drainage important owing to diversity of organisms:
 - If spontaneous drainage present or after surgical drainage
- Blood cultures if patient appears toxic

Imaging

- Mastoid plain radiographs:
 - Early stage of disease may show hazy or cloudy but intact mastoid
 - May reveal opacification or coalescence of the mastoid air cells or coalescence as disease progresses
 - Unreliable due to low sensitivity
- CT scan:
 - More useful, especially if abscess formation present
 - Can determine presence and extent of destruction of trabeculae as well as evaluate for the complications of mastoiditis
- MRI:
 - If intracranial involvement suspected but not confirmed by CT

Pediatric Considerations

- Conservative use of CT in children may be warranted
- The diagnosis can often be made on clinical grounds and avoids radiation exposure

Diagnostic Procedures/Surgery

Lumbar puncture:

- Cerebrospinal fluid evaluation for signs of meningitis

DIFFERENTIAL DIAGNOSIS

- Otitis media
- Cellulitis
- External otitis media
- Scalp infection with inflammation of posterior auricular nodes
- Rubella: Posterior auricular node enlargement
- Trauma to pinna or postauricular area

- Meningitis



TREATMENT

INITIAL STABILIZATION/THERAPY

- ABCs
- Airway management for signs of airway compromise
- 0.9% NS IV fluid bolus for hypotension/volume depletion

ED TREATMENT/PROCEDURES

- Initiate IV antibiotics
- Otolaryngologist consult for surgical drainage:
 - Drainage is the definitive therapy for acute or coalescent mastoiditis
 - Emergent drainage if the patient appears toxic
 - Types of surgical procedures:
 - Myringotomy drainage and tympanostomy tube placement
 - Mastoidectomy and drainage for severe extension (needed in ~ 50% of cases)

MEDICATION

- Initiate IV antibiotics:
 - Given increasing proportion of *S. aureus* as causative organism, consider including antistaphylococcal agent before culture results
 - Parenteral antibiotics can be switched to PO after patient afebrile for 36–48 hr
 - Consider antipseudomonal coverage when appropriate
- Administer pain medications:
 - NSAIDs
 - PO or parenteral narcotics

First Line

- Ceftriaxone: 1–2 g (peds: 50–75 mg/kg/24 h) IV q12–24 h
- Cefotaxime: 1–2 g (peds: 50–180 mg/kg/24 h) IV q4–6h

Second Line

- Ampicillin/sulbactam: 1.5–3 g IV q6h
- Chloramphenicol: 50–100 mg/kg/24 h IV or PO q6h
- Clindamycin: 600–2,700 mg/d IV div. q6–12h or 150–450 mg PO q6–8h (peds: 20–40 mg/kg/d IM/IV div. q6–8h or 10–25 mg/kg/d PO div. q6–8h)
- Ticarcillin/clavulanate: 3.1 g IV q4–6h
- Piperacillin/tazobactam: 3.375 g IV q6h
- Vancomycin: 1 g q8h (peds 40 mg/kg/24 h) IV q6–8h



FOLLOW-UP

DISPOSITION

Admission Criteria

- Clinical suspicion of acute or coalescent mastoiditis
- Subperiosteal abscess
- Toxic appearing

Discharge Criteria

Patients with acute or coalescent mastoiditis should not be discharged

Issues for Referral

- Otolaryngologist consult for possible surgical drainage
- Audiography should be performed after resolution of mastoiditis to assess hearing loss

FOLLOW-UP RECOMMENDATIONS

Patients should follow up with otolaryngologist after discharge, if not admitted

COMPLICATIONS

- Bezold abscess:
 - Extension of infection to soft tissue below pinna or behind the sternocleidomastoid muscle of neck after erosion through the mastoid tip
- Petrositis:
 - Spread of the infection to the petrous air cells
- Osteomyelitis of the calvarium
- Intracranial complications:
 - Subperiosteal abscess
 - Subdural empyema:
 - Extension of infection to CNS with empyema around the tentorium
 - Sinus thromboses

Pediatric Considerations

Even with conservative management of otitis media, a 10-yr analysis did not show a significant increase in cases of acute mastoiditis.

PEARLS AND PITFALLS

- It is important to maintain a high index of suspicion for mastoiditis in setting of persistent or untreated acute otitis media.
- Failure to recognize meningitis or intracranial involvement, which require more

aggressive management, is a pitfall

- Drainage is the definitive therapy

ADDITIONAL READING

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- Marx JA, Hockberger RS, Walls RM, et al. *Rosen's Emergency Medicine: Concepts and Clinical Practice.* 7th ed. St. Louis, MO: Mosby; 2009.
- Tamir S, Schwartz Y, Peleg U, et al. Acute mastoiditis in children: Is computed tomography always necessary? *Ann Otol Rhinol Laryngol.* 2009;118:565–569.
- Tamir S, Schwartz Y, Peleg U, et al. Shifting trends: Mastoiditis from a surgical to a medical disease. *Am J Otolaryngol.* 2010;31:467–471.



CODES

ICD9

- 383.00 Acute mastoiditis without complications
- 383.01 Subperiosteal abscess of mastoid
- 383.9 Unspecified mastoiditis

ICD10

- H70.009 Acute mastoiditis without complications, unspecified ear
- H70.019 Subperiosteal abscess of mastoid, unspecified ear
- H70.90 Unspecified mastoiditis, unspecified ear

MDMA POISONING

Mark B. Mycyk



BASICS

DESCRIPTION

- MDMA: 3,4-methylenedioxyamphetamine (“ecstasy”)
- Schedule I drug manufactured illegally
- Used recreationally:
 - Rave parties
 - Dance clubs
 - College campuses
- Onset of effects: 15–30 min after ingestion
- Duration of effects: 2–6 hr
- Pills commonly contain contaminants:
 - Caffeine
 - Ephedrine
 - Dextromethorphan
 - Ketamine
 - Related methylated amphetamines: 3,4-methylenedioxyamphetamine (MDA), 3,4-methylenedioxy-*N*-ethylamphetamine (MDEA), 3,4-methylenedioxy-*N*-butylamphetamine (MDBA), *para*-methoxyamphetamine (PMA)
- Pathophysiology:
 - Amphetamine-like structure stimulates catecholamine release.
 - Mescaline-like ring structure enhances serotonergic and dopaminergic activity.

ETIOLOGY

Deliberate or accidental ingestion of MDMA



DIAGNOSIS

SIGNS AND SYMPTOMS

- Overdose:
 - Altered mental status
 - Severe sympathomimetic symptoms
- Central nervous system:
 - Excitation
 - Coma
 - Seizures

- Cerebral edema
- Cardiovascular:
 - Hypertension (early)
 - Hypotension (late)
 - Palpitations
 - Ventricular tachycardia and ectopy
- Pulmonary:
 - Pulmonary edema
- Metabolic:
 - Hyponatremia
 - Hypoglycemia
 - Syndrome of inappropriate antidiuretic hormone
- Musculoskeletal:
 - Bruxism
 - Restlessness
 - Rigidity
- Renal:
 - Rhabdomyolysis
- Hepatic:
 - Jaundice
 - Hepatitis
- Hematologic:
 - Disseminated intravascular coagulation
- Gastrointestinal:
 - Vomiting
 - Diarrhea
 - Abdominal cramping
- Psychiatric:
 - Euphoria
 - Flight of ideas
 - Delirium/hallucinations
- Other:
 - Hyperthermia
 - Mydriasis
 - Nystagmus

ESSENTIAL WORKUP

- Diagnosis based on clinical presentation and an accurate history.
- Obtain core temperature.
- Exclude toxic coingestants or contaminants.

DIAGNOSIS TESTS & NTERPRETATION

Lab

- Electrolytes, BUN, creatinine, and glucose levels
- Prothrombin time, partial thromboplastin time, international normalized ratio
- Urine dip for blood and myoglobin
- Creatine phosphokinase level if rhabdomyolysis suspected
- Liver function tests for significant overdose or suspected hepatitis
- Urine toxicology screen to exclude coingestants:
 - May cause positive amphetamine and methamphetamine screen
- Quantitative MDMA levels rarely helpful

Imaging

- CXR if suspected aspiration pneumonia
- Head CT if suspected intracranial hemorrhage

Diagnostic Procedures/Surgery

ECG:

- Sinus tachycardia (most common)
- Dysrhythmias, conduction disturbances

DIFFERENTIAL DIAGNOSIS

- Cocaine overdose
- Amphetamine overdose
- Anticholinergic overdose
- Cathinone overdose (e.g., Bath salts)
- Serotonin syndrome
- Occult head injury
- Sepsis
- Thyroid storm
- Pheochromocytoma



TREATMENT

PRE HOSPITAL

- Transport all pills/pill bottles involved in overdose for identification in ED.
- Watch for MDMA paraphernalia:
 - Pacifiers
 - Glow sticks
 - Surgical masks

INITIAL STABILIZATION/THERAPY

ABCs:

- Airway control is essential.

- Administer supplemental oxygen.
- Intubate if indicated.
- IV access
- Naloxone, thiamine, dextrose (or Accu-Chek), if altered mental status

ED TREATMENT/PROCEDURES

- Supportive care
- Monitor core temperature and cardiac rhythm for at least 6 hr.
- Hydrate with 0.9% normal saline (NS) IV
- Hypertension:
 - _ Nitroprusside
 - _ Phentolamine
 - _ Esmolol
- Hypotension:
 - _ 0.9% NS IV bolus
 - _ Trendelenburg position
 - _ Pressors titrated to blood pressure
- Anxiety, restlessness, agitation:
 - _ Diazepam or lorazepam as needed
- Seizures:
 - _ Treat initially with benzodiazepines.
 - _ Phenobarbital for persistent seizures
- Rhabdomyolysis:
 - _ Hydrate aggressively with 0.9% NS IV
 - _ Consider sodium bicarbonate administration.
 - _ Hemodialysis if renal failure
- Hyperthermia:
 - _ Standard cooling measures
 - _ Treat agitation with benzodiazepines.

MEDICATION

- Diazepam: 5–10 mg (peds: 0.2–0.5 mg/kg) IV q10–15min
- Esmolol: 500 µg/kg IV bolus, then 50 µg/kg/min IV
- Lorazepam: 2–6 mg (peds: 0.05–0.1 mg/kg) IV q10–15min
- Naloxone: 0.4–2 mg (peds: 0.1 mg/kg; neonatal: 10–30 mg/kg) IV or IM
- Nitroprusside: 0.3 mg/kg/min to max. 10 µg/kg/min
- Phenobarbital: 10–20 mg/kg IV (loading dose)
- Phentolamine: 1–5 mg (peds: 0.02–0.1 mg/kg) IV bolus q5–10min
- Propofol: 0.5–1.0 mg/kg IV (loading dose), then 5–50 mg/kg/min (maintenance dose)



FOLLOW-UP

DISPOSITION

Admission Criteria

- Altered mental status
- Seizures
- Persistent cardiovascular instability
- Rhabdomyolysis
- Loss of behavioral control
- Disseminated intravascular coagulation

Discharge Criteria

Asymptomatic 6 hr after oral overdose

FOLLOW-UP RECOMMENDATIONS

- Substance abuse referral for patients with recreational drug abuse
- Patients with unintentional (accidental) poisoning require poison prevention counseling.
- Patients with intentional (e.g., suicide) poisoning require psychiatric evaluation.

PEARLS AND PITFALLS

- Always obtain a core temperature.
- Concomitant recreational drugs might not be present on a routine hospital drug screen.
- For persistent altered mental status, assess electrolytes for hyponatremia.
- Consider nontoxicologic causes for altered mental status.

ADDITIONAL READING

- Centers for Disease Control and Prevention. Ecstasy overdoses at a New Year's Eve rave—Los Angeles, California, 2010. *MMWR Morb Mortal Wkly Rep.* 2010;59(22):677–681.
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- Patel MM, Wright DW, Ratcliff JJ, et al. Shedding new light on the “safe” club drug: Methylenedioxymethamphetamine (ecstasy)-related fatalities. *Acad Emerg Med.* 2004;11(2):208–210.
- Rosenson J, Smollin C, Sporer KA, et al. Patterns of ecstasy-associated hyponatremia in California. *Ann Emerg Med.* 2007;49(2):164–171.

ICD9

969.72 Poisoning by amphetamines

ICD10

- T43.621A Poisoning by amphetamines, accidental (unintentional), initial encounter
- T43.623A Poisoning by amphetamines, assault, initial encounter
- T43.624A Poisoning by amphetamines, undetermined, initial encounter

MEASLES

Austen-Kum Chai

BASICS

DESCRIPTION

- Vaccine preventable, primarily childhood, infectious disease characterized by fever, cough, coryza, conjunctivitis, and erythematous maculopapular rash
- Also known as rubeola
- Incidence is low secondary to widespread immunization

ETIOLOGY

- Rubeola is a *morbillivirus*, a negative-strand (RNA) paramyxovirus
- Humans are the only known reservoir
- Highly contagious. Respiratory isolation should be initiated when suspected. Outbreaks seen in nonimmunized or underimmunized

Pregnancy Considerations

- Increased risk of spontaneous abortion and premature contractions if infected during pregnancy.
- Does not appear to cause birth defects.
- Women should not be vaccinated with MMR or MMRV during pregnancy.

Geriatric Considerations

Those born before 1957 are generally considered immune. However, those in health care should receive vaccination if serologic testing reveals negative titer.

Pediatric Considerations

- Measles, mumps, and rubella ± Varicella (MMR or MMRV) vaccine should be administered to children on or after 12 mo of age. A 2nd dose is administered at the age of 4–6 yr, before start of school.
- Catch-up doses should be separated by at least 4 wk between vaccinations.

DIAGNOSIS

SIGNS AND SYMPTOMS

- Incubation (10–12 days) before appearance of rash:
 - Transmission via direct contact or inhalation of infectious droplet
 - Children usually have incomplete or no immunization.
- Prodrome (1–7 days):
 - Fever, followed by mild respiratory illness, conjunctivitis, fever

- Koplik spots:
 - Small white to grayish-blue specks on buccal mucosa
 - Pathognomonic for rubeola
 - Transient. Appears 1–2 days before rash and disappears within 48 hr after onset of rash
- Active disease:
 - Cough, coryza, conjunctivitis (“three C’s”).
 - Fever of 101°F, usually higher. Fever beyond 3–4 days suggests measles related complication
 - Rash appears on day 3–7, lasting 4–7 days:
 - Begins on head and spreads centrifugally downward
 - Maculopapular blanching rash which becomes confluent. May have petechiae. Palm and soles rarely involved.
 - Clinical improvement seen in 48 hr of appearance of rash
 - Rash clears in 3–4 days and may desquamate as rash fades in order of appearance
- Complications:
 - Respiratory:
 - Cough may persist for 1–2 wk after measles infection.
 - Pneumonia (6%) seen most commonly in immunocompromised
 - Most common cause of fatality seen with measles
 - Laryngotracheobronchitis in patients <2 yr old
 - CNS:
 - Seizures <1%
 - Encephalitis
 - Encephalomyelitis:
 - *1–14 days after onset of rash. Due to post infection autoimmune response*
 - Fever, headache, vomiting, and stiff neck
 - Lethargy, stupor, and seizure followed by coma
 - Subacute sclerosing panencephalitis (SSPE):
 - *Very rare but serious complication that develops 7–10 yr after infection*
 - Insidiously progressive degeneration of CNS functions
 - Personality change, intellectual deterioration, motor and visual deficits, seizures, coma, and death
 - Cardiovascular:
 - Transient myocarditis, pericarditis, and conduction defects
 - Rarely clinically significant
 - Congestive heart failure in elderly patients
 - Thrombocytopenic purpura
 - Otitis media 7%
 - Sinusitis

- Diarrhea 8%, most common

ESSENTIAL WORKUP

- Diagnosis is based on clinical findings.
- Cough, coryza, and conjunctivitis with fever and subsequent rash

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CSF analysis for suspected encephalitis
- Viral isolation from blood, throat, nasopharynx, and urine for epidemiologic surveillance. Ideally within 7 and not more than 10 days of appearance of rash.
- Serologic tests for measles IgM and IgG titers, and PCR of measles virus RNA are available to confirm diagnosis

Imaging

Chest radiograph for suspected pneumonia

DIFFERENTIAL DIAGNOSIS

- Rubella
 - Milder course, postauricular nodes, pinker rash, no conjunctivitis
- Scarlet fever:
 - Sandpaper-textured rash, strawberry tongue, sore throat
- Infectious mononucleosis:
 - Serologic test available
- Roseola:
 - Rash appears after temperature falls.
- Erythema infectiosum (“fifth disease”):
 - No prodrome and without fever
 - Red, flushed cheeks with lace-like rash when fading
- Enterovirus:
 - No respiratory complaints
- Kawasaki disease:
 - Rash on palm and soles
- Secondary syphilis
- Toxic shock syndrome
- Drug reactions:
 - Usually without high fever and upper respiratory infection symptoms



TREATMENT

PRE HOSPITAL

Nonimmunized pre-hospital care personnel should be advised of potential risks described above.

- ABCs if indicated

ED TREATMENT/PROCEDURES

- Prevention with vaccination is cornerstone of therapy
- Antipyretics
- IV hydration if needed
- Isolate suspected cases
- Postexposure prophylaxis for the nonimmune:
 - _ Give MMR if <72 hr after exposure:
 - Avoid if pregnant or immunocompromised.
 - _ Immunoglobulin 0.25 mL/kg IM up to 15 mL (max.):
 - If given <6 days after exposure, may prevent or modify severity of symptoms
 - Indicated for susceptible household or other close contacts, particularly those <1 yr, pregnant women, or immunocompromised
 - For patients who receive immune globulin IV (IGIV) regularly, the usual dose of 400 mg/kg should be adequate for measles prophylaxis after exposure occurring within 3 wk of receiving IGIV.
 - Patients receiving IG should subsequently receive vaccine no sooner than 5–6 mo if not contraindicated.
- ABCs. Oxygenation and airway protection for:
 - _ Pneumonia
 - _ Encephalitis

MEDICATION

WHO recommends vitamin A once in a day for 2 days for children with measles where vitamin A deficiency is prevalent. It may reduce the risk of measles mortality:

- 50,000 IU for age <6 mo
- 100,000 IU for age 6–12 mo
- 200,000 IU for age >12 mo
- Parenteral and oral formulations are available in US



FOLLOW-UP

DISPOSITION

Admission Criteria

- Severe pneumonia
- Dehydration
- Encephalitis

- SSPE
- Immunocompromised patients:
 - AIDS
 - Immunosuppressive therapy
- Elderly patients with comorbid conditions

Discharge Criteria

Duration of infectivity:

- 4 days before symptoms and up to 4 days after onset of rash
- Immunocompromised are contagious for duration of illness

PEARLS AND PITFALLS

- One of the most highly communicable of infectious diseases; death occurs in 1–3/1,000 cases in US. Respiratory isolation in health care settings is a must
- Severely immunocompromised patients, those on immunotherapy, and pregnant patients should not receive MMR or MMRV vaccine.
- Characteristic rash may not develop in immunocompromised patients.

ADDITIONAL READING

- American Academy of Pediatrics. Measles. In: Pickering LK, ed. *Red Book 2012: The Report of the Committee on Infectious Diseases*. 27th ed. Elk Grove Village, IL: American Academy of Pediatrics; 2012:489–499.
- Centers for Disease Control and Prevention. Measles. In: Atkinson W, Wolfe S, Hamborsky J, eds. *Epidemiology and Prevention of Vaccine-Preventable Diseases (The Pink Book)*. 12th ed. Washington, DC: Public Health Foundation; 2012:173–192.
- Mason WH. Measles. In: Kliegman RM, ed. *Nelson Textbook of Pediatrics*, 19th ed. Philadelphia, PA: Saunders Elsevier; 2011:1069–1075.



See Also (Topic, Algorithm, Electronic Media Element)

<http://www.cdc.gov/measles>



CODES

ICD9

- 055.0 Postmeasles encephalitis
- 055.1 Postmeasles pneumonia
- 055.9 Measles without mention of complication

ICD10

- B05.0 Measles complicated by encephalitis

- B05.2 Measles complicated by pneumonia
- B05.9 Measles without complication

MECKEL DIVERTICULUM

Galeta C. Clayton



BASICS

DESCRIPTION

- Most common congenital abnormality of the GI tract
 - Results from incomplete obliteration of the omphalomesenteric duct
- True diverticula (contains all layers):
 - 50% contain normal ileal mucosa.
 - 50% contain either gastric (most common), pancreatic, duodenal, colonic, endometrial, or hepatobiliary mucosa.
- Rule of 2's:
 - 2% prevalence in general population
 - 2% lifetime risk for complications, decreasing with age
 - Symptoms commonly occur around 2 yr of age:
 - 45% of symptomatic patients < 2 yr old
 - Average length 2 in
 - Found within 2 ft of the ileocecal valve
- Male-to-female ratio approximately equal, but more often symptomatic in males
- Complications:
 - Obstruction and diverticulitis in adults
 - Hemorrhage and obstruction in children
 - Mean age 10 yr
 - Current mortality rate 0.0001%
 - Occurs more frequently in males
- Obstruction:
 - Diverticulum attached to the umbilicus, abdominal wall, other viscera, or is free and unattached, leading to:
 - Intussusception: Diverticulum is the leading edge.
 - Volvulus: Persistent fibrous band leads to bowel rotation.
- Diverticulitis:
 - Opening obstructed
 - Bacterial infection follows.
 - Presents like appendicitis (most common preoperative diagnosis with Meckel diverticulum)

Pediatric Considerations

- Most common cause of significant lower GI bleeding in children.
- Presents at age < 5 yr with episodic painless, brisk, and bright-red rectal bleeding.

ETIOLOGY

Remnant of the omphalomesenteric duct that typically regresses by week 7 of gestation.

DIAGNOSIS

SIGNS AND SYMPTOMS

- 3 different types of presentation:
 - *Rectal bleeding* due to hemorrhage, which results from mucosal ulcerations within the ectopic gastric tissue
 - *Vomiting* due to obstruction secondary to volvulus, intussusceptions, or intraperitoneal bands
 - *Abdominal pain* (appendicitis like) due to an inflamed or perforated diverticulum
- General:
 - Fever
 - Malaise
 - Weakness
 - Fatigue
- GI:
 - Classically painless rectal bleeding
 - Abdominal pain:
 - Location depends on cause
 - Appendicitis like
 - Vomiting
 - Distention
 - Changes in bowel movements
 - Hematochezia or melena (depending on briskness or location of diverticulum)
 - Peritonitis and septic shock (late complications)
- Cardiovascular:
 - Tachycardia (due to pain or blood loss)
 - Hypotension and shock (due to bleeding)

ESSENTIAL WORKUP

- May cause a variety of signs and symptoms:
 - < 10% diagnosed preoperatively
 - Consider in patients with recurrent nonspecific abdominal pain, nausea and vomiting, or rectal bleeding.
- History and physical exam narrow diagnosis, but will not give specific findings for Meckel diverticulum.
- Rectal exam mandatory
- Nasogastric (NG) tube placement to rule out upper GI bleed

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC:
 - _ Decreased hematocrit due to bleeding
 - _ Rarely a cause of chronic anemia
 - _ Leukocytosis with diverticulitis, perforation, or gangrene
- Electrolytes, BUN, creatinine, coagulation studies
- Type and screen/cross-match when significant GI bleeding.

Imaging

- CT abdomen/pelvis:
 - _ For suspected infection (appendicitis/diverticulitis) or bowel obstruction
- Abdominal radiographs:
 - _ Screening for bowel obstruction
 - _ Cannot diagnose Meckel diverticulum
- Tc-99m pertechnetate radioisotope scan (Meckel scan):
 - _ Noninvasive scan that identifies Meckel diverticulum containing heterotopic gastric mucosa
 - _ 90% accurate in children
 - _ 45% accurate in adults
- Small bowel enteroclysis:
 - _ 75% accuracy
 - _ Barium/methyl cellulose introduced through NG tube into distal duodenum or proximal jejunum
 - _ Increases the ability to detect Meckel diverticulum in adults
 - _ Diverticulum may be short and wide-mouthed, making diagnosis difficult.
- Barium enema:
 - _ Introduces fluid into distal small bowel
 - _ Look for diverticulum
- Angiogram for further evaluation of Meckel diverticulum if radioisotope scan and enteroclysis normal:
 - _ Blood supply is not always abnormal (vitelline artery).
- Ultrasound may be useful in nonbleeding presentations.
- Laparoscopic evaluation may provide both diagnosis and definitive treatment.
- ECG:
 - _ Eliminate myocardial ischemia as cause of abdominal pain.
- Colonoscopy:
 - _ Not useful in diagnosing Meckel diverticulum

DIFFERENTIAL DIAGNOSIS

- Adults:
 - _ Adhesions

- Appendicitis
- Arteriovenous malformation
- Bowel obstruction
- Diverticulitis
- Hemorrhoids
- Inflammatory bowel disease
- Internal hernias
- Intestinal polyps
- Intussusception
- Peptic ulcer disease
- Pseudomembranous colitis
- Volvulus
- Pediatric:
 - Adhesions
 - Anal fissures
 - Appendicitis
 - Atresia
 - Gastroenteritis
 - Hemolytic-uremic syndrome
 - Henoch–Schönlein purpura
 - Intestinal polyps
 - Intussusception
 - Malrotation
 - Milk allergy
 - Strictures
 - Volvulus



TREATMENT

PRE HOSPITAL

Establish IV access for patients with rectal bleeding or abdominal pain.

INITIAL STABILIZATION/THERAPY

- Stabilization followed by early surgical evaluation
- Hypotension:
 - Aggressive fluid resuscitation
 - Packed RBC (PRBC) transfusion with brisk rectal bleeding (more common in children)
 - Pressors for septic shock

ED TREATMENT/PROCEDURES

- GI bleeding:

- Fluid resuscitate and transfuse PRBC as indicated
- Foley to follow urine output
- NG tube to exclude brisk upper GI bleeding
- Surgical consult for surgical intervention as indicated
- Obstruction:
 - NG tube
 - Foley
 - Surgical consult
- Diverticulitis/perforation:
 - NPO
 - Preoperative antibiotics
 - Surgical consult
- Surgical intervention:
 - Symptomatic Meckel diverticula should be resected
 - Asymptomatic Meckel diverticula discovered incidentally at laparotomy in children should be resected

MEDICATION

- Ampicillin/sulbactam (Unasyn): 3 g (peds: 100–200 mg ampicillin/kg/24h) q8h IV
- Cefoxitin (Mefoxin): 1–2 g (peds: 100–160 mg/kg/24h) IV q6h
- Dopamine: 2–20 μ /kg/min IV



FOLLOW-UP

DISPOSITION

Admission Criteria

Presumptive diagnosis of Meckel diverticulum with diverticulitis, obstruction, intussusception, hemorrhage, or volvulus requires admission and surgical evaluation.

Discharge Criteria

None

FOLLOW-UP RECOMMENDATIONS

Postoperative surgical follow-up

PEARLS AND PITFALLS

- Painless, brisk, bright-red blood per rectum in an infant is often caused by Meckel diverticulum.
- Presents with a wide range of complications, including obstruction, intussusception, and hemorrhage.
- Often diagnosed in the OR for patients undergoing surgery for a presumptive

appendicitis.

- Rule of 2's:
 - 2% of the population
 - 2% risk of complications
 - Mostly <2 yr old
 - 2 in long
 - 2 ft from the ileocecal valve

ADDITIONAL READING

- McCollough M, Sharieff GQ. Abdominal pain in children. *Pediatr Clin North Am.* 2006;53(1):107–137.
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- Zani A, Eaton S, Rees CM, et al. Incidentally detected Meckel diverticulum: To resect or not to resect? *Ann Surg.* 2008;247(2):276–281.



See Also (Topic, Algorithm, Electronic Media Element)

- Abdominal Pain
- Appendicitis
- Bowel Obstruction
- Diverticulitis
- Intussusceptions



CODES

ICD9

751.0 Meckel's diverticulum

ICD10

Q43.0 Meckel's diverticulum (displaced) (hypertrophic)

MÉNIÈRE DISEASE

Amira M. Bass • Charles V. Pollack, Jr.



BASICS

DESCRIPTION

- Disease of the inner ear
- Classically unilateral ear involvement (may be bilateral in up to 40% of cases)
- Characterized by recurrent spontaneous and episodic vertigo, sensorineural hearing loss, “roaring” tinnitus, and aural fullness
- Estimated incidence about 15/100,000 in US
- Slight female > male (1.3:1)
- Positive family history up to 20%
- May develop at any age
 - Peak incidence is age 40–60 yr.
- Affects more Whites of Northern European descent than Africans or Blacks
- A benign disease without cure
- Can be associated with significant morbidity

ETIOLOGY

- Idiopathic
- Endolymphatic hydrops: Blockage of the endolymphatic sac and duct leading to endolymphatic outflow obstruction and increased hydraulic pressure within the endolymphatic system:
 - Increased pressure causes a break in the membrane that separates the perilymph (potassium [K]-poor extracellular fluid) and the endolymph (K-rich intracellular fluid)
 - The resultant chemical mixture bathes the vestibular nerve receptors, leading to a depolarization blockade and transient loss of function
- May be associated with structural abnormalities such as atrophy of the sac, hypoplasia of the vestibular aqueduct, narrowing of the endolymphatic duct, forwardly located lateral sinus causing compression, and obstruction of the endolymphatic sac
- Autoimmune processes suggested as an etiology, with immune complex deposition in endolymphatic sac and autoantibodies directed against endolymphatic sac
- Thought to have a genetic predisposition, leading to earlier age of onset and more severe symptoms
- Other proposed mechanisms are subclinical viral infection causing hydrops many decades later, and ischemia of the endolymphatic sac and the inner ear
- Need to differentiate Ménière syndrome from other disease processes that interfere with normal production or resorption of endolymph (e.g., thyroid disease, inner

ear inflammation due to syphilis, medication)



DIAGNOSIS

- Diagnosis based upon clinical symptoms and neurotologic evaluation
- Definitive diagnosis currently can only be made postmortem, though MRI holds potential for definitive diagnosis
- Diagnostic criteria (1995 American Academy of Otolaryngology guidelines):
 - At least 2 episodes of spontaneous and episodic vertigo, ≥ 20 min
 - At least 1 episode of hearing loss documented by audiogram
 - Tinnitus or aural fullness in the affected ear
 - Certain Ménière disease: Definite disease with histopathologic confirmation
 - Definite Ménière disease: 2 or more definitive episodes of vertigo with hearing loss, plus tinnitus, aural fullness, or both
 - Probable Ménière disease: Only 1 definitive episode of vertigo and the other symptoms and signs
 - Possible Ménière disease: Definitive vertigo with no associated hearing loss or hearing loss with nondefinitive disequilibrium

SIGNS AND SYMPTOMS

History

- Classical tetrad of symptoms:
 - Vertigo
 - Hearing loss
 - Tinnitus
 - Aural fullness
- Vertiginous attacks lasting minutes to hours, often associated with nausea and vomiting (96.2%)
- Sensorineural hearing loss is typically fluctuating and progressive
 - Low frequencies are affected more severely than high frequencies (87.7%)
 - Can result in permanent hearing loss at all frequencies
- Tinnitus is typically low pitch
- Aural fullness is described as pressure, discomfort, fullness sensation in unilateral ear
- Attacks reach maximum intensity within minutes, slowly subside over hours
- After the acute attack, patients generally feel tired, unsteady, and nauseated for hours to days
- Between episodes, some patients are completely symptom free
- Sudden, unexplained falls without loss of consciousness or associated vertigo may also occur
- Constellation of symptoms can vary from patient to patient
 - Auditory and vestibular symptoms may not be present simultaneously or in

the same pattern, particularly in the early phases of disease

- Close clustering of attacks may occur

Physical-Exam

- Exam results vary, depending upon the phase of disease
- During acute attack, patients are often in significant distress, diaphoretic, and pale
- Vital signs may show elevated blood pressure, pulse, and respiration
- Horizontal nystagmus
- Impaired hearing
- Pneumo-otoscopy may elicit symptoms or cause nystagmus
- Weber tuning fork test usually lateralizes away from the affected ear
- Rinne test usually indicates better air than bone conduction
- Positive Romberg test, with instability, especially when eyes are closed
- Must exclude central CNS lesion, peripheral pathology in ear (ruptured tympanic membrane, cholesteatoma, cerumen impaction, etc.)

ESSENTIAL WORKUP

- Complete history and neurologic exam
- Patients with central vertigo or focal neurologic findings require neuroimaging
- Focal findings include new unilateral hearing loss, usually with tinnitus

DIAGNOSIS TESTS & INTERPRETATION

Lab

When indicated:

- CBC
- Sedimentation rate
- Thyroid function
- Fasting lipid profiles
- Fasting blood glucose, hemoglobin A1c
- Treponemal antibody-absorption test
- Chemistry panel
- Urinalysis for proteinuria or hematuria

Imaging

- MRI with intratympanic gadolinium and views of internal auditory canal (typically outpatient)
- CT scan of temporal bone
- Standard lateral mastoid radiographs

Diagnostic Procedures/Surgery

- Audiometric assessment
- Bithermal caloric testing

- Transtympanic electrocochleography
- Electronystagmography

DIFFERENTIAL DIAGNOSIS

- Otologic:
 - Chronic suppurative otitis media
 - Benign positional vertigo
 - Acoustic neuroma
 - Vestibular neuronitis
 - Vestibular paroxysmia
 - Otosclerosis
 - Otic capsule dysplasia
 - Perilymphatic fistula
 - Labyrinthitis
- Systemic:
 - Vertebrobasilar insufficiency
 - Stroke and transient ischemic attack
 - Basilar artery thrombosis
 - Intracranial hemorrhage
 - Head trauma
 - CNS lesions (tumors)
 - Epilepsy
 - Multiple sclerosis
 - Paget disease
 - Diabetes
 - Concussive syndrome
 - Pseudotumor cerebri
 - Complex or vestibular migraine
 - Thyroid dysfunction
 - Drugs/medications
 - Autoimmune disorders (e.g., systemic lupus erythematosus)
 - Viral meningitis/encephalitis
 - Lyme disease
 - Neurosyphilis
 - Electrolyte imbalance
 - Panic attacks



TREATMENT

PRE HOSPITAL

- Vertigo and neurologic symptoms can represent a stroke
- Rapid transport to ED

- Protect patient from falling
- Maintain patient in comfortable position
- IV isotonic fluids for patients with vomiting
- Monitor for dysrhythmia

INITIAL STABILIZATION/THERAPY

- IV hydration with isotonic fluids
- IV benzodiazepines
- IV antiemetics

ED TREATMENT/PROCEDURES

Supportive therapy

MEDICATION

- Symptomatic:
 - Meclizine: 12.5–25 mg PO q8h
 - Diazepam: 5–10 mg PO/PR/IV
 - Lorazepam: 0.5–2 mg PO/IV/IM
 - Dimenhydrinate: 12.5–50 mg PO
 - Ondansetron: 4–8 mg IV/IM/PO
 - Metoclopramide: 10 mg IV/IM
 - Promethazine: 10–25 mg PO/PR/IV/IM
 - Prochlorperazine: 10 mg IV
- Therapeutic:
 - Hydrochlorothiazide: 25–50 mg PO daily
 - Triamterene: 100 mg PO daily
 - Acetazolamide: 250 mg PO daily
 - Furosemide: 20 mg PO daily
 - Prednisone: 1 mg/kg PO daily with taper over 7–14 days
 - Dexamethasone: 4 g/L transtympanic injection
 - Gentamicin transtympanic perfusion
 - Pressure pulse treatment
 - Surgery (surgical labyrinthectomy, vestibular neurectomy, sacculotomy)

First Line

- Diazepam or lorazepam
- Ondansetron for nausea, vomiting
- IV fluid

Second Line

- Meclizine
- Prochlorperazine



FOLLOW-UP

DISPOSITION

Admission Criteria

Patient refractory to acute control of vertigo and associated effects (e.g., dehydration from protracted vomiting)

Discharge Criteria

- Tolerate oral fluids
- Steady gait
- Normal neurologic exam
- Fall precautions
- Recurrent attacks are typical
- Dietary restrictions: Sodium, caffeine, chocolate, tobacco, and alcohol intake
- Patient needs to avoid driving, operating dangerous equipment, and working at heights until attacks have resolved and sedating medications have been withdrawn

Issues for Referral

- Persistent/intractable symptoms and medical treatment failures
- Presence of ear pathology

FOLLOW-UP RECOMMENDATIONS

- Proper education in terms of dietary control and avoidance techniques is helpful
- Vestibular rehabilitation can be helpful in teaching patients to cope with vertigo and imbalance
- Counsel regarding fall risks and avoiding dangerous tasks due to the unpredictable nature of the disease
- Refer to neurologist, otologist, and otolaryngologist for outpatient audiometry and electronystagmography testing

PEARLS AND PITFALLS

- Ménière disease typically presents with the classic tetrad of vertigo, hearing loss, tinnitus, and aural fullness
- Treatment focus is symptom relief, not cure
- Discharged patients should be referred to an outpatient neurologist, otologist, and otolaryngologist
- Inpatient care is generally unnecessary and is reserved for patients refractory to acute control of their symptoms or associated effects such as dehydration and vomiting
- Surgery is reserved for patients who fail medical therapy with intractable

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See Also (Topic, Algorithm, Electronic Media Element)

- Dizziness
- Labyrinthitis



CODES

ICD9

386.00 Meniere's disease, unspecified

ICD10

- H81.01 Meniere's disease, right ear
- H81.02 Meniere's disease, left ear
- H81.09 Meniere's disease, unspecified ear

MENINGITIS

Austen-Kum Chai • Patricia Shipley

BASICS

DESCRIPTION

CNS infection with inflammation of leptomeninges defined by an increased number of WBCs in the CSF often associated with fever, nuchal rigidity, headache, and altered mental status.

ETIOLOGY

- Bacterial:
 - Neonates: Group B *Streptococcus*, *Escherichia coli* and other enteric bacilli, *Listeria monocytogenes*
 - Children/adults: *Streptococcus pneumoniae*, *Neisseria meningitidis*, group B *Streptococcus* and gram-negative bacilli (< 3 yr)
 - Elderly/alcoholic: *S. pneumoniae*, gram-negative bacilli, *Listeria* spp.
 - Neurosurgical patients: *Staphylococcus* and gram-negative organisms
 - Transplant recipients and dialysis patients: Increased incidence of *Listeria* spp. infection
 - AIDS: Above, plus tuberculosis, fungal, syphilis
- Viral
- Fungal
- Chemical, drug, or toxin induced

DIAGNOSIS

SIGNS AND SYMPTOMS

- General:
 - Fever
 - Nuchal rigidity:
 - Kernig: Flexed knee resists extension (bilateral).
 - Brudzinski: Flexion of neck produces flexion at hips.
 - Kernig and Brudzinski signs are neither sensitive nor specific for meningitis.
 - Altered mental state, headache
 - Photophobia
 - Papilledema
 - Focal CNS abnormalities
 - Seizure, nonsimple
 - Petechial and palpable purpuric rash (meningococcal infection)

- Associated infections: Sinusitis, otitis media, pneumonia
- Infant/pediatric:
 - Fever or hypothermia
 - Lethargy
 - Weak suck
 - Vomiting
 - Dehydration
 - Respiratory distress
 - Apnea
 - Cyanosis
 - Bulging fontanel
 - Hypotonia
 - Meningismus often absent in < 1 yr old
- Elderly and immune compromised:
 - Confusion with or without fever
 - Less-striking symptoms overall

History

- Neonates: Prematurity, intrapartum complications as fever, prolonged rupture of membrane, antibiotic use, group B *Streptococcus* infection
- Adults: Recent travels
- Elderly: Pneumococcal vaccination status
- Immunologic incompetency suggested by frequent infections
- Recent trauma or ENT, facial, or neurologic surgery
- Shunt

ESSENTIAL WORKUP

- Treat immediately based on clinical suspicion
- Blood cultures. Give antibiotic therapy if at all possible after blood cultures but before other diagnostic procedures if patient is unstable.
- Routine CT before lumbar puncture (LP) not always required. Generally indicated with:
 - Immune deficiency/HIV
 - History of CNS disease (abscess, bleed, mass lesion, stroke, shunt)
 - History of seizure < 7 days
 - Focal neurologic deficit
 - Altered level of consciousness
 - Age > 60 yr
 - Papilledema
- LP: Every suspected meningitis patient unless contraindicated:
 - May delay LP when:
 - Risk for herniation (see above)
 - Unstable patient

- Thrombocytopenia or bleeding diathesis
- Spinal epidural abscess
- Overlying soft tissue infection
- CSF analysis:
 - _ Tube 1: Cell count and differential
 - _ Tube 2: Protein and glucose
 - _ Tube 3: Gram stain, culture, and sensitivity
- May add acid-fast bacillus smear, TB culture, India ink and fungal cultures, VDRL, cryptococcal antigen as needed
 - _ Tube 4: Repeat cell count or save for additional tests.
 - _ Check for elevated opening pressure: Normal up to 200 mm H₂O
 - _ Latex agglutination (optional):
 - Useful if other tests are not diagnostic
 - Best if urine and blood also tested
 - Detects: *Meningococcus*, *Pneumococcus*, group B *Streptococcus*, *Haemophilus influenzae*, *E. coli*, *Cryptococcus*
 - _ Polymerase chain reaction (optional):
 - Useful for virus (especially herpes simplex) and bacteria: *N. meningitidis*, *S. pneumoniae*, *H. influenzae* A and B
 - _ CSF interpretation:
 - Culture is diagnostic
 - >4 WBC/mL in CSF is highly sensitive for meningitis for age >3 mo and >9 WBC/mL for infants 29–90 days.
 - Cell count may be normal in HIV/AIDS.
 - Neonate: Up to 20–25 WBC/mL and protein up to 150 mg/dL in term and up to 100 mg/dL in preterm neonate may be normal.
 - _ Typical bacterial meningitis:
 - CSF glucose <40 mg/dL. Also ratio of CSF to blood glucose <0.6.
 - WBC >500/mL (usually 1,000–20,000). However, significantly fewer WBC count may be seen in the early course of the disease.
 - Differential >80% polymorphonuclear neutrophils (PMNs) is suggestive.
 - CSF protein >200 mg/dL. Normally <50 mg/dL.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Blood cultures (2 sets) before antibiotics
- Urine culture and urinalysis
- CBC with differential and platelets
- Electrolytes/glucose:
 - _ Calculate CSF glucose to serum glucose ratio

- Assess for metabolic acidosis, SIADH
- BUN/creatinine for medication dosing
- Prothrombin time, partial thromboplastin time, and platelet: Particularly in patients with petechiae or purpura:
 - Obtain before LP in severe sepsis or disseminated intravascular coagulation
- Toxicology studies as needed

Imaging

- CT: See essential workup section above.
- CXR: Pneumonia, TB if suspected

DIFFERENTIAL DIAGNOSIS

- Encephalitis
- Brain, spinal, epidural abscess
- Febrile seizure
- CNS/systemic lupus erythematosus cerebritis
- Intracranial bleed
- Primary or metastatic CNS malignancy
- Stroke
- Venous sinus thrombophlebitis
- Trauma
- Toxic/metabolic



TREATMENT

PRE HOSPITAL

- IV, O₂, and transport. ABCs
- Administer prophylactic antibiotics to any close personal contacts of patient diagnosed with meningococcal meningitis:
 - Adults:
 - Rifampin: 600 mg PO BID for 2 days; *or*
 - Ciprofloxacin: 500 mg PO single dose; *or*
 - Ceftriaxone: 250 mg IM (if pregnant)
 - Children:
 - Rifampin: 5 mg/kg if < 1 mo old and 10 mg/kg if > 1 mo old, BID for 4 doses

INITIAL STABILIZATION/THERAPY

- Isolate patient as appropriate.
- ABCs. Treat seizures.

ED TREATMENT/PROCEDURES

- Ideally perform LP and give antibiotic ± steroids promptly.
- If LP is delayed, give antibiotic ± steroids empirically before LP.
- If CT is indicated prior to LP, empiric antibiotic ± steroids should be given prior to CT.
- Steroids: If given, should be given prior to, or concurrently with, administration of antibiotics.
- Antibiotics:
 - _ Obtain blood cultures before antibiotics.
 - _ Do not delay giving antibiotics to obtain LP or CT unless absolutely necessary.
- IV (or IM) empiric antibiotics for presumed bacterial Infection:
 - _ Neonates:
 - 0–7 days old: Ampicillin 50–100 mg/kg q6h + gentamicin 2.5 mg/kg q8–12h
 - >7 days old: Ampicillin 50–100 mg/kg q6–8h; + cefotaxime 50 mg/kg q6h or gentamicin 2.5 mg/kg q8h
 - Add acyclovir 10–20 mg/kg q8h for suspected herpes simplex encephalitis.
 - _ Age 1–3 mo:
 - Ampicillin 50–100 mg/kg q6h; + ceftriaxone 75 mg/kg load, then 50 mg/kg q12h thereafter or cefotaxime 50 mg/kg q6h; + vancomycin 15 mg/kg q8h (if cephalosporin-resistant *S. pneumoniae* prevalent) ± dexamethasone (0.15 mg/kg q6h for 4 days)
 - _ Children > 3 mo:
 - Ceftriaxone 100 mg/kg/d or 50 mg/kg q12h or cefotaxime 50 mg/kg q6h + vancomycin 15 mg/kg q8h ± dexamethasone 0.15 mg/kg q6h for 4 days
 - Immune deficient: Add gentamicin 2.5 mg/kg q8h or amikacin 7.5 mg/kg q12h or 5 mg/kg q8h.
 - CNS surgery: Vancomycin 15 mg/kg q8h; + meropenem 40 mg/kg q8h or ceftazidime 50 mg/kg q8h or cefepime 50 mg/kg q8h
 - Penetrating head trauma: Vancomycin 15 mg/kg q8h; + cefepime 50 mg/kg q8h or ceftazidime 50 mg/kg q8h or meropenem 40 mg/kg; + gentamicin 2.5 mg/kg q8h or amikacin 5–10 mg/kg q8h
 - _ Adults:
 - Ceftriaxone 2 g q12h or cefotaxime 2 g q4–6h; + vancomycin 15–20 mg/kg q8–12h (not to exceed 2 g/dose or 60 g/kg/d); + dexamethasone (15 mg/kg) up to 10 g q6h IV, continue for 4 days if causative agent is *S. pneumoniae*
 - >50 yr: Add ampicillin 2 g q4h to above regimen for Listeria coverage
 - Immune impaired: Vancomycin 15–20 mg/kg q8–12h + ampicillin 2 g q4h; + meropenem 2 g q8h or cefepime 2 g q8h

- CNS surgery, shunt, head trauma: Vancomycin 15–20 mg/kg q8–12h; + meropenem 2 g
- Vancomycin dosing for patients with normal renal function: 50–89 kg (1 g q12h), 90–130 kg (1.5 g q12h), > 130 kg (2 g q12h)
- Other medication considerations:
 - _ Dexamethasone:
 - Benefits are not conclusive.
 - May be beneficial for children with *H. influenzae* meningitis and may be beneficial in children >6 wk and adults with *S. pneumoniae meningitis*. May reduce neurologic sequelae
 - Give before or with antibiotics in patient with altered mental status, focal neurologic deficit, papilledema, or CNS trauma, surgery, or space-occupying lesion. Give if CSF is cloudy, has positive Gram stain, or > 1,000 WBC/mm³.
 - _ Penicillin allergy (severe):
 - Aztreonam or chloramphenicol may be used in place of cephalosporins.
 - Do not delay therapy for lesser allergy history.
 - _ Vancomycin:
 - Add when concerned about penicillin-resistant pneumococcal infection.
 - _ Acyclovir if suspect herpes simplex virus encephalitis

MEDICATION

- Acyclovir: 30 mg/kg/d q8h IV (Neonate: 20 mg/kg/d q8h IV)
- Amikacin: Peds: 7.5 mg/kg q12h or 5 mg/kg q8h IV. Newborn: Load 10 mg/kg followed by 7.5 mg/kg q12h IV
- Ampicillin: 2 g q4h (peds: 50–100 mg/kg q6h–q8h) IV, max. 12 g/d
- Aztreonam: 2 g (peds: 30 mg/kg) q6–8h, max. 6–8 g/d IV
- Bactrim: 5–10 mg/kg trimethoprim q12h IV
- Cefepime: 2 g q8h, max. 6 g/d IV
- Cefotaxime: 2 g (peds: 50 mg/kg) q6h, max. 8–12 g/d IV
- Ceftazidime: 2 g q8h, max. 6 g/d IV
- Ceftriaxone: 2 g (peds: 50–75 mg/kg) q12h, max. 4 g/d IV
- Chloramphenicol: 1–1.5 g (peds: 12.5 mg/kg) q6h, max. 4–6 g/d IV
- Dexamethasone: 10 mg (peds: 0.15 mg/kg) q6h IV for 4 days
- Gentamicin: Peds: 2.5 mg/kg q8h IV
- Meropenem: 2 g (peds 40 mg/kg) q8h IV, max. 6 g/d
- Tobramycin: Peds: 2.5 mg/kg q8h IV
- Vancomycin: 1–2 g q8–12h IV (peds: 15 mg/kg q8h)
- Vancomycin and aminoglycosides: Adjust for renal function and serum concentration levels.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Known or suspected bacterial infection
- Immune-compromised host
- Any toxic-appearing patient

Discharge Criteria

- Clear viral infection. Controlled symptoms.
- Thorough and specific discharge instructions
- Careful follow-up plan discussed with primary care physician prior to discharge

PEARLS AND PITFALLS

- Meningitis generally does not present as uncomplicated febrile seizure in children.
- Failure to diagnose or delay in treatment of meningitis results in catastrophic outcome for patients, and not infrequently, negative medicolegal consequences for the physicians involved.

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See Also (Topic, Algorithm, Electronic Media Element)

Seizures



CODES

ICD9

- 320.2 Streptococcal meningitis
- 320.9 Meningitis due to unspecified bacterium
- 322.9 Meningitis, unspecified

ICD10

- G00.2 Streptococcal meningitis
- G00.9 Bacterial meningitis, unspecified
- G03.9 Meningitis, unspecified

MENINGOCOCCEMIA

Brian D. Euerle



BASICS

DESCRIPTION

- Bacterial illness caused by *Neisseria meningitidis*
- Several forms of illness may occur
- Mild meningococemia
- Overwhelming meningococcal sepsis
- Meningococcal meningitis
- Chronic/occult meningococemia
- Septic arthritis
- Acquired from close contact with an infected individual or an asymptomatic carrier
- Intimate kissing and cigarette smoking are independent risk factors.

ETIOLOGY

- *N. meningitidis*:
 - Serotypes A, B, C, D, H, I, K, L, X, Y, Z, 29E, and W135
 - Serotype B is most common in US
 - Majority of infections caused by A, B, C, X, Y, and W135
- Bacteria attach to and enter nasopharyngeal epithelial cells.
- Bacteria spread from the nasopharynx through the bloodstream via entry of vascular endothelium.
- Most circulating meningococci are eliminated by the spleen.
- Meningococci produce an endotoxin (lipooligosaccharide):
 - Involved in pathogenesis of the skin, adrenal manifestations, and vascular collapse
- Human oropharynx/nasopharynx is the only reservoir.
- Carrier usually has developed immunity to serotype-specific antibody (not immune to all serotypes):
 - Age < 5 yr: 1% carrier rate
 - Age 20–40 yr: 30–40% carrier rate
 - Lower rate of immunity in children, which is reflected by the higher rates of infection
- Most common in fall and spring
- Increased incidence in military recruits and close living conditions
- Epidemics—ages 5–9 yr most/earliest affected



DIAGNOSIS

SIGNS AND SYMPTOMS

- “Mild” meningococemia:
 - _ Most common
 - _ Preceded by upper respiratory infection
 - _ Fever, chills, myalgias/artralgias, malaise
 - _ Often self-limited, resolving in several days
 - _ Can progress to meningitis (mortality rate 2–10%) or overwhelming sepsis without meningitis
- Overwhelming meningococcal sepsis:
 - _ 10% of overall meningococemia cases
 - _ High mortality rate (20–60%)
 - _ Most deaths occur in 1st 48 hr
 - _ Sudden onset of illness and rapid progression of clinical course
 - _ Initial presentation may be mild:
 - Mild tachycardia
 - Mild tachypnea/respiratory symptoms
 - Mild hypotension
 - _ Fever, chills, vomiting, headache, rash, muscle tenderness
 - _ Toxic appearing
 - _ Infants: Lethargy, poor feeding, bulging fontanel
 - _ Rash:
 - Combination of purpura/ecchymosis
 - May later exhibit coalescence, necrosis/sloughing of the involved skin (purpura fulminans)
 - Petechiae (over skin, mucous membranes, conjunctivae) seen in 50–60%
 - Macules
 - Papules (scrapings of papules demonstrate the organism on Gram stain)
 - _ Deteriorate quickly over several hours:
 - Hypotension/shock
 - Acidosis
 - Acute respiratory distress syndrome (ARDS)
 - Disseminated intravascular coagulation (DIC)
 - _ Meningitis may or may not be present.
 - _ Waterhouse–Friderichsen syndrome:
 - Bilateral hemorrhagic destruction of adrenal glands
 - Vasomotor collapse
 - _ Acute renal failure:
 - From prolonged hypotension (low renal perfusion causing acute tubular necrosis)
- Chronic meningococemia:
 - _ Uncommon

- Well appearing
- Recurrent fevers, chills, arthralgias over weeks to months
- Intermittent rash—painful on the extremities
- Migratory polyarthritits
- Splenomegaly (20%)
- Meningococcal meningitis:
 - Headache
 - Fever
 - Neck stiffness
 - Confusion
 - Lethargy
 - Obtundation
- Septic arthritis:
 - Occurs during active meningococemia
 - Multiple joints involved
 - Joint pain, redness, swelling, effusion, fever, chills
 - Extremely limited or no range of motion
- Other meningococcal infections:
 - Occur with meningococcal infection elsewhere
 - Conjunctivitis—may occur alone
 - Sinusitis
 - Panophthalmitis
 - Urethritis
 - Salpingitis
 - Prostatitis
 - Pneumonia
 - Myocarditis/pericarditis:
 - Occurs late in onset
 - Usually associated with serogroup C

History

Progression of illness is variable and classifies illness into mild, overwhelming, and chronic.

Physical-Exam

- Tachycardia
- Hypotension, which may be mild initially
- Progressive, rapid deterioration
- Respiratory failure with ARDS picture
- Petechial rash 50–80%:
 - Involves axillae, flanks, wrists, ankles

ESSENTIAL WORKUP

- Do not allow workup (including delay in lumbar puncture) to postpone resuscitation and administration of antibiotics in suspected cases of meningococemia.
- Suspect diagnosis in setting of dramatic clinical presentation.
- Gram stain and culture of:
 - Peripheral blood, CSF, sputum, urine, joint aspirate, or petechial/papular scrapings
 - Gram stain: Intracellular or extracellular gram-negative diplococci

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC:
 - Elevated WBCs initially; later may be suppressed in severe disease
 - Decreased platelet count when large areas of purpura/petechiae or DIC
- Electrolytes, BUN, creatinine, glucose
- CSF:
 - Gram stain, culture, protein and glucose, cell count with differential
 - Consistent with bacterial infection in meningococcal meningitis
- Arterial blood gases for acidosis, hypoxia
- Fibrinogen levels, fibrin degradation products, prothrombin time, partial thromboplastin time if DIC suspected
- Throat/nasopharyngeal swab:
 - Positive swab does not establish the diagnosis of meningococemia.
- Analysis of buffy-coat layer of peripheral blood for bacteria if sepsis is suspected
- Blood culture:
 - Often negative with chronic meningococemia
 - Positive in mild and overwhelming meningococemia
- Immunoassays (beware false negatives)
- Polymerase chain reaction, especially useful when antibiotics given before specimen collection

Imaging

CXR: For ARDS/pneumonia

Diagnostic Procedures/Surgery

Amputations and débridement of necrotic tissue and/or extremities may be necessary.

DIFFERENTIAL DIAGNOSIS

- Viral exanthem
- Vasculitis
- Mycoplasma
- Rocky Mountain spotted fever

- Toxic shock syndrome
- Henoch–Schönlein purpura
- Idiopathic thrombocytopenic purpura
- Dengue fever
- Disseminated gonococcal infection
- Influenza
- *Streptococcus* group A and B
- Thrombotic thrombocytopenic purpura



TREATMENT

PRE HOSPITAL

Postexposure prophylaxis needed for pre-hospital personnel in close contact with patient

INITIAL STABILIZATION/THERAPY

- Wear mask and gloves, observe droplet precautions.
- Notify department of health.
- ABCs
- Immediate endotracheal intubation for severe acidosis, hypoxia, or decreased mental status:
 - Hyperventilate to treat acidosis (target PCO₂ about 25 mm Hg)
- Treat hypotension:
 - 0.9% normal saline bolus of 20 mL/kg; cautious rehydration with ARDS, CHF
 - Begin dopamine or norepinephrine (epinephrine if no response) if hypotensive after 2 L of IV fluids.
- Naloxone, thiamine, dextrose (Accu-Chek) for altered mental status
- Initiate IV antibiotics:
 - 1st line: High-dose penicillin (proven meningococemia) or 3rd-generation cephalosporin (broader coverage pending definitive diagnosis)
 - 2nd line: Ampicillin
 - 3rd line: Chloramphenicol (penicillin-allergic patients)

ED TREATMENT/PROCEDURES

- Overwhelming meningococcal sepsis
- Severe acidosis (pH <7–7.1 or serum HCO₃ <8–10):
 - Administer IV NaHCO₃ along with hyperventilation.
- Insert Foley catheter to monitor urine output.
- Place in respiratory isolation.
- High-dose steroids:
 - To protect against cranial nerve injury in the setting of ongoing infection

(controversial)

- Administer with adrenal gland injury.
- DIC treatment:
 - Administer fresh-frozen plasma and platelet transfusions.
 - Heparin is not indicated unless significant thrombotic complications are evident clinically (e.g., cyanosis or cold digits, low urine output despite adequate volume status, and blood pressure).
- Prophylaxis options for close contacts:
 - Ideally, prophylaxis should be given within 1st 24 hr.
 - 10-day window of observation
 - Serogroup-specific vaccine as adjunct only
- Vaccine:
 - Vaccine recommended in military recruits, travelers to endemic areas, complement-deficient or asplenic patients, 1st-year college dormitory residents
 - Vaccine recommended routinely for ages 11–18 yr

Pregnancy Considerations

The safety of meningococcal vaccine is unclear in pregnancy.

MEDICATION

First Line

- Cefotaxime: 2 g (peds: 50 mg/kg) IV q6h
- Ceftriaxone: 2 g (peds: 50 mg/kg) IV q12h
- Penicillin G: 4 MU (peds: 250,000 U/kg/24 h) IV q4h

Second Line

- Ampicillin: 2–3 g (peds: 200–400 mg/kg/24 h) IV q6h
- Chloramphenicol: 50–100 mg/kg/24 h IV q6h (max. 4 g/d)
- Prophylaxis:
 - Single-dose ceftriaxone:
 - 125 mg IM for age <15 yr
 - 250 mg IM for age >15 yr
 - Ciprofloxacin: 500 mg PO (adults)
 - Rifampin: 600 mg (peds: 5–10 mg/kg) PO BID for 2 days
 - Azithromycin 500 mg PO single dose (not routinely used)
- Dexamethasone: 0.15 mg/kg IV for pediatric meningitis
- Dopamine: 5–20 ug/kg/min IV titrate to blood pressure (BP)
- Epinephrine: 2–10 ug/min IV titrate to BP
- Heparin: 3,000–5,000 U (peds: 80 U/kg) IV bolus followed by 600–1,000 U/h (peds: 18 U/kg/h) IV drip

- Hydrocortisone (Solu-Cortef): 100 mg (peds: 2 mg/kg) bolus IV for adrenal insufficiency q8h
- Meningococcal polysaccharide 0.5 mL IM × 1
- Meningococcal vaccine 0.5 mL SC × 1
- Norepinephrine: 0.5–30 ug/min IV titrate to BP
- Sodium bicarbonate: 2–5 mEq/kg (peds: 0.5–1 mEq/kg) IV over 30 min to 4 hr



FOLLOW-UP

DISPOSITION

Admission Criteria

- ICU admission for overwhelming sepsis with respiratory isolation
- Respiratory isolation admission for mild meningococemia

Discharge Criteria

Prophylaxis for close patient contacts

Issues for Referral

- Consider transfer to tertiary care center, as multisystem organ failure is common.
- Late neurologic, cardiovascular, and orthopedic complications may necessitate follow-up with specialists.

FOLLOW-UP RECOMMENDATIONS

- Complete antibiotic course.
- Respiratory precautions may be discontinued after 24 hr.
- All close contacts need prophylaxis.

PEARLS AND PITFALLS

- Notify department of health in any suspected case.
- Watch for late development of pericardial tamponade.
- Do not wait to give antibiotics.

ADDITIONAL READING

- Apicella M. *Neisseria meningitidis*. In: Mandell GL, Bennett JE, Dolin R, eds. *Principles and Practice of Infectious Disease*. 7th ed. Philadelphia, PA: Churchill Livingstone Elsevier; 2010:2737–2752.
- Cramer JP, Wilder-Smith A. Meningococcal disease in travelers: Update on vaccine options. *Curr Opin Infect Dis*. 2012;25(5):507–517.
- Pace D, Pollard AJ. Meningococcal disease: Clinical presentation and sequelae. *Vaccine*. 2012;30(suppl 2):B3–B9.

- [Rosenstein R, Perkins BA, Stephens DS, et al. Meningococcal disease. *N Engl J Med.* 2001;344\(18\):1378–1388.](#)



See Also (Topic, Algorithm, Electronic Media Element)

- [Meningitis](#)
- [Sepsis](#)



CODES

ICD9

036.2 Meningococcemia

ICD10

- [A39.2 Acute meningococcemia](#)
- [A39.3 Chronic meningococcemia](#)
- [A39.4 Meningococcemia, unspecified](#)

MERCURY POISONING

Keri L. Carstairs • David A. Tanen



BASICS

DESCRIPTION

Mercury:

- 3 forms: Elemental, inorganic salts, and organic
- Reacts with sulfhydryl groups, causing enzyme inhibition and alterations in cellular membranes
- Binds to phosphoryl, carboxyl, amide, and amine groups of enzymes

ETIOLOGY

- Exposure is usually through the GI tract and inhalation and less frequently dermal exposure.
- Exposure through manufacturing of chlorine and caustic soda, diuretics, antibacterial agents, antiseptics, thermometers, batteries, fossil fuels, plastics, paints, jewelry, lamps, explosives, fireworks, vinyl chloride, and pigments
- Exposure through taxidermy, photography, dentistry, mercury mining
- Contaminated seafood



DIAGNOSIS

SIGNS AND SYMPTOMS

- Naturally occurring mercury is converted into 3 primary forms, each with its toxicologic effects:
- Elemental mercury:
 - _ Symptoms from inhalation occur within hours:
 - Cough and dyspnea, which may progress to pulmonary edema
 - Metallic taste, salivation
 - Weakness, nausea, diarrhea, fever, headaches, visual disturbances
 - _ Subcutaneous deposits may present as granulomas or abscesses.
 - _ IV exposure presents with symptoms consistent with pulmonary embolization.
 - _ Relatively nontoxic from oral ingestion, although appendicitis has been reported
- Inorganic mercurial salt ingestion:
 - _ Caustic GI injury:
 - Abdominal pain with nausea, vomiting, and diarrhea
 - Metallic taste, sore throat
 - Hemorrhagic gastroenteritis with hematochezia and hematemesis

- Acute tubular necrosis
- Acrodynia (pink disease):
 - Idiosyncratic, occurs mainly in children
 - Painful extremities
- Pink discoloration with desquamation
- Organic mercury ingestion:
 - Historically, infants exposed in womb are most severely affected (e.g., Minamata Bay, Japan)
 - May see GI symptoms acutely
 - Delayed CNS toxicity predominates and may take weeks to months to manifest:
 - Paresthesias
 - Ataxia
 - Paralysis
 - Visual field constriction
 - Dysarthria
 - Hearing loss
 - Mental deterioration
 - Death

History

- Ask about possible workplace, environmental, or accidental exposure to mercurial products.
- Document the patient's ingestion of seafood over the last few weeks.

Physical-Exam

- Elemental mercury:
 - Cough progressing to respiratory distress if inhaled or intravenously injected
 - Ataxia
 - Subcutaneous nodules or granulomas if injected
- Inorganic mercury:
 - Oral burns
 - Abdominal tenderness
 - Heme-positive stools
- Organic mercury:
 - CNS abnormalities:
 - Progressive cognitive deterioration

ESSENTIAL WORKUP

- Good history for workplace or environmental exposure
- Physical exam looking for:
 - Respiratory distress

- Caustic GI injury
- Neuropsychiatric impairment
- Lab tests:
 - Renal failure
 - Urine and blood mercury levels:
 - Not reliable with recent seafood ingestion

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Inorganic mercury exposure:
 - CBC
 - Electrolytes, BUN, creatinine, glucose
 - 24-hr urine mercury collection:
 - Normal urine levels < 20 mg/dL
 - Whole-blood mercury level:
 - Normal blood < 10 mg/dL
- Organic mercury exposure:
 - CBC with peripheral smear
 - Electrolytes, BUN, creatinine, glucose
 - Whole-blood mercury level:
 - Normal blood < 10 mg/dL

Imaging

- Chest radiograph:
 - For noncardiac pulmonary edema
 - Evidence of IV mercury in pulmonary vascular tree
- Abdominal radiograph:
 - For presence of mercury with intentional oral ingestion
- Head CT:
 - May detect cerebellar atrophy

Diagnostic Procedures/Surgery

Lumbar puncture in the workup of altered mental status

DIFFERENTIAL DIAGNOSIS

- Multisystem involvement is often confused with other heavy-metal intoxications.
- Cerebrovascular accident
- Senile dementia, Alzheimer disease
- Parkinson disease
- Peptic ulcer disease
- Gastrointestinal bleeding
- Pancreatitis

- Sepsis
- Acute respiratory distress syndrome



TREATMENT

PRE HOSPITAL

- Remove from toxin exposure.
- Decontamination:
 - Wash exposed skin.
- For altered mental status:
 - Dextrose
 - Thiamine
 - Naloxone (Narcan)
 - Oxygen

INITIAL STABILIZATION/THERAPY

- Secure ABCs and monitoring.
- 0.9% NS
- IV fluid resuscitation for hypotension:
 - Blood transfusion for significant gastrointestinal hemorrhage
- Naloxone, D₅₀W, thiamine for altered mental status

ED TREATMENT/PROCEDURES

- Elemental mercury:
 - For inhalation exposure, observe closely for several hours for development of noncardiogenic pulmonary edema.
 - Ingested elemental mercury passes through normal intestinal tract with minimal absorption.
 - Consider chelation for symptomatic patients with oral dimercaptosuccinic acid (DMSA).
 - For subcutaneous nodules/abscess, perform an incision and drainage.
- Inorganic mercury salt ingestion:
 - Administer activated charcoal.
 - Aggressive 0.9% NS IV fluid resuscitation/blood products for hypovolemic shock:
 - Hydrate and maintain urine output (1 mL/kg/h).
 - Chelate symptomatic patients:
 - IM dimercaprol (British anti-Lewisite [BAL])
 - Oral DMSA efficacy may be limited secondary to caustic GI injury.
- Organic mercury:
 - Administer activated charcoal.
 - Chelate with oral DMSA.

MEDICATION

First Line

- Dextrose: D₅₀W 1 amp: 50 mL or 25 g (peds: D₂₅W 2–4 mL/kg) IV
- Dimercaprol (BAL): 5 mg/kg IM q4h for 48 hr, then 2.5 mg/kg q6h for 48 hr, then 2.5 mg/kg q12h for 7 days
- DMSA: 10 mg/kg PO q8h for 5 days, then q12h for 2 wk
- Naloxone (Narcan): 2 mg (peds: 0.1 mg/kg) IV/IM initial dose
- Thiamine (vitamin B₁): 100 mg (peds: 50 mg) IV or IM

Second Line

- D-penicillamine:
 - _ Adult: 250 mg PO QID for 7–14 days
 - _ Peds: 5–7 mg/kg PO QID for 7–14 days
- 2,3-Dimercapto-1-propanesulfonate:
 - _ IV or PO formulations. Contact your poison center at 1-800-222-1222 for availability.



FOLLOW-UP

DISPOSITION

Admission Criteria

Acutely symptomatic patients:

- Any evidence of respiratory compromise
- Ingestion of inorganic mercury salt that may lead to a caustic GI injury
- Renal impairment
- Any patient starting chelation therapy

Discharge Criteria

- Asymptomatic patient with history of ingestion of elemental mercury and intact intestinal tract
- Patient with history of inhalation exposure to elemental mercury who remain asymptomatic after 6 hr of observation

Issues for Referral

- Medical toxicology referral for symptomatic patients or where chelation is considered
- Gastroenterology for caustic GI injury
- Pulmonary/ICU care for patients with symptomatic inhalational injury
- Neurology in the evaluation of progressive cerebral deterioration

- Poison center for all suspected exposures

FOLLOW-UP RECOMMENDATIONS

- For discharged patients with possible workplace or environmental exposures, follow up with their primary care provider for results of 24-hr urine or whole-blood mercury levels.
- Outpatient referral to medical toxicology for suspected or confirmed cases
- For the asymptomatic patient, have the patient refrain from eating seafood for 2 wk before repeating the 24-hr urine for mercury.

PEARLS AND PITFALLS

- Obtain a good history for workplace, environmental or accidental exposure in patients with gastrointestinal and/or neuropsychiatric complaints.
- Monitor patients for at least 6 hr if they were exposed to inhalational elemental mercury.
- Ingestion of inorganic mercurial salts can lead to significant caustic GI injury.
- Lab tests may yield false positives especially in patients who eat seafood.

ADDITIONAL READING

- Clarkson TW, Magos L, Myers GJ. The toxicology of mercury—current exposures and clinical manifestations. *N Engl J Med.* 2003;349:1731–1737.
- Rocha JB, Aschner M, Dórea JG, et al. Mercury toxicity. *J Biomed Biotechnol.* 2012;2012:831890.
- Young-Jin S. Mercury. In: Flomenbaum NE, Goldfrank LR, Hoffman RS, et al., eds. *Goldfrank's Toxicologic Emergencies.* 9th ed. New York, NY: McGraw-Hill; 2010.



See Also (Topic, Algorithm, Electronic Media Element)

- Respiratory Distress
- Caustic Ingestion
- Renal Failure
- Psychosis, Medical vs. Psychiatric



CODES

ICD9

- 961.2 Poisoning by heavy metal anti-infectives
- 976.0 Poisoning by local anti-infectives and anti-inflammatory drugs
- 985.0 Toxic effect of mercury and its compounds

ICD10

- T37.8X1A Poisoning by oth systemic anti-infect/parasit, acc, init
- T49.0X1A Poisoning by local antifung/infect/inflamm drugs, acc, init
- T56.1X1A Toxic effect of mercury and its compounds, accidental (unintentional), initial encounter

MESENTERIC ISCHEMIA

Rashid F. Kysia



BASICS

DESCRIPTION

- Decreased or occluded blood flow through the mesenteric vessels leading to ischemic or infarcted bowel
- Can be from arterial or venous blockage, or low flow states.
- 1 in 1,000 of all hospital admissions
- 1–2% of all admissions for abdominal pain:
 - Most cases occur in patients > 50 yr.
 - Mortality as high as 60–70%, particularly if diagnosis/presentation delayed > 24 hr

ETIOLOGY

- Acute mesenteric arterial embolism:
 - 50% of cases of acute mesenteric ischemia
 - Mean age 70 yr
 - Emboli most commonly arise in left atria or ventricle, from a dysrhythmia, valvular lesions, or ventricular thrombus from a prior MI
 - Typically lodge 3–10 cm distal to the origin of the superior mesenteric artery (SMA):
 - Preserves blood flow to proximal small and large bowel
 - Risk factors include dysrhythmia (especially atrial fibrillation), valvular heart disease, prior MI, aortic aneurysm, or dissection.
- Mesenteric artery thrombus:
 - SMA thrombus in 15% of cases of acute mesenteric ischemia
 - Rare in other vessels
 - Develops from plaque rupture of mesenteric atherosclerotic disease
 - 50–80% may have longstanding intestinal angina (chronic mesenteric ischemia).
 - Risk factors include age, atherosclerotic disease, HTN.
- Mesenteric venous thrombosis:
 - 5–15% of cases of acute mesenteric ischemia
 - Subacute/indolent presentation
 - 20–40% mortality
 - Typically occurs in younger patients with underlying hypercoagulable state
 - Risk factors include:
 - Hypercoagulable state (lupus, protein C and S deficiency)
 - Sickle cell disease

- Antithrombin III deficiency
- Malignancy (particularly portal)
- Pregnancy
- Sepsis
- Renal failure on dialysis
- Estrogen therapy
- Recent trauma or inflammatory conditions
- Nonocclusive mesenteric ischemia:
 - 20–30% of cases of acute mesenteric ischemia
 - Occurs in low cardiac output states with decreased mesenteric blood flow
 - Risk factors include CHF, sepsis, hypotension, hypovolemia, diuretic use, recent surgery (especially cardiac), or recent vasopressor requirement.
 - Poorer survival rates
- Chronic mesenteric ischemia:
 - “Intestinal angina”:
 - Postprandial, diffuse abdominal pain occurring ~1 hr after eating, lasts 1–2 hr
 - Patients may develop food aversions and eat small meals to avoid pain.
- Uncommon causes:
 - Spontaneous mesenteric arterial dissection
 - Median arcuate ligament syndrome—compression of the celiac axis or SMA by the arcuate ligament of the diaphragm
 - Extrinsic compression from tumors
 - Medications:
 - Digitalis
 - Ergotamine
 - Cocaine
 - Pseudoephedrine
 - Vasopressin



DIAGNOSIS

SIGNS AND SYMPTOMS

- Sudden-onset, severe, diffuse abdominal pain in acute ischemia:
 - Pain out of proportion to exam:
 - Patients may have relatively benign abdominal exam despite severe pain.
- Nausea
- Vomiting
- Diarrhea
- Occult GI bleeding

- Elderly patients can have nonspecific symptoms such as altered mental status, tachypnea, or tachycardia.
- Late findings:
 - _ Peritoneal signs owing to irreversible bowel ischemia
 - _ Abdominal distention
 - _ Hypoactive bowel sounds

History

Rapidity of onset of pain

Physical-Exam

Abdominal pain out of proportion to physical exam during the acute phase of illness

ESSENTIAL WORKUP

Maintain a high index of suspicion in patients >50 yr old with unexplained abdominal pain.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Often nonspecific and nondiagnostic
- CBC:
 - _ Elevated WBC count (90% > 15,000)
- Chemistry panel:
 - _ Approximately 50% have a metabolic acidosis.
- Amylase:
 - _ Elevated amylase found in 50% of cases
- Creatine phosphokinase (CPK) may be elevated.
- Lactate:
 - _ Elevated in 90% of patients
 - _ Indicative of advanced tissue damage, may not be elevated early in ischemic course.
 - _ High levels correlate with mortality.

Imaging

- Flat and upright abdominal radiographs:
 - _ Often obtained to rule out acute obstruction or perforation
 - _ Frequently normal
 - _ Late findings:
 - Thumbprinting from bowel wall edema and hemorrhage
 - Pneumatosis intestinalis: Air in bowel wall from tissue necrosis
 - Pneumobilia is a late finding associated with poor outcomes
- Abdominal CT scan:

- Can detect bowel wall edema, pneumatosis
- Newer helical and multidetector CT (MDCT) scanners can directly visualize mesenteric vascular anatomy and localize sites of occlusion
- MDCT angiography is more frequently the imaging modality of choice
- MRI:
 - Excellent images of mesenteric vasculature especially with MR angiography
 - Acquisition time and availability limits utility
- Angiography:
 - Historically the gold standard diagnostic modality, now being replaced by MDCT
 - Allows for direct visualization of emboli and administration of vasodilating or fibrinolytic agents
 - Invasive, time-consuming, and potentially nephrotoxic
- Doppler US:
 - Can detect decreased blood flow in SMA but more helpful in chronic mesenteric ischemia
 - For optimal results the patient should be NPO for 8 hr, limiting the utility of this study in the ED

DIFFERENTIAL DIAGNOSIS

- Bowel obstruction
- Volvulus
- GI malignancy
- Diverticulitis
- Inflammatory bowel disease
- Peptic ulcer disease
- Perforated viscus
- Cholecystitis
- Ascending cholangitis
- Pancreatitis
- Appendicitis
- Abdominal aortic aneurysm
- MI
- Renal stones



TREATMENT

PRE HOSPITAL

Initiate fluid replacement for dehydrated or hypotensive patients.

INITIAL STABILIZATION/THERAPY

- Airway, breathing, and circulation management (ABCs) with fluid resuscitation as

needed

- Caution:
 - Early diagnosis and intervention is critical to decrease mortality.

ED TREATMENT/PROCEDURES

- General measures:
 - Nasogastric suction to decompress the stomach and bowel
 - NPO
 - Electrolyte replacement as needed
 - Cardiac monitor for dysrhythmia
 - Consider invasive cardiac monitoring if patient is unstable
 - Monitor urine output
 - Analgesics
 - Broad-spectrum antibiotics to cover bowel flora (may need to adjust dose if concomitant renal failure):
 - Piperacillin/tazobactam
 - Ampicillin/sulbactam
 - Ticarcillin/clavulanate
 - Alternatives include imipenem, meropenem, 3rd-generation cephalosporins + metronidazole
 - Anticoagulation with heparin
 - Surgical consultation: All patients with peritoneal signs should have exploratory laparotomy.
- Specific therapies:
 - Papaverine 30–60 mg/h intra-arterial:
 - Phosphodiesterase inhibitor causes mesenteric vasodilatation.
 - Administered through angiography catheter
 - Intra-arterial thrombolytics can be used.
 - Surgical revascularization often indicated
- Caution:
 - Avoid vasoconstrictive medications, which may worsen ischemia:
 - If vasopressors are needed, use agents with less impact on mesenteric perfusion—consider dobutamine, low-dose dopamine, milrinone.

MEDICATION

- Ampicillin/sulbactam: 3 g IV q6h (peds: 100–200 mg/kg/d)
- Heparin sulfate: 80 U/kg IV bolus followed by 18 U/kg/h infusion
- Metronidazole: 1 g IV bolus followed by 500 mg IV q6h (peds: 12 mg/kg IV bolus, then 7.5 mg/kg IV q6h)
- Piperacillin/tazobactam: 3.375 g IV q6h (peds: 240–400 mg/kg/d)
- Ticarcillin/clavulanate: 3.1 g IV q4–6h



FOLLOW-UP

DISPOSITION

Admission Criteria

Admit all patients with mesenteric ischemia.

Discharge Criteria

None

FOLLOW-UP RECOMMENDATIONS

Surgical consultation

PEARLS AND PITFALLS

- Aggressive pursuit of diagnosis is mandatory.
- Mortality rises to 80% when the diagnosis is made > 24 hr after symptom onset.
- Early surgical evaluation for emergent operative intervention is mandatory.

ADDITIONAL READING

- Cangemi JR, Picco MF. Intestinal ischemia in the elderly. *Gastroenterol Clin North Am.* 2009;38:527–540.
- Krupski WC, Selzman CH, Whitehill TA. Unusual causes of mesenteric ischemia. *Surg Clin North Am.* 1997;77(2):471–499.
- Lewiss RE, Egan DJ, Shreves A. Vascular abdominal emergencies. *Emerg Med Clin North Am.* 2011;29(2):253–272.
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- Tekwani T, Sikka R. High-risk chief complaints III: Abdomen and extremities. *Emerg Med Clin North Am.* 2009;27(4):747–765.



See Also (Topic, Algorithm, Electronic Media Element)

Abdominal Pain



CODES

ICD9

- 557.0 Acute vascular insufficiency of intestine
- 557.1 Chronic vascular insufficiency of intestine

ICD10

- K55.0 Acute vascular disorders of intestine
- K55.1 Chronic vascular disorders of intestine

METACARPAL INJURIES

Davut J. Savaser • David Palafox

BASICS

DESCRIPTION

- Most metacarpal injuries are caused by crush injuries, a direct blow with hand vs. object, or burns.
- Most common fracture is boxer's fracture of distal 5th metacarpal neck.

DIAGNOSIS

SIGNS AND SYMPTOMS

- Pain or swelling at the site of injury
- Deformity at the site of injury
- Malalignment of the distal tip of the finger on flexion indicates rotational deformity.
- Lines drawn down the longitudinal axis of each digit in flexion normally should converge on the scaphoid volarly.
- Limitation of movement secondary to pain and anatomic deformity

ALERT

Have a high suspicion for “fight bite.” This injury is the direct blow of a closed fist against a human tooth:

- Concern is violation of the extensor sheath, metacarpophalangeal (MCP) joint, or metacarpal head by a tooth, with subsequent infection by oral flora.

History

Not all patients are truthful as to cause of injury.

ESSENTIAL WORKUP

Exam should pay specific attention to skin integrity and alignment of the distal phalanges in flexion and extension.

DIAGNOSIS TESTS & INTERPRETATION

Imaging

- Hand radiographs when fracture suspected, and/or to rule out opaque foreign body
- Special radiographic views (CT) of the proximal metacarpals and the carpometacarpal joints may be necessary for patients with a suggestive physical

exam and no definite fracture on a standard 3-view series.

DIFFERENTIAL DIAGNOSIS

Fracture of the metacarpal may be accompanied by dislocation of adjacent phalanges or carpal bones.



TREATMENT

PRE HOSPITAL

- Most do not require EMS transport solely for metacarpal injury.
- Cautions:
 - Metacarpal injuries should be splinted in position of comfort.

INITIAL STABILIZATION/THERAPY

- Other, more serious injuries should be treated 1st.
- Immobilize hand pending evaluation.
- Lacerations should be cleaned as soon as possible, and consideration should be given to the possibility of foreign body.
- Thermal burns are treated with early analgesia.

ED TREATMENT/PROCEDURES

- Elevation, rest, and intermittent application of ice for the 1st 24 hr are appropriate treatment for all hand injuries (RICE).
- Boxer's fractures usually have some volar flexion of the distal fragment:
 - Reduction should be attempted for volar angulation of 40° or more.
 - Fractures of the 4th and 5th metacarpals that are stable and with no significant rotational component can be treated with a padded ulnar gutter splint.
- Fractures of the index and middle finger metacarpals are more difficult to stabilize:
 - Radial gutter splint and early orthopedic referral
- Thumb metacarpal fractures are uniformly complicated and all should be referred early to a hand surgeon or orthopedist:
 - Place in thumb spica splint.
- Dislocations should be reduced immediately and splinted; metacarpal dislocations are rare and frequently need open reduction and pinning.
- Appropriate splinting position for the MCP joint is the intrinsic plus, or “cobra” position (20–30° wrist extension):
 - MCP joint as close to 90° of flexion as possible
 - Proximal interphalangeal (PIP) and distal interphalangeal (DIP) joints in extension
- Antibiotics for oral flora should be started early for any open injury to the metacarpals suspicious for injury against a tooth, and may require curettage of the impaction site in the operating room.

- Simple torus (buckle) fractures may be splinted and may be followed by a primary care physician.

MEDICATION

- Check for tetanus status and vaccinate per immunization schedule.
- Silvadene cream or bacitracin ointment is appropriate for thermal burn injury.
- Analgesics may be necessary; NSAIDs or hydrocodone is usually sufficient.
- For human bites or dirty wounds, administer amoxicillin/clavulanate (Augmentin), or:
 - A cephalosporin or other penicillinase-resistant antibiotic given parenterally is appropriate.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Open fractures or dislocations require urgent surgical intervention and should be admitted.
- All thumb metacarpal fractures or dislocations should be seen by an orthopedist or hand surgeon because of the special importance of the thumb in all activities of the hand.
- Infection from a bite wound requires prompt orthopedic consultation, admission for irrigation, débridement, and IV antibiotics.

Discharge Criteria

- Patients with a stable transverse or oblique fracture in a good splint may be discharged for early orthopedic follow-up.
- Metacarpal–carpal dislocations are usually unstable enough to require surgery even if reduction is achieved, but this may be semiurgent rather than emergent.
- If a metacarpal fracture produces impaired range of motion or misalignment of the finger, the patient will require surgical repair in the 1st several days after injury.

Pediatric Considerations

Epiphyseal injuries mandate orthopedic referral.

PEARLS AND PITFALLS

With all metacarpal injuries assure proper rotational alignment.

ADDITIONAL READING

- American College of Radiology, Expert Panel on Musculoskeletal Imaging. Acute

Hand and Wrist Trauma. 2001.

- Chudnofsky CR, Byers SE. *Clinical Procedures in Emergency Medicine: Splinting Techniques*. 5th ed. Philadelphia, PA: Saunders Elsevier; 2010.
- Harrison B, Holland P. Diagnosis and management of hand injuries in the ED. *Emerg Med Pract*. 2005;7(2):1–28.



CODES

ICD9

- 815.00 Closed fracture of metacarpal bone(s), site unspecified
- 815.04 Closed fracture of neck of metacarpal bone(s)
- 927.20 Crushing injury of hand(s)

ICD10

- S62.309A Unsp fracture of unsp metacarpal bone, init for clos fx
- S62.368A Nondisp fx of neck of oth metacarpal bone, init for clos fx
- S67.20XA Crushing injury of unspecified hand, initial encounter

METHANOL POISONING

Michelle M. Troendle • Kirk L. Cumpston

BASICS

DESCRIPTION

- Colorless, volatile liquid
- Absorbed in 30–60 min
- Metabolized by liver
- Half-life 4–8 hr
- Mechanism:
 - Inebriating
 - Nontoxic
 - Metabolites of formaldehyde and formic acid produce toxic effects.
 - Inhibits cytochrome oxidase.
- Formic acid:
 - Determines degree of acidosis, visual symptoms, and mortality
 - Directly toxic to retinal and optic nerve tissue
- Methanol metabolism:
 - Step 1: Methanol is converted to formaldehyde by liver enzyme alcohol dehydrogenase.
 - Step 2: Formaldehyde is then rapidly converted by aldehyde dehydrogenase to formic acid.
 - Step 3: Formic acid is degraded to carbon dioxide and water by folate-dependent mechanism.
 - Steps 1 and 3 are rate-limiting steps.

ETIOLOGY

Common sources of methanol:

- Wood alcohol
- Windshield washer fluid (> 60% cases)
- Inhalational abuse of carburetor cleaners
- Fuel antifreeze solutions
- Formalin
- Gasoline
- Paint solvents
- Household cleaners
- Sterno cans
- Moonshine
- Model airplane fuel
- Photocopying fluid

- Perfumes



DIAGNOSIS

SIGNS AND SYMPTOMS

- GI:
 - Anorexia
 - Nausea/vomiting
 - Abdominal pain
- CNS:
 - Headache
 - Dizziness
 - Confusion
 - Inebriation
 - Coma
 - Seizures
- Ophthalmologic:
 - Blurry/hazy vision
 - Photophobia
 - “Snowfield vision”
 - Blindness
 - Central scotoma

History

- Intentional or unintentional methanol ingestion
- No history, but a patient with an unexplained high anion gap metabolic acidosis
- Elevated unexplained osmol gap

Physical-Exam

- Optic disc:
 - Hyperemia or pallor
 - Papilledema
- Afferent pupillary defect
- Tachypnea
- Altered mental status

ESSENTIAL WORKUP

- History of all substances ingested
- Inquire about visual symptoms.
- Funduscopic exam
- Drawn simultaneously:
 - Arterial blood gas

- Serum methanol, ethylene glycol, isopropyl alcohol, and ethanol levels
- Electrolytes, BUN, creatinine, and glucose
- Measured serum osmolality (by freezing-point depression is preferred)

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Calculate anion gap = $(\text{Na}^+) - (\text{Cl}^- + \text{HCO}_3^-)$:
 - Normal = 8–12.
- Determine serum osmol gap:
 - Osmol gap = measured osmolality – calculated osmolarity:
 - Calculated osmolarity = $2(\text{Na}^+) + \text{glucose}/18 + \text{BUN}/2.8 + \text{ethanol (in mg/dL)}/4.6$.
- Osmol gap:
 - Screens for methanol (methanol is osmotically active, toxic metabolites are not)
 - Most sensitive early in poisoning and normalizes as methanol is metabolized or with concurrent ethanol ingestion
 - Traditionally an osmol gap > 10 is considered indication for ruling out occult methanol ingestion. However, potentially toxic serum concentrations of methanol can be present with osmol gap < 10 .
 - A negative osmol gap DOES NOT rule out a methanol exposure.
 - Ethanol has higher affinity for alcohol dehydrogenase than methanol. With concurrent ethanol ingestion, osmol gap tends to be larger and acidosis tends to be less severe because relatively less methanol has been converted to acid-producing metabolites.
- Serum methanol concentrations *confirm* methanol poisoning:
 - Late after ingestion, no parent compound (methanol) may be detected and severe high anion gap metabolic acidosis will be present.
- Ethanol concentration may have clinical implications and is pertinent in interpreting lab tests.

Imaging

CT brain

DIFFERENTIAL DIAGNOSIS

- Increased osmol gap:
- *ME DIE A*:
 - Methanol
 - Ethanol
 - Diuretics/diluents (mannitol, glycerin, sorbitol, propylene glycol)
 - Isopropyl alcohol

- Ethylene glycol
- Acetone, ammonia
- Elevated anion gap metabolic acidosis: *A CAT MUDPILES:*
 - Alcoholic ketoacidosis
 - Cyanide, CO, H₂S, others
 - Acetaminophen
 - Antiretrovirals (NRTI)
 - Toluene
 - Methanol, metformin
 - Uremia
 - Diabetic ketoacidosis
 - Paraldehyde, phenformin, propylene glycol
 - Iron, INH
 - Lactic acidosis
 - Ethylene glycol
 - Salicylate, acetylsalicylic acid (ASA; aspirin), starvation ketosis



TREATMENT

PRE HOSPITAL

- Transport all possibly ingested substances.
- Dermal decontamination of a methanol spill by clothing removal, irrigation with soap and water
- Monitor airway and CNS depression.

INITIAL STABILIZATION/THERAPY

- Airway, breathing, and circulation (ABCs)
- Dextrose, naloxone, and thiamine for altered mental status
- Prevent further methanol absorption:
 - Gastric lavage with nasogastric tube:
 - Likely not helpful because of rapid absorption of methanol and delay in presentation > 1 hr
 - Activated charcoal:
 - For potential coingestants
 - Poorly adsorbs methanol
 - Aspiration risk for patients with altered mental status

ED TREATMENT/PROCEDURES

- Prevent methanol conversion to toxic metabolites with fomepizole (preferable) or ethanol infusion
- Fomepizole (4-MP, Antizol):
 - Competitive inhibitor of alcohol dehydrogenase

- Indications:
 - Intentional methanol ingestion
 - Accidental methanol ingestion of more than a sip
 - Altered mental status or visual symptoms associated with unexplained osmol gap and/or elevated anion gap metabolic acidosis
- Initiate before serum methanol level returns if intentional ingestion or more than a sip.
- Continue until methanol level is < 25 mg/dL.
- Advantages:
 - No need for continuous infusion
 - No inebriation/CNS depression
 - Ease of dosing
 - No hypoglycemia, no hyponatremia, no hyperosmolality
 - No checking serum concentrations
 - Reduced nursing care and monitoring
 - Occult methanol exposure can often be ruled out before 2nd dose is needed.
- Disadvantages:
 - Blurry vision
 - Transient elevation of liver function tests
- Ethanol therapy:
 - Not FDA approved for treatment of methanol
 - Ethanol has greater affinity than methanol for alcohol dehydrogenase:
 - Slows metabolism to formaldehyde and formic acid by competitive inhibition
 - Ethanol is the 2nd-choice antidote if fomepizole is not available.
 - Initiate before methanol level returns if potentially toxic ingestion is highly suspected or confirmed by history:
 - Therapeutic range is 100 mg/dL.
 - Continue until methanol level is < 25 mg/dL.
 - Indications for ethanol therapy:
 - Intentional methanol ingestion
 - Accidental methanol ingestion of more than a sip
 - Altered mental status or visual symptoms associated with unexplained osmol gap and or elevated anion gap metabolic acidosis
 - Advantages:
 - Easily accessible
 - Oral and IV routes
 - Disadvantages:
 - CNS depression especially in children
 - Respiratory depression
 - Hyponatremia or hypernatremia
 - Hypoglycemia

- Hyperosmolarity
- Continuous infusion
- Frequent lab testing
- Contraindicated in pregnancy
- Pancreatitis
- Gastritis
- Enhance elimination of methanol and toxic metabolites with hemodialysis:
 - _ Decreases elimination half-life of methanol
 - _ Removes formaldehyde and formic acid
 - _ Indications:
 - Ingestion of >1 mL/kg of 100% methanol
 - Ophthalmologic manifestations
 - Severe metabolic acidosis unresponsive to bicarbonate therapy
 - Persistent electrolyte or metabolic acidosis
 - Renal insufficiency
 - Serum methanol level >25 mg/dL
 - _ Continue hemodialysis until methanol level approaches <25 mg/dL and the metabolic acidosis has resolved.
- Folic acid and folinic acid (leucovorin):
 - _ Folic acid: Cofactor required for conversion of formic acid to carbon dioxide and water
 - _ Supplemental folate important in malnourished individuals (alcoholics)
- Correct acid–base abnormalities:
 - _ Sodium bicarbonate for severe acidosis (pH <7.1)
 - _ The goal of the sodium bicarbonate drip is to maintain a normal serum pH.

MEDICATION

- Activated charcoal: 1 g/kg PO
- Dextrose: D₅₀W 1 amp: 50 mL or 25 g (peds: D₂₅W 2–4 mL/kg) IV
- Fomepizole:
 - _ Loading dose: 15 mg/kg slow infusion over 30 min
 - _ Maintenance dose: 10 mg/kg q12h for 4 doses, then 15 mg/kg q12h until methanol levels reduced <25 mg/dL
 - _ Dosing related to hemodialysis:
 - Do not administer dose at beginning of dialysis if last dose was <6 hr previously.
 - Administer next dose if last dose was >6 hr previously.
 - Dose q4h during dialysis.
 - If time between last dose and end of dialysis was <1 hr from last dose, do not administer new dose.
 - If time between last dose and end of dialysis was 1–3 hr from last dose, administer 1/2 of next scheduled dose.
 - If time between last dose and end of dialysis was >3 hr from last

dose, administer next scheduled dose.

- Ethanol:
 - Oral: 50% ethanol solution (100-proof liquor) via nasogastric tube:
 - Loading dose 2 mL/kg
 - Maintenance dose 0.5 mL/kg/h
 - Maintenance dose during hemodialysis 1 mL/kg/h
 - IV: 10% ethanol in D₅W:
 - Loading dose 8 mL/kg over 30–60 min
 - Maintenance infusion 2 mL/kg/h
 - Maintenance infusion during hemodialysis 4 mL/kg/h
- Folic acid: 50 mg IV q4h for 24 hr
- Sodium bicarbonate: 1–2 mEq/kg in 1 L of D₅W with 40 mEq KCl at 250 mL/h



FOLLOW-UP

DISPOSITION

Admission Criteria

- Significant historical methanol ingestion even if initially asymptomatic
- ICU admission for seriously ill patients
- Transfer to another facility if hemodialysis or antidote is indicated but not readily available.

Discharge Criteria

Asymptomatic patient with isolated methanol ingestion if serum methanol level is < 25 mg/dL; normal acid/base status and electrolytes.

FOLLOW-UP RECOMMENDATIONS

Psychiatric follow-up for suicidal/depressed patients

PEARLS AND PITFALLS

- An osmol gap < 10 mmol/L does not rule out a methanol exposure.
- If you have a patient with an elevated anion gap and methanol exposure is in the differential diagnosis, administer fomepizole immediately and confirm exposure with a serum concentration.
- If you cannot confirm a methanol exposure, or do not have hemodialysis capabilities 24/7, or have no antidote, transfer the patient to a facility that has all of these capabilities.
- Not all patients will have an elevated osmol and anion gap. Early presenters will have an osmol gap only, because methanol is osmotically active, and there are no toxic metabolites yet. Late presenters may have an anion gap only, because the

osmotically active parent compound has metabolized to the toxic acidotic metabolites. Patients who present in between will have a combination of an anion gap and an osmol gap.

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- Leikin J, Paloucek F. Fomepizole. *Leikin and Paloucek's Poisoning and Toxicology Handbook*. 4th ed. New York, NY: Lexi-Comp; 2008:989.



CODES

ICD9

980.1 Toxic effect of methyl alcohol

ICD10

- T51.1X1A Toxic effect of methanol, accidental (unintentional), initial encounter
- T51.1X2A Toxic effect of methanol, intentional self-harm, initial encounter
- T51.1X4A Toxic effect of methanol, undetermined, initial encounter

METHEMOGLOBINEMIA

Navneet Cheema



BASICS

DESCRIPTION

- Iron molecule in hemoglobin is oxidized from ferrous (Fe^{2+}) to ferric (Fe^{3+}) state resulting in a form of hemoglobin that cannot transport oxygen.
- Oxygen-carrying capacity of blood is reduced and cyanosis is generally present with significant levels.
- Normal methemoglobin levels are $\leq 1\%$; symptoms usually occur with levels $> 20\%$.
- More serious with coexisting anemia
- Methemoglobin:
 - Decreases total oxygen-carrying capacity (functional anemia)
 - Shifts hemoglobin oxygen-dissociation curve to the left, impairing O_2 release to tissues
 - Maintained at physiologic level (1–2%) by nicotinamide adenine dinucleotide (NADH)-methemoglobin (cytochrome B_5) reductase in red blood cells (RBCs)
- Congenital methemoglobinemia:
 - NADH-methemoglobin (cytochrome B_5) reductase deficiency (homozygous or heterozygous)
 - Heterozygous hemoglobin M and other abnormal hemoglobins
- Acquired methemoglobinemia results from oxidant stress on RBCs:
 - Some methemoglobin-inducing agents are direct oxidants (e.g., nitrites)
 - Many substances produce oxidant injury via *N*-hydroxylamine metabolites.
 - Methemoglobinemia may be delayed relative to initial substance exposure.
- Many methemoglobin-inducing agents also cause Heinz body hemolytic anemia (HA):
 - Caused by oxidant injury of RBC proteins
 - Glucose-6-phosphate dehydrogenase (G6PD)–deficient patients have higher risk.
 - Patients with methemoglobinemia should be worked up for HA.
- Methemoglobinemia may serve as marker for genetic abnormalities:
 - Heterozygous NADH-methemoglobin (cytochrome B_5) reductase deficiency

ETIOLOGY

- Cyanide (CN) antidote kit:
 - Induces methemoglobinemia via amyl and sodium nitrite

- CN will preferentially complex with methemoglobin, which can then be chelated by sodium thiosulfate.
- Nitrates/nitrites:
 - Nitrites (NO₂)
 - Nitrates (NO₃) (e.g., nitroglycerine, via metabolic conversion to nitrites)
 - Nitric oxide (NO)
- Dyes:
 - Aniline dyes
 - Methylene blue (excessive)
- Antiparasitic drugs (high potential for MetHb formation):
 - Dapsone
 - Primaquine
 - Chloroquine
- Local anesthetics (high potential for MetHb formation):
 - Benzocaine
 - Lidocaine
 - Prilocaine
- Analgesics:
 - Phenazopyridine (Pyridium)
 - Phenacetin
- Antibiotics:
 - Nitrofurantoin
 - Sulfones
 - Sulfonamides
- Others:
 - Metoclopramide
 - Naphthalene (mothballs)
 - Paraquat (herbicide)
 - Arsine gas (AsH₃)
 - Chlorates (ClO₄)
 - Phenols (e.g., dinitrophenol, hydroquinone)

DIAGNOSIS

SIGNS AND SYMPTOMS

- Central cyanosis, refractory to oxygen administration:
 - Cyanosis evident at methemoglobin (MetHb) of 10–15% of total hemoglobin in nonanemic patient (or 1.5 g of MetHb/dL blood)
- Dyspnea/tachypnea
- Chest pain/dysrhythmias
- Syncope

- Altered mental status with levels > 50%

History

- Exposure to methemoglobin-inducing agent
- All substances ingested and time(s) of ingestion
- G6PD deficiency
- Medical conditions vulnerable to impaired oxygen delivery (e.g., coronary artery disease)

Physical-Exam

- Cyanosis
- Emphasis on mental status and cardiovascular findings
- Icterus or dark-colored urine with accompanying HA

ESSENTIAL WORKUP

- Pulse oximetry is *inaccurate* in methemoglobinemia:
 - MetHb interferes with pulse oximetry measurement of hemoglobin oxygen saturation.
 - Saturation decreases to ~ 85% with increasingly more severe methemoglobinemia.
 - Pulse oximetry cannot be used to guide management.
- ABG for:
 - Methemoglobin level
 - Carboxyhemoglobin level
 - PaO₂ and PaCO₂
- ECG

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Blood classically described as chocolate colored
- CBC with manual differential count and smear analysis for evidence of HA

Imaging

CXR to rule out other pulmonary pathology

DIFFERENTIAL DIAGNOSIS

- Hypoxia:
 - CHF
 - COPD
 - Pulmonary embolism
- Irritant gas exposure

- Blue discoloration:
 - Hypoxia
 - Sulfhemoglobinemia
 - CN poisoning
 - Hydrogen sulfide poisoning
 - Excess methylene blue administration
 - Tellurium toxicity
 - Skin contact/staining with blue dye



TREATMENT

PRE HOSPITAL

- Bring to hospital all substances patient may have ingested.
- Question witnesses and observe scene for household products and other potential coingestants:
 - Document and relay findings to emergency medical staff.
- Commercial or industrial sites:
 - Obtain relevant material safety data sheets (MSDSs) if available to identify commercial or chemical products.
 - Avoid dermal exposures.

INITIAL STABILIZATION/THERAPY

- ABCs:
 - Cardiac monitor
 - Isotonic crystalloids as needed for hypotension
- Naloxone, thiamine, and dextrose (D₅₀W) as indicated for altered mental status
- Supplemental oxygen

ED TREATMENT/PROCEDURES

- Decontamination:
 - If owing to acute ingestion/overdose within previous 1–2 hr, and protective airway reflexes are intact, administer 50–100 g of activated charcoal PO.
- Remove source of oxidant stress.
- Methylene blue:
 - Indications:
 - Asymptomatic with levels > 30%
 - Symptomatic patients with levels > 10–20%, especially if comorbid diseases are present
 - Expect transient worsening of saturations on pulse oximetry after methylene blue is administered:
 - Interferes with pulse oximetry measurement and no specific intervention required

- Use with caution in patients with glucose-6 pyruvate decarboxylase deficiency:
 - May cause hemolysis
- If no improvement with methylene blue, consider that source of oxidant stress is not eliminated, or that sulfhemoglobinemia is present:
 - Sulfhemoglobin is sulfur molecule bound to hemoglobin. Presents similar to methemoglobin, but is self-limited and not responsive to methylene blue.
- RBC transfusion:
 - May be necessary to increase blood oxygen-carrying capacity
 - Consider in the presence of HA.
- Exchange transfusion:
 - Especially with neonates/infants
- Hyperbaric oxygen therapy:
 - Increases oxygen delivery to tissues by allowing more oxygen to be dissolved in the blood, independent of hemoglobin.
 - Use in life-threatening methemoglobinemia if immediately available.

Pediatric Considerations

- Children may develop significant methemoglobinemia from apparently minor ingestions.
- Symptoms delayed several hours after ingestion, so prolonged observation necessary
- Neonates are also at higher risk of methemoglobinemia (owing to decreased stores of NADH methemoglobin reductase).

MEDICATION

- Dextrose 50%: 25 g (50 mL) (peds: 0.5–1 g/kg of dextrose) IV for hypoglycemia
- Methylene blue: 0.1–0.2 mL/kg 1% solution IV over 5 min (adults and peds)
 - May repeat if no improvement in 1 hr
 - Doses of 0.3 to 1 mg/kg IV have been effective in neonates. Has been used IO over 3–5 min.
- Naloxone: 0.4–2 mg (peds: 0.1 mg/kg) IV, may repeat up to 10 mg for suspected opioid intoxication
- Thiamine: 100 mg (peds: 1 mg/kg) IM or IV



FOLLOW-UP

DISPOSITION

Admission Criteria

- Severely symptomatic patients
- Patients requiring multiple doses of methylene blue

- Dapsone may cause prolonged recurrent methemoglobinemia

Discharge Criteria

Methemoglobin levels < 20% and falling with no symptoms or comorbid disease

Issues for Referral

Toxicology consult for significant exposures

FOLLOW-UP RECOMMENDATIONS

Occupational medicine referral for work-related exposures

PEARLS AND PITFALLS

- Pulse oximetry is *inaccurate* in methemoglobinemia.
- Obtain an ABG.
- Administer methylene blue for significant levels/symptoms.

A special thanks to Dr. Gerald Maloney who contributed to the previous edition.

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CODES

ICD9

289.7 Methemoglobinemia

ICD10

- D74.0 Congenital methemoglobinemia
- D74.8 Other methemoglobinemias
- D74.9 Methemoglobinemia, unspecified

MITRAL VALVE PROLAPSE

Liudvikas Jagminas



BASICS

DESCRIPTION

- Bulging of 1 or both of the mitral valve leaflets into the left atrium during systole
- Occurs when the leaflet edges of the mitral valve do not coapt
- Commonly due to abnormal stretching of 1 of the mitral valve leaflets during systole:
 - Myxomatous proliferation of the spongiosa layer within the valve causing focal interruption of the fibrosa layer
 - Excessive stretching of the chordae tendineae, leading to traction on papillary muscles
- Theoretical explanations for associated chest pain:
 - Focal ischemia from coronary microembolism due to platelet aggregates and fibrin deposits in the angles between the leaflets
 - Coronary artery spasm
- Mitral regurgitation (MR) may occur in some patients.
- Age of onset is 10–16 yr
- Female > male (3:1)
- Typically benign in young women, whereas men > 50 yr tend to have serious sequelae and more often develop severe regurgitation requiring surgical intervention
- Can be identified by ECG in 2–4% of the general population and in 7% of autopsies
- A variety of neuroendocrine and autonomic disturbances occur in some patients
- Genetics:
 - Strong hereditary component
 - Sometimes transmitted as an autosomal dominant trait with varying penetrance

ETIOLOGY

- Marfan syndrome
- Relapsing polychondritis
- Ehlers–Danlos syndrome (i.e., types I, II, IV)
- Osteogenesis imperfecta
- Pseudoxanthoma elasticum
- Stickler syndrome
- Systemic lupus erythematosus
- Polyarteritis nodosa

- Polycystic kidney disease
- von Willebrand syndrome
- Duchenne muscular dystrophy



DIAGNOSIS

SIGNS AND SYMPTOMS

Separated into 3 categories:

- Symptoms related to autonomic dysfunction
- Symptoms related to the progression of MR
- Symptoms that occur as a result of an associated complication (i.e., stroke, endocarditis, or arrhythmia)

History

- Palpitations in up to 40% of cases:
 - Usually ventricular premature beats or paroxysmal supraventricular tachycardia
 - Up to 40% have symptoms of dysautonomia
- Chest pain occurs in 10%:
 - Sharp, localized, of variable duration, and nonexertional
 - Rarely may respond to nitroglycerin
- Panic attacks
- Anxiety
- Fatigue
- Depression in up to 70%
- Nervousness
- Migraine headaches
- Irritable bowel
- Syncope/presyncope:
 - Occurs in 0.9% of patients
- Orthostasis
- Dyspnea and fatigue relatively uncommon

Physical-Exam

- Mid to late systolic click at the cardiac apex:
 - Standing or Valsalva moves click closer to S1.
 - S1 may be accentuated when prolapse occurs early in systole.
 - Squatting moves click closer to S2.
- Late systolic murmur
- Skeletal abnormalities are observed in 2/3 of patients:
 - Asthenic body habitus: Height-to-weight ratio > normal
 - Arm span > height (dolichostenomelia)

- Scoliosis or kyphosis
- Pectus excavatum
- Arachnodactyly
- Joint hypermobility
- Hypomastia
- Cathedral palate

ESSENTIAL WORKUP

- History and auscultation of a midsystolic click are often sufficient to make the diagnosis.
- Echocardiography confirms the diagnosis when clinical information is insufficient.

DIAGNOSIS TESTS & INTERPRETATION

Lab

Not required to establish the diagnosis

Imaging

- EKG:
 - Usually normal
 - Occasionally ST-T wave depression and inversion in leads III and aVF
 - Prolonged QT interval or prominent Q waves
 - Premature atrial and ventricular contractions
- CXR:
 - Typically normal
 - If MR is present, may show both left atrial and ventricular enlargement
 - Calcification of the mitral annulus in patients with Marfan syndrome
- Echocardiography:
 - Classic MVP: The parasternal long-axis view shows > 2 mm superior displacement of the mitral leaflets into the left atrium during systole, with a leaflet thickness of at least 5 mm.
 - Nonclassic MVP: Displacement is > 2 mm, with a maximal leaflet thickness of < 5 mm.
 - Other ECG findings that should be considered as criteria are leaflet thickening, redundancy, annular dilatation, and chordal elongation.
 - Minor criteria:
 - Isolated mild to moderate superior systolic displacement of the posterior mitral leaflet
 - Moderate superior systolic displacement of both mitral leaflets

Diagnostic Procedures/Surgery

Cardiac studies may be indicated in patients with chest pain when the etiology is uncertain.

DIFFERENTIAL DIAGNOSIS

- MI/ischemia
- Hypertrophic cardiomyopathy with obstruction
- Idiopathic hypertrophic subaortic stenosis
- Tachyarrhythmias
- Atrial fibrillation/flutter
- Ventricular septal defect
- Papillary muscle dysfunction
- Hypokalemia
- Hypomagnesemia
- Valvular heart disease
- Pheochromocytoma
- Anemia
- Thyrotoxicosis
- Pregnancy
- Toxicity from cocaine, amphetamines, or other sympathomimetics
- Ventricular tachycardia
- WPW syndrome
- Rheumatic endocarditis
- Anxiety/panic disorder
- Stress
- Menopause



TREATMENT

PRE HOSPITAL

- ABCs
- IV access
- Supplemental oxygen
- Cardiac monitoring
- Pulse oximetry

INITIAL STABILIZATION/THERAPY

- Cardiac monitoring
- Supplemental oxygen
- IV catheter placement

ED TREATMENT/PROCEDURES

- Medications generally are not necessary. β -blockers may be helpful if palpitations are severe.
- Antiplatelet agents (aspirin, aspirin with extended-release dipyridamole, or clopidogrel) are indicated for patients with transient ischemic attack or stroke

symptoms.

- Orthostatic hypotension and presyncope symptoms may be treated with sodium chloride tablets; however, if this treatment is not successful, fludrocortisone may be used.
- Magnesium supplementation may improve symptoms of the classic MVP syndrome.
- Significant MR in the setting of HTN (systolic blood pressure > 140 mm Hg) may be improved with the use of ACE inhibitors.
- β -Blockers:
 - Patients with tachycardia or severely symptomatic chest pain
- Digoxin is an alternative for supraventricular tachycardia and prevention of chest pain and fatigue.
- Antibiotic prophylaxis:
 - When performing surgical procedures (e.g., contaminated wound repair, abscess incision and drainage)
 - Indicated in the following settings:
 - Presence of a murmur
 - Evidence of nontrivial MR on Echocardiogram
 - Men > 45 yr with valve thickening
 - Prophylaxis is not recommended for patients who have an isolated click without a murmur or for patients without evidence of MR on an echocardiogram or previous history of endocarditis.

MEDICATION

First Line

- Amoxicillin: 2 g PO 1 hr before the procedure (peds: 50 mg/kg PO 1 hr before procedure)
- Ampicillin: 2 g IV/IM 30 min before the procedure (peds: 50 mg/kg IV/IM 30 min before the procedure)
- Clindamycin: 600 mg PO 1 hr before procedure (peds: 20 mg/kg PO 1 hr before procedure; not to exceed 600 mg)
- Propranolol: 1–3 mg IV at 1 mg/min, 80–640 mg/d PO (peds: 1–4 mg/kg/d PO div. BID/QID)
- Isoproterenol: 0.02–0.06 mg IV \times 1, 0.01–0.02 mg IV or 2–20 mg/min infusion
- Atenolol: 0.3–2 mg/kg/d PO, max. 2 mg/kg/d

Second Line

- Digoxin: 0.5–1 mg IV/IM div. 50% initially then 25% \times 2 q6–12h or 0.125–0.5 mg/d PO
- Fludrocortisone: 0.05–0.10 mg/d PO



FOLLOW-UP

DISPOSITION

Admission Criteria

- Severe MR
- Severe chest pain with ischemic symptoms
- Syncope or near syncope
- Life-threatening dysrhythmias
- Cerebral ischemic events, including transient ischemic attack

Discharge Criteria

- Asymptomatic
- No lab abnormalities
- No significant MR or dysrhythmias

Issues for Referral

- Cardiology consultation is warranted in cases of ventricular dysrhythmia or risk of sudden death, as well as when symptoms of severe MR are present.
- Cardiothoracic surgery follow-up is recommended for consideration of valve replacement or repair
 - Symptomatic patients
 - Atrial fibrillation
 - Ejection fraction < 50–60%
 - Left ventricular end-diastolic dimension > 45–50 mm
 - Pulmonary systolic pressure > 50–60 mm Hg
- Valve repair rather than replacement is preferred to avoid the need for anticoagulation.
- Pilots with mitral valve prolapse may develop MR under positive G force and be at risk for dysrhythmia or syncope.

Pediatric Considerations

Dysrhythmias, sudden death, and bacterial endocarditis have been reported.

Geriatric Considerations

- Often present in an atypical manner:
 - More likely to have holosystolic murmurs and a greater degree of MR.
- Heart failure may be presenting symptom complex associated with ruptured chordae tendineae.

Pregnancy Considerations

MVP does not predispose women to any increased risk during pregnancy.

FOLLOW-UP RECOMMENDATIONS

- Repeat evaluations are necessary every 3–5 yr to identify any progression of disease.
- Infective endocarditis prophylaxis is indicated in patients with MVP and MR while undergoing at-risk procedures.
- Coronary artery anomalies should be excluded in patients with chest pain before they participate in sports.
- Patients with MVP and a murmur should avoid high-intensity competitive sports in the following settings:
 - Syncope associated with dysrhythmia
 - A family history of sudden death associated with MVP
 - Significant supraventricular or ventricular dysrhythmias
 - Moderate to severe MR

PEARLS AND PITFALLS

- The diagnosis of MVP should not be an excuse to terminate further diagnostic evaluation of patients with symptoms of chest pain, palpitations, dyspnea, or syncope.
- MVP is the 3rd most common cause of sudden death in athletes.

ADDITIONAL READING

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CODES

ICD9

424.0 Mitral valve disorders

ICD10

I34.1 Nonrheumatic mitral (valve) prolapse

MOLLUSCUM CONTAGIOSUM

Evan Small • Deepi G. Goyal

BASICS

DESCRIPTION

- Molluscum contagiosum (MC) is a generally benign human disease characterized by multiple small, painless, pearly lesions.
- MC appears on epithelial surface and spreads through close contact or autoinoculation.
- Confined to the skin and mucous membranes
- 5–20% of patients with HIV have coinfection with MC.
- Found worldwide with an incidence of 2–8%, with higher distribution in tropical areas

ETIOLOGY

- MC is caused by a double-stranded DNA poxvirus of the *Molluscipox* genus
- Transmission in children is by direct skin-to-skin contact, fomites, or pool or bath water.
- Transmission in adults is most often by sexual contact; autoinoculation is common at any age.
- There are rare reports of transmission to infants during childbirth.

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Incubation period: 14–50 days
- Patients are usually asymptomatic, with occasional pruritus or tenderness.
- 10–25% of patients may have eczematous reaction surrounding the lesions.
- Untreated lesions in immunocompetent hosts usually resolve within several months but can last up to 5 yr.

Physical-Exam

- Lesions are smooth-surfaced, firm, spherical papules 2–6 mm in diameter.
- May be flesh colored, white, translucent, or light yellow
- Lesions have a waxy, curd-like core composed of collagen–lipid-rich material containing large numbers of maturing virions
- Distinctive central umbilication in 25%
- Atypical presentations include nonumbilicated, persistent, disseminated, or giant

lesions, usually in the setting of immunosuppression.

- Distribution:
 - Children:
 - Face
 - Trunk
 - Extremities
 - Healthy adults:
 - Genitals
 - Lower abdomen
 - Occasionally perioral
 - Rarely on palms and soles
- MC is commonly seen with HIV infection, causing atypical involvement of face, neck, and trunk, lesions to 1.5 cm, and a progressive course. Lesions may also appear with initiation of highly active antiretroviral therapy (HAART) as a manifestation of the immune reconstitution inflammatory syndrome.
- Occasional intraocular or periocular involvement presenting as trachoma or chronic follicular conjunctivitis

ESSENTIAL WORKUP

- History and careful skin exam
- Skin biopsy for confirmation
- Lesions in adult men necessitate evaluation for an immunocompromised state.
- MC in children is rarely associated with immunodeficiency, and usually no further evaluation is needed.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Test for immunocompromised state if no clear etiology:
 - CBC with differential
 - HIV if indicated
- If anogenital lesions:
 - Consider syphilis, hepatitis C, HIV

Diagnostic Procedures/Surgery

Skin biopsy for confirmation

DIFFERENTIAL DIAGNOSIS

- Basal cell carcinoma
- Histiocytoma
- Keratoacanthoma
- Intradermal nevus
- Darier disease

- Nevoxanthoendothelioma
- Syringoma
- Epithelial nevi
- Sebaceous adenoma
- Atopic dermatitis
- Dermatitis herpetiformis
- Mycosis fungoides
- Jessner lymphocytic infiltration
- *Cryptococcus neoformans*



TREATMENT

PRE HOSPITAL

Maintain universal precautions.

INITIAL STABILIZATION/THERAPY

Not applicable in routine cases.

ED TREATMENT/PROCEDURES

- Aimed at destruction or removal of virus-infected epithelial cells and is indicated to prevent autoinoculation and transmission:
 - Intervention is not always indicated: Lesions are self-limited in immunocompetent hosts.
 - Untreated immunocompromised patients are at greater risk for secondary inflammation and bacterial infections
- If treatment is necessary, consider referral to dermatology.
- If dermatology referral is not an option, physical treatment modalities generally most effective:
 - Curettage after local anesthesia with EMLA (eutectic mixture, lidocaine, prilocaine) or ethyl chloride
 - Cryotherapy with liquid nitrogen
 - Podophyllin, trichloroacetic acid, cantharidin, tretinoin, and cidofovir applied topically are variably effective.
 - Repeatedly applying adhesive tape to the lesions as a means of removing the superficial epidermis
- Griseofulvin and methisazone orally for extensive disease have given mixed results.
- HAART has been effective in reducing incidence in HIV-infected patients.
- Topical imiquimod has shown effectiveness in several small studies.
- Examine sexual partners for MC and other sexually transmitted diseases:
 - Patients should avoid contact sports, swimming pools, shared baths and towels, scratching, and shaving until lesions have resolved.

- Re-examine treated patients for recurrence every 2–4 wk; 2–4 treatments are often needed to clear lesions completely.
- Discourage picking and scratching lesions, a common habit, as it may lead to scarring or pigment alteration.

MEDICATION

- Cantharidin 0.9% solution with equal parts of acetone and flexible collodion: Apply topically 1–3 treatments every 7 days or until resolution.
- Imiquimod 5%: Apply topically daily for 3–5 consecutive days for 16 wk.
- Podophyllin (podofilox 0.5%): Apply topically q12h for 3 days, withhold for 4 days; repeat 1-wk cycle up to 4 times until resolved.
- Tretinoin 0.1%: Apply topically q12h for 10 days or until resolution of lesions.
- Trichloroacetic acid (50–80%): Apply and cover with bandage 5–6 days.
- Oral cimetidine (40 mg/kg/d) in 2 div. doses for 2 mo has been used to treat extensive infections; however, further study is needed to determine efficacy.



FOLLOW-UP

DISPOSITION

Admission Criteria

Widespread disease with extensive superinfection in an immunocompromised host

Discharge Criteria

Patients without extensive superinfection may be safely treated as outpatients.

Issues for Referral

Consider referral to dermatology if treatment or confirmatory testing is necessary.

FOLLOW-UP RECOMMENDATIONS

Re-examine treated patient for recurrence every 2–4 wk.

PEARLS AND PITFALLS

- Active nonintervention is an option in immunocompetent hosts.
- Search for an immunocompromised state if no clear etiology.
- Physical destruction of lesions is often most effective treatment vs. medication.

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CODES

ICD9

078.0 Molluscum contagiosum

ICD10

B08.1 Molluscum contagiosum

MONOAMINE OXIDASE INHIBITOR POISONING

James W. Rhee



BASICS

DESCRIPTION

- Primarily for depression
- Selegiline, a selective monoamine oxidase B inhibitor, is sometimes used to treat Parkinson disease, and also comes in a transdermal preparation.
- Monoamine oxidase inhibitor (MAOI) pharmacologic actions:
 - Disruption of equilibrium between endogenous monoamine synthesis and degradation, resulting in:
 - Increased neural norepinephrine levels
 - Downregulation of several receptor types
 - Inhibition of irreversible (noncompetitive) enzyme
 - Inhibition of other B₆-containing enzymes
- MAO: Principal inactivator of neural bioactive amines:
 - MAO A:
 - Present in the gut and liver
 - Protects against dietary bioactive amines
 - MAO B:
 - Present in neuron terminals and platelets
 - Sympathomimetic amines: Types of bioactive amines

ETIOLOGY

- MAOI overdose:
 - Toxicopharmacology poorly understood
 - MAO inhibitors: Amphetamine-like in structure:
 - Early: Indirect sympathomimetic effect
 - Late: Sympatholytic response (hypotension)
- MAOI hypertensive crisis syndrome:
 - Results from impaired norepinephrine degradation and large norepinephrine release precipitated by an indirect- or mixed-acting sympathomimetic agent
 - Common precipitants: Tyramine, cocaine, amphetamines
- Serotonin syndrome (SS):
 - Commonly results from exposure to combinations of agents that affect serotonin metabolism or action
 - Increases serotonin synthesis: Tryptophan
 - Increase serotonin release:
 - Indirect- and mixed-acting sympathomimetic agents and dopamine

receptor agonists

- Decrease serotonin reuptake:
 - o Selective serotonin reuptake inhibitors
 - o Tricyclic antidepressants
 - o Newer antidepressants: Trazodone, nefazodone, venlafaxine
 - o Phenylpiperidine opioids: Meperidine, dextromethorphan, tramadol, methadone, propoxyphene
- Direct serotonin receptor agonists:
 - o Buspirone, sumatriptan, lysergic acid diethylamide
- Decrease serotonin breakdown:
 - o MAOIs
- Increases nonspecific serotonin activity:
 - o Lithium

DIAGNOSIS

SIGNS AND SYMPTOMS

- MAOI overdose:
 - Delayed onset (6–12 hr)
 - Initial hypertension with headache
 - Hyperadrenergic activity:
 - o Tachycardia
 - o Hypertension
 - o Mydriasis
 - o Agitation
 - Neuromuscular excitation:
 - o Nystagmus
 - o Hyperreflexia
 - o Tremor
 - o Myoclonus
 - o Rigidity
 - o Seizures
 - Hyperthermia
 - Associated complications:
 - o Rhabdomyolysis
 - o Renal failure
 - o Disseminated intravascular coagulation (DIC)
 - o Acute respiratory distress syndrome (ARDS)
- MAOI hypertensive crisis syndrome (MAOI interaction with drug or food):
 - Hypertension
 - Tachycardia or bradycardia
 - Hyperthermia

- Headache, usually occipital
- Altered mental status
- Intracranial hemorrhage
- Seizures
- SS:
 - Increased neuromuscular activity
 - Increased deep tendon reflexes:
 - Lower extremity may be greater than upper
 - Tremor
 - Myoclonus
 - Rigidity (when severe)
 - Autonomic nervous system hyperactivity:
 - Hyperthermia
 - CNS:
 - Agitation
 - Hallucinations
 - Delirium
 - Coma
 - Diarrhea
 - SS vs. neuroleptic malignant syndrome (NMS):
 - Both present along a spectrum of severity (mild to severe)
 - Onset: Hours (SS) vs. days (NMS)
 - Gastrointestinal symptoms: May be present (SS) vs. absent (NMS)
 - Only drug/medication history may differentiate in many cases

History

- Time of ingestion
- Bottle available
- Intentional or accidental
- Coingestions

Physical-Exam

- Neuromuscular hyperactivity:
 - Myoclonus
 - Rigidity
 - Tremors
 - Hyperreflexia
- Autonomic hyperactivity:
 - Tachycardia or bradycardia
 - Fever
 - Diaphoresis
- Altered mental status:

- Agitation, confusion, or excitement

ESSENTIAL WORKUP

- History of ingested substances
- Rectal temperature monitoring as indicated
- Blood pressure/cardiac monitoring

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Urinalysis:
 - Blood
 - Myoglobin
- Electrolytes, BUN/creatinine, glucose:
 - Hypoglycemia may contribute to altered mental status.
 - Acidosis may accompany severe toxicity.
 - Rhabdomyolysis may cause renal failure.
 - Hyperkalemia—life-threatening consequence of acute renal failure
- Coagulation profile to monitor for potential DIC:
 - INR, prothrombin time, partial thromboplastin time, platelets
- Creatinine kinase:
 - Markedly elevated in rhabdomyolysis
- Urine toxicology screen:
 - May be positive for amphetamines, given the structural similarities between some MAOIs and amphetamines
- Aspirin and acetaminophen levels if suicide attempt a possibility
- Arterial blood gas

Imaging

- Chest radiograph:
 - ARDS
- Head CT if significant headache or altered mental status or focal neurologic signs:
 - Subarachnoid hemorrhage, intracerebral bleed

Diagnostic Procedures/Surgery

Lumbar puncture for:

- Suspected meningitis (headache, altered mental status, hyperpyrexia)
- Suspected subarachnoid hemorrhage and CT normal

DIFFERENTIAL DIAGNOSIS

- Hyperthermia:
 - Infection
 - Hyperthyroidism

- Heat stroke
- Anatomic thalamic dysfunction
- NMS
- Malignant hyperthermia
- Malignant catatonia
- Ethanol or drug withdrawal
- Anticholinergic toxicity
- Sympathomimetic overdose
- Cocaine-associated delirium/rhabdomyolysis
- Salicylate toxicity
- Theophylline toxicity
- Nicotine toxicity
- Hypertension:
 - Hypoglycemia
 - Carcinoid syndrome
 - Pheochromocytoma
 - Accelerated renovascular hypertension
 - Ethanol or drug withdrawal
 - Sympathomimetic toxicity



TREATMENT

PRE HOSPITAL

- Patient may be uncooperative or violent.
- Secure IV access.
- Protect from self-induced trauma.

INITIAL STABILIZATION/THERAPY

- Airway, breathing, and circulation (ABCs)
- IV access and fluid resuscitation if hypotensive
- Oxygen
- Cardiac monitor
- Naloxone, thiamine, D50W (or Accu-Chek) if altered mental status

ED TREATMENT/PROCEDURES

- Gastrointestinal decontamination:
 - In potential life-threatening ingestions gastric lavage may be carefully considered if within *1 hr* of ingestion
 - Administer activated charcoal
- Hyperthermia:
 - Benzodiazepines if agitated
 - Active cooling if temperature > 40°C:

- Tepid water mist
 - Evaporate with fan
- Paralysis:
 - Indicated if muscle rigidity and hyperactivity contributing to persistent hyperthermia
 - Nondepolarizing agent (e.g., vecuronium)
 - Avoid succinylcholine
 - Intubation; mechanical ventilation
- Administer acetaminophen.
- Apply cooling blankets.
- Severe, malignant hypertension:
 - Nitroprusside (for MAOI overdose)
 - Calcium-channel blocker or phentolamine (for MAOI + food interaction)
 - Use short-acting IV agent that can be rapidly “turned off.”
- Hypotension:
 - Initially bolus with isotonic crystalloid solution
 - If no response, administer norepinephrine.
 - Dopamine may be ineffective
- Dysrhythmias (premorbid sign in MAOI overdose):
 - Treatment based on dysrhythmia
- Seizures:
 - Benzodiazepines
 - Barbiturates if benzodiazepines unsuccessful
 - Pyridoxine for refractory seizures
- Rigidity:
 - Benzodiazepines
 - Paralysis with vecuronium, endotracheal intubation, and mechanical ventilation
- ARDS:
 - Oxygen
 - Intubation and positive end-expiratory pressure as indicated
- DIC:
 - Fresh-frozen plasma
 - Platelets
 - Whole-blood transfusions
- Rhabdomyolysis:
 - IV isotonic crystalloid solution
 - Maintain hydration to ensure adequate urine output
- Specific treatment for SS:
 - Mainstay: Supportive care, discontinuation of offending agents
 - Nonselective serotonin antagonist:
 - Cyproheptadine

ALERT

Phentolamine *contraindicated in MAOI overdose* (results in unopposed β -agonist)

MEDICATION

- Activated charcoal: 1–2 g/kg PO
- Cyproheptadine: 4–8 mg PO/nasogastric tube q1–4h until therapeutic response; max. daily dose: 0.5 mg/kg (peds: 0.25 mg/kg/d; max. 12 mg/d; safety not established age < 2 yr)
- Dextrose: D50W 1–2 amp (50–100 mL or 25–50 g) (peds: D25W 2–4 mL/kg) IV push (IVP)
- Diazepam: 5–10 mg (peds: 0.1 mg/kg slowly) increments IVP
- Lorazepam: 1–2 mg increments IVP
- Nitroprusside: 0.3–10 μ /kg/min IV
- Norepinephrine: 2–4 μ /kg/min (peds: 0.05–0.1 μ /kg/min) IV
- Phentolamine: 5 mg (peds: 0.05–0.2 mg/kg/dose) increments IVP
- Sodium bicarbonate: Bolus 1–2 mEq/kg IVP; adult infusion: 3 amp (50 mEq per amp) sodium bicarbonate in 1,000 mL D5W at 2–3 mL/kg/h IV
- Vecuronium: 0.1 mg/kg IVP



FOLLOW-UP

DISPOSITION

ALERT

MAOI toxicity can occur in delayed fashion necessitating an extended observation period

Admission Criteria

- All MAOI overdose patients require admission to a monitored unit for 24 hr.
- ICU admission for seriously ill patients

Discharge Criteria

- Resolved mild hypertensive syndrome or resolved mild serotonin syndrome may be discharged after several hours of ED observation.

Issues for Referral

Intentional overdoses should receive a psychiatry consult for suicide attempt.

FOLLOW-UP RECOMMENDATIONS

Following significant MAOI toxicity, medications need to be reassessed to prevent future crises.

PEARLS AND PITFALLS

- Delayed onset of 6–12 hr prior to symptoms
- Linezolid and methylene blue are MAOIs.
- Phentolamine is contraindicated in MAOI overdose secondary to unopposed β -agonist.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Sympathomimetic Poisoning



CODES

ICD9

969.01 Poisoning by monoamine oxidase inhibitors

ICD10

- T43.1X1A Poisoning by monoamine-oxidase-inhibitor antidepressants, accidental (unintentional), initial encounter
- T43.1X2A Poisoning by monoamine-oxidase-inhibitor antidepressants, intentional self-harm, initial encounter
- T43.1X4A Poisoning by MAO inhib antidepressants, undetermined, init

MONONUCLEOSIS

Steven C. Rogers • Alberto Cohen-Abbo

BASICS

DESCRIPTION

- Results in most cases from infection with the Epstein–Barr virus (EBV) (a herpesvirus):
 - Non-EBV causes of infectious mononucleosis (IM):
 - Cytomegalovirus (CMV)
 - Adenovirus
 - Hepatitis A
 - Herpesvirus 6
 - HIV
 - Rubella
 - *Toxoplasma gondii*
 - Group A β -hemolytic streptococci
- > 90% of adults on serologic testing demonstrate prior infection with EBV:
 - Most do not recollect specific IM symptoms
- Mode of transmission is close or intimate contact particularly with saliva from “shedders” who may or may not be symptomatic:
 - Nickname “kissing disease”
 - Viral shedding in saliva can persist intermittently for life
 - May occur after transfusions/transplants
- Incubation period: 4–6 wk
- Immunologic response:
 - T-cells response:
 - T-cell response is responsible for an elevated absolute lymphocyte count and the associated clinical symptoms and complications
 - Subtype of the T-cell lineage, cytotoxic CD8 cells (Downey cells), contain eccentrically placed and lobulated nuclei with vacuolated cytoplasm: The “atypical lymphocytes” seen on differential
 - B-cell response:
 - EBV infects and replicates in B-cells
 - B-cells are then transformed into plasmacytoid cells that secrete immunoglobulins
 - IgM antibody secreted: The heterophile antibody which is reactive against red cell antigens
- Mortality from IM is rare, but may occur due to the following complications:
 - Airway edema
 - Neurologic complications

- Secondary bacterial infection
- Splenic rupture
- Hepatic failure
- Myocarditis
- EBV infection has also been strongly linked to African Burkitt lymphoma and nasopharyngeal carcinoma

Pediatric Considerations

- In children < 4 yr, infection with EBV is often asymptomatic
- In children who do become symptomatic, there is propensity toward atypical presentations:
 - Neutropenia, pneumonia, and varied rashes
 - Mesenteric lymphadenopathy and splenomegaly can cause the illness to present with abdominal pain and be confused with appendicitis.
 - Infants and toddlers can present with only irritability and failure to thrive so must be considered when no other source can be identified

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Typically an insidious onset over several days to weeks but may be abrupt onset
- Prodromal fatigue, malaise, arthralgias, and myalgias with a biphasic or “waxing and waning” course
- Prominent or “worst ever” sore throat and fever. Airway edema may be reported as difficulty breathing or respiratory distress.
- Swollen lymph nodes
- Headache
- Significant abdominal pain is uncommon but when present should raise concern about marked splenic enlargement or splenic rupture.
- Varied rashes can be seen in 18–34% of children and adolescents (not associated with antibiotics)
- Administration of ampicillin or amoxicillin in patients with IM is associated with development of a rash

Physical-Exam

- Malaise and/or fatigue (90–100%)
- Pharyngitis (65–85%) and tonsillar enlargement
- Fever (80–95%)
- Eyelid edema (15–35%)
- Symmetric tender lymphadenopathy (100%)

- Hepatomegaly (15–25%)
- Splenomegaly (50–60%)
- Nonspecific rashes
- Morbilliform rash can be seen if the patient has been given ampicillin or amoxicillin:
 - Typically develops 5–9 days after the onset of antibiotic therapy (should not be interpreted as a penicillin allergy)
- Petechia can occur on the skin or at the junction between the hard and the soft palate.
- Complications found on exam:
 - Airway compromise due to edema (1–5%)
 - Severe abdominal tenderness may be due to splenic rupture (may also cause referred pain to left shoulder)
 - Jaundice (~5%) due to hepatitis or hepatic failure
 - Hepatitis is the most common complication
 - Neurologic findings consistent with:
 - Encephalitis or cerebellitis
 - Aseptic meningitis
 - Guillain–Barré syndrome
 - Optic neuritis
 - Bell palsy
- Anemia (pallor): May be due to hemolytic anemia, thrombocytopenia, agranulocytosis, hemophagocytic lymphohistiocytosis (HLH)
- Orchitis
- Neck tenderness and/or limited range of motion due to pain: Secondary bacterial soft tissue infection such as retropharyngeal or peritonsillar abscesses
- Signs of shock: May be due to dehydration or a secondary anaerobic sepsis

DIAGNOSIS TESTS & INTERPRETATION

Lab

- WBC with differential:
 - Typically a modest elevation in total WBC between 10,000 and 20,000, which peaks during week 2 of the illness but occasionally can be 30,000–50,000
- Lymphocyte count—findings suggestive of IM:
 - >50% lymphocytes on differential
 - Absolute lymphocyte count >4,500
 - Elevated lymphocyte count with >10% atypical lymphocytes (up to 90% of patients)
- Liver function tests:
 - Elevated with transaminases up to 3 times normal found in 80–85% of patients in the 1st 2 wk

- Significant elevations in bilirubin to the point of causing clinical jaundice in ~5% of cases
- Monospot test detects presence of heterophile antibodies:
 - Moderately sensitive (85%) and highly specific (practically 100%)
 - Rarely false positives can occur with CMV, leukemia, lymphoma, rubella, hepatitis, HIV, or lupus
 - Most patients develop heterophile antibodies after ~1 wk of illness
 - Small percentage of patients (<10%) never develop heterophile antibodies
 - Heterophile antibodies peak at 2–5 wk and may persist for several months
 - Positive test relates to a titer >1:40
 - Results likely to be negative in children <4 yr old
- Testing does exist for EBV-specific antibodies but is expensive, time consuming, and rarely needed
 - Useful in patients with atypical/severe cases or when monospot testing is negative and confirmation of IM is desired
 - Acute infection is indicated by antibodies (IgG, IgM) against viral capsid antigens (VCAs) without antibodies against the Epstein–Barr nuclear antigen (EBNA) which are only present during the latency period 3–4 wk after onset of illness
 - Past infection indicated by negative IgM and positive EBNA

Imaging

Sonography or CT scan of abdomen for significant abdominal pain to identify splenic rupture and to ensure no signs of appendicitis

DIFFERENTIAL DIAGNOSIS

Divided into infectious and noninfectious causes:

- Infectious:
 - Adenovirus
 - CMV
 - Streptococcal pharyngitis
 - HIV
 - Rubella
 - Hepatitis A, B, C
 - Diphtheria in nonimmunized populations
 - Mumps
 - Toxoplasmosis
- Noninfectious:
 - Leukemia
 - Lymphoma
 - Medication-induced syndrome—phenytoin, sulfa drugs



TREATMENT

PRE HOSPITAL

ALERT

- Follow standard universal precautions
- ABCs. Assess airway patency
- Initiate IV hydration with normal saline if patient is dehydrated

INITIAL STABILIZATION/THERAPY

- ABC management. Airway edema may require intervention
- If possible, avoid placing patient in the same general area as post-transplant and other immunocompromised patients

ED TREATMENT/PROCEDURES

- Supportive therapy:
 - Hydration with IV or PO fluids
 - Antipyretics for fever control
 - Analgesics for pain of sore throat
- Steroids (methylprednisolone, prednisone, or dexamethasone) if there is significant pharyngeal/tonsillar edema with concern about impending airway obstruction. May also be considered for massive splenomegaly, myocarditis, hemolytic anemia, or HLH. Treatment is controversial and theoretically may be associated with increased risk of secondary infections or malignant disease
- Antiviral therapy has not been proven to effect clinical course but emerging research may suggest potential benefits
- Antibiotics if concerned for bacterial superinfection:
 - Avoid ampicillin because of associated rash
- Counsel patient on athletic activity limitations (*see follow-up recommendations*)

Pediatric Considerations

Advise parents of athletic activity limitations (*see follow-up recommendations*)

MEDICATION

- Methylprednisolone: 125 mg IV (peds: 2 mg/kg IV up to adult dose)
- Prednisone: 20–40 mg PO daily for 7 days (peds: 1 mg/kg up to adult dose) with subsequent tapering
- Dexamethasone: 12–16 mg PO (peds: 0.3 mg/kg up to adult dose)



FOLLOW-UP

DISPOSITION

Admission Criteria

- Significant airway edema that represents any level of potential airway compromise
- Neurologic or severe hematologic/hepatic complications
- Inability to take PO
- Pain control

Discharge Criteria

- No airway compromise
- Mild hematologic complications or mild hepatitis
- Ability to take PO fluids
- Fever usually resolved within 10 days and lymph nodes and spleen within 4 wk; fatigue may continue for several weeks, although it may go on for 2–3 mo

Issues for Referral

- Infectious disease consultation may be useful if serology is not conclusive
- Significant complications or persistent symptoms

FOLLOW-UP RECOMMENDATIONS

- Contact sports, physical education, or other strenuous exercise should be avoided in the 1st 3 wk regardless of spleen size or current symptoms.
- After the initial 3-wk period patients need repeat outpatient evaluation to determine if they are able to return to full activity. Those with concerns of persistent symptoms including splenomegaly may require further studies (i.e., ultrasound) to determine when they are safe to return to full activity

PEARLS AND PITFALLS

- Although usually self-limited, significant complications occur and require consultation
- Treatment with steroids may be useful but is controversial due to potential increased risk of complications

ADDITIONAL READING

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CODES

ICD9

075 Infectious mononucleosis

ICD10

- B27.00 Gammaherpesviral mononucleosis without complication
- B27.10 Cytomegaloviral mononucleosis without complications
- B27.90 Infectious mononucleosis, unspecified without complication

MRSA, COMMUNITY ACQUIRED

Benjamin S. Heavrin

BASICS

DESCRIPTION

- Methicillin-resistant *Staphylococcus aureus* (MRSA) has historically been a pathogen endemic within healthcare settings, usually affecting the elderly and chronically ill. This strain of *S. aureus* has been termed “healthcare-associated MRSA” (HA-MRSA).
- Throughout the past decade, MRSA has become an increasingly common pathogen among younger, healthier populations who do not have a healthcare-related exposure history. This type of MRSA pathogen has been termed “community-acquired MRSA” (CA-MRSA).
- CA-MRSA is the most common cause of skin and soft tissue infections seen in the ED
- While CA-MRSA may cause skin and soft tissue infection, it may also lead to severe multisystem disease, including sepsis and necrotizing pneumonia

Geriatric Considerations

HA-MRSA (see below) is a different genotypic form of MRSA that frequently causes morbidity among the elderly, especially those living within extended-care facilities or those with healthcare-related exposures.

ETIOLOGY

- *S. aureus* is a gram-positive cocci frequently colonizing the skin
- MRSA refers to a specific strain of *S. aureus* that has resistance against the antimicrobial properties of numerous antibiotics, including methicillin
- Prisoners, athletes, soldiers, children in daycare, IV drug users, and those with prior treatment for MRSA or exposure to MRSA are at highest risk for colonization and subsequent infection.

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Skin and soft tissue infections:
 - Increasing redness
 - Pain
 - Warmth

- Swelling
- Fever
- Chills
- Malaise
- Sepsis/pneumonia:
 - Weakness
 - Dyspnea
 - Fever
 - Rigors
 - Productive cough
 - Chest pain
- Inquire about prior diagnosis of MRSA infections, MRSA exposures, and family members or close contacts with a history of MRSA, as such a patient is at risk for CA-MRSA infection.

Physical-Exam

- Skin and soft tissue infections:
 - Abscess: Tender, raised boil with underlying induration and fluctuance
 - Cellulitis: Warm erythema possibly with lymphangitic streaking
- Sepsis:
 - Vital sign abnormalities including tachycardia and hypotension, respiratory failure, mental status changes, petechiae, systemic signs of toxicity
- Pneumonia:
 - Tachypnea, crackles, retractions, hypoxia
 - Alveolar opacities on chest radiographs

Pediatric Considerations

MRSA is the leading cause of skin and soft tissue infections among children presenting to the emergency department.

ESSENTIAL WORKUP

- Abscess:
 - I&D with packing and prompt follow-up is warranted for abscess
 - Microbiology often performed for antibiotic sensitivity given the changing antimicrobial resistance patterns
- Sepsis:
 - Source identification, including blood culture/urine culture, CXR, is indicated as resuscitation begins
- Pneumonia:
 - Chest radiographs and continuous vital sign monitoring is indicated

DIAGNOSIS TESTS & NTERPRETATION

Lab

- Skin and soft tissue infections:
 - Bacterial culture is often warranted to monitor for CA-MRSA resistance patterns
- Sepsis and pneumonia:
 - Blood, urine, and body fluid cultures. CBC, CMP to assess for organ dysfunction

Imaging

- Bedside US:
 - Abscess: Anechoic fluid collection
 - Cellulitis: “Cobblestoning” within the soft tissue
- CXR:
 - Indicated for patients with presumed sepsis, systemic illness, or pneumonia

Diagnostic Procedures/Surgery

Cultures of skin and soft tissue infections are frequently obtained to monitor microbiology and antimicrobial resistance patterns should a patient fail a course of therapy.

DIFFERENTIAL DIAGNOSIS

- Other skin and soft tissue infections:
 - Pathogens beyond MRSA which cause abscesses and cellulites should be considered (i.e., streptococcus)
- Necrotizing fasciitis
- Contact dermatitis
- Deep vein thrombosis
- Spider/insect bite
- Drug reaction

ALERT

Empiric antimicrobial treatment of skin and soft tissue infections should cover for common skin pathogens beyond MRSA (i.e., streptococcus)



TREATMENT

PRE HOSPITAL

- Contact precautions for all providers if MRSA is suspected
- IV access and fluid resuscitation if sepsis is suspected

INITIAL STABILIZATION/THERAPY

Begin resuscitation and administer early empiric antibiotics if pneumonia, fasciitis, or

sepsis is suspected:

- Include early coverage with antibiotics effective against MRSA

ED TREATMENT/PROCEDURES

- Skin and soft tissue infections:
 - Abscess:
 - I&D with packing
 - Antibiotics may not be necessary if there is no evidence for deep tissue infection or cellulitis
 - Cellulitis:
 - Cellulitis caused by CA-MRSA in a healthy, well-appearing patient may be treated with oral antibiotics in the outpatient setting
 - Ill appearing patients, patients with underlying medical conditions, and patients failing outpatient therapy require IV antibiotics with coverage against CA-MRSA
- Sepsis and pneumonia:
 - Early administration of broad-spectrum antibiotics that cover against MRSA should be given promptly if the patient is at risk for CA-MRSA

MEDICATION

ALERT

Review antimicrobial resistance patterns of CA-MRSA within your community prior to choosing a specific antibiotic regimen, as many antibiotics listed below may not be 100% effective against CA-MRSA.

First Line

- Bactrim:
 - Adults: Bactrim DS 160/800 PO BID
 - Children: 10 mg/kg PO BID
- Clindamycin:
 - Adults: 150–450 mg PO QID
 - Children: 5 mg/kg PO/IV TID–QID
- Doxycycline:
 - Adults: 100 mg PO BID
 - Children: 2.2 mg/kg PO BID
- Vancomycin:
 - Adults: 1 g IV q8–12h
 - Children: 15 mg/kg IV q8–12h

Second Line

- Rifampin:
 - Should not be used as monotherapy due to inducible resistance

- Adults: 300 mg PO BID
- Children: 10–20 mg/kg/d in 2 div. doses PO for 5 days; not to exceed 600 mg/d
- Linezolid:
 - Adults: 600 mg PO/IV q12h
 - Children: 10 mg/kg PO/IV q8h

Pregnancy Considerations

Avoid the use of tetracyclines in pregnancy



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients with signs/symptoms of bacteremia, progressive infection, or systemic illness should be admitted:
 - Fever, chills, lymphangitic streaking
- Patients with underlying comorbid diseases such as diabetes or immunodeficiency should be admitted
- Individuals who have failed a course of outpatient therapy should be admitted and given IV antibiotics effective against MRSA

Discharge Criteria

Healthy, well-appearing patients with simple skin and soft tissue infections may be followed in the outpatient setting.

Issues for Referral

MRSA infection refractory to multiple medications may require infectious disease consultation.

FOLLOW-UP RECOMMENDATIONS

- All skin and soft tissue infections should be re-evaluated within 24–48 hr to monitor for clinical improvement.
- Individuals failing outpatient therapy require hospital admission and IV antibiotics.

PEARLS AND PITFALLS

- CA-MRSA is the most common cause of skin and soft tissue infections seen in the ED.
- CA-MRSA is a rare but serious cause of rapidly progressive pneumonia and sepsis.

- Antibiotic resistance patterns are dynamic and vary widely across geographic boundaries.
- Be cautious with long-term use of tetracyclines in children.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Abscess
- Cellulitis
- Pneumonia
- Sepsis



CODES

ICD9

041.12 Methicillin resistant *Staphylococcus aureus* in conditions classified elsewhere and of unspecified site

ICD10

- A41.02 Sepsis due to Methicillin resistant *Staphylococcus aureus*
- A49.02 Methicillin resis staph infection, unsp site

MULTIPLE MYELOMA

Nicole M. Franks

BASICS

DESCRIPTION

- Normal cells transform into myeloma cells at the hematopoietic stem cell level.
- Pathologic derangements:
 - Tumor cells within marrow lead to bone destruction and cytopenia.
 - Immunodeficiency develops secondary to suppression of normal immune functions.
 - Myeloma proteins lead to hyperviscosity and amyloidosis.
 - Multifactorial renal failure
- Plasma cell secretions activate osteoclasts, leading to:
 - Bone lysis, pathologic fractures, and neurologic impairment
 - Hypercalcemia (exacerbated by impaired renal function)
- Anemia due to marrow infiltration and renal insufficiency
- Immunocompromised due to:
 - Decrease in the number of normal immunoglobulins
 - Qualitative and quantitative defects in T- and B-cell subsets
 - Granulocytopenia
 - Decreased cell-mediated immunity
- Hyperviscosity secondary to protein accumulation:
 - Leads to high-output congestive heart failure
- Myeloma light chains accumulate in the renal epithelial cells and destroy the entire nephron.
- Clinical signs such as anemia, renal insufficiency, or lytic bone lesions
- Complications:
 - Pathologic fractures
 - Hypercalcemia
 - Renal failure
 - Recurrent infection
 - Anemia
 - Spinal cord compression (10% of all multiple myeloma [MM] patients)

ETIOLOGY

- Incidence: 4/100,000 population:
 - 1% of all cancers
 - 15% of all hematopoietic malignancies
 - 10,000 deaths/yr
- Mean age at diagnosis is 70 yr

- Slightly higher incidence in men and African Americans (reason unknown)

Pediatric Considerations

- Rarely seen in children.
- <2% in patients <40 yr of age

DIAGNOSIS

SIGNS AND SYMPTOMS

- Bone pain predominates (with secondary disuse or neurologic sequelae):
 - Ribs/sternum
 - Spine
 - Clavicle
 - Skull
 - Shoulder
 - Hip
- Constitutional symptoms:
 - Anemia
 - Weakness
 - Fatigue
 - Recurrent infection
 - Weight loss
- Asymptomatic (20%):
 - MM found on follow-up of routine blood screening
- Multiple bouts of sepsis secondary to the encapsulated organisms (*Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Staphylococcus*).

ESSENTIAL WORKUP

- CBC, ESR, electrolytes, BUN, creatinine, urinalysis
- Plain radiographs related to bone pain:
 - Skeletal survey: Lateral skull, AP/lateral spine, AP of pelvis, humerus, and femur
- CT or MRI for persistent bone pain with negative plain radiographs
- Confirmation of diagnosis:
 - Serum and urine protein electrophoresis
 - Serum and urine protein immunofixation (diagnostic when electrophoresis is normal or nonspecific)
 - Vitamin D levels
 - Bone marrow biopsy

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC:
 - Normochromic, normocytic anemia
 - Thrombocytopenia
 - Leukocytosis
- “Rouleaux” formation on peripheral blood smear (stacks of red blood cells)
- Electrolytes, BUN, creatinine, glucose:
 - Renal insufficiency
- Serum calcium:
 - Hypercalcemia due to bone resorption
- Urinalysis:
 - Dipstick selects for albumin and not light-chain proteinuria.
 - False-negative screening urinalysis for Bence Jones protein is common.
- Elevated erythrocyte sedimentation rate (ESR)
- Urinary and serum electrophoresis show a monoclonal protein spike:
- Quantitative screening for light chain is diagnostic.

Imaging

- Plain radiographs demonstrate:
 - Lytic bone lesions
 - Pathologic fractures
- CT:
 - More sensitive for small lesions
 - Can differentiate malignant from benign vertebral compression fractures in non-MRI candidates
- MRI:
 - Preferred to detect spinal compression or soft-tissue plasmacytomas
- PET with MR or CT: May have future role in surveying response to treatment
- Technetium pyrophosphate bone scan:
 - Lights up bone deposition
 - False-negative scan with MM due to an uncoupling of bone absorption and deposition that results in a negative bone scan even when lytic lesions are present
- Bone marrow biopsy: Increase in plasma cells
- Cytogenetic screening may offer prognostic significance.

DIFFERENTIAL DIAGNOSIS

- Monoclonal gammopathy of undetermined significance
- Amyloidosis
- Chronic lymphocytic leukemia
- Non-Hodgkin lymphoma
- Waldenström macroglobulinemia
- Bone marrow plasmacytosis includes collagen vascular disease, cirrhosis, immune complex disease, viral illness, and papular mucinosis.



TREATMENT

PRE HOSPITAL

Immobilize appropriately patients with MM who present with back pain or neurologic symptoms:

- Presume to have a pathologic spinal fracture

INITIAL STABILIZATION/THERAPY

Recognition and treatment of:

- Hypercalcemia
- Renal failure
- Sepsis
- Spinal cord compression
- Anemia

ED TREATMENT/PROCEDURES

- Opiate analgesics are the mainstay of therapy in ED (NSAIDS may worsen renal insufficiency).
- Splint pathologic fracture; immobilize pathologic spine fractures.
- Aggressive normal saline hydration with bisphosphonate therapy for hypercalcemia
- Symptomatic anemia may be managed with transfusions or erythropoietin therapy.
- Hematology/oncology consultation for chemotherapy—administer on inpatient/outpatient basis:
 - Early or asymptomatic stages do not need treatment.
 - Chemotherapy in early stage shows no benefit.
 - Melphalan and prednisone combination chemotherapy is the most common treatment:
 - Symptom relief and decrease in M protein levels in up to 70% of patients
 - Alternative chemotherapy includes cyclophosphamide with or without prednisone or VAD (vincristine, doxorubicin [Adriamycin], and dexamethasone).
- Prolonged melphalan use may lead to a secondary leukemia.
- High-dose chemotherapy with stem cell transplantation has shown promise.
- Thalidomide is useful for salvage therapy.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Refractory pain requiring systemic analgesics
- Life-threatening complications of MM, including acute renal failure, hypercalcemia, sepsis, spinal cord compression, hyperviscosity, neutropenia, and cardiac tamponade

Discharge Criteria

Pain controlled with oral analgesics

Issues for Referral

- Oncology referral for all patients regardless of stage of disease discovery
- Neurosurgery and orthopedic referral for persistent vertebral pain that may require percutaneous vertebroplasty or kyphoplasty

PEARLS AND PITFALLS

- Infectious complications are the major cause of morbidity and mortality such that febrile illness should be treated with empiric therapy for common respiratory and urinary tract infections.
- Consider diagnosis of multiple myeloma for any persistent neurologic complaints or unknown mobility in the elderly.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Anemia
- Hypercalcemia
- Renal Failure
- Sepsis



CODES

ICD9

203.00 Multiple myeloma, without mention of having achieved remission

ICD10

C90.00 Multiple myeloma not having achieved remission

MULTIPLE SCLEROSIS

Richard S. Krause

BASICS

DESCRIPTION

- Pathophysiology: Recurrent episodes of CNS demyelination:
 - Signs and symptoms depend on location of lesions and timing of demyelination
- Multiple sclerosis (MS) occurs in distinct patterns:
 - *Relapsing recurring MS*: 2 or more episodes lasting ≥ 24 hr separated by ≥ 1 mo
 - *Primary progressive MS*: Slow or stepwise progression over at least 6 mo
 - *Secondary progressive MS*: Initial exacerbations and remissions followed by slow progression over at least 6 mo
 - *Stable MS*: No progression (without treatment) over at least 18 mo

ETIOLOGY

- MS is a chronic demyelinating disease of CNS:
 - Etiology is not well understood
- Presumed to be T cell-mediated autoimmune disease
- There is evidence for a viral trigger
- Plaques in white matter:
 - Characterized by infiltrate of T cells and macrophages
- Persons of northern European origin most often affected (in US)
- Increased prevalence is seen moving away from equator

DIAGNOSIS

SIGNS AND SYMPTOMS

- Initial attacks usually (~85%) represent single lesions, are abrupt in onset, and are seen in characteristic patterns (in order of decreasing frequency):
 - Optic neuritis:
 - Eye pain exacerbated by movement progressing to visual loss
 - Paresthesias (or changed sensory level) in 1 limb
 - Limb (usually leg) weakness
 - Diplopia:
 - Intranuclear ophthalmoplegia from lesion of medial longitudinal fasciculus
 - Results in unilateral or bilateral paralysis of adduction of eye on horizontal gaze

- Trigeminal neuralgia
- Urinary retention
- Vertigo
- Transverse myelitis:
 - Acute onset of motor and sensory findings at specific spinal cord level
 - Often associated with bladder or bowel incontinence
 - Can be early manifestation of MS
 - Unusual initial manifestations include psychosis, aphasia, etc.
 - Initial symptoms may be minor and only be recognized as due to MS in retrospect
- Symptoms typically develop abruptly (minutes to hours) and last 6–8 wk
- Most common in young women of Northern European descent:
 - Increased risk in 1st-degree relatives
 - Peak age: 30 yr
 - Female-to-male ratio: 2:1
- Pain is an uncommon symptom in MS:
 - Exceptions: Trigeminal neuralgia, early optic neuritis

Physical-Exam

Physical exam: Focused on “hard” neurologic signs:

- Focal neurologic deficits
- Afferent pupillary defect
- Internuclear ophthalmoplegia
- Sensory level or sphincter disturbance (transverse myelitis)

ESSENTIAL WORKUP

- MS is suspected based on history and physical exam
- Definitive diagnosis is not typically made in ED:
 - Requires observation over time and confirmatory testing

ALERT

Infection, especially with fever, is a common cause of MS exacerbation

DIAGNOSIS TESTS & INTERPRETATION

Lab

CSF analysis:

- CSF gel electrophoresis reveals “oligoclonal bands” not present in serum
 - Oligoclonal bands are found in $\geq 95\%$ of patients with clinically definite MS

Imaging

MRI most useful imaging test:

- Lesions appear as areas of high signal, in cerebral white matter or spinal cord on

T2-weighted images

- Abnormal in almost all patients who have clinically diagnosed MS
- McDonald diagnostic criteria for MS include specific MRI findings
- May also see plaques on CT (less sensitive)

Diagnostic Procedures/Surgery

Sensory-evoked potential testing:

- Not an ED test

DIFFERENTIAL DIAGNOSIS

- Signs and symptoms of MS are usually *focal*:
 - Diffuse symptoms (seizures, syncope, and dementia) are seldom due to MS
- Cerebrovascular accident and transient ischemic attack:
 - Usually older patients with risk factors for atherosclerotic disease or atrial fibrillation
- Systemic lupus erythematosus:
 - CNS involvement usually in setting of known disease and usually nonfocal
- Sarcoid:
 - CNS manifestations usually with known disease and lung involvement
- Lyme disease:
 - May mimic MS
 - Seek history of rash and tick exposure in geographic areas of high risk
 - Lyme titers may aid in diagnosis
- Psychiatric illness is diagnosis of exclusion
- Postinfectious or postimmunization demyelination may mimic MS, usually in children
- Guillain–Barré is usually ascending and symmetric and progresses over hours to days
- MS unlikely in patients with:
 - Normal neurologic exam
 - Abrupt hemiparesis (stroke)
 - Aphasia (stroke)
 - Pain predominating
 - Very brief symptoms (seconds to minutes)
 - Age < 10 or > 50 yr



TREATMENT

INITIAL STABILIZATION/THERAPY

Fever in MS patients is treated aggressively because it can worsen the manifestations of MS

ED TREATMENT/PROCEDURES

- Acute optic neuritis:
 - High-dose parenteral steroids
 - Oral steroids *relatively contraindicated* (has been reported to increase recurrence risk)
- Exacerbations:
 - High-dose IV methylprednisolone (up to 1 g/day) or other parenteral corticosteroid
 - High-dose oral steroids are sometimes used (except for optic neuritis)
- Symptomatic treatment:
 - Spasticity: Baclofen, tizanidine
 - Tremor: Clonazepam
 - Urinary symptoms:
 - Diagnose and treat infection
 - Self-catheterization
 - Oxybutynin may promote continence
 - Trigeminal neuralgia: Carbamazepine
 - Fatigue, general weakness: Amantadine, methylphenidate, and modafinil have been used
 - Depression: SSRIs are effective

MEDICATION

- Amantadine: 100 mg PO BID
- Baclofen: 10 mg PO TID initially; may increase to 25 mg PO TID
- Carbamazepine: 100 mg PO BID to 200 mg PO QID
- Clonazepam: 0.5 mg/d PO, increase in 0.5-mg increments and up to 3 times a day
- Methylprednisolone: 1 g IV daily (1st-line treatment)
- Modafinil: 100–200 mg PO daily in AM
- Oxybutynin: 5 mg PO BID to TID
- Tizanidine: 2–8 mg PO TID



FOLLOW-UP

DISPOSITION

Admission Criteria

- Acute exacerbation that requires IV therapy
- Patients unable to care for themselves due to severity of their illness
- Another condition requiring inpatient treatment cannot be effectively ruled out

Discharge Criteria

- *Suspected MS*: Patients may be referred for outpatient evaluation if their general

condition permits and other serious conditions requiring admission have been effectively ruled out

- *Complication of known MS*: Discharge if effective outpatient treatment is available for complication or exacerbating factor

FOLLOW-UP RECOMMENDATIONS

Patients with suspected MS should be referred to their primary care provider or a neurologist for further evaluation

PEARLS AND PITFALLS

- Signs and symptoms of MS are usually focal
- Diffuse symptoms are rarely MS
- Oral steroids are contraindicated
- Treat fever in MS patients aggressively

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Cerebrovascular Accident
- Guillain–Barré Syndrome
- Lyme Disease



CODES

ICD9

340 Multiple sclerosis

ICD10

MUMPS

Austen-Kum Chai

BASICS

DESCRIPTION

Vaccine preventable infectious diseases characterized by swelling of salivary glands, in particular the parotid glands.

ETIOLOGY

- Rubulavirus, single stranded RNA virus, in the Paramyxovirus family
- Humans only known reservoir

Pediatric Considerations

- Mumps vaccine with measles and rubella \pm varicella (MMR or MMRV) should be administered to children on or after 12 mo of age. 2nd dose is usually administered at the age of 4–6 yr, before start of school.
- Catch-up vaccination should include 2 doses separated by at least 4 wk between vaccinations.
- Systemic symptoms and serious complications are less common in children when compared to adults with the infection.

Pregnancy Considerations

Infection during 1st trimester of pregnancy is associated with increased spontaneous abortion. Although mumps virus may cross the placenta, there is no evidence that mumps virus causes congenital malformation.

Geriatric Considerations

Adults born before 1957 are considered to have been exposed to mumps and are considered immune. However, during outbreaks, healthcare workers born before 1957 and without lab evidence of immunity to mumps should receive 2 doses of the MMR vaccine.

DIAGNOSIS

SIGNS AND SYMPTOMS

- Incubation period from exposure to onset of symptoms (14–18 days):
 - Viral transmission is via respiratory droplets, saliva, contact with contaminated fomites
 - Replication in nasopharynx and regional lymph nodes
 - Viremia to glands as salivary, pancreas, testes, ovaries and also to meninges

- Contagious 1–7 days prior to onset of symptoms and 6 days afterward
- Often history of none or incomplete immunization
- Active illness (1–10 days):
 - Nonspecific prodromal symptoms such as low-grade fever, headache, malaise, myalgia, anorexia, otalgia, jaw pain up to 48 hr prior to presentation of parotitis
 - Up to 20% of infections are asymptomatic but still contagious
 - Up to 50% of cases have nonspecific symptoms of upper respiratory tract infection along with fever, malaise, anorexia, and headache without apparent salivary gland swelling.
 - *Parotitis* (30–40% of patients):
 - Most common manifestation of mumps. Present in 95% of symptomatic cases of mumps
 - Painful and tender unilateral or bilateral (90% of the time) enlargement of parotid gland
 - May begin as earache or pain at angle of jaw
 - Other salivary gland may be affected.
 - Stensen's duct is often edematous and erythematous and exudes a clear fluid.
 - Skin overlying swollen gland is nonerythematous.
 - Symptoms decrease after 1 wk and resolve by 10th day.
 - Considered contagious until swelling resolves
 - *Orchitis* (20–50% of postpubertal males):
 - Most common complication in postpubertal males
 - May occur alone, before, during, but most commonly, after parotitis
 - Unilateral or bilateral (up to 30%)
 - Abrupt, painful, tender swelling with nausea, vomiting, and fever
 - Pain and swelling resolve in 1 wk
 - Testicular atrophy in up to 50% of patients
 - Sterility rare
 - *Oophoritis* (5% of postpubertal females):
 - May mimic appendicitis if right-sided
 - Fertility not impaired
 - Pancreatitis (2–5%):
 - May occur without any other manifestations of mumps
 - Fever, nausea, vomiting, and epigastric pain
 - May see transient hyperglycemia
 - May be complicated by pseudocyst formation and shock
 - CNS involvement:
 - Aseptic meningitis (10–15% of patients)
 - Usually resolves without sequelae in 3–10 days
 - Encephalitis (very rare)
 - Sensorineuronal deafness (80% unilateral) with permanent hearing

- impairment
- Cerebellar ataxia
- Transverse myelitis
- Other:
 - Myocarditis (rarely with symptomatic involvement)
 - Glomerulonephritis
 - Polyarthralgia and arthritis
 - Thrombocytopenic purpura
 - Ocular complaints
 - Thyroiditis
 - Mastitis

ESSENTIAL WORKUP

Diagnosis is based on clinical findings of parotitis and associated signs, symptoms, and complications.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Lab tests as needed
- Cerebrospinal fluid (CSF) for symptomatic CNS involvement
- Hyperamylasemia usually due to parotitis and supports diagnosis
- Mumps RNA detection using PCR assays, mumps viral cultures, or enzyme immunoassays for mumps IgM antibody or significant rise in IgG titers between acute and convalescent specimens:
 - Provides definitive diagnosis
 - Viral culture may be from blood, throat swab, salivary gland secretions, CSF, or urine. PCR provides rapid confirmation of mumps in CSF
 - Not indicated unless need to confirm diagnosis in absence of parotitis

DIFFERENTIAL DIAGNOSIS

- Bacterial parotitis:
 - Commonly *Staphylococcus aureus*
 - Erythematous and tender parotid gland
 - Usually in elderly or immunocompromised
- Calculus parotid:
 - Stone may be palpable or may be seen on sialogram (CT)
- Cervical adenitis
- Tumors:
 - Older patients
 - History of indolent course
- Testicular torsion
- Bacterial epididymo-orchitis

- Other viral cause of parotitis; influenza A, parainfluenza, cytomegalovirus, coxsackieviruses, HIV



TREATMENT

PRE HOSPITAL

Nonimmunized pre-hospital care personnel exposed to mumps should be advised of potential risks.

INITIAL STABILIZATION/THERAPY

IV fluids for vomiting/dehydration

ED TREATMENT/PROCEDURES

- Prevention with mumps vaccination is cornerstone of therapy
- Supportive therapy:
 - Antipyretics
 - Analgesia:
 - Acetaminophen, NSAIDs, narcotics (for severe pain)
 - IV fluids and antiemetics for vomiting and dehydration
 - Ice pack
 - Scrotal support and bed rest for orchitis
- Isolation, droplet precaution, of infected patients



FOLLOW-UP

DISPOSITION

Admission Criteria

- Seriously ill who require supportive care
- Vomiting and dehydration
- Encephalitis, meningitis
- Severe pancreatitis
- Isolate admitted patients

Discharge Criteria

- Virtually all patients
- Contagious until about a week after onset of symptoms

PEARLS AND PITFALLS

- Mumps virus is the only cause of epidemic parotitis.
- Vaccines are highly effective, and when correctly given confer 90% immunity.

MMR should not be given to pregnant women or immunosuppressed or immunocompromised individuals.

- Mumps virus is endemic to many parts of the world and may pose a risk to travelers without immunity to mumps.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

www.cdc.gov/mumps



CODES

ICD9

- 072.0 Mumps orchitis
- 072.3 Mumps pancreatitis
- 072.9 Mumps without mention of complication

ICD10

- B26.0 Mumps orchitis
- B26.3 Mumps pancreatitis
- B26.9 Mumps without complication

MUNCHAUSEN SYNDROME

Sophie Galson • Richard E. Wolfe



BASICS

DESCRIPTION

- A neurotic disorder in which the patient fakes signs or symptoms without tangible personal benefit other than to experience the sick role.
- Most dramatic form of chronic factitious disorder with a predominance of physical findings.
- The nature of the disorder resists rigorous study but possible risk factors include:
 - _ Males
 - _ Less severe factitious disorders are more common in women
 - _ Unmarried
 - _ Age in the forties
 - _ Personality disorder
 - _ A history of sadistic and rejecting parents
 - _ A history of chronic childhood illness

ETIOLOGY

- Factitious disorder:
 - _ 3 DSM-IV diagnostic criteria:
 - Intentional production of physical or psychological signs
 - Motivation to assume the sick role
 - Absence of external incentives
 - Predominance of symptoms rather than physical findings
- Classic Munchausen syndrome:
 - _ Most severe and chronic form of factitious disorders
 - _ Predominantly physical findings
- Clinical clusters:
 - _ Self-induced infection
 - _ Simulated specific illnesses with no actual disorder
 - _ Chronic wounds
 - _ Self-medication

Pediatric Considerations

- Munchausen by proxy:
 - _ The patient's illness is caused by the caregiver, not the patient
 - _ The motivation for the caregiver's behavior is to assume the sick role by proxy
 - _ The caregiver inflicts injury or induces illness in their charge, usually a child

- Commonly parents (mostly mothers)
- May simulate injury and disease in a number of ways:
 - Inflicts injury
 - Induces Illness
 - Fabricates symptoms
 - Exaggerates symptoms of the child's illness causing overaggressive medical interventions
- The perpetrator usually refuses to acknowledge the deception
- Cessation of the symptoms when the patient and caregiver are separated

Geriatric Considerations

Caregivers of elderly patients may also be perpetrators in Munchausen by proxy

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Inappropriate or bizarre use of the ED
- Frequent visits
- Numerous hospital admissions
- Peregrination: Travel from hospital to hospital
- Pseudologia fantastica:
 - Intricate and colorful stories associated with the presentation
- Alteration of biographical information:
 - Use of aliases
 - Change date of birth by 1 digit
- Escalating demands for diagnostic testing and therapeutic interventions
- Hostility toward the health care providers when questioned
- Evasiveness regarding details of the presenting complaint
- The patient provides excessive medical documentation
- Masochistic acceptance of painful procedures
- The patient appears more comfortable than is likely considering the disease
- The patient demonstrates unusually strong medical knowledge
- Frequent homelessness and significant wandering between cities and states
- An absence of close interpersonal relationships
- Self-medication
- Abdominal complaints with history of repeated negative laparotomies (laparotomaphilia migrans)
- Witnessed intentional acts to fake illness:
 - Inappropriate ingestion of medication to reproduce physical findings
 - Injection of contaminants (feces, bacteria, sputum, corrosives)

- Self-induced wounds
- Swallowing blood to simulate a GI hemorrhage
- Self-phlebotomy
- Instrument tampering

Physical-Exam

- Fever:
 - Factious from manipulation of thermometer
 - Induced from injection of contaminants
- Self-induced wounds
- Chronic wounds
- Multiple scars
- Foreign bodies in wounds, ear canals, urethra

ESSENTIAL WORKUP

- Diligent detective work is needed:
 - Retrieval of records from other hospitals
 - Call on family members to discuss past history for inconsistencies and excessive use
 - Call personal physician for background and to coordinate information
 - Search patient's room and belongings to establish the method of deception
- Conclusive proof of faking disease is needed to make the diagnosis

DIAGNOSIS TESTS & NTERPRETATION

Lab

- Direct observation of the patient when obtaining tests to prevent faking results
- Commonly faked lab results:
 - Hemocult positive stool
 - Hematuria (intentionally dripping blood into urine sample)
 - Hypoglycemia (self-administration of insulin)
- Abnormal results from self-medication:
 - Low hematocrit (ingestion of warfarin or self-phlebotomy)
 - Elevated INR (ingestion of warfarin)
 - Thyroid function tests (ingestion of thyroxine)
 - Low serum glucose (injection of insulin or ingestion of sulfonylurea)
- Evidence of intent to fake illness:
 - Testing stool for phenolphthalein may detect laxative abuse
 - Serum C-peptide with high insulin levels:
 - Low C-peptide: Exogenous administration of insulin
 - Elevated C-peptide: Endogenous hypoglycemia or sulfonylurea ingestion

Imaging

Do not rely on imaging brought by the patient

Diagnostic Procedures/Surgery

Avoid unless clear objective findings indicate the necessity of a procedure

DIFFERENTIAL DIAGNOSIS

- True illness:
 - Primary illness unrelated to a psychiatric disorder
- Secondary to a comorbid condition associated with factitious disorders:
 - Secondary to self-destructive acts in patients with dementia, psychotic disorders, or mental retardation
 - Secondary to diagnostic and therapeutic procedures
- Malingering:
 - Clear-cut secondary gain
- Conversion disorder:
 - Deficits of the voluntary motor or sensory neurologic system that are not consciously produced
- Somatization disorder (hysteria, Briquet syndrome):
 - Symptoms that involve multiple organs, that varies over time, and are not consciously produced
- Other neurotic disorders:
 - Anxiety
 - Depression



TREATMENT

PRE HOSPITAL

INITIAL STABILIZATION/THERAPY

Treatment should be limited to stabilization of life or limb threats caused by acts of self-harm

ED TREATMENT/PROCEDURES

- Identify objective physical illness and treat as appropriate
- Document history and findings suggestive of factitious illness
- List of all the aliases, addresses, and date of births that the patient is known to use
- Summarize the patient's known modus operandi (the factitious histories and behaviors that he or she has presented with)
- Ensure that the information will be communicated or available to all doctors who are likely to come into contact with the patient
- Confrontation of the patient in the ED is controversial and should only occur when

unambiguous evidence is gathered

- Report Munchausen syndrome by proxy to child protective services

MEDICATION



FOLLOW-UP

DISPOSITION

Admission Criteria

- Injuries and disease caused by self-harm
- Munchausen by proxy:
 - When the diagnosis is suspected but there is not enough evidence to have child protective services take custody
- Observation to collect evidence of faking disease:
 - May also allow setting to rule out rare organic etiologies
- To establish a long-term plan to prevent future self-harm and iatrogenic adverse events
- Psychiatric admission may be of benefit, but it is rarely accepted by the patient

Discharge Criteria

- Medical stability
- Not an active threat to harm self
- Appropriate referral for medical and psychiatric follow-up arranged

Issues for Referral

- May offer psychiatric referral as a method of dealing with stress caused by illness
- Psychiatric providers located directly in medical settings (e.g., primary care physician office) may be more accepted. Overall, this is a chronic illness with poor prognosis

FOLLOW-UP RECOMMENDATIONS

Maintain contact between the patient and an identified provider for that patient.

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See Also (Topic, Algorithm, Electronic Media Element)

Abuse, Pediatric



CODES

ICD9

301.51 Chronic factitious illness with physical symptoms

ICD10

- F68.11 Factitious disorder w predom psych signs and symptoms
- F68.12 Factitious disorder w predom physical signs and symptoms
- F68.13 Factitious disord w comb psych and physcl signs and symptoms

MUSHROOM POISONING

Michael E. Nelson • Timothy B. Erickson

BASICS

DESCRIPTION

- Amanitin/phalloidin:
 - Species:
 - *Amanita phalloides* (“death cap”)
 - *Amanita virosa/Amanita verna* (“destroying angel”)
 - *Galerina marginata, Galerina venenata*
 - Mechanism:
 - Cyclopeptide toxins inhibit RNA polymerase 2, which kills GI epithelium, hepatocytes, nephrocytes
- Gyromitrin:
 - Species:
 - *Gyromitra esculenta* (“false morels”)
 - Other *Gyromitra* spp.
 - Mechanism:
 - Inhibits pyridoxal phosphate
 - Damage to RBCs, hepatocytes, neurons
- Muscarine:
 - Species:
 - *Inocybe* (several species)
 - *Clitocybe* (several species)
 - Mechanism:
 - Parasympathomimetic
- Coprine:
 - Species:
 - *Coprinus atramentarius* (“inky caps”)
 - Mechanism:
 - Blocks acetaldehyde dehydrogenase
 - Causes disulfiram-like reaction if mixed with alcohol
- Ibotenic acid/muscimol:
 - Species:
 - *Amanita pantherina* (“the panther”)
 - *Amanita muscaria* (“fly agaric”)
 - Mechanism:
 - GABA agonists
- Psilocin/psilocybin:
 - Species:

- *Psilocybe* and *Panaeolus* spp. as well as others
- Mechanism:
 - Similar structure to lysergic acid diethylamide, effect serotonin receptor
- Gastric irritants:
 - Many various mushrooms, including those normally considered edible
- Orellanine:
 - Species:
 - *Cortinarius* (several species)
 - Mechanism:
 - Direct renal toxicity
- *Tricholoma equestre* (“man on horse”):
 - Rhabdomyolysis-inducing mushrooms
 - Unidentified myotoxin

DIAGNOSIS

SIGNS AND SYMPTOMS

- Amanitin/phalloidin:
 - Nausea
 - Vomiting
 - Abdominal cramps
 - Bloody diarrhea
 - Clinical course:
 - Onset of symptoms delayed 6–36 hr with development of GI symptoms
 - Transient latent phase may last 2 days (no pain/symptoms)
 - Can progress to hepatic or renal failure and death in 2–6 days
 - Most lethal mushroom toxins
- Gyromitrin:
 - 1st 5–10 hr:
 - Abdominal cramps
 - Nausea/vomiting
 - Watery diarrhea
 - Later symptoms:
 - Weakness
 - Cyanosis
 - Confusion
 - Seizures
 - Coma
- Muscarine:
 - Cholinergic symptoms include:

- Miosis
- Salivation
- Lacrimation
- Sweating
- Diarrhea
- Flushed skin
- Nausea
- Bradycardia
- Bronchoconstriction
- Onset usually within 1 hr (may be delayed)
- Coprine:
 - Disulfiram-like reaction within minutes to hours when combined with alcohol:
 - Flushing
 - Sweating
 - Nausea/vomiting
 - Palpitations
- Ibotenic acid/muscimol:
 - Relatively rapid onset of 30–120 min
 - GABA agonist effects include:
 - Hallucinations
 - Dysarthria
 - Ataxia
 - Somnolence/coma
 - Glutamatergic effects (mainly pediatrics):
 - Seizures
 - Muscle cramps/myoclonic movements
- Psilocin/psilocybin:
 - Rapid onset, usually resolves in 6–12 hr
 - Visual hallucinations
 - Alteration of perception
 - Mydriasis
 - Tachycardia
 - Fever and seizures in children
- Gastric irritants:
 - Group of toxins that cause nausea, vomiting, intestinal cramps, and watery diarrhea
 - Onset 30 min to 3 hr, usually resolved in 6–12 hr
- *Amanita smithiana*:
 - Nausea/vomiting
 - Headache
 - Sweating
 - Chills

- Low-back pain
- Polydipsia
- Clinical course:
 - May progress to oliguria and acute renal failure
 - Markedly delayed onset of symptoms (2–14 days)
- *T. equestre*:
 - Acute rhabdomyolysis:
 - Myalgias/arthralgias
 - Hematuria/dark urine
 - Decreased urine output
 - Dehydration

History

- Time of ingestion
- Time of symptom onset
- Quantity ingested
- Preparation: Raw or cooked
- Picked in the wild or store-bought
- Coingestants, other mushrooms
- Alcohol/drug use history
- Symptoms of family members, friends

Physical-Exam

- Vital signs
- Changes in mental status
- Pupillary response
- Cardiopulmonary exam
- Abdominal exam
- Neurologic exam

ESSENTIAL WORKUP

- Mushroom description:
 - Pileus (cap); margin shape
 - Stipe (stem)
 - Lamellae (gills)
 - Veil
 - Annulus (ring)
 - Volva
- Store mushroom in brown paper bag for future identification:
 - < 3% of cases result in an exact mushroom identification.
 - Digital photography and electronic image transfer to poison control center or regional mycologist

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC
- Prothrombin time (PT), partial thromboplastin time (PTT)
- Electrolytes, BUN, creatinine, glucose
- Urinalysis
- LFTs, creatine phosphokinase (CPK)
- Imaging
- Spore print: Mycologist needed for specific genus/species interpretation

DIFFERENTIAL DIAGNOSIS

- Very broad differential
- Gastroenteritis
- Hepatitis/acetaminophen hepatotoxicity
- Acute renal failure (many causes)
- Rhabdomyolysis (many causes)
- Cholinergic syndrome (e.g., organophosphates)
- Anticholinergic syndrome
- Seizures (many causes)



TREATMENT

PRE HOSPITAL

Bring any unconsumed mushrooms or mushroom pieces to hospital to aid in diagnosis:

- Refrigerate specimens if possible, place in brown paper bag.

INITIAL STABILIZATION/THERAPY

- ABCs
- Establish IV 0.9% NS saline
- Monitor
- Naloxone, D₅₀W (or Accu-Chek), and thiamine for altered mental status

ED TREATMENT/PROCEDURES

General Measures

- Decontamination:
 - Activated charcoal (50–100 g)
 - Gastric decontamination if early after ingestion and patient:
 - Has not yet vomited.
 - Has normal mental and respiratory status
 - Is not undergoing hallucinations

- Fluid rehydration and electrolyte replacement as necessary
- Call local poison control center at 800-222-1222 and request mycologist—digital picture may be electronically sent for identification.
- Obtain specimens (vomit if needed) for identification.

Mushroom-specific Therapy

- Amanitin/phalloidin:
 - _ Administer activated charcoal PO q2–4h.
 - _ Hypoglycemia and elevated PT:
 - Signs of liver failure
 - Administer fresh-frozen plasma and vitamin K for coagulation disorders with active bleeding.
 - _ Administer calcium in presence of hypocalcemia.
 - _ Liver transplant for severe hepatic necrosis
 - _ Consider N-acetylcysteine, high-dose penicillin G, or silibinin if available (thioctic acid controversial)
- Gyromitrin:
 - _ Treat seizure with benzodiazepines.
 - Administer pyridoxine (vitamin B₆) in severely symptomatic patients.
 - _ Treat liver dysfunctions as outlined for amanitin/phalloidin group.
 - _ Dialysis for renal failure
- Muscarine:
 - _ Administer atropine in severe cases.
- Coprine:
 - _ Self-limited toxicity—supportive care
 - _ Avoid syrup of ipecac (contains alcohol)
 - _ β -Blockers for cardiac dysrhythmias
- Ibotenic acid/muscimol:
 - _ Usually self-limited toxicity
 - _ Provide supportive care
 - _ Monitor for hypotension
 - _ Treat moderate symptoms with benzodiazepines, if severe anticholinergic symptoms; consider physostigmine.
- Psilocin/psilocybin:
 - _ Self-limited toxicity
 - _ Dark, quiet room and reassurance
 - _ Benzodiazepines for agitation
 - _ External cooling measures if needed in children
- GI Irritants:
 - _ When poisoning from above groups not suspected, administer fluids and antiemetics.
 - _ Provide supportive care

- Orellanine and *A. smithiana*:
 - Closely monitor BUN, creatinine, electrolytes, and urine output.
 - Forced diuresis with Lasix contraindicated
 - Diuresis with alkalinization of urine with NaHCO₃ if signs of rhabdomyolysis
 - Hemodialysis/renal transplantation may be needed.
- *T. equestre* (“man on horse”):
 - Fluid hydration
 - Check and follow CPK.
 - Monitor urine output.

MEDICATION

- Activated charcoal slurry: 1–2 g/kg up to 100 g PO
- Atropine: 0.5 mg (peds: 0.02 mg/kg) IV; repeat 0.5–1 mg IV (peds: 0.04 mg/kg) q10min if secretions recur, to max. 1 mg/kg in children and 2 mg/kg in adults
- Dextrose: D₅₀W 1 amp: 50 mL or 25 g (peds: D₂₅W 2–4 mL/kg) IV
- Diazepam (benzodiazepine): 5–10 mg (peds: 0.2–0.5 mg/kg) IV
- Lorazepam (benzodiazepine): 2–6 mg (peds: 0.03–0.05 mg/kg) IV
- Naloxone (Narcan): 2 mg (peds: 0.1 mg/kg) IV or IM initial dose
- Physostigmine: 0.5–2 mg IM or IV in adults
- Propranolol: 1 mg (peds: 0.01–0.1 mg/kg) IV
- Pyridoxine: 25 mg/kg IV over 30 min
- Thiamine (vitamin B₁): 100 mg (peds: 50 mg) IV or IM



FOLLOW-UP

DISPOSITION

Admission Criteria

- All symptomatic patients:
 - Protracted vomiting, dehydration, liver or renal toxicity, or seizures
- Transfer to tertiary medical center for early signs of renal or hepatic failure.
- Symptomatic infants and young children found with mushrooms:
 - Assume ingestion.
- ICU admission for known ingestion of an amanitin-containing mushroom:
 - Early liver service consultation

Discharge Criteria

Asymptomatic during 6–8 hr with 24 hr of close home observation and close follow-up (if reliable caregivers)

Issues for Referral

Potential liver or renal transplantation

FOLLOW-UP RECOMMENDATIONS

Drug detoxification programs if chronic recreational use

PEARLS AND PITFALLS

- There are old mushroom pickers, and bold mushroom pickers; but there are no old, bold mushroom pickers.
- Symptoms with late onset (> 6 hr) generally indicate more lethal toxins.
- Lack of proper mycologic identification
- Timely organ transplant referrals when indicated

ADDITIONAL READING

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CODES

ICD9

988.1 Toxic effect of mushrooms eaten as food

ICD10

T62.0X1A Toxic effect of ingested mushrooms, accidental, init

MYASTHENIA GRAVIS

Douglas W. Lowery-North

BASICS

DESCRIPTION

- Antibody-mediated condition that results in painless, fatigable muscle weakness
- Ocular or generalized:
 - Ocular (eyelids and extraocular) muscle weakness:
 - Most common initial symptom (60%)
 - ~80% of myasthenia gravis (MG) patients who present with ocular weakness initially will progress to general weakness within 2 yr.
 - Generalized:
 - Usually affects proximal limbs, axial muscle groups such as neck, face, bulbar muscles
- Acute or subacute, with relapses and remissions
- Associated with thymoma in 15% and thymic hyperplasia in 65%
- Myasthenic crisis:
 - Respiratory failure or inability to protect airway due to weakness
 - Triggers:
 - Infection
 - Surgery
 - Trauma
 - Pregnancy
 - Medication changes (e.g., rapid tapering of steroids)
 - Difficult to distinguish from cholinergic crisis resulting from excessive doses of acetylcholinesterase (AChE) inhibitors:
 - Cholinergic crisis may also include muscarinic effects such as sweating, lacrimation, salivation, and GI hyperactivity in addition to weakness.

EPIDEMIOLOGY

- Pediatric MG is rare and distinct:
 - Congenital MG: Genetic defect
 - Juvenile MG: Autoimmune disorder
 - Transient neonatal MG: Postdelivery complication from placental transfer of maternal antibodies
- Adult MG has bimodal distribution:
 - 1st peak in 2nd and 3rd decades affecting mostly women
 - 2nd peak in 6th and 7th decades affecting men

ETIOLOGY

- Antibody-mediated attack on nicotinic acetylcholine receptors
- Up to 20% of patients may be acetylcholine receptor antibody (AChR Ab) negative
- Penicillamine can cause MG as well as other autoimmune conditions
- Many medications may worsen myasthenic weakness:
 - Aminoglycosides, macrolides, quinolones, antimalarials
 - Local anesthetics
 - Antidysrhythmics (propafenone, quinidine, procainamide)
 - β -Blockers, calcium-channel blockers
 - Anticonvulsants (phenytoin, carbamazepine)
 - Antipsychotics (phenothiazine, atypicals)
 - Neuromuscular blocking agents
 - Iodine-containing radiocontrast



DIAGNOSIS

SIGNS AND SYMPTOMS

Fluctuating focal weakness

History

- Symptoms worsen with repeated activity:
 - Improve with rest
- Ocular weakness:
 - Diplopia
 - Ptosis while driving or reading
- Bulbar and facial muscle weakness:
 - Trouble chewing, speaking, swallowing
 - Inability to keep jaw closed after chewing
 - Slurred, nasal speech
- Limb weakness:
 - Difficulty climbing stairs, rising from chair, reaching up with arms

Physical-Exam

- Ocular findings:
 - Ptosis, diplopia
 - Inability to keep eyelid shut against resistance
 - No pupillary changes
- Bulbar and facial findings:
 - Ask patient to count to 100; look for changes in speech.
 - Decreased facial expression
 - Head droop
- Limb findings:
 - Repetitive testing of proximal muscles or small muscles of hand results in

weakness.

- Reflex and sensory exam are normal.

ESSENTIAL WORKUP

- Assess for respiratory compromise
- Search for secondary triggers (e.g., infectious source)

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC
- Electrolytes
- LFTs
- Thyroid function tests
- Anti-AChR Ab:
 - Positive in 90% with generalized disease
 - Positive in 50% with ocular disease
- Other tests for initial diagnosis:
 - Antistriated muscle antibody
 - Antinuclear antibody
 - Rheumatoid factor
 - ESR

Imaging

- Head CT or MRI to rule out compressive lesions causing cranial nerve findings
- Chest CT with contrast to look for associated thymoma
- CXR as needed to evaluate for infectious source

Diagnostic Procedures/Surgery

- Edrophonium (Tensilon) test:
 - Short-acting AChE inhibitor
 - A positive test produces rapid, short-lived (2–5 min) improvement in strength
 - Sensitivity 95% in generalized MG and 86% in ocular MG
 - False positives possible with Lambert–Eaton, Guillain–Barré, MS, botulism, and others
 - Keep patient on cardiac monitor during test.
 - Atropine at bedside for possible bradycardia
 - Suction at bedside for possible increased secretions
- Ice test:
 - Place ice on eyelid for 2 min.
 - Improvement in ptosis suggests MG.

DIFFERENTIAL DIAGNOSIS

- Amyotrophic lateral sclerosis
- Botulism
- Electrolyte abnormalities
- Graves disease
- Guillain–Barré syndrome
- Hyperthyroidism
- Inflammatory muscle disorders
- Intracranial mass lesions
- Lambert–Eaton syndrome
- Multiple sclerosis
- Periodic paralysis
- Tick paralysis



TREATMENT

PRE HOSPITAL

Attention to airway management

INITIAL STABILIZATION/THERAPY

Myasthenic crisis:

- Most important is early intubation and mechanical ventilation.
- Signs of impending failure:
 - Vital capacity < 20 mL/kg
 - Negative inspiratory pressure > -30 cm H₂O
 - Negative expiratory pressure < 40 cm H₂O
- Considerations regarding paralytics:
 - Decreased sensitivity to depolarizing agents may necessitate higher dose; consider doubling the usual dose of succinylcholine.
 - Nondepolarizing agents can cause extended paralysis; consider halving the usual dose.
 - Others recommend midazolam, etomidate, or thiopental instead.

ED TREATMENT/PROCEDURES

- Treat infections aggressively.
- Search for and remove triggers.
 - Careful medication history.
- Myasthenic crisis may require plasmapheresis or IV gamma globulin (IVIG).
 - Plasmapheresis: Remove 1–1.5 plasma volume each session \times 5 sessions
 - IVIG: 0.4 mg/kg/d \times 5 days
- Initiate high-dose corticosteroids.
- Discontinue AChE inhibitors while intubated.

- Atropine for AChE inhibitor effects (bradycardia, GI symptoms, increased bronchial or oral secretions)

MEDICATION

First Line

- Edrophonium (Tensilon): 2 mg IV over 15–30 sec; if no effect after 45 sec, can give 2nd dose of 3 mg IV. If still no response, final dose of 5 mg IV can be given (total 10 mg).
- Prednisolone 1 mg/kg/d for crisis
- Atropine for cholinergic crisis 0.5 mg IV or IM

Second Line

Other medications that may be initiated by neurologist:

- Prednisone, AChE inhibitors, azathioprine, mycophenolate, mofetil, cyclosporine, tacrolimus, rituximab



FOLLOW-UP

DISPOSITION

Admission Criteria

- New-onset myasthenic symptoms
- Diagnosis unclear, but myasthenia a possibility
- Myasthenic patients with worsening symptoms
- Myasthenic crisis or questionable respiratory status mandates admission to ICU.

Discharge Criteria

Myasthenic patients who are improving can be considered for discharge in consultation with neurology.

FOLLOW-UP RECOMMENDATIONS

Any discharged patient should have neurology follow-up arranged.

PEARLS AND PITFALLS

- Search for signs of myasthenic crisis in any MG patient who presents to the ED.
- Search carefully for secondary conditions in patients with worsening MG.
- Place patient on cardiac monitor and keep atropine and suction at bedside when performing edrophonium test.

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See Also (Topic, Algorithm, Electronic Media Element)

- Amyotrophic Lateral Sclerosis
- Botulism
- Guillain–Barré Syndrome
- Hyperthyroidism
- Multiple Sclerosis

The author gratefully acknowledges Kelley Ralph’s contribution for the previous edition of this chapter.



CODES

ICD9

- 358.00 Myasthenia gravis without (acute) exacerbation
- 358.01 Myasthenia gravis with (acute) exacerbation
- 775.2 Neonatal myasthenia gravis

ICD10

- G70.00 Myasthenia gravis without (acute) exacerbation
- G70.01 Myasthenia gravis with (acute) exacerbation
- P94.0 Transient neonatal myasthenia gravis

MYOCARDIAL CONTUSION

Sean Patrick Nordt

BASICS

DESCRIPTION

- Also known as blunt cardiac injury
- Pathologically characterized by discrete and well-demarcated area of hemorrhage
- Usually subendocardial
- May extend in pyramidal transmural fashion
- Most commonly involves anterior wall of right ventricle or atrium due to anatomic location

ETIOLOGY

- Blunt trauma to chest:
 - High-speed deceleration accidents
 - May occur in accidents with speeds as low as 20–35 mph
- Auto–pedestrian injuries
- Falls
- Prolonged closed-chest cardiac massage
- Heart may be compressed between sternum and vertebrae.
- Heart strikes sternum during deceleration.
- Heart is damaged by abdominal viscera upwardly displaced by force on abdomen.
- Concussive forces (e.g., explosion)
- Associated conditions:
 - Life-threatening dysrhythmias
 - Cardiogenic shock/CHF
 - Hemopericardium with tamponade
 - Valvular/myocardial rupture
 - Intraventricular thrombi
 - Thromboembolic phenomena
 - Coronary artery occlusion from intimal tearing or adjacent hemorrhage and edema may rarely occur.

DIAGNOSIS

SIGNS AND SYMPTOMS

- Clinical picture is varied and nonspecific:
 - Chest pain
 - Cardiogenic shock
 - Subtle EKG changes without clinical symptoms

- Most common sign is tachycardia out of proportion to degree of trauma or blood loss.
- Friction rub may occur rarely.
- Retrosternal chest pain unrelieved by nitroglycerin:
 - Often delayed up to 24 hr
 - May respond to oxygen
- Evidence of significant thoracic trauma:
 - Contusions, abrasions
 - Palpable crepitus
 - Sternal fracture alone with normal EKG and negative serial troponin I does not predict BCI
 - Visible flail segments
- Other injuries may mask signs and symptoms of myocardial contusion.

History

- Mechanism of injury (e.g., MVA, fall, explosion, missile to chest wall)
- Any syncope or loss of consciousness suggests possible dysrhythmia.
- Crush injury

Geriatric Considerations

Obtain and consider pre-existing cardiac disease and concurrent medications in elderly patients following blunt cardiac injury.

Pediatric Considerations

Due to increased compliance of pediatric chest wall, significant cardiac compression and contusion may be present with minimal or no external signs of trauma.

Physical-Exam

Complete physical exam as in any trauma patient:

- Evaluate for jugular venous distention (JVD)
- Decreased or muffled heart sounds
- Extra heart sounds
- Crepitus
- Pulsus paradoxus
- Evidence of chest wall trauma

ESSENTIAL WORKUP

- No single diagnostic study (other than autopsy findings!) confirms presence of myocardial contusion.
- EKG:
 - Best initial screening tool
 - Most common rhythm is sinus tachycardia (70%).

- Normal EKG does not rule out myocardial damage.
- EKG changes may be subtle or include nonspecific findings such as ST changes, right bundle branch block, and premature atrial and ventricular contractions.
- At least 1 repeat EKG is recommended because changes may occur over time.
- Serious dysrhythmias may result in hemodynamic instability:
 - Atrial fibrillation/atrial flutter
 - Ventricular tachycardia/ventricular fibrillation (commotio cordis)
- Troponin I is now the recommended screening lab test over CK-MB to be interpreted with EKG.
- Echocardiography should be performed on all patients with any EKG changes, elevated troponin I or troponin T.
- Transesophageal ECG (TEE) more sensitive than transthoracic ECG (TTE) but technically more difficult and time-consuming.
- Multidetector CT angiography or MRI may be of benefit.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Troponin I is the preferred lab test.
- Troponin T less sensitive than troponin I.
 - Levels should be sent on all patients where BCI suspected.
 - Should be repeated at 6–8 hr after injury.
 - Any elevation requires admission.
- Cardiac troponins are more specific than CK-MB for cardiac injury.
- CK-MB no longer routinely recommended.

Imaging

- Radiographs, CT, MRI detect associated injuries:
 - Pulmonary contusion
 - Rib or sternal fractures
 - Acute pulmonary edema
 - No specific findings in cardiac contusion
- Focused assessment with sonography for trauma (FAST) should be performed on all patients to assess pericardium and possible concurrent intra-abdominal injuries.
- ECG:
 - Generally regarded as best imaging study for detecting cardiac contusion
 - Detects wall-motion abnormalities and effusions
 - Allows direct visualization of cardiac chambers and valves
 - May not visualize small (possibly clinically insignificant) contusions
 - TEE preferable to TTE if patient stable enough for procedure.
 - TTE may be performed although may also visualize great vessels.

- Radionuclide ventriculography:
 - Has been largely abandoned owing to wide availability of ECG
- Thallium²⁰¹ scintigraphy (single photon emission CT [SPECT]):
 - Sensitive and specific to left ventricular injury
 - Unable to evaluate right ventricle, which is most commonly injured

Diagnostic Procedures/Surgery

- Pericardiocentesis:
 - For treatment of cardiac tamponade, preferably under US guidance
- Thoracotomy:
 - Consider in patient with acute cardiac arrest or decompensation in ED or after unsuccessful pericardiocentesis

DIFFERENTIAL DIAGNOSIS

- Cardiac rupture
- Tamponade
- Valvular damage
- Other traumatic chest wall injury
- Angina or MI



TREATMENT

PRE HOSPITAL

Pre-hospital personnel should convey accurate information to emergency department personnel:

- Mechanism of injury
- Motor vehicle status
- Steering wheel and dashboard damage
- Use of restraint devices
- Vehicle speed
- Patient position
- Time to extrication
- Any loss of consciousness

INITIAL STABILIZATION/THERAPY

Manage airway and resuscitate as indicated:

- Oxygen:
 - IV access
 - Cardiac monitoring

ED TREATMENT/PROCEDURES

- Dysrhythmias may be treated with same pharmacologic agents used for

nontraumatic dysrhythmias:

- Supraventricular tachycardia:
 - Adenosine or calcium channel blocker if patient not hypovolemic
- Bradycardia:
 - Atropine
 - Pacing
- Ventricular dysrhythmias:
 - Electrical conversion
 - Amiodarone
 - Lidocaine
 - Procainamide
- Cardiac arrest:
 - Epinephrine
 - Atropine
 - Other interventions as appropriate
- Rapid atrial fibrillation or flutter:
 - Diltiazem, or digoxin if patient not hypotensive
- Prophylactic treatment of dysrhythmias is not indicated.
- Cardiogenic shock caused by myocardial contusion:
 - Judicious fluid administration
 - Inotropic support (dopamine or dobutamine)
 - Intra-aortic balloon counterpulsation may be necessary.

MEDICATION

- Medications used in cardiac contusion are supportive for dysrhythmias or hemodynamic compromise secondary to injury.
- There is no primary therapy for cardiac contusion.
- Adenosine: 6 mg rapid IVP (peds: 0.05–0.1 mg/kg rapid IVP), may repeat 12 mg q1–2min twice if no response
- Amiodarone: Load 150 mg IV over 10 min (peds: 5 mg/kg), then 1 mg/min for 6 hr, then 0.5 mg/min (peds: 5 µg/kg/min)
- Atropine: 0.5–1 mg (peds: 0.02 mg/kg/ dose, min. 0.1 mg) IV or endotracheal tube (ET)
- Digoxin: Load 0.5 mg (peds: 0.02 mg/kg) IV, then 0.25 mg (peds: 0.01 mg/kg) IV q6h for 2 more doses
- Diltiazem: 0.25 mg/kg IV for both adults and peds over 2 min, may rebolus 0.35 mg/kg (adult and peds) 15 min later
- Dobutamine: 2–15 µg/kg/min (adults and peds)
- Dopamine: 2–20 µg/kg/min (adults and peds)
- Epinephrine: 1 mg (peds: 0.01 mg/kg) IV or ET for cardiac arrest (1:10,000 solution)
- Lidocaine: Load 1 mg/kg IV, then 0.5 mg/kg q8–10min to max. 3 mg/kg (adults and peds); infusion 1–4 mg/min (peds: 20–50 µg/kg/min) IV

- Procainamide: start at 3 to 6 mg/kg/dose over 5 minutes not to exceed 100 mg to a titrated maximum of 15 mg/kg/loading dose
- Verapamil: 0.1–0.3 mg/kg up to 5–10 mg IV over 2 min (not approved in children)



FOLLOW-UP

DISPOSITION

- Adverse outcomes, particularly dysrhythmias, are uncommon but generally occur within 1st 24 hr.
- No single test or combination of tests will accurately predict which patients can be discharged safely from ED:
 - All patients in whom diagnosis is seriously being entertained should be admitted to a monitored setting.

Admission Criteria

- EKG abnormalities
- Cardiac enzyme abnormalities
- Hemodynamic instability
- Other studies suggesting cardiac contusion
- Admit to monitored unit for close observation.

Discharge Criteria

Asymptomatic patients with no EKG abnormalities or dysrhythmia and with normal cardiac enzymes after 6–8 hr period may be discharged.

Issues for Referral

Immediate surgical consultation:

- Suspected myocardial wall rupture
- Suspected valve or papillary muscle rupture
- Suspected septal rupture
- Coronary artery thrombosis
- Pericardial effusion
- Cardiac tamponade

FOLLOW-UP RECOMMENDATIONS

Discharged patients:

- Should have follow-up within 24 hr of injury

PEARLS AND PITFALLS

- Obtain EKG in patients following chest wall trauma.
- Perform FAST exam on all patients to assess pericardium.

- External signs of chest wall trauma should increase concern of blunt cardiac injury.
- Pediatric patients may have little or no external signs of chest wall trauma.
- Do not administer thrombolytics to patients with ST elevation MI following trauma.
- Negative troponin I and normal EKG make significant blunt cardiac injury unlikely.

ADDITIONAL READING

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CODES

ICD9

861.01 Contusion of heart without mention of open wound into thorax

ICD10

- S26.01XA Contusion of heart with hemopericardium, initial encounter
- S26.11XA Contusion of heart without hemopericardium, initial encounter
- S26.91XA Contusion of heart, unspecified with or without hemopericardium, initial encounter

MYOCARDITIS

Liudvikas Jagminas



BASICS

DESCRIPTION

- An inflammatory change in the heart muscle characterized by myocyte necrosis and subsequent myocardial destruction
- Direct cytotoxic effect of causative agent followed by a secondary immune response
- True incidence is unknown because many cases are asymptomatic.
- Autopsy studies have demonstrated evidence of myocarditis in 1–7% of cases and > 50% in HIV patients.
- Male > female (1.5:1)
- Average age of patients with myocarditis is 42 yr.
- Major cause of unexpected sudden death (15–20% of cases) < 40 yr old

ETIOLOGY

- Viral:
 - Enteroviruses (coxsackie B)
 - Adenovirus
 - Herpesvirus (including cytomegalovirus [CMV])
 - Hepatitis C
 - Influenza
 - Echovirus
 - Herpes simplex virus
 - Varicella-zoster
 - Epstein–Barr virus
 - Cytomegalovirus
 - Mumps
 - Rubeola
 - Variola/vaccinia
 - Yellow fever
 - Rabies
 - HIV
- Bacteria:
 - Diphtheria
 - Tuberculosis
 - Brucellosis
 - Psittacosis
 - Meningococcus

- Mycoplasma
- Group A streptococcus
- Protozoa:
 - Leishmaniasis
 - Malaria
 - Toxoplasmosis in the immunocompromised host
 - *Treponema cruzi* (Chagas disease):
 - Most common cause of heart failure and myocarditis worldwide
 - 20 million persons infected in Central and South America
 - Trichinosis
 - Trypanosomiasis
- Spirochetes:
 - *Borrelia burgdorferi*, the spirochete agent in Lyme disease
 - Syphilis
- Rickettsial:
 - Scrub typhus
 - Rocky Mountain spotted fever
 - Q fever
- Fungal:
 - Candidiasis
 - Aspergillosis
 - Cryptococcosis
 - Histoplasmosis
 - Actinomycosis
 - Helminthic
 - Trichinosis
 - Echinococcosis
 - Schistosomiasis
 - Cysticercosis
- Drugs:
 - Acetaminophen
 - Ampicillin
 - Chemotherapeutic agents (anthracyclines)
 - Cocaine
 - Hydrochlorothiazide
 - Lithium
 - Methyldopa
 - Penicillin
 - Sulfamethoxazole
 - Sulfonamides
 - Zidovudine
 - Radiation
 - Hypersensitivity

- Heavy metals
- Hydrocarbons
- Carbon monoxide
- Arsenic
- Autoimmune disorders:
 - Systemic lupus erythematosus (SLE)
 - Wegener granulomatosis
 - Kawasaki disease
 - Giant cell arteritis
 - Sarcoidosis
- Peripartum cardiomyopathy
- Bites/stings:
 - Scorpion
 - Snake
 - Black widow venom



DIAGNOSIS

SIGNS AND SYMPTOMS

Arrhythmias (18%), dyspnea (72%), and chest pain (35%)

History

- Fatigue
- Myalgias/arthralgias
- Malaise
- Fever
- Chest pain:
 - Reported in 35%
 - Most commonly pleuritic, sharp, stabbing, precordial
- Dyspnea on exertion is common.
- Orthopnea and shortness of breath if congestive heart failure (CHF) is present
- Palpitations are common
- Acute coronary syndrome due to local spasm & inflammation
- Syncope:
 - May signal high-grade aortic valve block or risk for sudden death from VT/VF

Physical-Exam

- Fever
- Tachypnea
- Tachycardia:
 - Often out of proportion to fever

- Cyanosis
- Hypotension:
 - Due to left ventricular dysfunction
 - Uncommon in the acute setting and indicates a poor prognosis when present
- Bibasilar crackles
- Rales
- Jugular venous distention (JVD)
- Peripheral edema
- Hepatomegaly
- Ascites
- S₃ or a summation gallop if significant biventricular involvement
- Intensity of S₁ may be diminished
- Murmurs of mitral or tricuspid regurgitation
- Pericardial friction rub if associated with pericarditis

Pediatric Considerations

- Most common cause of heart failure in previously healthy children
- Particularly infants, present with nonspecific symptoms:
 - Fever
 - Respiratory distress
 - Poor feeding or, in cases with CHF, sweating while feeding
 - New onset murmur
 - Cyanosis in severe cases

ESSENTIAL WORKUP

- Physical exam
- EKG
- CXR

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Cardiac enzymes
 - Troponin T: Low levels can be used to exclude myocarditis
 - Troponin I specificity is 89%; sensitivity is 34%
 - Creatinine kinase (elevated MB) may be elevated from myocardial necrosis
- Erythrocyte sedimentation rate (ESR) is elevated in 60% during the acute phase.
- Leukocytosis is present in 25%.
- Viral titers; cultures rarely positive
- Mycoplasma, antistreptolysin titers, cold agglutinin titer
- Hepatitis panels

- Lyme titer
- Monospot testing
- CMV serology
- Blood cultures

Imaging

- EKG:
 - Sinus tachycardia most frequent finding
 - Transient, nonspecific ST- and T-wave changes
 - Atrial and ventricular dysrhythmias
 - Heart block and conduction defects:
 - 20% have a conduction delay.
 - 20% have a left bundle branch block.
- CXR:
 - Normal cardiac silhouette
 - Pulmonary edema
 - Pleural effusion
- Echocardiogram:
 - Impairment of left ventricular systolic and diastolic function
 - Segmental wall motion abnormalities
 - Impaired ejection fraction
 - Pericardial effusion
 - Ventricular thrombus has been identified in 15% of patients
- Gallium⁶⁷ and Indium¹¹¹-labeled antimyosin antibody scans
- Gadolinium-enhanced MRI:
 - Indicate cardiac inflammation and myocyte necrosis
- Cardiac MRI:
 - Abnormal signal areas correlate with regions of myocarditis
 - Reported 76% sensitivity, 96% specificity, and 85% diagnostic accuracy
 - Considered in patients in whom the diagnosis is unclear and endocardial biopsy is planned

Diagnostic Procedures/Surgery

- Right ventricular endomyocardial biopsy:
 - Appropriate in heart transplant recipients
 - Polymerase chain reaction (PCR) amplification of viral genome in endomyocardial tissue
- PCR identification of a viral infection from pericardial fluid, or other body fluid sites

DIFFERENTIAL DIAGNOSIS

- Acute MI

- Acute and chronic pulmonary embolus
- Aortic dissection
- Adrenal insufficiency
- Environmental challenges
- Esophageal perforation/rupture/tear
- Hyperpyrexia
- Hypothermia
- Kawasaki disease
- Pericarditis
- Pneumonia
- Viral
- Bacterial
- Sepsis
- Severe hypothyroidism and hyperthyroidism
- Toxin-mediated disease



TREATMENT

ALERT

- Avoid sympathomimetic and β -blocker drugs.
- Patients presenting with Mobitz II or complete heart block require pacemaker placement.

INITIAL STABILIZATION/THERAPY

- ABCs
- Supplemental oxygen
- Cardiac monitor
- Pulse oximetry
- IV access

ED TREATMENT/PROCEDURES

- Treat dysrhythmias.
- Transthoracic or transvenous pacing for symptomatic heart block
- Supplemental oxygen
- ACE inhibitors (captopril):
 - _ Reduce afterload and inflammation.
- Digoxin:
 - _ CHF or atrial fibrillation
- Diuretics (furosemide, bumetanide)
- Hyperimmunoglobulin therapy in CMV-associated myopericarditis.
- NSAIDs contraindicated in early and acute-phase myocarditis
- Heparin and warfarin for patients with depressed LV function or intracardiac

thrombus

Pediatric Considerations

- IV immunoglobulin is an effective treatment option in pediatric viral myocarditis.
- Improved LV function and trend toward better survival

MEDICATION

- Captopril:
 - Adult dose: Initial dose 6.25 mg; can titrate to 50 mg/dose
 - Pediatric dose:
 - Infants: 0.15–0.3 mg/kg/dose (max. 6 mg/kg)
 - Children: 0.5–1 mg/kg/24h
- Digoxin:
 - Adult dose: Load: 0.4–0.6 mg IV, then 0.1–0.3 mg q6–8h. Maintain: 0.125–0.5 mg/d IV/PO
 - Pediatric dose:
 - < 2 yr: 15–20 µg/kg IV
 - 2–10 yr: 10–15 µg/kg IV
 - > 10 yr: 4–5 µg/kg IV
- Furosemide:
 - Adult dose: 20–80 mg/d PO/IV/IM; titrate up to 600 mg/d for severe edematous states
 - Pediatric dose: 1–2 mg/kg PO; not to exceed 6 mg/kg; do not administer > q6h 1 mg/kg IV/IM slowly under close supervision; not to exceed 6 mg/kg
- Immunoglobulin IV (Gamimune, Gammagard, Gammar-P, Sandoglobulin):
 - Adult dose: 2 g/kg IV over 2–5 days



FOLLOW-UP

DISPOSITION

Admission Criteria

Symptomatic patients with myocarditis:

- New-onset
- CHF
- Dysrhythmia
- Mobitz II or complete heart block
- Embolic events
- Cardiogenic shock

Discharge Criteria

Asymptomatic patient with no evidence of dysrhythmia or cardiac dysfunction

Issues for Referral

Cardiac transplant for patients with intractable CHF:

- Approximately 50% of patients die within 5 yr of diagnosis.
- Best prognosis for lymphocytic myocarditis

PEARLS AND PITFALLS

- Careful physical exam for signs of CHF and pericarditis is paramount.
- EKG should be obtained when considering the diagnosis and is especially sensitive for pediatric cases.
- Patients with evidence of dysrhythmia, CHF, or thromboembolism must be admitted.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Congestive Heart Failure



CODES

ICD9

- 074.23 Coxsackie myocarditis
- 422.91 Idiopathic myocarditis
- 429.0 Myocarditis, unspecified

ICD10

- B33.22 Viral myocarditis
- I40.0 Infective myocarditis
- I51.4 Myocarditis, unspecified

NASAL FRACTURES

David W. Munter



BASICS

DESCRIPTION

- Fractures of nasal skeleton are the most common body fractures.
- Most nasal fractures are result of blunt trauma, frequently from motor vehicle crashes, sports injuries, and altercations.
- Lateral forces are more likely to cause displacement than straight-on blows.
- Characteristics that suggest associated injuries:
 - History of trauma with significant force
 - Loss of consciousness
 - Findings of facial bone injury
 - Frontal bone crepitus
 - CSF leak

ETIOLOGY

- The vast majority of nasal fractures are from direct trauma
- Altercations account for most nasal fractures in adults
- Direct blows, especially sports, account for most nasal fractures in children



DIAGNOSIS

SIGNS AND SYMPTOMS

- Nasal deformity, asymmetry, swelling, or ecchymosis
- Epistaxis
- Periorbital ecchymosis (“raccoon eyes”) from damage to branches of ethmoidal artery:
 - May indicate nasofrontoethmoid complex injury
- Palpable sharp edges, depressions, or other irregularities suggest nasal fracture.
- Crepitus or mobility of skeletal parts on palpation
- Septal hematoma:
 - Bluish fluid-filled sac overlying nasal septum
 - Critical to detect because it must be drained
 - Failure to drain can result in necrosis of the septum
- Flattening of nasal root and widening of intercanthal distance (telecanthus):
 - Indicative of serious nasofrontoethmoid complex injury
- Clear rhinorrhea indicates possible CSF leak:
 - Rhinorrhea may be delayed.
- Loss of sense of smell suggests significant injury.

- Tear duct injuries may be present with abnormal tearing.
- Associated eye injuries:
 - Subconjunctival hemorrhage
 - Hyphema
 - Retinal detachments

History

- Direct blow
- Associated injuries or symptoms
- Presence of epistaxis
- Changes in vision or smell

Physical-Exam

- Thorough physical exam with visual inspection and palpation is vital.
- It is critical to identify a septal hematoma:
 - Bluish bulging mass on nasal septum
- Septal deviation
- Epistaxis or intranasal laceration
- Examine closely for telecanthus:
 - Intercanthal width > 30–35 mm
 - Wider than width of 1 eye
 - May indicate nasofrontoethmoid fracture
 - Usually associated with depressed nasal bridge
- CSF rhinorrhea:
 - Indicates more serious underlying facial bone or skull fracture
 - CSF mixed with blood will often cause double ring sign when placed on filter paper, although this sign is not 100% reliable.

ESSENTIAL WORKUP

If concern for anything other than a simple nasal fracture:

- Evaluate nasolacrimal duct for patency:
 - Instill fluorescein into eye and look for it in nasopharynx under inferior turbinate.
 - Absence implies duct injury.
- Eyelash traction test:
 - Grasp eyelashes on eyelid and pull laterally:
 - If eyelid margin does not become taut or “bow string,” then medial portion of tendon has been disrupted.
 - This test is performed on both upper and lower eyelids.
 - Possible for only 1 portion of tendon to be selectively injured

DIAGNOSIS TESTS & NTERPRETATION

Lab

Coagulation studies if on anticoagulants with uncontrolled epistaxis

Imaging

- Nasal radiographs are rarely indicated:
 - _ Normally do not alter initial or subsequent management
 - _ Gross deformities will need referral.
 - _ Fractures without deformity will be treated conservatively regardless of radiographic findings.
 - _ Patients with associated facial bone deformity, crepitus, or tenderness may require radiographs.
- CT is test of choice if facial bone, nasofrontoethmoid, or depressed skull fractures are suspected; have low threshold for ordering CT if other injuries are suspected.

DIFFERENTIAL DIAGNOSIS

- Other facial injuries such as orbital, frontal sinus, maxillary sinus, or cribriform plate fractures
- Nasofrontoethmoid fracture



TREATMENT

PRE HOSPITAL

- Management of airway takes precedence.
- Nasotracheal intubation is contraindicated.
- Consider orotracheal intubation or cricothyroidotomy if definitive airway control is needed.
- Cervical spine precautions are indicated if there is associated trauma.
- Epistaxis can normally be controlled with direct pressure; pinch nares together.

INITIAL STABILIZATION/THERAPY

- Airway management with orotracheal intubation or cricothyroidotomy:
 - _ Nasotracheal intubation is contraindicated.
- Cervical spine precautions
- Other injuries take precedence.

ED TREATMENT/PROCEDURES

- Abrasions and lacerations:
 - _ Proper cleansing of facial wounds is essential.
 - _ Lacerations may be sutured.
- Epistaxis must be controlled if it does not stop spontaneously:
 - _ Anesthetize/vasoconstrict with topical cocaine, lidocaine, or neosynephrine spray.

- Identify bleeding source; cauterize anterior source if necessary.
- Pack nares with petroleum jelly, impregnated gauze, or any number of commercial packs.
- Posterior packs are rarely needed.
- Prophylactic antibiotics to prevent sinus infection are indicated if packed: Amoxicillin, amoxicillin/clavulanate, or trimethoprim–sulfamethoxazole or azithromycin in penicillin allergic patients.
- Displaced fractures do not need reduction in ED unless airway is compromised.
- Generally recommended to allow swelling to abate and reduce fracture in 3–5 days, although there are many specialists who recommend local anesthesia and immediate reduction.
- Septal hematoma must be drained immediately in ED:
 - Anesthetize with topical cocaine or lidocaine and vascular constriction with neosynephrine.
 - Attempt to aspirate with 18G to 20G needle on 3-mL syringe.
 - Rolling cotton swab down septum may facilitate drainage.
 - Holding mucosa down against cartilage must be done to prevent reaccumulation.
 - This can be done with petroleum jelly gauze packing.
 - Both nares should be packed to ensure adequate pressure:
 - Packing is left in place for 3–5 days or until follow-up with ear, nose, and throat (ENT).
 - Prophylactic antibiotics are prescribed.

MEDICATION

- Amoxicillin: 500 mg PO TID (peds: 40 mg/kg PO div. TID)
- Amoxicillin/clavulanate: 500/125–875/125 mg PO BID (peds: 40 mg/kg/d of amoxicillin PO BID)
- Azithromycin: 500 mg PO day 1 followed by 250 mg PO daily for 4 additional days (peds: 10 mg/kg PO day 1, followed by 5 mg/kg PO days 2–4)
- Cocaine: Topical 4%
- Lidocaine: 1–2% without epinephrine
- Neosynephrine nasal spray
- Trimethoprim–sulfamethoxazole: Double strength (DS) PO BID (peds: 40 mg/kg/d sulfamethoxazole PO BID)

IN PATIENT CONSIDERATIONS

Admission Criteria

- Most nasal fractures do not require admission.
- Admit patients with nasoethmoid fractures or more significant craniofacial injuries.

Discharge Criteria

- No evidence of significant head, neck, or other injuries.
- Epistaxis controlled
- Reliable companion or caregiver

Pediatric Considerations

- Follow up with specialist sooner because fibrous union begins in only 3–4 days
- Consider contacting child protective services if any suspicion of nonaccidental trauma:
 - History does not fit injury.
 - Always consider nonaccidental trauma as potential mechanism of injury.
- Fractures are rare in children; nasal injuries in children are more likely to be cartilaginous.
- Significant injuries in children are not always fully appreciated.



FOLLOW-UP

FOLLOW-UP RECOMMENDATIONS

- Follow up with ENT, plastic surgery, or oral maxillofacial (OMF) surgeon in 3–5 days for management:
 - Patients with septal hematoma should follow up in 24 hr for re-evaluation after drainage.
- Return for signs of clear rhinorrhea, difficulty breathing, fever, or signs associated with head injury.

PEARLS AND PITFALLS

- The absence of a septal hematoma must be documented in every case.
- Every patient discharged with nasal packing should be placed on antistaphylococcal antibiotics.
- Consider cribriform plate fractures in patients with clear rhinorrhea after nasal injury.
- Have a low threshold for ordering facial bone CT if there is suspicion for associated injuries or fractures.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Epistaxis
- Facial Fractures



CODES

ICD9

- 801.00 Closed fracture of base of skull without mention of intra cranial injury, unspecified state of consciousness
- 802.0 Closed fracture of nasal bones
- 920 Contusion of face, scalp, and neck except eye(s)

ICD10

- J34.89 Other specified disorders of nose and nasal sinuses
- S02.2XXA Fracture of nasal bones, init encntr for closed fracture
- S02.19XA Oth fracture of base of skull, init for clos fx

NECK INJURY BY STRANGULATION/HANGING

David Della-Giustina • Karen Della-Giustina

BASICS

DESCRIPTION

- Strangulation:
 - Ligature: Material used to compress structures of neck
 - Manual: Physical force used to compress structures of neck
 - Postural: Airway obstruction from body weight (over an object) or position (typically in infants)
- Hanging is a form of strangulation:
 - Complete (judicial type): Victim's entire body is suspended off the ground
 - Incomplete (nonjudicial): Some part of victim's body contacts the ground
 - Typical: The point of suspension is placed centrally over the occiput.
 - Atypical: The point of suspension is in any position other than over the central occiput.
 - Intentional: Suicide, homicide, autoerotic, "the choking game"
 - Accidental: Often children or clothing caught in machinery
 - Near-hanging: Survival following nonjudicial hanging

ETIOLOGY

- Hanging (judicial):
 - Victim is dropped a distance at least equal to his or her height
 - Forceful distraction of head from torso results in a decapitation type of injury (fracture of cervical spine and transection of spinal cord)
- Hanging (nonjudicial):
 - Typically occurs from a lower height
 - Injuries mimic nonjudicial strangulation
- Strangulation:
 - External neck pressure causes cerebral hypoxia secondary to venous and arterial obstruction.
 - Pressure on neck structures may cause airway, soft tissue, and vascular injuries.
 - Cervical spine injuries are uncommon except with judicial-type hanging.
- Death:
 - Secondary to mechanical closure of blood vessels or airway
 - Secondary to cardiac arrest from extreme bradycardia due to increased vagal tone from carotid sinus pressure
 - Secondary to direct neurologic injury to the spinal cord
 - Secondary to pulmonary complications in near-hanging victims

- Secondary to cerebral hypoxia

COMMONLY ASSOCIATED CONDITIONS

- Cervical spine injury
- Hypoxic cerebral injury
- Arterial or venous dissection/thrombosis
- Hyoid bone fracture:
 - Typically seen in nonjudicial strangulation
- Cricoid cartilage disruption (rare)
- Thyroid cartilage disruption:
 - More common in nonjudicial strangulation deaths
- Phrenic nerve injury
- Airway edema
- Aspiration pneumonitis (may be late)
- Neurogenic pulmonary edema (may be late):
 - Due to massive central sympathetic discharge
- Postobstructive pulmonary edema (may be rapid onset):
 - Due to negative intrapleural pressure resulting from inspiration against an external airway obstruction
- Air embolism:
 - Consider when SC air and vascular injuries are present



DIAGNOSIS

SIGNS AND SYMPTOMS

- Airway disruption:
 - SC emphysema
 - Dyspnea
 - Dysphonia or stridor
 - Loss of normal cartilaginous landmarks
- Cervical spine injury:
 - Respiratory arrest
 - Paralysis
- Neurologic injury:
 - Hoarseness
 - Dysphagia
 - Altered mental status
 - Neurologic deficit
- Pulmonary sequelae:
 - Respiratory distress
 - Pulmonary edema, ARDS, pneumonia
- Soft tissue injury:
 - Abrasions, contusions, ecchymoses, ligature, or hand marks

- Vascular injuries:
 - Expanding hematoma
 - Pulse deficits or bruits
 - Evidence of cerebral infarction
 - Tardieu spots: Petechial hemorrhages of the skin, mucous membranes, and conjunctiva cephalad to the ligature marks

Pediatric Considerations

- Structures of neck are more cartilaginous and mobile than in adults
- More resistant to crush injuries and fractures
- Rapid airway compromise can occur with relatively little edema of soft tissues secondary to smaller airway diameter.

History

- Strangulation method:
 - Patient position:
 - To determine mechanism of strangulation
 - Predict potential injuries
 - Higher fall implies greater force:
 - Decapitation-type injury more common
 - Knot position:
 - Arterial occlusion more likely in typical hanging
 - Ligature material:
 - Elasticity limits force applied
 - Venous occlusion may still produce unconsciousness and death
- Circumstance:
 - Accidental, suicide/homicide, NAT, sexual, “choking game”

Physical-Exam

- ABCs:
 - Airway or respiratory compromise
 - C-spine precautions
- Disability:
 - Coma, AMS, neurologic deficit, paralysis
- Secondary survey:
 - Assess for traumatic injury to the neck:
 - Soft tissue, aero-digestive, vascular
 - Other traumatic injury due to fall, self-inflicted wounds (suicidal), injury sustained in conflict (homicidal/NAT)

ESSENTIAL WORKUP

- CT of the cervical spine through T1

- CT scan of the head:
 - For cerebral hemorrhage, subarachnoid hemorrhage, hematoma, edema, and evidence of hypoxic injury
- CT angiography of the neck:
 - For thrombosis and intimal dissection
- Plain radiography:
 - CXR to evaluate for SC emphysema, aspiration pneumonitis, and pulmonary edema
- Pulse oximetry
- Cardiac monitor

DIAGNOSIS TESTS & NTERPRETATION

Lab

- ABG (may be considered):
 - Evaluate for evidence of hypoxia or respiratory compromise.
- Hematocrit for significant blood loss
- Type and cross-match in anticipation of transfusion for vascular injuries.
- Coagulation profile for significant blood loss or coagulopathy
- Toxicology studies (ASA/APAP/ETOH):
 - Consider for suicide-related ingestions

Imaging

- MRI of the neck:
 - High sensitivity of MRI for soft tissue injury, bone and cartilaginous injury.
 - Superior to CT in diagnosis of soft tissue injury.
- Arteriography:
 - Definitive evaluation for potential vascular injuries

Diagnostic Procedures/Surgery

- Fiberoptic endoscopy:
 - Allows direct visualization for evaluation of aero-digestive injury
 - May aid in intubation
- Surgical exploration

DIFFERENTIAL DIAGNOSIS

Etiology of strangulation:

- Accidental, homicidal, suicidal, NAT, auto-erotic, choking game



TREATMENT

PRE HOSPITAL

- ABCs
- Early and aggressive airway management: Oxygen, suction, intubation, as indicated:
 - Remove ligature.
- Cardiac monitor
- Cervical spine stabilization:
 - Patient position, strangulation method, drop involved, knot location, signs of foul play

INITIAL STABILIZATION/THERAPY

- ABCs
- Aggressive airway management with cervical spine precautions is paramount:
 - Early intubation for respiratory compromise
 - Supplemental oxygen
 - Cricothyrotomy or tracheostomy may be required if severe maxillofacial injuries are present:
 - Avoid cricothyrotomy if hematoma over cricothyroid membrane or evidence of cricotracheal disruption is seen.
 - Arrange for emergent tracheostomy in above scenario (see Larynx Fracture).
- Control bleeding with application of direct pressure:
 - Do not explore in the ED

ED TREATMENT/PROCEDURES

- IV access
- Consult otolaryngologist or trauma surgeon in management of neck soft tissue injuries.
- Consult vascular surgery in management of vascular injuries.
- Consult neurology for suspected cerebral ischemic insults (thrombosis, embolism, dissection).
- Supportive care for suspected elevated intracranial pressure/cerebral edema:
 - Elevate head of bed.
 - Ensure adequate oxygenation and cerebral perfusion.
 - Prevent secondary neurologic injury/insult.
 - Consult neurosurgery for intracranial pressure monitoring and surgery as indicated.
- Neck injury with SC emphysema:
 - Assume that mucosa of upper airway communicates with deep tissues of neck.
 - Administer antibiotics.
- Laryngeal edema:
 - Consider steroids.
- Evaluate for associated injuries or harm:

- Consider ingestions in suicidal cases.
- Report suspected nonaccidental injuries in children.

MEDICATION

- Hypoxic brain injury:
 - Mannitol: 0.25–1 g/kg/dose IV (consider for elevated intracranial pressure; not routinely used in pediatric cases)
 - Hypertonic saline: Dosing regimens vary (consider for elevated intracranial pressure)
 - Phenytoin: 15–20 mg/kg IV (loading dose) as needed for seizures
- Neck injury with SC emphysema:
 - Ampicillin/sulbactam: 1.5–3 g (peds: 100–400 mg/kg/d) IV q6h
 - Clindamycin: 600 mg (peds: 25–40 mg/kg/d) IV q8h
- Airway edema:
 - Dexamethasone: 0.5–2 mg/kg/d (peds: 0.25–0.5 mg/kg) IV q6h



FOLLOW-UP

DISPOSITION

Admission Criteria

- Admit patients with strangulation or hanging-mechanism injuries to a monitored setting to observe for airway or neurologic compromise (may have delayed onset).
- Surgical correction of laryngeal, esophageal, or vascular neck injuries
- Altered level of consciousness, new neurologic deficit, coma
- Respiratory distress:
 - Supportive care for pulmonary edema, ARDS, pneumonia
- All patients with suspected suicidal or homicidal strangulation injury should have psychiatric or social work consultation.
- For pediatric patients:
 - Suspected nonaccidental trauma, concern for safety in the home

Discharge Criteria

Only patients without strangulation or hanging injuries may be discharged after appropriate observation in the ED to ensure absence of airway compromise, vascular injury, neurologic deficit, and suicidal/homicidal ideation.

FOLLOW-UP RECOMMENDATIONS

- Neuropsychiatric evaluation:
 - Consider in evaluation for hypoxic encephalopathy
- Psychiatry/psychology:
 - Suicidal or homicidal patients

- Auto-erotic or “choking game” patients for medical/cognitive/behavioral therapy
- Surgical follow-up:
 - As indicated, based on injuries sustained

PEARLS AND PITFALLS

- Cervical spine injury is uncommon in nonjudicial hanging victims:
 - Cerebral hypoxia is the probable cause of death in the majority of victims.
- Aggressive airway management is paramount.
- Thoroughly evaluate for associated injuries.
- Consider admission for observation of all strangulation/hanging victims.
- Prognosis:
 - GCS on arrival does not predict prognosis.
 - Poor prognosis is suggested by:
 - Anoxic brain injury on head CT
 - Increased hanging time
 - Cardiac arrest at the scene AND on arrival to the ED

ADDITIONAL READING

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- Nichols SD, McCarthy MC, Ekeh AP, et al. Outcome of cervical near-hanging injuries. *J Trauma*. 2009;66:174–178.



CODES

ICD9

994.7 Asphyxiation and strangulation

ICD10

- T71.161A Asphyxiation due to hanging, accidental, initial encounter
- T71.162A Asphyxiation due to hanging, intentional self-harm, initial encounter

- T71.163A Asphyxiation due to hanging, assault, initial encounter

NECK TRAUMA, BLUNT, ANTERIOR

Alfred A. Joshua



BASICS

DESCRIPTION

- Blunt anterior neck trauma may result in various injuries to structures in the neck:
 - Vascular:
 - Carotid artery injury (internal, external, common carotid)
 - Vertebral artery injury
 - Intramural hematoma, intimal tear, thrombosis, and pseudoaneurysm
 - Hemorrhage or neck hematoma
 - Laryngotracheal:
 - Laryngeal injuries: Fracture of hyoid bone, thyroid cartilage, cricoid cartilage, cricotracheal separation
 - Vocal cord disruption
 - Dislocation of arytenoid cartilage
 - Tracheal injuries: Hematoma or transection
 - Pharyngoesophageal:
 - Pharynx: Hematoma, perforation
 - Esophagus: Hematoma, perforation
 - Nervous system:
 - Thoracic sympathetic chain wraps around carotid artery: Disruption can result in Horner's syndrome
 - Vagus nerve and recurrent laryngeal nerve
 - Cervical nerve roots and spinal cord
 - Cervical spine:
 - Fracture of vertebral body, transverse process, spinous process, etc.
 - Dislocation

ETIOLOGY

- Motor vehicle accidents (most common cause):
 - Unrestrained occupants involved in frontal collisions may strike neck on dashboard or steering wheel: “Padded dash syndrome”
 - Shoulder harness (seatbelt) can also cause shearing injury to anterior neck.
- Assault: Blows to anterior neck from fists, kicks, or objects
- “Clothesline injury”:
 - Motorcycle, snowmobile, jet ski, or all-terrain vehicle (ATV)
 - Drivers strike neck on cord or wire suspended between 2 objects.
- Strangulation

Pediatric Considerations

- Head is proportionally larger in children, increasing risk of acceleration–deceleration injury to neck
- Intraoral blow to soft palate may cause carotid thrombosis (popsicle in mouth of child who falls, pushing the object into soft palate).

DIAGNOSIS

SIGNS AND SYMPTOMS

- Presentation varies depending on mechanism of injury and structures involved:
 - Vascular injury:
 - Hemorrhage, ecchymosis, edema
 - Carotid bruit or thrill (pathognomic for vascular injury)
 - Neurologic deficits (often delayed)
 - Laryngotracheal injury:
 - Voice changes, hoarseness, aphonia
 - Dyspnea, inspiratory stridor, labored breathing, “air hunger”
 - SC emphysema, tenderness to palpation
 - Pharyngoesophageal injury (rarely isolated):
 - Dysphagia, odynophagia, hematemesis, blood in saliva
 - Tenderness to palpation
 - Infection, sepsis (delayed presentation)
 - Neurologic injury:
 - Central or peripheral nervous system deficits

History

- Detailed history (if patient is able to provide) based on signs and symptoms:
 - Cover all structures of the neck, as well as structures outside the neck (neck trauma usually associated with injures to chest, head, etc.)

Physical-Exam

- Ensure airway protection and patency.
- Inspect neck for hemorrhage, hematoma, ecchymosis, edema, or distortion of anatomy.
- Auscultate for carotid bruits, stridor.
- Palpate to detect tenderness or SC emphysema.
- Neurologic exam to detect evidence of ischemic event, spinal cord injury, or peripheral nerve damage
- Complete physical exam to detect associated injuries to the chest, abdomen, etc.

ESSENTIAL WORKUP

Depends on history and physical exam findings

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Type and cross-match
- Baseline CBC
- BUN/creatinine may be needed prior to radiologic testing (contrast with CT or MRI)

Imaging

- Cervical spine and lateral neck radiographs
- Limited value but may show subglottic narrowing, prevertebral soft tissue swelling, SC air, fractured calcified larynx
- CXR to rule out associated injury to thorax (pneumothorax, pneumomediastinum, etc.)
- Carotid duplex US is a noninvasive, rapid screening test for arterial injury:
 - Sensitivity as high as 92% in retrospective studies for dissection, operator-dependent, poor visualization above carotid bifurcation
- CT may be used in stable patients to evaluate laryngotracheal injury, cartilage disruption, or cervical spine injury.
- CT angiography:
 - Low sensitivity (50%) and high specificity (99%) on initial studies with early-generation CT scanner compared with angiography for carotid and vertebral artery injury, may have improved rates of detection with newer-generation CT scanners
- Magnetic resonance arteriography (MRA):
 - Low sensitivity (49%) and high specificity (99%) on initial studies with MRA compared with angiography for carotid and vertebral artery injury
 - 4-vessel angiography is considered gold standard for evaluation of arterial injury.
- Indications for angiography:
 - Presence of carotid bruit
 - Expanding neck hematoma
 - Neurologic deficit without intracranial pathology on CT
 - Horner's syndrome
 - Decreased level of consciousness

Diagnostic Procedures/Surgery

- Unstable patients must go directly to surgery.
- Laryngotracheal injuries:
 - Fiberoptic laryngoscopy can visualize subglottic airway, facilitate intubation, assess airway patency and injury.
- Esophageal injuries:

- Initial study of choice: Gastrografin swallow study (less pleural irritation with extravasation) or barium swallow study
- Indications for endoscopy:
 - Odynophagia
 - Hematemesis or blood in saliva
 - SC emphysema

DIFFERENTIAL DIAGNOSIS

- Peripheral or CNS injury
- Cervical spine injury
- Associated head or thoracic trauma



TREATMENT

PRE HOSPITAL

- Airway must be vigilantly monitored:
 - Edema or expanding hematoma can progress to airway compromise.
- Orotracheal intubation preferred 1st-line technique for airway control
- Clinical signs of respiratory distress:
 - Stridor
 - Air hunger
 - Labored breathing
 - Expanding neck hematoma
- Blind nasotracheal intubation should be avoided:
 - Owing to anatomy distortion and risk of hematoma rupture
- Cervical spine must be stabilized.

INITIAL STABILIZATION/THERAPY

Airway management with cervical spine control:

- Immediate intubation indicated for patients with signs of airway compromise or impending compromise
- Cricothyroidotomy or emergent tracheostomy may be needed if oral intubation is unsuccessful.
- Contraindicated if bruising or hematoma noted over thyroid/cricoid cartilage
- Bleeding into pharynx can be reduced by packing throat with heavy gauze after airway is secured by intubation.
- Unstable patients must go directly to OR.

ED TREATMENT/PROCEDURES

- Surgical consultation should be obtained for patients with suspicion of vascular, tracheal, or esophageal injury.
- Immediate surgical repair is required for symptomatic vascular injury, tracheal injury, pharyngeal, or esophageal injury.

- Laryngeal injury may not require immediate surgical repair.
- Anticoagulation is recommended for vascular injuries due to consequent luminal narrowing and thrombosis:
 - Results in improved neurologic outcomes
 - Requires surgical consultation prior to initiation of therapy

MEDICATION

- Anticoagulation (see above)
- Prophylactic antibiotics recommended in presence of an esophageal injury to prevent abscess formation (anaerobic coverage):
 - Cefoxitin: 2 g (peds: 80–160 mg/kg/d div. q6h) IV q8h *or*
 - Clindamycin: 600–900 mg (peds: 25–40 mg/kg/d div. q6–8h) IV q8h *or*
 - Penicillin G: 2.4 million U/d (peds: 150,000–250,000 U/kg/d) IV q4–6h, + metronidazole
 - Metronidazole: 1 g load, then 500 mg (peds: 30 mg/kg/d div. q12h) IV q6h



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients who are symptomatic, have abnormal studies, or have significant blunt trauma mechanism must be admitted and observed for at least 24 hr.
- Patients with suspicion of airway or vascular injury must be admitted to ICU.

ALERT

Patients on anticoagulation medications should be observed in ED for 6 hr from injury to look for signs of delayed neck hematoma.

Discharge Criteria

Only patients with most trivial injuries who have negative studies may be discharged from ED after thorough evaluation.

FOLLOW-UP RECOMMENDATIONS

Patients should be given return precautions to the ED for delayed signs of vascular, tracheal, neurologic injury.

PEARLS AND PITFALLS

- Vascular injuries frequently have delayed presentation.
- Look for vascular injuries in blunt neck trauma patients with neurologic deficit and normal head CT.
- Always prepare for difficult airway and have specialty intervention (anesthesia,

ENT) on standby (if available).

ADDITIONAL READING

- Miller PR, Fabian TC, Bee TK, et al. Blunt cerebrovascular injuries: Diagnosis and treatment. *J Trauma*. 2001;51(2):279–285.
- Miller PR, Fabian TC, Croce MA, et al. Prospective screening for blunt cerebrovascular injuries: Analysis of diagnostic modalities and outcomes. *Ann Surg*. 2002;236(3):386–393.
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- Ullman E. Blunt neck trauma. In: Wolfson AB, Hendey GW, Ling LJ, et al., eds. *Harwood-Nuss' Clinical Practice of Emergency Medicine*. Philadelphia, PA: Lippincott Williams & Wilkins; 2005.



CODES

ICD9

- 900.00 Injury to carotid artery, unspecified
- 900.89 Injury to other specified blood vessels of head and neck
- 959.09 Injury of face and neck

ICD10

- S15.009A Unspecified injury of unspecified carotid artery, initial encounter
- S15.109A Unspecified injury of unspecified vertebral artery, initial encounter
- S19.80XA Other specified injuries of unspecified part of neck, initial encounter

NECK TRAUMA, PENETRATING, ANTERIOR

Angela Pham

BASICS

DESCRIPTION

- Wound severity gauged by violation of platysma muscle
- Neck is divided into 3 zones
 - Zone I: Between clavicles and cricoid cartilage
 - Involves vessels, lungs, trachea, esophagus, thyroid
 - Penetrating trauma in this zone carries highest mortality owing to injury to thoracic structures.
 - Zone II: Between cricoid cartilage and angle of mandible
 - Involves vessels, trachea, esophagus, C-spine, and spinal cord
 - Injuries are most common in this zone due to it being most exposed region.
 - Zone III lies above angle of mandible to base of skull
 - Injuries are difficult to access surgically

Pediatric Considerations

Larynx is located higher in neck and receives better protection from mandible and hyoid bone.

ETIOLOGY

- Gunshot wounds
- Stab wounds
- Miscellaneous (e.g., glass shards, metal fragments, animal bites)

DIAGNOSIS

SIGNS AND SYMPTOMS

- Vascular:
 - Active/persistent hemorrhage or hematoma
 - Pulse deficit
 - Horner's syndrome (carotid injury)
 - Vascular bruit or thrill
 - Venous air embolism
- Aerodigestive:
 - Respiratory distress
 - Stridor
 - Hemoptysis

- Tracheal deviation
- SC emphysema
- Pneumothorax
- Sucking wound
- Hoarseness, aphonia, dysphonia
- Dysphagia/odynophagia
- Neurologic:
 - Central or peripheral nervous system deficits

History

- Wounds across midline increase injury significance
- Stab wound
 - Size of instrument
 - Mostly low-energy penetration
- Gunshot wound
 - Type of gun used
 - Long range vs. close range

Physical-Exam

- Careful exam of wound to determine extent of injury and whether it penetrates platysma
- Wounds should never be probed blindly:
 - May result in uncontrolled hemorrhage

ESSENTIAL WORKUP

- Platysma violation
 - No: Wound care, discharge
 - Yes:
 - Unstable: Emergent airway, OR
 - Stable: Workup depends on zone violation

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Type and cross-match.
- Baseline CBC, chem panel, coags

Imaging

- Lateral neck radiograph to evaluate soft tissue injury and detect foreign bodies
- Chest radiograph to detect hemopneumothorax, mediastinal air
- Zone I:
 - Angiography: Gold standard to evaluate vessel injury, invasive
 - Helical CT angiography: Speed, noninvasive

- Aware of streak artifact from shoulder, poor view of subclavian vessels
- Esophagram with water soluble contrast or dilute barium:
 - Low sensitivity
 - Combine with esophagoscopy to exclude injury.
 - Indications: Wound approaches/crosses midline, SC air
- Zone II:
 - Asymptomatic: Observation
 - Symptomatic: OR
- Zone III:
 - Symptomatic: Angiography or CT angiogram

Diagnostic Procedures/Surgery

- Bronchoscopy can be helpful in evaluating tracheal injury.
- Surgical consult for all wounds that penetrate platysma muscle
 - Surgical exploration:
 - Expanding or pulsatile hematoma
 - Active bleeding
 - Absence of peripheral pulses
 - Hemoptysis
 - Horner's syndrome
 - Bruit
 - SC emphysema
 - Respiratory distress
 - Air bubbling through wound

DIFFERENTIAL DIAGNOSIS

- Peripheral or CNS injury
- Cervical spine injury
- Associated head or thoracic trauma



TREATMENT

PRE HOSPITAL

- Frequent suctioning to clear airway of blood, secretions, or vomitus
- 2 large-bore IVs
- High-flow O₂ should be provided
- BVM should be avoided for potential distortion of neck anatomy and airway compromise due to forced air through tracheolaryngeal wound into tissues
- Airway must be vigilantly monitored, as edema or expanding hematoma can progress to airway compromise.
- Indications for early oral intubation:

- Clinical signs of respiratory distress
- Stridor
- Air hunger
- Labored breathing
- Expanding neck hematoma
- Nasotracheal intubation has not been proven to worsen penetrating wounds

ALERT

- Occlusive dressings should be applied to lacerations over major veins to prevent air embolism.
- Cervical spine immobilization in the absence of focal neurologic deficits is not indicated
 - Blocks direct visualization/palpation of neck; increases likelihood of missing life-threatening signs

INITIAL STABILIZATION/THERAPY

- Emergent intubation is indicated:
 - Patients who are in respiratory distress or comatose.
 - Be aware of voice change or odynophagia
 - Patients who are stable without evidence of respiratory distress may be managed aggressively with prophylactic intubation or observed closely with airway equipment at bedside.
 - Orotracheal intubation with rapid-sequence induction is method of choice for securing airway in penetrating neck trauma.
 - Blind nasotracheal intubation is contraindicated with apnea, severe facial injury, or airway distortion.
 - Fiberoptic bronchoscopic intubation is advantageous as patient may stay awake, allows direct visualization of vocal cords and injuries.
 - Percutaneous transtracheal ventilation may be useful when oral or nasotracheal intubation fails:
 - This is contraindicated in cases of upper airway obstruction.
 - May cause barotrauma
 - Cricothyroidotomy contraindicated if significant hematoma overlying cricothyroid membrane
 - Tracheostomy is warranted in this setting
 - Breathing:
 - Zone I injury can cause pneumothorax or subclavian vein injury and hemothorax:
 - May require needle decompression and tube thoracostomy
- Circulation:
 - External hemorrhage:
 - Control with direct pressure.
 - If failed, insert and inflate Foley catheter balloon within wound to

- tamponade bleeding
 - Blind clamping of vessels is contraindicated owing to risk of further neurovascular injury.
- Uncontrolled bleeding or hemodynamic instability: Send directly to OR.
- After intubation, throat can be packed with heavy gauze to tamponade bleeding.
- Hemothorax: Tube thoracostomy

ED TREATMENT/PROCEDURES

- Nasogastric tube should *not* be placed because of risk of rupturing pharyngeal hematoma.
- Prophylactic antibiotics are recommended (cefoxitin, clindamycin, penicillin G + metronidazole).
- Tetanus prophylaxis

MEDICATION

- Cefoxitin: 2 g (peds: 80–160 mg/kg/d div. q6h) IV q8h *or*
- Clindamycin: 600–900 mg (peds: 25–40 mg/kg/d div. q6–8h) IV q8h *or*
- Penicillin G: 2.4 million U/d (peds: 150,000–250,000 U/kg/d) IV q4–6h, + metronidazole
- Metronidazole: 1 g load, then 500 mg (peds: 30 mg/kg/d div. q12h) IV q6h

IN PATIENT CONSIDERATIONS

Admission Criteria

- All patients with penetrating neck trauma should be admitted and observed for at least 24 hr.
- Observation must take place in facility capable of providing definitive surgical care.
- Patients with injuries suggesting airway or vascular injury must be admitted to ICU.

Discharge Criteria

- Asymptomatic patients who have negative studies may be discharged after 24 hr of observation.
- Patients with wounds superficial to the platysma may be discharged directly from the ED

PEARLS AND PITFALLS

- Failure to anticipate difficulties in airway management
- Failure to recognize impending airway compromise

ADDITIONAL READING

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- Woo K, Magner DP, Wilson MT, et al. CT angiography in penetrating neck trauma reduces the need for operative neck exploration. *Am Surg*. 2005;71(9):754–758.
- Wolfson AB. *Harwood-Nuss' Clinical Practice of Emergency Medicine*. Philadelphia, PA: Lippincott Williams & Wilkins; 2005.



CODES

ICD9

- 874.01 Open wound of larynx, without mention of complication
- 874.8 Open wound of other and unspecified parts of neck, without mention of complication
- 874.9 Open wound of other and unspecified parts of neck, complicated

ICD10

- S11.011A Laceration without foreign body of larynx, initial encounter
- S11.81XA Laceration w/o foreign body of oth part of neck, init encntr
- S11.90XA Unsp open wound of unspecified part of neck, init encntr

NECROTIZING SOFT TISSUE INFECTIONS

Adam Z. Barkin

BASICS

DESCRIPTION

- Necrotizing soft tissue infections (NSTI) are infections of any layer of the skin associated with necrotizing changes
 - Usually spreads rapidly along tissue planes
- Characterized by:
 - Widespread fascial and muscle necrosis with relative sparing of the skin
 - High mortality
 - Systemic toxicity
- Crepitant anaerobic cellulitis:
 - Necrotic soft tissue infection with abundant connective tissue gas
- Progressive bacterial gangrene:
 - Slowly progressive erosion affecting the total thickness of skin but not involving deep fascia
- Nonclostridial myonecrosis (synergistic necrotizing cellulitis):
 - Aggressive soft tissue infection of skin, muscle, SC tissue, and fascia
- Fournier gangrene:
 - Mixed aerobic–anaerobic soft tissue necrotizing fasciitis of the skin of the scrotum and penis in men and the vulvar and perianal skin in women
- *Necrotizing fasciitis*:
 - Progressive, rapidly spreading infection with extensive dissection and necrosis of the superficial and deep fascia
- Accounts for 500–1,500 cases per year in US
- Often difficult to recognize
- Incidence increases with:
 - Age
 - Smoking
 - Chronic systemic disease:
 - Diabetes
 - Obesity
 - Peripheral vascular disease
 - Alcohol abuse
 - IV drug use
- 24–34% mortality
- Also high morbidity:
 - Amputations
 - Renal failure

ETIOLOGY

- Conditions that lead to the development of NSTIs:
 - Local tissue trauma with bacterial invasion
 - Local ischemia and reduced host defenses:
 - More frequently in diabetics, alcoholics, immunosuppressed patients, IV drug users, and patients with peripheral vascular disease
- Type I NSTI:
 - Polymicrobial
 - Anaerobic and aerobic
 - Include Fournier gangrene and Ludwig angina
 - After surgical procedures
 - Existing diabetes, peripheral vascular disease, chronic kidney disease, alcohol abuse
 - Compromised immune system
 - Represent 80% of NSTIs
 - Strep species are most common aerobes
 - Also staph, enterococci, and gram-negative rods
 - Bacteroides are most common anaerobes
- Type II NSTI:
 - Monomicrobial
 - Typically aerobic Streptococcus
 - Often young, healthy patients
 - Most common cause of “flesh eating” disease
 - Methicillin-resistant Staphylococcus aureus (MRSA) species are becoming more common
- Type III NSTI
 - Least common NSTI (< 5%)
 - Rapidly progressive
 - Clostridial myonecrosis is an example
 - Usually following penetrating wounds or crush injuries
 - Also can be seen after black tar heroin injection, skin popping, intestinal surgery, obstetrical complications
- Bacteria involved include:
 - Group A β_2 -hemolytic streptococcus (GABHS)
 - Group B streptococcus
 - Staphylococci
 - Enterococci
 - Bacillus
 - Pseudomonas
 - *Escherichia coli*
 - Proteus
 - Klebsiella

- Enterobacter
- Bacteroides
- *Pasteurella multocida*
- Clostridium sp.
- Vibrio sp.
- Aeromonas sp.
- Fungi

Pediatric Considerations

- Neonates: Omphalitis and circumcision are predisposing factors.
- Risk factors for children:
 - Chronic illness
 - Surgery
 - Recent varicella infection (58-fold increased risk of GABHS NSTI)
 - Congenital and acquired immunodeficiencies



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Fever
- Altered mental status
- Chronic medical conditions
- IV drug use
- Skin:
 - Rapid progression of pain and swelling of involved area
 - In 1st 24 hr, rapid development of local swelling, heat, erythema, and tenderness
 - 24–48 hr: Purple and blue discoloration, blisters and bullae develop (often hemorrhagic)
 - Foul-smelling thin fluid (from necrosis of fat and fascia)

Physical-Exam

- Systemic toxicity:
 - Fever
 - Tachycardia
 - Tachypnea
 - Hypotension
 - Altered mental status
- Pain out of proportion to physical findings
- Skin:

- Erythema
- Tense edema
- Grayish or other discolored wound drainage
- Vesicles or bullae
- Necrosis
- Ulcers
- Crepitus (pathognomonic but present in only 10–37% of cases)
- Pain that extends past margin of infection

Pediatric Considerations

- Most common presenting symptoms
 - Localized pain (97%)
 - Rash (73%)
 - Hypotension, altered mental status, and other signs of shock are much less common

ESSENTIAL WORKUP

- Diagnosis can be difficult
- Careful exam for the aforementioned signs and symptoms in high-risk patients
- NSTIs must be suspected in patients who appear very ill and have pain out of proportion to physical findings
- Diagnosis may require incision and probing of tissue

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC with differential
- Electrolytes
- BUN and creatinine
- Disseminated intravascular coagulation panel
- Calcium level: Hypocalcemia can develop from extensive fat necrosis
- Gram stain and aerobic/anaerobic cultures of wound or tissue biopsy

Imaging

- X-rays to detect soft tissue gas: Pathognomonic, but present in only 39–57% of cases
- CT scan:
 - May be more helpful than plain x-rays in detecting SC air
 - May also identify deep abscess or other cause of infection
- MRI:
 - Can delineate extent of spread of the infection
- US:
 - Fascial thickening

- Fluid in the fascial plane
- SC soft tissue edema

ALERT

Imaging of any kind should never delay surgical debridement

Diagnostic Procedures/Surgery

- All patients with suspected NSTI must undergo surgical debridement
- Deep incisional biopsy and cultures are the gold standard for diagnosis

DIFFERENTIAL DIAGNOSIS

- Cellulitis
- Gas gangrene



TREATMENT

PRE HOSPITAL

- IV fluid resuscitation
- Manage airway as necessary.

INITIAL STABILIZATION/THERAPY

Manage airway and resuscitate as indicated:

- Rapid-sequence intubation as needed
- Supplemental oxygen, monitor, evaluate for acid–base disturbances
- IV access, CVP line may be needed
- Aggressive volume expansion including crystalloid, plasma, packed RBCs, and albumin

ED TREATMENT/PROCEDURES

- Antibiotics: Broad coverage of aerobic gram-positive and gram-negative organisms and anaerobes
- Acceptable combination therapy:
 - Penicillin or cephalosporin + an aminoglycoside or fluoroquinolone + anaerobic coverage with either clindamycin or metronidazole
- Treat methicillin-resistant Staphylococcus aureus (MRSA) until excluded:
 - Vancomycin
 - Linezolid
 - Daptomycin
- Surgical consultation:
 - Early debridement of all necrotic tissue with fasciotomy and drainage of fascial planes is paramount
- Hyperbaric oxygen as an adjunct:
 - Early transfer to hyperbaric facility may result in greater tissue salvage

- IV immunoglobulin (IVIG):
 - Controversial
 - May be beneficial in NSTI caused by group A streptococcal infection
- Observe for major complications including acute respiratory distress syndrome, renal failure, myocardial irritability, and DIC

ALERT

Clindamycin therapy should be initiated as soon as possible when group A strep infection is suspected

MEDICATION

- Ceftriaxone: 2 g (peds: 100 mg/kg/24 h; max. 4 g) IV q24h
- Ciprofloxacin: 400 mg IV q12h
- Clindamycin: 900 mg (peds: 40 mg/kg/d q6h) IV q8h
- Daptomycin: 4 mg/kg IV q24h
- Gentamicin: 2 mg/kg (peds: 2 mg/kg IV q8h) IV q8h
- Doxycycline: 100 mg IV q12h
- Imipenem/cilastatin: 250–1,000 mg IV q6–8h
- Levofloxacin: 750 mg IV q24h
- Linezolid: 600 mg PO/IV q12h (peds: 30 mg/kg/d PO/IV div. q8h)
- Meropenem: 1 g (peds: 20–40 mg/kg up to 2 g/dose) IV q8h
- Metronidazole: 500 mg (peds: Safety not established) IV q8h
- Penicillin G: 24 million U q24h (peds: 250,000 IU/kg/24h) IV q4–6h
- Piperacillin/tazobactam 3.375–4.5 g (peds: 240 mg/kg/d of piperacillin div. q8h) IV q6h
- Tigecycline: Start 100 mg IV × 1; 50 mg IV q12h
- Vancomycin: 10–15 mg/kg IV q12h (peds: 10–15 mg/kg IV q6–8h)

First Line

- Type I infections:
 - Piperacillin/tazobactam + clindamycin + ciprofloxacin/levofloxacin
 - Imipenem/cilastatin
 - Meropenem
- Type II infections:
 - Clindamycin + penicillin (or linezolid or vancomycin)
- Type III infections:
 - Clindamycin + penicillin
- Type IV infections:
 - Doxycycline



FOLLOW-UP

DISPOSITION

Admission Criteria

- All patients with an NSTI *must be admitted* for surgical debridement and IV antibiotics
- Early hyperbaric oxygen therapy may be an important adjunct

Discharge Criteria

No patient with NSTI should be discharged

Issues for Referral

After stabilization with antibiotics and surgical debridement, consider referral for hyperbaric oxygen treatment as an adjunct.

PEARLS AND PITFALLS

- The clinician must have a high index of suspicion for NSTI, as initial skin findings may be unimpressive
- Pain out of proportion to exam may be a key finding
- Mortality will be near 100% if treatment is ONLY with antimicrobials
- Scoring systems for NSTI have limited utility
- 4 tenets of treating NSTI:
 - Fluid resuscitation and management of metabolic disturbances
 - Early antimicrobial therapy
 - Surgical debridement
 - Treating organ failure

ADDITIONAL READING

- Anaya DA, Bulger EM, Kwon YS, et al. Predicting death in necrotizing soft tissue infections: A clinical score. *Surg Infect (Larchmt)*. 2009;10:517–522.
- Anaya DA, Dellinger EP. Necrotizing soft-tissue infection: Diagnosis and management. *Clin Infect Dis*. 2007;44:705–710.
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- Lancerotto L, Tocco I, Salmaso R, et al. Necrotizing fasciitis: Classification, diagnosis, and management. *J Trauma Acute Care Surg*. 2012;72:560–566.
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See Also (Topic, Algorithm, Electronic Media Element)

- Cellulitis

- Erysipelas
- MRSA, Community Acquired
- Gangrene



CODES

ICD9

- 608.83 Vascular disorders of male genital organs
- 728.86 Necrotizing fasciitis
- 785.4 Gangrene

ICD10

- I96 Gangrene, not elsewhere classified
- N49.3 Fournier gangrene
- M72.6 Necrotizing fasciitis

NECROTIZING ULCERATIVE GINGIVITIS

Stephen K. Epstein • Laura B. Glicksman

BASICS

DESCRIPTION

- Periodontal disease
- Characterized by the “punched-out” appearance of the gingival papillae
- Synonym(s):
 - Acute necrotizing ulcerative gingivitis
 - Trench mouth
 - Vincent disease
 - Fusospirochetal gingivitis
- Not contagious
- Occurs most often in children and young adults in developing nations
- Mainly occurs in sub-Saharan Africa
- Rare; seen mostly in severely immunocompromised patients
- Males > females
- Can progress to more advanced disease:
 - Necrotizing stomatitis:
 - Similar to necrotizing ulcerative gingivitis with extension to the tongue and buccal mucosa
 - Necrotizing ulcerative periodontitis:
 - Similar to necrotizing ulcerative gingivitis with periodontal attachment loss and alveolar bone involvement
 - Orofacial gangrene

ETIOLOGY

- Caused by an overgrowth of oral flora
- *Prevotella intermedia*
- Spirochetes
- Predisposing factors (not required for diagnosis):
 - Poor oral hygiene/gingivitis
 - Immunodeficiencies (e.g., HIV)
 - Immunosuppression
 - Malnutrition
 - Smoking
 - Emotional and physical stress
 - Possible association with direct contact to certain chemicals (e.g., MDMA or ecstasy)

SIGNS AND SYMPTOMS

- Essential clinical features:
 - Painful gingival lesions
 - “Punched-out,” crater-like ulcers of the papillae
 - Ulcers bleed easily or spontaneously
- Nonessential clinical features:
 - “Pseudomembrane” of necrotic debris covering the ulcerated area
 - Foul breath
 - Fever/malaise

History

- Acute, generalized oral pain
- Bleeding gums:
 - Spontaneous or with minimal manipulation
- Foul breath
- Malaise
- Low-grade fever

Physical-Exam

- Loss of interdental papillae (key clinical feature)
- “Punched-out,” crater-like ulcers of the papillae
- Necrotic debris often present over ulcerated surfaces
- “Pseudomembrane” of inflammatory and necrotic cells
- Covers ulcerative lesions
- Leaves a bleeding surface when removed
- Lymphadenopathy, particularly submandibular
- Foul breath
- Low-grade fever

ESSENTIAL WORKUP

- Consider systemic disease:
 - Neutropenia
 - HIV
- Other reasons for immunosuppression or immunocompromise
- Rule out complications:
 - Progression to necrotizing stomatitis or ulcerative periodontitis
 - Lesions extending to periodontal ligament and alveolar bone
 - Alveolar bone destruction
 - Progression to orofacial gangrene (noma)

DIAGNOSIS TESTS & INTERPRETATION

Lab

Lab tests not clinically helpful

Imaging

Generally not indicated

DIFFERENTIAL DIAGNOSIS

- Other diseases rarely have the essential clinical feature of “punched-out” interdental papillae with ulcerations.
- Acute herpetic gingivostomatitis:
 - Affects entire gingival, not just papillae
 - Low-grade fever commonly present
 - Contagious
- Viral:
 - Viral infections: Epstein–Barr, varicella zoster virus
- Thrush
- Actinomycosis
- Streptococcal/gonococcal gingivitis/stomatitis
- Secondary syphilis
- Diphtheria
- Vesiculobullous disease
- Pemphigoid
- Pemphigus
- Oral lichen planus
- Systemic lupus erythematosus
- Trauma:
 - Toothpicks
 - Vigorous toothbrushing/flossing
- Immunocompromise:
 - Leukemia
 - Agranulocytosis (malignant neutropenia)
 - HIV



TREATMENT

INITIAL STABILIZATION/THERAPY

IV fluids for dehydration

ED TREATMENT/PROCEDURES

- Administer systemic and topical pain management:

- Narcotics rarely necessary
- Viscous lidocaine
- Debride pseudomembrane:
 - Use gauze or cotton-tipped applicator soaked in diluted H₂O₂
- Antibiotics (penicillin/metronidazole or clindamycin) when indicated:
 - Fever
 - Lymphadenopathy
 - Consider broad-spectrum antibiotics, antifungals, and antivirals in the immunosuppressed patient
- Institute outpatient therapy:
 - Remove predisposing factors
 - Dilute hydrogen peroxide rinses
 - Chlorhexidine gluconate (Peridex)
 - Antibiotics if indicated
 - Avoid irritants (spicy foods, hot beverages)
 - Analgesics for pain control
 - Improve oral hygiene with daily brushing and flossing of teeth

MEDICATION

First Line

- Oral rinses:
 - Chlorhexidine gluconate (Peridex): 15 mL swish/spit BID
 - Hydrogen peroxide (3% solution diluted in half): Rinse up to 12 times daily
- Viscous lidocaine
- Pain control:
 - NSAIDs (e.g., ibuprofen), acetaminophen

Second Line

- Metronidazole: 250–750 mg (peds: 30 mg/kg/24h) PO QID × 7 days
- Penicillin VK: 500 mg (peds: <12 yr, 25–50 mg/kg/24h) PO QID. × 10 days
- Clindamycin: 300 mg PO (peds: 6–8 mg/kg/24h) TID
- Narcotic pain control



FOLLOW-UP

DISPOSITION

Admission Criteria

- Extensive disease with systemic signs
- Severe dehydration/inability to tolerate PO fluids
- Evidence of orofacial gangrene (noma): Infection of mouth/face:

– 70% mortality with no treatment

Discharge Criteria

Able to maintain hydration

FOLLOW-UP RECOMMENDATIONS

Urgent referral to a dentist or periodontist for deep scaling and debridement

PEARLS AND PITFALLS

- Consider HIV or immunosuppression
- If untreated, can progress rapidly

ADDITIONAL READING

- Bermejo-Fenoll A, Sánchez-Pérez A. Necrotising periodontal diseases. *Med Oral Patol Oral Cir Bucal*. 2004;9(suppl):108–114.
- Califano JV. Position paper: Periodontal diseases of children and adolescents. *J Periodontol*. 2003;74:1696–1704.
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- Parameter on acute periodontal diseases. American Academy of Periodontology. *J Periodontol*. 2000;71(5 suppl):863–866.
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CODES

ICD9

- 101 Vincent's angina
- 526.4 Inflammatory conditions of jaw
- 528.1 Cancrum oris

ICD10

- A69.0 Necrotizing ulcerative stomatitis
- A69.1 Other Vincent's infections
- M27.2 Inflammatory conditions of jaws

NEEDLESTICK

Gordon S. Chew

BASICS

DESCRIPTION

- Mechanisms of exposure to blood or body fluid:
 - Percutaneous
 - Mucous membrane
 - Skin
- General prevention:
 - Universal precautions
 - Avoid recapping of needles
 - Wear gloves: Decreases amount of blood exposure by 50%
 - Double gloving
 - Follow body–substance isolation protocols.
 - Hepatitis B virus vaccination
- Risk factors:
 - Risk of seroconversion from a single needlestick exposure without prior immunization:
 - Hepatitis B virus: 37–62% from HB_sAg-positive and HB_eAg-positive source, 23–37% from HB_sAg-positive and HB_eAg-negative source
 - Hepatitis C virus: 1.8%
 - HIV: Blood 0.3%, mucous membrane 0.09%
 - Infectiousness of various body fluids for HIV:
 - Plasma/serum: 10–5,000 ppm
 - CSF: 10–1,000 ppm
 - Semen: 10–50 ppm
 - Vaginal secretions, urine, saliva, tears, breast milk: <1 ppm
 - Factors affecting risk:
 - Viral load
 - Actual injection volume
 - Type and size of needle
 - Portal of entry (depth of inoculation)
 - Duration of contact
 - Level of disease in source patient
 - Host susceptibility
 - Barriers (e.g., through gloves)

DIAGNOSIS

SIGNS AND SYMPTOMS

History

Exposure to blood or body fluid:

- Date, time, circumstances, details of exposure, source
- Immunizations

DIAGNOSIS TESTS & INTERPRETATION

Women with body fluid exposure who are considering antiviral therapy must have serum or urine pregnancy testing.

Lab

To be done with occupational health if possible:

- Baseline serology for HIV (enzyme immunoassay, western blot), hepatitis B virus, hepatitis C virus (anti-HCV), ALT. Assess adequacy of hepatitis B virus vaccination.
 - HIV-Ab, HCV-Ab, HB_sAg, HB_sAb titer
- Obtain consent from source patient for HIV (consider rapid HIV-antibody test), hepatitis B virus, hepatitis C virus (anti-HCV) testing.
 - HIV-Ab, HCV-Ab, HB_sAg

Imaging

Not applicable unless concerned for retained tissue foreign body

DIFFERENTIAL DIAGNOSIS

Principally concerned with transmission of hepatitis B virus, hepatitis C virus, and HIV



TREATMENT

PRE HOSPITAL

ALERT

- Pre-hospital personnel should always maintain universal precautions to prevent needlestick or other body fluid exposure.
- Patients with exposure should be evaluated within hours for prophylactic therapy.

INITIAL STABILIZATION/THERAPY

- Copious cleaning, wound care
- Direct and immediate referral to occupational health, when available, to ensure strictest confidentiality in lab testing and treatment
- In the ED, after hours, patients with needlestick exposure must be triaged with high priority. It is important to initiate prophylactic therapy quickly after exposure.

ED TREATMENT/PROCEDURES

- If referral to occupational health is unavailable, initiate prophylactic therapy in ED.
- Tetanus prophylaxis if necessary
- HIV:
 - Begin basic vs. expanded antiretroviral prophylaxis regimen after considering HIV status of source and severity of exposure. Some organizations advocate only the expanded 3-drug regimen. Treat for 28 days.
 - CDC guidelines: For less severe percutaneous exposure, if source patient is:
 - HIV negative: No prophylaxis
 - Unknown source: Consider basic regimen
 - Patient with risk factors: Consider basic regimen
 - HIV positive, low viral load: Recommend basic regimen
 - HIV positive, high viral load: Recommend expanded regimen ≥ 3 drugs
 - CDC guidelines: For more severe percutaneous exposure, if source patient is:
 - HIV negative: No prophylaxis
 - Unknown source: Consider basic regimen
 - Patient with risk factors: Consider basic regimen
 - HIV positive, low viral load: Recommend expanded regimen 3 drugs
 - HIV positive, high viral load: Recommend expanded regimen ≥ 3 drugs
 - CDC guidelines: For less severe mucous membrane or nonintact skin exposure, if source patient is:
 - HIV negative: No prophylaxis
 - Unknown source: No prophylaxis
 - Patient with risk factors: No prophylaxis
 - HIV positive, low viral load: Consider basic regimen
 - HIV positive, high viral load: Recommend basic regimen
 - CDC guidelines: For more severe mucous membrane or nonintact skin exposure, if source patient is:
 - HIV negative: No prophylaxis
 - Unknown source: Consider basic regimen
 - Patient with risk factors: Consider basic regimen
 - HIV positive, low viral load: Recommend basic regimen
 - HIV positive, high viral load: Recommend expanded regimen ≥ 3 drugs
 - CDC preferred basic regimen:
 - Zidovudine (AZT) + lamivudine (3TC); sold as combination drug Combivir; *or*
 - Tenofovir DF (TDF) + emtricitabine (FTC);
 - Zidovudine (AZT) + emtricitabine (FTC); *or*

- Lamivudine (3TC) + tenofovir DF (TDF); *or*
- CDC alternative basic regimen:
 - Lamivudine (3TC) + stavudine (d4T); *or*
 - Emtricitabine (FTC) + stavudine (d4T) *or*
 - Lamivudine (3TC) + didanosine (ddI) *or*
 - Emtricitabine (FTC) + didanosine (ddI)
- CDC preferred expanded regimen: Basic regimen and:
 - Lopinavir/ritonavir (Kaletra)
- CDC alternative expanded regimen: Basic regimen and:
 - Atazanavir (ATV) ± ritonavir (RTV) *or*
 - Fosamprenavir ± ritonavir (RTV) *or*
 - Indinavir (IDV) ± ritonavir (RTV) *or*
 - Saquinavir (SQV) + ritonavir (RTV) *or*
 - Nelfinavir *or*
 - Efavirenz *or*
 - Consider others after expert consultation: These include abacavir, delavirdine, zalcitabine, nevirapine, enfuvirtide.
- Counseling to prevent secondary infection:
 - Safer sex advice
 - Avoid becoming pregnant
 - Do not donate blood/tissue.
 - Do not breast-feed.
- Hepatitis B virus:
 - Known HB_sAg-positive source:
 - Complete vaccination confirmed by titer: No prescription
 - Unvaccinated: Hepatitis B immune globulin ASAP, begin hepatitis B virus vaccine series.
 - Nonresponder to vaccine: Hepatitis B immune globulin ASAP, may repeat in 30 days; consider revaccination with 3-dose series.
 - Unknown responder to vaccine with inadequate titer: Hepatitis B immune globulin ASAP, vaccine booster
 - Known HB_sAg-negative source:
 - Vaccinated: No prescription
 - Unvaccinated: Begin hepatitis B virus vaccine series
 - Unknown source:
 - Complete vaccination confirmed by titer: No prescription
 - Unvaccinated: Begin vaccine series. If high-risk exposure, consider hepatitis B immune globulin
 - Nonresponder to vaccine: Hepatitis B immune globulin ASAP with revaccination 3-dose series. If high-risk exposure, repeat hepatitis B immune globulin in 30 days.
 - Unknown responder to vaccine with inadequate titer: Vaccine booster

and recheck titer in 1–2 mo

- Hepatitis C virus:
 - Use of immunoglobulins or antivirals (interferon, ribavirin) inconclusive as prophylaxis, but possibly beneficial if initiated early when infection evident

MEDICATION

- HIV:

- Zidovudine:
 - 300 mg PO BID or 200 mg PO TID
 - Side effects: GI symptoms, headache, fatigue, myalgias, marrow suppression, seizure
- Zidovudine should be taken in conjunction with lamivudine
- Lamivudine:
 - 300 mg PO QD or 150 mg PO BID
 - Side effects: GI symptoms, headache, fatigue, neuropathy, congestion, cough (caution with trimethoprim/sulfamethoxazole)
- Combivir (combination zidovudine + lamivudine) (300 mg + 150 mg tab):
 - 1 tablet PO BID
 - Side effects: See side-effect profiles of zidovudine and lamivudine
- Emtricitabine:
 - 200 mg/d PO
 - Side effects: Rash, hyperpigmentation
 - Emtricitabine must be taken in conjunction with efavirenz and zidovudine
- Tenofovir DF:
 - 300 mg/d PO
 - Side effects: GI symptoms, headache, fatigue, neuropathy, dizziness
 - Tenofovir must be taken in conjunction with efavirenz and emtricitabine
- Didanosine:
 - <60 kg 250 mg/d PO as delayed-release
 - Side effects: Pancreatitis, GI symptoms, lactic acidosis, neuropathy
- Stavudine:
 - 60 kg 40 mg PO BID or
 - If wt <60 kg, then 30 mg PO BID
 - Side effects: Peripheral neuropathy, GI symptoms, headache, pancreatitis, elevated liver function tests, neutropenia, anemia
- Lopinavir/ritonavir (Kaletra) (200 mg + 50 mg cap):
 - 2 capsules PO BID
 - Side effects: GI symptoms, hyperlipidemia
- Atazanavir:
 - 400 mg/d PO

- If used with tenofovir, then decrease to 300 mg/d PO and add ritonavir 100 mg/d PO
- Side effects: Be wary with medications that prolong PR interval, hyperbilirubinemia
- Fosamprenavir:
 - 1,400 mg PO BID
 - If used with ritonavir, then decrease to 1,400 mg/d PO or 700 mg PO BID
 - Side effects: GI symptoms, rash, drug interactions, depression
- Indinavir:
 - 800 mg + ritonavir 100 mg PO BID, in combination with (lamivudine + zidovudine) or (emtricitabine + zidovudine) or (lamivudine + tenofovir) or (emtricitabine + tenofovir);
 - If used with ritonavir, then decrease to 800 mg PO BID
 - Side effects: Nephrolithiasis, hyperbilirubinemia, GI symptoms
- Saquinavir:
 - 1,000 mg + ritonavir 100 mg PO BID
 - Side effects: GI symptoms, hepatitis
- Nelfinavir:
 - 1,250 mg PO BID
 - Side effects: Potential carcinogenic and teratogenic warning, GI symptoms, weakness, rash
- Efavirenz:
 - Alternate expanded regimen for HIV postexposure prophylaxis: 600 mg PO in combination with (lamivudine + zidovudine) or (emtricitabine + zidovudine) or (lamivudine + tenofovir) or (emtricitabine + tenofovir)
 - Side effects: Stevens–Johnson syndrome, rash, sleep disruption, dizziness, psychiatric, teratogen
- For some of the antiretroviral agents, the oncogenic and teratogenic effects are unknown.
- NRTIs and NtRTIs can result in lactic acidosis with hepatic steatosis.
- All can have serious drug interactions that lead to significant harm or death.
- Hepatitis B:
 - Hepatitis B immune globulin: 0.06 mL/kg IM
 - Hepatitis B virus booster: Unit-dose vial



FOLLOW-UP

DISPOSITION

Admission Criteria

Admission not necessary

Discharge Criteria

Manage as outpatients with appropriate follow-up in occupational medicine clinic

ADDITIONAL READING

- Beltrami EM, Williams IT, Shapiro CN, et al. Risk and management of blood-borne infections in health care workers. *Clin Microbiol Rev.* 2000;13:385–407.
- Panlilio AL, Cardo DM, U.S. Public Health Service, et al. Updated U.S. Public Health Service guidelines for the management of occupational exposures to HIV and recommendations for postexposure prophylaxis. *MMWR Recomm Rep.* 2005;54(RR-9):1–17.
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- National Clinicians' Post-Exposure Prophylaxis Hotline: Phone (888) 448-4911; Available at [Http://www.nccc.ucsf.edu](http://www.nccc.ucsf.edu). Accessed on January 29, 2013.
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CODES

ICD9

- V01.79 Contact with or exposure to other viral diseases
- V07.8 Other specified prophylactic or treatment measure
- V15.85 Personal history of contact with and (suspected) exposure to potentially hazardous body fluids

ICD10

- Z20.5 Contact with and (suspected) exposure to viral hepatitis
- Z20.6 Contact w and (suspected) exposure to human immunodef virus
- Z77.21 Contact w and exposure to potentially hazardous body fluids

NEONATAL JAUNDICE

Michele M. Chetham



BASICS

Produced by an imbalance between rates of bilirubin production and bilirubin elimination:

- Newborns have higher rate of bilirubin production than adults because of increased RBC mass and shorter RBC life span.
- Newborns, especially preterm infants, have rate limitations in hepatic conjugation and biliary excretion of bilirubin, increased enterohepatic circulation, and diminished bilirubin binding to albumin- and bilirubin-binding protein.

DESCRIPTION

- In most newborns, this represents *physiologic jaundice* and is not pathologic:
 - Bilirubin normally increases from 1.5 mg/dL in cord blood to a mean of 6.5 mg/dL on day 3, followed by a gradual decline to levels of <1.5 mg/dL by day 10 or 12 of life.
- Serum bilirubin may rise to levels exceeding neuroprotective defenses, causing bilirubin tissue binding in basal ganglia, hippocampus, brainstem nuclei, and cerebellum:
 - *Bilirubin-induced neurologic dysfunction (BIND)* caused by increasingly severe hyperbilirubinemia from mild dysfunction to acute bilirubin encephalopathy (ABE) and kernicterus.
 - *ABE* describes the acute manifestations of bilirubin toxicity seen in the 1st wk after birth.
 - *Kernicterus*: Chronic form of BIND, with significant mortality or permanent sequelae including choreoathetoid type of cerebral palsy, gaze abnormalities, hearing loss, and dental dysplasia.
 - Rate of progression of BIND depends on rate of increase of bilirubin levels, duration of hyperbilirubinemia, albumin-binding reserves, unbound bilirubin level, host susceptibility, and comorbidities.
 - Death is due to respiratory failure and progressive coma or intractable seizures.
- Risk factors for severe hyperbilirubinemia:
 - Jaundice observed in 1st 24 hr
 - Predischarge total serum bilirubin (TSB) or transcutaneous bilirubin (TcB) (see “Diagnosis”) in high-risk or high–intermediate-risk zone
 - Lower gestational age, 35–38 wk
 - Exclusive breastfeeding, especially if inadequate with excessive weight loss
 - Isoimmune or other hemolytic disease
 - Sibling with neonatal jaundice

- Cephalohematoma or excessive bruising
- Ethnicity: East Asian
- Severe hyperbilirubinemia is associated with perinatal factors: Low birth weight, macrosomia from maternal diabetes, infection, polycythemia
- Hyperbilirubinemia neurotoxicity risk factors:
 - Isoimmune hemolytic disease
 - G6PD deficiency
 - Asphyxia
 - Sepsis
 - Acidosis
 - Albumin < 3 mg/dL
- Postphototherapy (post-PT) bilirubin rebound to bilirubin levels of concern may occur:
 - At high risk are newborns < 37 wk gestation, patients with hemolytic disease, patients treated for < 72 hr.

ETIOLOGY

- Unconjugated hyperbilirubinemia:
 - Physiologic jaundice
 - Jaundice in breastfed infants:
 - *Breastfeeding failure jaundice*: Exaggeration of physiologic jaundice due to inadequate ingestion/production of sufficient volume of breast milk in the 1st wk of life
 - *Breast milk jaundice*: Begins days 3–5, peaks within 2 wk but lasts up to 8 wk; caused by increased β -glucuronidase in breast milk
 - May be exacerbated by dehydration
 - Specific hemolytic conditions:
 - Blood group isoimmunization due to ABO, Rh, and minor blood group incompatibility; ABO is most common: Rh disease is unusual (RhoGAM prevents).
 - Red cell enzyme deficiencies: G6PD deficiency
 - Red cell membrane defects: Hereditary spherocytosis and elliptocytosis
 - Sepsis: Bacterial, viral, or protozoal
 - Birth trauma:
 - Increased heme load from resolving cephalohematoma or ecchymosis
 - Polycythemia:
 - Caused by maternal–fetal transfusion
 - Fetal–fetal transfusion
 - Infants of diabetic mothers
 - Congenital hypothyroidism
 - Defective hepatic conjugation:
 - Gilbert syndrome (familial partial defect in glucuronyl transferase)

activity) is benign.

- Crigler–Najjar syndrome (congenital absence of glucuronyl transferase), lifelong unconjugated hyperbilirubinemia
- Intestinal obstruction such as ileus, functional or anatomic, increases enterohepatic circulation
- Conjugated hyperbilirubinemia:
 - Failure of hepatic excretion of conjugated bilirubin
 - Causes include neonatal hepatitis, congenital biliary atresia, extrahepatic biliary obstruction, shock liver from neonatal asphyxia, neonatal hemosiderosis

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Sleepiness, poor intake, and inadequate urine output may be present.
- Early phase of ABE:
 - Feeding difficulties with poor suck; decreased urine output
 - Fussiness, irritability, hypotonia
 - Lethargy with altered awake–sleep pattern
- Intermediate phase of ABE:
 - High-pitched cry, irritability
 - Increased tone with backward arching of neck (retrocollis) and trunk (opisthotonos) alternating with hypotonia
 - Fever
- Signs of advanced ABE:
 - Pronounced retrocollis–opisthotonos
 - Semicoma, seizures
 - Bicycling movements

Physical-Exam

- Yellowish discoloration of skin, sclera, and body fluids due to bilirubin deposition. Indicates elevated serum bilirubin level.
- Evidence of dehydration: Mottled, prolonged capillary refill
- Increasing levels of bilirubin affect skin color progressing in cephalocaudal direction:
 - Blanch skin with digital pressure to reveal underlying skin color
 - Face: Bilirubin levels > 6–8 mg/dL
 - Feet: Bilirubin levels > 12–15 mg/dL
 - Visual diagnosis of jaundice is unreliable, especially in darkly pigmented infants.

- Neurologic dysfunction is identified by abnormal tone—hypotonia, hypertonia, or variability; setting sun sign—sclera visible below upper eyelid.
- Clues to contributing conditions

ESSENTIAL WORKUP

- Clinical diagnosis considering risk factors
- TSB mandatory in any infant with suspected or obvious jaundice
- Initial TSB should be fractionated into indirect (unconjugated) and direct (conjugated) bilirubin

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Interpret TSB according to infant's age in hours, not days, to determine risk and need for treatment. Chart progression
- Identify TSB level or TcB
- TcB correlates well with TSB if available.
- Further evaluation is recommended for these newborns with jaundice:
 - _ Occurs in the 1st 24 hr of life
 - _ Persists beyond the 1st wk of life
 - _ TSB levels reach level to initiate *intensive* PT
 - _ Conjugated bilirubin is $> 10\%$ or > 2 mg/dL.
 - _ Any signs of ABE
- Serum albumin, electrolytes, BUN, Cr, calcium
- CBC with differential and RBC morphology
- Reticulocyte count
- Maternal and infant blood type
- Direct Coombs test on cord blood:
 - _ Hospital routines vary: Some will test newborns from all type O mothers.
 - _ If not available, direct Coombs test on infant's blood
- Sepsis evaluation in ill-appearing infant
- Further inpatient workup is directed at suspected cause:
 - _ Red cell enzyme assay: G6PD
 - _ Liver function tests
 - _ Urine-reducing substances
 - _ Metabolic or endocrine studies

Imaging

- Evaluation for obstructive liver disease (direct hyperbilirubinemia)
- MRI scan of brain with abnormal globus pallidus is pathognomonic of kernicterus; not indicated for emergency management.

DIFFERENTIAL DIAGNOSIS

- See “Etiology.”
- Essential to differentiate unconjugated from conjugated hyperbilirubinemia.



TREATMENT

ALERT

- Severe newborn hyperbilirubinemia with signs of encephalopathy requires immediate treatment, as outcome is related in part to duration of exposure.
- Initiate PT when TSB exceeds threshold level based on age-in-hours nomogram and risk factors.

INITIAL STABILIZATION/THERAPY

0.9% normal saline 20 mL/kg bolus if signs of volume depletion.

ED TREATMENT/PROCEDURES

- Treatment guidelines for infants ≥ 35 wk gestation based on TSB plotted vs. age in hours for infants by risk group (below).
- Higher risk are 35–37 6/7 wk + risk factors.
- Medium risk are ≥ 38 wk + risk factors, or 35–37 6/7 wk and well.
- Lower risk are ≥ 38 wk and well.
- Risk factors: Isoimmune hemolytic disease, G6PD deficiency, asphyxia, significant lethargy, temperature instability, sepsis, acidosis, albumin < 3 g/dL
- Depending upon the risk group, hospitalization for *intensive* PT is indicated when TSB (mg/dL) is above:

AGE	High	Medium	Low
12 hr	6	7.5	9
24 hr	7.5	9.5	11.5
36 hr	9.5	11.5	13
48 hr	11	13	15
60 hr	12.5	14.5	16.5
72 hr	13.5	15	18
96 hr	14.5	17	20
5–7 days	15	18	21

- BiliTool is an online calculator (<http://bilitool.org/>) for risk stratification.
- *Intensive* PT involves use of high level of irradiance delivered to as much of infant’s surface area as possible (overhead light source and *bili blanket* beneath) per light source manufacturer. Eyes must be shielded.

- *Intensive* PT should decrease TSB > 0.5 mg/dL/h. Begin as soon as possible.
- Indications for *exchange transfusions* (ET) are also determined by age in hours and risk stratification, and lack of response to PT in consultation with neonatology. Exchange requires irradiated blood and albumin infusion.
- *ET* is often recommended regardless of TSB level if infant shows signs of ABE. NICU admission and monitoring.
- If isoimmune hemolytic disease, consider IV immunoglobulin 0.5–1 g/kg over 2 hr if TSB level is nearing exchange criteria.
- If any delay in admission/transfer, initiate *intensive* PT in ED.
- Treat comorbid disease (sepsis, liver dysfunction, polycythemia, hypothyroidism)
- Encourage increased frequency of feeding with breast milk or formula; supplemental dextrose–water is not useful. May need to enter supplementation or IV fluids.
- *Breastfeeding failure and breast milk jaundice*:
 - Most infants can continue to breastfeed.
 - Encourage mothers to nurse at least 8–12 times per day for 1st several days.
 - Supplementation with formula and/or IV fluids may be temporarily required.
 - 2–3 day cessation of breastfeeding is recommended for infants with breast milk jaundice and levels not responding to PT.
 - Encourage mother to maintain lactation by use of breast pump or manual expression during period of cessation.
- *Physiologic jaundice*: Reassurance and arrange appropriate follow-up

MEDICATION

First Line

IV immunoglobulin 0.5–1 g/kg over 2 hr in isoimmune hemolytic disease if TSB level is nearing exchange criteria and not responding to intensive PT.

Second Line

- Phenobarbital increases bilirubin conjugation and excretion slowly; may adversely impact cognitive development; not routinely used
- Ursodeoxycholic acid increases bile flow and is useful in the treatment of cholestatic jaundice.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Infants requiring *intensive* PT

- Evidence of significant anemia, sepsis, dehydration, or evidence of obstructive liver disease requires hospitalization for diagnostic evaluation and treatment
- Rapid transport to NICU; transport PT if transport time > 30 min

Discharge Criteria

- Stable infant with hyperbilirubinemia not requiring PT
- Stable term infant with uncomplicated nonhemolytic hyperbilirubinemia with no risk factors and TSB 2–3 mg/dL below levels recommended for *intensive* PT; may have *home* PT arranged if appropriate timely follow-up can be ensured.
- Direct communication with primary care provider and neonatal consultant.

Issues for Referral

Breastfeeding failure: Lactation consultants are available at many hospitals.

FOLLOW-UP RECOMMENDATIONS

Follow-up with primary care provider:

- Within 12 hr: Stable infant with hyperbilirubinemia not requiring PT and with no risk factors
- Within 8 hr: Stable infant with uncomplicated nonhemolytic hyperbilirubinemia with home PT arranged

PEARLS AND PITFALLS

- TSB must be interpreted according to the newborn's age in hours, not days, and with regard for risk factors for severe hyperbilirubinemia.
- PT needs to be initiated when the TSB exceeds the threshold level.
- Infant feeding and hydration must be assessed and corrected.

ADDITIONAL READING

- Maisels MJ, Bhutani VK, Bogen D, et al. Hyperbilirubinemia in the newborn infant > or = 35 wk gestation: An update with clarifications. *Pediatrics*. 2009;124:1193–1198.
- Schwartz HP, Haberman BE, Ruddy RM. Hyperbilirubinemia: Current guidelines and emerging therapies. *Pediatr Emerg Care*. 2011;27(9):884–889.



See Also (Topic, Algorithm, Electronic Media Element)

- Neonatal Sepsis
- Online access to AAP guidelines available at: <http://aappolicy.aappublications.org/cgi/content/full/pediatrics;114/1/2970> Factors, in order to prevent *BIND dysfunction*.



CODES

ICD9

- 774.6 Unspecified fetal and neonatal jaundice
- 774.7 Kernicterus of fetus or newborn not due to isoimmunization
- 774.39 Other neonatal jaundice due to delayed conjugation from other causes

ICD10

- P57.9 Kernicterus, unspecified
- P59.3 Neonatal jaundice from breast milk inhibitor
- P59.9 Neonatal jaundice, unspecified

NEONATAL SEPSIS

Lazaro Lezcano

BASICS

DESCRIPTION

Mechanism

- Life-threatening infection of the newborn, rarely occurring as late as 3 mo of age
- Overwhelmingly bacterial:
 - Rarely viral or fungal infection
 - Organisms usually present in the maternal perineal flora
- Occurs in 3–5 newborns per 1,000 live births
- Risk factors:
 - Perinatal:
 - History of recent fever ($> 37.5^{\circ}\text{C}$)
 - UTI
 - Chorioamnionitis
 - Prolonged rupture of membranes (> 18 hr)
 - Foul lochia
 - Uterine tenderness
 - Intrapartum asphyxia
 - Neonatal:
 - Prematurity
 - Fetal tachycardia (> 180 beats/min)
 - Male
 - Twinning (especially 2nd twin)
 - Developmental or congenital immune defects
 - Administration of IM iron
 - Galactosemia
 - Congenital anomaly (urinary tract, asplenia, myelomeningocele, sinus tract)
 - Omphalitis

ETIOLOGY

Sepsis

- Bacterial:
 - Group B Streptococcus
 - *Escherichia coli*
 - *Listeria monocytogenes*

- Coagulase-negative Staphylococcus
- *Treponema pallidum*
- Viral:
 - Herpes simplex is a common viral etiology.
 - Enterovirus
 - Adenovirus
- Fungi:
 - *Candida* species
- Protozoa:
 - Malaria
 - *Borrelia*

Meningitis

- Bacterial:
 - Group B Streptococcus
 - *E. coli* type K1
 - *L. monocytogenes*
 - Other streptococci
 - Nontypeable *Haemophilus influenzae*
 - Coagulase-positive and coagulase-negative Staphylococcus
 - Less commonly: Klebsiella, Enterobacter
 - *Pseudomonas*, *T. pallidum*, and *Mycobacterium tuberculosis*
 - *Citrobacter diversus* (important cause of brain abscess)
 - *Additional pathogens: Mycoplasma hominis* and *Ureaplasma urealyticum*
- Viral:
 - Enteroviruses
 - Herpes simplex virus (type 2 more commonly)
 - Cytomegaloviruses
 - *Toxoplasma gondii*
 - Rubella
 - HIV
- Fungi:
 - *Candida albicans* and other fungi



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Nonspecific history:
 - “Not acting normal”
 - Feeding poorly

- Irritable or lethargic
- General:
 - Toxic appearing
 - Altered mental status: Irritable or lethargic
 - Apnea or bradycardia
 - Mottled, ashen, cyanotic, or cool skin

Physical-Exam

- Vital signs:
 - Hyperthermia/hypothermia
 - Tachypnea
 - Tachycardia
 - Prolonged capillary refill time
- Abdominal distention
- Jaundice
- Bruising or prolonged bleeding
- Sepsis syndrome in the neonate:
 - Septic shock
 - Hypoglycemia
 - Seizures
 - Disseminated intravascular coagulation (DIC)
 - If untreated, cardiovascular collapse and death

ESSENTIAL WORKUP

- Sepsis evaluation followed by empiric antibiotics and support
- Determine a source for the infection.
- Identify metabolic abnormalities.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Bedside glucose determination
- CBC:
 - WBCs elevated or suppressed
 - Shift to the left
 - Thrombocytopenia
- C-reactive protein (CRP)
- Urinalysis
- Cultures as soon as the diagnosis is entertained:
 - Blood, CSF, catheterized or suprapubic urine, stool
- Lumbar puncture:
 - May need to delay if hemodynamically unstable
 - Cell count, protein, glucose, culture, Gram stain

- Serum glucose needed to exclude hypoglycemia
- Arterial blood gas and oximetry
 - Metabolic acidosis is common.
- Electrolytes and calcium:
 - Hyponatremia
 - Hypocalcemia
- DIC panel:
 - Coagulopathy is a late complication.
 - Monitor PT, PTT and fibrinogen-split products

Imaging

CXR to rule out pneumonia

DIFFERENTIAL DIAGNOSIS

- Heart disease:
 - Hypoplastic left heart syndrome
 - Myocarditis
- Metabolic disorders:
 - Hypoglycemia
 - Adrenal insufficiency (congenital adrenal hyperplasia)
 - Organic acidoses
 - Urea cycle disorders
- Intussusception
- Child abuse
- CNS:
 - Intracranial hemorrhage
 - Perinatal asphyxia
- Neonatal jaundice
- Hematologic emergencies:
 - Neonatal purpura fulminans
 - Severe anemia
 - Methemoglobinemia
 - Malignancy (congenital leukemia)



TREATMENT

PRE HOSPITAL

Cautions

- Ventilatory support if obtunded, apneic, or respiratory distress
- IV access
- Continuous monitoring

ED TREATMENT/PROCEDURES

- Implement empiric treatment for neonatal sepsis if presentation at all consistent, particularly if any risk factors are present.
- Administer antibiotics:
 - Ampicillin and gentamicin or cefotaxime
 - Add vancomycin if the patient's condition continues to deteriorate or any suggestion of *Streptococcus pneumoniae*.
 - Cefotaxime may be substituted for gentamicin.
- Support for septic shock if present

MEDICATION

- Ampicillin: 200 mg/kg/d q6h IV/IM for infant > 2 kg birth weight and > 2 wk old; 150 mg/kg/d q8h if < 7 days old
- Cefotaxime: 150 mg/kg/d q6h IV/IM for infants > 2 kg birth weight and > 1 wk old; 150 mg/kg/d q8h IV/IM if 8–28 days old; 100 mg/kg/d IV/IM q12h if 0–7 days old
- Gentamicin: 2.5 mg/kg/dose q8h IV/IM if postconceptual age > 37 wk and > 7 days old; 2.5 mg/kg/dose q12h if < 7 days old
- Vancomycin: 15 mg/kg/dose IV q8h if postconceptual age > 37 wk and > 7 days old; 15 mg/kg IV q12h if < 7 days old



FOLLOW-UP

DISPOSITION

Admission Criteria

- All patients with suspected sepsis are admitted to the hospital for supportive care, IV antibiotic therapy, and close monitoring.
- All children < 1 mo with a fever are generally admitted even in the absence of significant suspicion of sepsis. Older children are admitted based upon the clinical presentation.

Initial Stabilization

- Airway management indicated if obtundation, apnea, or respiratory distress
- IV access to administer fluids and pressors as needed
- Continuous monitoring

ADDITIONAL READING

- American Academy of Pediatrics. *Red Book: 2012 Report of the Committee on Infectious Diseases*. 29th ed. Elk Grove Village, IL: American Academy of Pediatrics; 2012.

- Edwards MS. Postnatal bacterial infections. In: Martin RJ, Fanaroff AA, Walsh MC, eds. *Neonatal-Perinatal Medicine. Diseases of the Fetus and Infant*. 9th ed. St. Louis, MO: Mosby; 2011:793–829.
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- Van de Hoogen A, Gerards LJ, Verboon-Macielek MA, et al. Long-term trends in the epidemiology of neonatal sepsis and antibiotic susceptibility of causative agents. *Neonatology*. 2010;97(1):22–28.
- Young TE, Mangum B. *Neofax 2011: A Manual of Drugs Used in Neonatal Care*. 24th ed. Montvale, NJ: Physicians' Desk Reference; 2011:2–105.



CODES

ICD9

- 038.0 Streptococcal septicemia
- 038.42 Septicemia due to escherichia coli [E. coli]
- 771.81 Septicemia [sepsis] of newborn

ICD10

- P36.0 Sepsis of newborn due to streptococcus, group B
- P36.4 Sepsis of newborn due to Escherichia coli
- P36.9 Bacterial sepsis of newborn, unspecified

NEPHRITIC SYNDROME

Maureen L. Joyner



BASICS

DESCRIPTION

- Acute glomerulonephritis (AGN) is acute inflammatory damage to glomerulus, associated with:
 - Abrupt onset of hematuria with or without RBC casts
 - Acute renal failure manifested by edema, hypertension, azotemia, decline in urine output
 - Variable proteinuria
 - Active urine sediment (RBC casts)
- Exact mechanism of AGN unclear:
 - Combination of autoimmune reactivity to specific antigens at renal glomeruli
 - Characterized by crescent formation secondary to nonspecific injury at the glomerular wall

ETIOLOGY

- Poststreptococcal glomerulonephritis (PSGN):
 - A postinfectious cause of acute nephritic syndrome, resulting from group A β -hemolytic streptococci
 - Considered a nonsuppurative complication (antibiotic treatment does not prevent this complication)
 - Occurs when immune complexes create hump-shaped subepithelial deposits in renal glomeruli
 - Most commonly affects patients between ages 3 and 15 yr but can occur at any age
 - Incidence of nephritis is 5–10% after pharyngitis and 25% after skin infections.
 - Consider PSGN in the setting of new-onset proteinuria, RBC casts, edema, and any recent infection.
 - Latent period between infection and onset of nephritis helps differentiate between PSGN and IgA nephropathy:
 - 1–3 wk in pharyngeal infection
 - 2–4 wk in cutaneous infection
 - Renal biopsy is usually not necessary for diagnosis.
 - Low complement (C3) for 6–8 wk
 - Can progress to severe renal failure if underlying infection goes untreated
 - Prognosis:

- Excellent; > 95% recover spontaneously with normalization of renal function within 6–8 wk, even with dialysis.
- Hematuria usually resolves in 3–6 mo.
- Transient nephrotic phase in 20% of patients during resolution of illness
- End-stage renal disease occurs < 5%
- Rapidly progressive glomerulonephritis (RPGN) is rare, occurring in < 1% cases.
- Most cases resolve spontaneously with no long-term sequelae.
- Other infectious sources of glomerulonephritis (GN):
 - Sepsis, pneumonia, endocarditis, viruses, HIV
 - Pulmonary, intra-abdominal, or cutaneous infections
 - Syphilis, leprosy, schistosomiasis, and malaria
 - Goal: Treat underlying infection.
- Hepatitis virus–related glomerular disease:
 - Can present with either nephritic or nephrotic symptoms
 - Causes membranoproliferative GN
 - Complements remain low indefinitely (compared to PSGN)
- Noninfectious causes of GN (due to immune complex formation):
 - Systemic lupus erythematosus, Henoch–Schönlein purpura, vasculitis, Wegener granulomatosis
 - Goodpasture syndrome
- IgA nephropathy (IgA-N)
 - Most common cause of AGN (> 25%) worldwide
 - Antibody–antigen causes immune complex deposition of IgA and C3
 - Complement levels are usually normal.
 - IgA-N has different presentations:
 - Gross hematuria following upper respiratory infection (URI)
 - Microscopic hematuria with proteinuria
 - Hematuria during viral illness or after exercise
 - Prognosis is related to serum creatinine, BP, and proteinuria.
 - 50% of patients with proteinuria may develop progressive renal disease.
 - ACE inhibitors or angiotensin-receptor blockers (ARBs) may help
- RPGN:
 - Certain patients with AGN may progress rapidly to renal failure.
 - Hallmarks are crescents on renal biopsy.
- Hereditary nephritis
 - Alport syndrome

SIGNS AND SYMPTOMS

- Hematuria:
 - Abrupt onset gross hematuria in 30–40% (coffee- or cola-colored urine)
- Edema:
 - Periorbital edema
 - Generalized edema more common in infants and children
- Infectious source or recent infection: Upper respiratory tract or skin common, e.g., PSGN
- Symptoms of congestive heart failure:
 - 40% occurrence in patients > 60 yr
 - Rare in children
- Arthritis, arthralgias, and various skin rashes: PSGN, systemic disease
- Nonspecific manifestations:
 - Malaise
 - Weakness
 - Anorexia
 - Nausea/vomiting

History

- Recent URI or skin or other infection
- Change in urine color

Physical-Exam

- Hypertension
- Edema

ESSENTIAL WORKUP

Urinalysis with sediment evaluation to detect:

- RBCs, proteinuria, and RBC casts
- RBC casts are diagnostic of an active glomerular inflammation.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC:
 - Anemia (seen in more chronic cases of GN or other systemic disease)
 - Acute leukocytosis (may suggest infectious process)
- Basic metabolic panel:
 - Assess baseline renal function
 - Check for electrolyte abnormalities
 - GFR will be normal or nearly normal
- Urinalysis:
 - RBCs +/- casts, protein

- Serum albumin
- Cultures (throat, skin, urine, blood):
 - As clinically suspected for infectious source
 - Streptozyme
- Serum complement level (C3): Decreased in infectious endocarditis, shunt nephritis, and PSGN
- Streptococcal antibodies:
 - Antistreptolysin (ASO), antistreptokinase (ASK), antideoxyribonuclease B (ADNase B), antinicotinyl adenine dinucleotidase (ANADase), and antihyaluronidase (AH)
 - ASO more reactive in pharyngeal infections
 - ADNase B, ANADase, and AH more reactive in cutaneous infections
 - ASK elevated in recent hemolytic Streptococcus infections
 - Titers do not correlate with prognosis of disease
- Urine osmolality, sodium, creatinine
- 24-hr urine collection:
 - Proteinuria initially present in 5% of children, 20% of adults with PSGN

Imaging

- Renal ultrasound: Kidney size abnormality
- Chest radiograph: Cardiomegaly, pulmonary edema, infection

Diagnostic Procedures/Surgery

Renal biopsy:

- Generally not done for PSGN, as symptoms typically resolve after a brief illness
- Recommended if atypical features of PSGN, persistently abnormal complement levels, persistent hypertension, and proteinuria > 3 g/d
- Facilitates diagnosis for other causes of nephritis

DIFFERENTIAL DIAGNOSIS

- (See “Glomerulonephritis” for further information on types of GN)
- Renal:
 - Primary glomerular disease
- Systemic:
 - Goodpasture syndrome
 - Vasculitis
 - Henoch–Schönlein purpura
- Other (rare):
 - Hemolytic-uremic syndrome
 - Thrombotic thrombocytopenic purpura
 - Acute hypersensitivity interstitial nephritis
 - Serum sickness



TREATMENT

PRE HOSPITAL

Support ABCs

INITIAL STABILIZATION/THERAPY

ABCs

ED TREATMENT/PROCEDURES

- Antibiotics for streptococcal infection:
 - Penicillin (erythromycin, if penicillin allergic)
- Restrict salt and fluid intake
- Administer loop diuretics (furosemide)
- Restore urine flow in oliguric patients:
 - Mannitol
- Treat pulmonary edema:
 - Oxygen
 - Morphine
 - Loop diuretics
- Stabilize BP to decrease proteinuria, retard progression of GN:
 - ACEIs, ARBs
 - Hypertensive emergency: Nitroprusside or other titratable antihypertensive medication
- Hemodialysis for:
 - Severe hyperkalemia
 - Fluid overload
 - Uremia
 - Severe acidosis
 - Correct electrolyte abnormalities

MEDICATION

- Erythromycin: 250–500 mg (peds: 30–50 mg/kg/d) PO q6h for 7–10 days
- Furosemide: 20–80 mg (peds: 1–6 mg/kg) PO daily/BID
- Lisinopril (ACEI): 10–40 mg (peds: > 6 yr: 0.07 mg/kg) PO daily
- Losartan (ARB): 25–100 mg (peds: > 6 yr: 0.7 mg/kg) PO daily
- Mannitol: 12.5–100 g (peds: 0.25–0.5 g/kg) IV:
 - May use single or repeat dosing; consider test dosing 1st.
- Morphine sulfate: 0.1 mg/kg/dose IV q4h
- Nitroprusside: 0.3–4 µg/kg/min IV
 - Titrate to goal mean arterial pressure for hypertensive emergency.
- Penicillin:
 - Benzathine penicillin: 1.2 million U (peds: 0.3–0.9 million U, based on weight) IM as single dose

- Penicillin VK: 250–500 mg (peds: < 12 yr 25–50 mg/kg/d) PO q6–8h for 10 days
- Other agents, including fish oil (ω -3 fatty acids for anti-inflammatory effects) and immunosuppressive agents (glucocorticoids, cyclophosphamide), may be used in consultation with specialists.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Evidence of infectious cause for GN
- Oliguria, anuria
- Uremia
- Elevated creatinine
- Edema
- Electrolyte abnormalities
- Severe hypertension
- CHF

Discharge Criteria

Mild cases of clinical nephritis in healthy patients with:

- No comorbid illness
- Strict supervision/monitoring of symptoms, diet, urine output, and medication
- Close follow-up with PMD and nephrology referral

Issues for Referral

Nephrology:

- Within 2–3 days

FOLLOW-UP RECOMMENDATIONS

- Adherence to antibiotic and antihypertensive therapy, as indicated
- Restrict salt and fluid intake.

PEARLS AND PITFALLS

- Diagnosis is confirmed by biopsy showing characteristic crescent formation within renal glomeruli.
- Must obtain thorough history of ongoing or recent infections as possible etiology of nephritis
- IgA nephropathy is most common cause of nephritis.
- Patients require aggressive management of BP and volume status.

ADDITIONAL READING

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- Kanjanabuch T, Kittikowit W, Eiam-Ong S. An update on acute postinfectious glomerulonephritis worldwide. *Nat Rev Nephrol.* 2009;5:259–269.
- Kunz R, Friedrich C, Wolbers M, et al. Meta-analysis: Effect of monotherapy and combination therapy with inhibitors of the renin angiotensin system on proteinuria in renal disease. *Ann Intern Med.* 2008;148(1):30–48.



See Also (Topic, Algorithm, Electronic Media Element)

- Acute Renal Failure
- Glomerulonephritis
- Nephrotic Syndrome



CODES

ICD9

- 580.0 Acute glomerulonephritis with lesion of proliferative glomerulonephritis
- 580.9 Acute glomerulonephritis with unspecified pathological lesion in kidney
- 583.2 Nephritis and nephropathy, not specified as acute or chronic, with lesion of membranoproliferative glomerulonephritis

ICD10

- N00.9 Acute nephritic syndrome with unsp morphologic changes
- N01.9 Rapidly progr nephritic syndrome w unsp morphologic changes
- N05.9 Unsp nephritic syndrome with unspecified morphologic changes

NEPHROTIC SYNDROME

Maureen L. Joyner

BASICS

DESCRIPTION

- Diseases causing defect in glomerular filtration barrier, producing proteinuria:
 - Proteinuria > 3 g in 24 hr
 - Hypoalbuminemia (serum albumin < 3 g/dL)
 - Peripheral edema due to hypoalbuminemia
 - Hypogammaglobulinemia
 - Hyperlipidemia (fasting cholesterol > 200 mg/dL)
- Urine fat (oval fat bodies, fatty/waxy casts)
- Glomerular basement membrane altered by:
 - Immune complexes
 - Nephrotoxic antibodies
 - Nonimmune mechanisms
 - Result: More permeable glomerular membranes and excretion of albumin and large proteins

PATHOPHYSIOLOGY

- Proteinuria due to increased filtration within renal glomeruli
- Edema due to sodium retention and hypoalbuminemia
- Postural hypotension, syncope, and shock due to severe hypoalbuminemia
- Hyperlipidemia due to hepatic lipoprotein synthesis stimulated by decreased plasma oncotic pressure
- Cumulative thromboembolism risk increased if:
 - Hypovolemia
 - Low serum albumin
 - High protein excretion
 - High fibrinogen levels
 - Low antithrombin III levels

ETIOLOGY

- Due to primary renal or systemic diseases
- Membranous nephropathy:
 - Primary cause of nephrotic syndrome in adults
 - Other causes include chronic infection (hepatitis B virus, hepatitis C virus, autoimmune disorders).
 - Renal biopsy shows involvement of all glomeruli.
 - Women have better prognosis.
 - 30% may slowly progress to renal failure.

- Renal vein thrombosis causes sudden loss of renal function.
- Treat with steroids and cytotoxic agents in severe cases.
- Minimal change disease:
 - Most common cause (90%) of nephrotic syndrome in children
 - Other causes: Idiopathic, NSAIDs, paraneoplastic syndrome associated with malignancy (often Hodgkin lymphoma)
 - Best prognosis among all nephrotic syndromes
 - Good response to steroids
- Focal segmental glomerulosclerosis (FSGS):
 - Young patients (15–30 yr) with nephrotic syndrome
 - Presents with high BP, renal insufficiency, proteinuria, microscopic or gross hematuria.
 - Causes include HIV, heroin abuse, obesity, hematologic malignancies.
 - Primary FSGS responds to steroids.
 - Secondary FSGS treated with ACE inhibitors (ACEI)
 - Collapsing FSGS usually seen in HIV patients
- Membranoproliferative glomerulonephritis:
 - May present with nephrotic, non-nephrotic, or nephritic sediment
 - Complement levels are persistently low
 - Supportive care: Steroids may be helpful in children.
 - Aspirin and dipyridamole may slow progression.
- Diabetes mellitus/diabetic nephropathy:
 - Most common secondary cause of nephrotic range proteinuria in adults
 - Microalbuminuria (30–300 mg/24hr) is primary indicator of renal disease.
 - Worsening of renal function in 5–7 yr
 - Does not cause rapid decline in renal function
 - Strict control of blood sugar and ACEI therapy slow progression.
- Monoclonal gammopathies:
 - Include amyloidosis, multiple myeloma, and light-chain nephropathy
 - Renal manifestations include proteinuria, nephrotic syndrome, nephritic syndrome, and acute renal failure.
 - Lab findings include pseudo hyponatremia, low anion gap, hypercalcemia, and Bence Jones proteinuria.
 - Congo red stain of amyloid shows apple green birefringence in polarized light.
 - Supportive care: Steroids and melphalan have some benefit.
- Systemic lupus erythematosus (SLE):
 - Can present initially as a nephritic process, with progression to nephrotic syndrome
- HIV-associated nephropathy:
 - FSGS is most common nephropathy.
 - Collapsing glomerulopathy in seropositive HIV carriers with supernephrotic syndrome results in end-stage renal failure that is rapidly progressive

(months).

- Other causes include pre-eclampsia, hepatitis, and drug reactions (culprits include NSAIDs, gold, penicillamine).

DIAGNOSIS

SIGNS AND SYMPTOMS

- Many patients are asymptomatic.
- Proteinuria
- Peripheral edema:
 - Mild pitting edema to generalized anasarca with ascites
- Hyperlipidemia
- Lipiduria (urine fatty casts and oval fat bodies)
- Postural hypotension, syncope, shock
- Hypertension
- Hematuria:
 - Microscopic or gross hematuria (secondary to renal vein thrombosis)
- Renal insufficiency to acute renal failure in some cases
- Tachypnea, tachycardia, with or without hypotension:
 - Acute onset: Suggests pulmonary embolus (PE), secondary to renal or deep venous thrombosis and hypercoagulable state
 - Up to 30% occurrence of PE in membranous glomerulonephritis
 - Chronic or exertional tachypnea due to:
 - Pulmonary edema
 - Pleural effusions
 - Infection risk due to immunosuppressive treatment and frequent exposure to infections such as Pneumococcus
 - Ascites
- Protein malnutrition

History

- Systemic disease such as diabetes, SLE, HIV
- Use of NSAIDs, gold, or penicillamine
- History of unintentional weight gain (due to fluid retention)
- History of “foamy” appearance of urine

Physical-Exam

Varies depending on degree of hypoalbuminemia, hemodynamic status, and etiology of nephrotic syndrome:

- Edema
- Hypotension/hypertension
- Shock

ESSENTIAL WORKUP

Urinalysis:

- Dipstick protein largely positive:
 - Urine specific gravity > 1.025 lowers the diagnostic significance of proteinuria.
- Microscopic analysis for urinary casts and the presence of cellular elements:
 - Oval fat bodies
 - Free lipid droplets

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC + differential:
 - Anemia common
 - Leukocytosis: Infection
 - Leukopenia: Neoplastic disease or sepsis
 - Thrombocytopenia: Liver disease
- PT/PTT, international normalized ratio:
 - Coagulation profiles abnormal with concurrent liver disease
- D-dimer, fibrinogen, antithrombin III
 - Suspected thromboembolic event:
 - Often patients are asymptomatic with PE or renal vein thrombosis; therefore need high clinical suspicion.
- 24-hr urine protein, total protein to creatinine ratio
- Serum albumin: < 3 g/dL
- Serum total protein
- Basic metabolic panel with Ca, Mg, P
- Lipid profile: Elevated total cholesterol, LDL, and VLDL
- Additional lab tests may be necessary for systemic diseases:
 - Examples include antinuclear antibody, serum and urine protein electrophoresis, hepatitis profile, syphilis, cryoglobulins, complement levels

Imaging

Renal US:

- Used in suspected secondary causes of nephrotic syndrome

Diagnostic Procedures/Surgery

- Renal biopsy:
 - Definitive test for patients who do not respond to a short course of corticosteroids
 - Helps discern primary vs. secondary pathology
- Renal angiography, CT scan, or MRI for suspected renal vein thrombosis

DIFFERENTIAL DIAGNOSIS

Proteinuria resulting from other causes:

- Renal parenchymal disease:
 - Chronic renal disease
 - Mechanical nephropathy (outlet obstruction/reflux)
 - Acute pyelonephritis
 - Sickle cell disease
- Other causes:
 - CHF
 - Essential hypertension
 - Acute febrile illness
 - Pregnancy (pre-eclampsia)
 - Severe obesity



TREATMENT

PRE HOSPITAL

Support ABCs

INITIAL STABILIZATION/THERAPY

ABCs:

- Supplemental oxygen if respiratory distress
- IV fluids:
 - For decreased BP or orthostatic hypotension due to decreased intravascular volume
 - Active rehydration in the presence of severe hypotension, shock

ED TREATMENT/PROCEDURES

- Control edema:
 - Restrict sodium intake: 2 g NaCl/d
 - Loop diuretic (furosemide): Titrate dose until response seen
 - Thiazides and potassium-sparing diuretics
 - Goal: *Slow* diuresis:
 - Aggressive diuresis can precipitate acute renal failure due to hypovolemia and increase the risk of thromboembolic complications.
- Thromboembolic prevention/treatment:
 - Heparin: 80 IU/kg bolus followed by 18 IU/kg drip IV for thromboembolic event
 - Prophylactic anticoagulation now considered acceptable when level of hypoalbuminemia is extremely low (< 2.5 g/dL): Goal INR 1.8–2
 - Consider low-dose aspirin 81 mg
 - Support stockings

- Plasmapheresis, for severe cases
- Glucocorticosteroid: Mainstay of treatment for primary nephrotic syndrome
- ACEIs/ARBs: Decreases proteinuria, prevents worsening of renal function:
 - Adverse effects of ACEI include renal failure and hyperkalemia.
- Cholesterol-lowering agents/dietary manipulation (e.g., bile acid resin, statins)
- Other agents to be considered, under supervision of a specialist:
 - Cytotoxic agents/cyclosporine
 - Recombinant erythropoietin for anemia

MEDICATION

- Enoxaparin (Lovenox): 30–40 mg (peds: 0.5–0.75 mg/kg) SC q12h
- Furosemide: 20–80 mg (peds: 1–6 mg/kg) PO daily/BID
- Heparin: 80 IU/kg bolus followed by 18 IU/kg/h drip IV
- Lisinopril (ACEI): 10–40 mg (peds: > 6 yr: 0.07 mg/kg) PO daily
- Losartan (ARB): 25–100 mg (peds: > 6 yr: 0.7 mg/kg) PO daily
- Metolazone: 5–20 mg (peds: 0.2–0.4 mg/kg) PO daily
- Prednisone: 5–60 mg (peds: 0.5–2 mg/kg) PO daily



FOLLOW-UP

DISPOSITION

Admission Criteria

- Moderate to severe heart failure, ascites, respiratory compromise
- Signs of comorbid illness, such as undiagnosed malignancy, poorly controlled diabetes, immunocompromised patients
- Acute renal failure
- Evidence of thromboembolic event

Discharge Criteria

- Patients with no comorbid disease, normal vital signs, and normal blood work
- Close follow-up with a nephrologist for further evaluation and treatment is mandatory.

Issues for Referral

Nephrology:

- Routine follow-up for BP and disease management
- Renal biopsy for appropriate patients

FOLLOW-UP RECOMMENDATIONS

- In addition to nephrology, patients should follow up with rheumatology, infectious disease, hematology/oncology, or endocrine specialist (dependent on underlying

disorder contributing to nephritic syndrome).

- Strict BP control and attention to low-cholesterol diet allow for best prognosis in long-term disease management.

PEARLS AND PITFALLS

- Characterized by proteinuria, hypoalbuminemia, and peripheral edema
- Most common causes are minimal change disease in pediatric patients and diabetic nephropathy in adults.
- May present along spectrum from hypertensive to severe hypotension and shock; maintain high index of suspicion in the appropriate setting.
- Consider associated risks of thromboembolic disease.

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See Also (Topic, Algorithm, Electronic Media Element)

- Acute Renal Failure
- Glomerulonephritis
- Nephritic Syndrome

The author gratefully acknowledges the contribution of Anwer Hussain.



CODES

ICD9

- 581.1 Nephrotic syndrome with lesion of membranous glomerulonephritis
- 581.3 Nephrotic syndrome with lesion of minimal change glomerulonephritis
- 581.9 Nephrotic syndrome with unspecified pathological lesion in kidney

ICD10

- N04.0 Nephrotic syndrome with minor glomerular abnormality
- N04.2 Nephrotic syndrome w diffuse membranous glomerulonephritis
- N04.9 Nephrotic syndrome with unspecified morphologic changes

NEUROLEPTIC MALIGNANT SYNDROME

Daniel L. Beskind



BASICS

DESCRIPTION

- Life-threatening neurologic disorder most often caused by an adverse reaction to a neuroleptic or antipsychotic medication.
- Mortality can be as high as 20%
- May develop any time during therapy with neuroleptics—from a few days to many years:
 - Most often occurs in the 1st mo of therapy
- Muscular rigidity and tremor resulting from dopamine blockade in the nigrostriatal pathway
- Hyperthermia due to central dopamine receptor blockage in the hypothalamus.
- More likely in the setting of benzodiazepine withdrawal
- May be indistinguishable from other causes of drug-induced hyperthermia (malignant hyperthermia, serotonin syndrome, anticholinergic toxins, or sympathomimetic poisoning)
- Most episodes resolve within 2 wk after cessation of offending agent.
- Diagnostic criteria:
 - Development of elevated temperature and severe muscle rigidity in association with use of antipsychotic/neuroleptic medication
 - 2 or more of the following:
 - Diaphoresis
 - Dysphagia
 - Tremor
 - Incontinence
 - Altered mental status (range from confusion to coma)
 - Mutism
 - Tachycardia
 - Elevated labile BP
 - Leukocytosis
 - Lab evidence of muscle injury
 - Symptoms are not caused by another disease process

ETIOLOGY

- Rare complication of treatment with neuroleptics:
 - Phenothiazines
 - Chlorpromazine (Thorazine)
 - Fluphenazine (Modecate)

- Prochlorperazine (Compazine)
- Promethazine (Phenergan)
- Metoclopramide (Reglan)
- Butyrophenones
 - Haloperidol
 - Droperidol
- Atypical antipsychotics
 - Risperidone (Risperdal)
 - Olanzapine (Zyprexa)
 - Quetiapine (Seroquel)
 - Clozapine (Clozaril)
 - Aripiprazole (Abilify)
- Occurs in ~1% of patients treated with neuroleptics (especially haloperidol)
- Has also been associated with abrupt withdrawal from dopamine agonists in Parkinson disease
- SSRIs or lithium along with neuroleptic medication may be associated with an increased risk
- Risk factors:
 - Rapid drug loading
 - High-dose antipsychotics
 - High-potency antipsychotics
 - IV administration of drug
 - Dehydration
 - Prior neuroleptic malignant syndrome (NMS)
 - Preceding extreme psychomotor agitation or catatonia
 - Infection or surgery



DIAGNOSIS

SIGNS AND SYMPTOMS

- Life-threatening condition
- Hallmarks of the disease:
 - Hyperthermia (temperature may be as high as 106–107°F, 41°C)
 - Altered level of consciousness—stupor
 - Significant skeletal muscle rigidity—”lead-pipe rigidity.”
 - Autonomic instability
 - Tachycardia
 - Labile BP
 - Tachypnea
 - Profuse sweating
 - Dysrhythmias

History

- Neuroleptic use
- Discontinuation of antiparkinsonian drugs
- Change in mental status

Physical-Exam

- Fever
- Tachycardia, labile BP
- Delirium
- Muscle rigidity
- Diaphoresis

ESSENTIAL WORKUP

- An accurate history (especially current medications) and physical exam confirm the diagnosis.
- Creatine phosphokinase, WBC determination, liver function tests, and iron level

DIAGNOSIS TESTS & NTERPRETATION

Lab

- Electrolytes, glucose
- BUN, creatinine
- PT/PTT, urinalysis, and urine myoglobin
- Creatine kinase
- LFTs (lactate dehydrogenase, aspartate transaminase, alkaline phosphatase, etc.)
- Venous blood gas (VBG)

Imaging

CT scan, EEG if the cause of altered level of consciousness is unclear

Diagnostic Procedures/Surgery

Lumbar puncture to rule out other causes of fever or altered mental status

DIFFERENTIAL DIAGNOSIS

Related disorders

- Malignant hyperthermia
- Serotonin syndrome
- Anticholinergic poisoning
- Sympathomimetic poisoning (cocaine, methamphetamine)
- Drug intoxication toxicity (PCP, ecstasy MDMA)
- Withdrawal from intrathecal baclofen therapy

Unrelated disorders

- CNS infection (meningitis, encephalitis)

- Tetanus
- Heat stroke
- Acute dystonia
- Strychnine poisoning
- Vascular CNS event
- Thyrotoxicosis
- Rabies
- Alcohol withdrawal
- Seizures
- Pheochromocytoma
- Acute porphyria
- Acute hydrocephalus
- Acute spinal cord injury
- Systemic infections (e.g., pneumonia, sepsis)



TREATMENT

PRE HOSPITAL

- Ventilation may be difficult because of chest wall rigidity
- Cool the patient and treat seizures if they occur
- Check fingerstick glucose

INITIAL STABILIZATION/THERAPY

- Airway intervention and circulatory support as needed
- IV, supplemental O₂, cardiac monitor
- Immediate IV benzodiazepines (diazepam, lorazepam, midazolam):
 - May require repeated large doses
- If symptoms are not controlled within a few minutes, rapid sequence intubation (RSI) and neuromuscular blockade are necessary:
 - Nondepolarizing neuromuscular blockers (vecuronium, rocuronium, pancuronium) are preferable to succinylcholine.
- Measures to control hyperthermia:
 - Ice packs
 - Mist and fan
 - Cooling blankets
 - Ice water gastric lavage
- Aggressive IV fluid therapy with lactated Ringer solution or normal saline

ED TREATMENT/PROCEDURES

- Relief of muscle rigidity
- Benzodiazepines are the drug of choice
- Bromocriptine is a dopamine agonist that may play a role in longer-term

management

- Dantrolene is a direct skeletal muscle relaxant that may play a role in longer-term management
- Neither bromocriptine nor dantrolene has a rapid onset and neither has been shown to alter outcome
- Amantadine has dopaminergic and anticholinergic effects and can be used as an alternative to bromocriptine
- Discontinue neuroleptics
- Recognize complications (rhabdomyolysis, respiratory failure, acute renal failure)

MEDICATION

First Line

- Diazepam: 5 mg IV q5min
- Lorazepam: 1 mg IV q5min
- Midazolam 1 mg IV q5min
- Rocuronium: 600–1,200 µg/kg IV × 1 for RSI
- Pancuronium: 60–100 µg/kg IV × 1 for intubation

Second Line

- Bromocriptine: 5–10 mg PO TID–QID (start 2.5 mg)
- Dantrolene: 1 mg/kg IV q4–6h × 24–48 hr
- Amantadine: 100 mg PO BID



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients with NMS should be admitted
- Patients will often require intensive care

FOLLOW-UP RECOMMENDATIONS

Patients and families must be counseled on the future use of any drug that may trigger NMS

PEARLS AND PITFALLS

- Maintain high clinical suspicion for NMS in patients on neuroleptics with mental status changes, rigidity, fever, or dysautonomia
- Must rule out other causes of fever and altered mental status (i.e., meningitis, encephalitis)

- Medication history is essential when considering NMS
- Discontinuing causative agent is the key step in treatment
- Aggressive supportive care is essential

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CODES

ICD9

333.92 Neuroleptic malignant syndrome

ICD10

G21.0 Malignant neuroleptic syndrome

NEUROLEPTIC POISONING

Molly C. Boyd • Timothy D. Heilenbach



BASICS

DESCRIPTION

- Neuroleptics (antipsychotics) used for management of:
 - Psychotic disorders
 - Agitation
 - Dementia in the elderly
 - Autism and behavioral problems in children
 - Eating disorders
 - Antiemetic
 - Migraine headaches
- Acute overdose:
 - Symptoms usually mild to moderate
 - CNS and cardiovascular symptoms predominate
 - CNS depression, seizure, and coma possible
- Dystonic reactions (dystonia):
 - Most common adverse effect
 - Can occur at any time, often within 48 hr of starting medication
- Akathisia:
 - Patient has motor restlessness and feels a need to pace or move constantly
 - Occurs within hours to weeks of starting medication
- Neuroleptic malignant syndrome (NMS):
 - Idiosyncratic, life-threatening event
 - Can occur at any time but most commonly in overdose, dose increase, and during the 1st wk of usage
- Tardive dyskinesia:
 - Movement disorder usually affecting patients after years of taking neuroleptics
 - Treated by decreasing, discontinuing, or changing the drug

ETIOLOGY

- Typical neuroleptics (phenothiazines, butyrophenones) strongly antagonize dopaminergic receptors, these include:
 - Haloperidol (Haldol)
 - Chlorpromazine (Thorazine)
 - Prochlorperazine (Compazine)
 - Thioridazine (Mellaril)
 - Fluphenazine (Prolixin)

- Promethazine (Phenergan)
- Droperidol (Inapsine)
- Hydroxyzine (Atarax)
- Typical neuroleptics also have varying degrees of antagonism for histamine, muscarinic, and α -adrenergic receptors.
- Atypical neuroleptics have weaker dopaminergic antagonism and moderate serotonergic antagonism, these include:
 - Asenapine (Saphris)
 - Aripiprazole (Abilify)
 - Clozapine (Clozaril)
 - Paliperidone (Invega)
 - Risperidone (Risperdal)
 - Olanzapine (Zyprexa)
 - Quetiapine (Seroquel)
 - Ziprasidone (Geodon)

DIAGNOSIS

SIGNS AND SYMPTOMS

- Acute overdose:
 - Symptom onset within 6 hr, 9 hr with aripiprazole, up to 24 hr with extended-release formulations (paliperidone)
 - Can be delayed if anticholinergic symptoms predominate
 - CNS:
 - Ranges from mild sedation to coma
 - Anticholinergic delirium possible
 - Extrapyrarnidal symptoms (dystonia, akathisia)
 - Seizures
 - Cardiovascular:
 - Tachycardia (anticholinergic)
 - Hypotension (antiadrenergic)
 - QT prolongation
 - Torsade de pointes (rare)
 - Respiratory:
 - Respiratory depression
 - Loss of airway reflexes
 - GI:
 - Constipation
 - Dry mouth
 - Genitourinary:
 - Urinary retention
- Dystonic reactions:

- Involuntary muscle spasms of face, neck, back, and limbs
- Dramatic appearance is frightening to patient and family
- Laryngeal dystonia is a rare form that may cause stridor and dyspnea.
- NMS:
 - Occurs in < 1% of patients, 30% mortality
 - Severe hyperthermia
 - Skeletal muscle rigidity
 - Altered mental status
 - Autonomic dysfunction
 - Electrolyte disturbance
 - Rhabdomyolysis
- Agranulocytosis:
 - Seen with clozapine and olanzapine
 - Occurs with chronic treatment
- Diabetes:
 - Hyperglycemia, new-onset diabetes, and DKA have all been reported with initiation of neuroleptics.

ESSENTIAL WORKUP

- Monitor vital signs with significant ingestions.
- Cardiac monitor
- Pulse oximetry
- Core body temperature for hyperthermia

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Electrolytes, BUN, creatinine, glucose, LFTs
- CBC for clozapine overdose, WBC can be elevated in NMS
- Creatine phosphokinase (CPK) levels if NMS suspected, agitation, or prolonged immobilization
- Serum drug screen with drug levels for possible coingestions based on history:
 - Aspirin
 - Acetaminophen
 - Lithium
 - Valproate
 - Phenytoin
 - Phenobarbital
- Urine toxicologic screens are rarely helpful
 - False-negatives and false-positives can be misleading
- Quantitative levels are rarely available and not helpful in acute management

Imaging

- ECG:
 - QT prolongation
 - QRS prolongation (rare)
- Head CT:
 - Indicated for significant mental status change

DIFFERENTIAL DIAGNOSIS

- Serotonin syndrome
- Malignant hyperthermia (if recent anesthesia)
- Antidepressant overdose
- Anticholinergic crisis
- Sympathomimetic overdose
- Opioid overdose
- Occult head injury
- Endocrine disorder
- Sepsis
- Heat stroke



TREATMENT

PRE HOSPITAL

Bring medication bottles when transporting patient to hospital.

INITIAL STABILIZATION/THERAPY

Airway, breathing, and circulation management (ABCs):

- Administer supplemental oxygen.
- Consider naloxone, thiamine, D₅₀ (or check blood glucose) for altered mental status
- Intubate if respiratory depression

ED TREATMENT/PROCEDURES

- Supportive care is the mainstay of treatment
- Decontamination:
 - Administer single dose of activated charcoal if ingestion within 1 hr
 - Do not give charcoal to patient with unprotected airway
 - Use NG tube for charcoal only if pt is intubated
 - Consider whole bowel irrigation if large amounts of extended-release formulation ingested (paliperidone)
 - Hemodialysis unlikely to be helpful due to high degree of protein binding
 - Consider lipid emulsion therapy for cardiovascular collapse
- Hypotension:
 - 0.9% normal saline (NS) IV fluid bolus

- Treat resistant hypotension with norepinephrine or phenylephrine
- Dopamine may be ineffective
- Ventricular dysrhythmias:
 - Class IA, IB, and III antidysrhythmics can potentiate cardiotoxicity. Lidocaine can be used in refractory cases
 - Magnesium for prolonged QT
 - Cardioversion if hemodynamically unstable
 - Consider intralipid (20% lipid emulsion) for cardiovascular collapse
 - For asymptomatic QTc prolongation, replete potassium, calcium, and magnesium to normal levels
 - QRS prolongation (>120 msec) should be treated with sodium bicarbonate therapy
- Dystonic reactions:
 - Administer diphenhydramine or benztropine mesylate.
 - Treatment should be continued for 3 days to prevent recurrence.
- NMS:
 - Recognition and cessation of neuroleptics is critical.
 - Active cooling for hyperthermia
 - Aggressive benzodiazepines for agitation
 - Severe cases may require bromocriptine (dopamine agonist) or dantrolene (a direct-acting muscle relaxant)
 - Consider intubation and neuromuscular blockade
- Seizures:
 - Treat initially with diazepam or lorazepam.
 - Phenobarbital for persistent seizures
 - There is no role for phenytoin in toxin-induced seizures
- Anticholinergic delirium:
 - Benzodiazepines are 1st-line agents
 - Physostigmine can be used with caution
 - Physostigmine is contraindicated in a patient with dysrhythmias, heart block, or interval prolongation on EKG

MEDICATION

- Activated charcoal: 1–2 g/kg PO
- Benztropine mesylate: 1–2 mg IV or PO
- Bromocriptine: 2.5–10 mg q8h PO
- Dantrolene: 2–3 mg/kg/d as continuous infusion (10 mg/kg max.)
- Diazepam: 5–10 mg IV q10–15min
- Diphenhydramine: 25–50 mg IV (1 mg/kg)
- Lidocaine 1–2 mg/kg followed by infusion
- Lipid emulsion (20%) 1.5 mL/kg bolus followed by 0.25 mL/kg/min infusion for 30–60 min, may repeat bolus for persistent hemodynamic compromise
- Lorazepam: 2–4 mg (peds: 0.03–0.05 mg/kg) IV q10–15min

- Magnesium sulfate: 1–2 g IV over 5–15 min
- Norepinephrine: 1–2 µg/kg/min IV titrate to BP
- Phenobarbital: 10–20 mg/kg IV (loading dose); monitor for respiratory depression
- Physostigmine 0.5 mg IV q3–5min



FOLLOW-UP

DISPOSITION

Admission Criteria

- Overdose with CNS sedation, agitation, dysrhythmias, or vital sign abnormalities to monitored bed or ICU
- NMS require ICU care
- New-onset diabetes (secondary to neuroleptic use) with severe hyperglycemia and/or ketoacidosis.

Discharge Criteria

- Asymptomatic after 6 hr of observation
- Longer observation required for aripiprazole and paliperidone ingestion as well as ingestion of extended release formulations

Issues for Referral

- Patients with unintentional (accidental) poisoning require poison prevention counseling.
- Patients with intentional (e.g., suicide) poisoning require psychiatric evaluation.
- New-onset diabetes requires primary care/endocrine follow-up.

FOLLOW-UP RECOMMENDATIONS

- Psychiatric referral for intentional overdoses
- Primary care follow-up for accidental ingestions or medication side effect follow-up

PEARLS AND PITFALLS

- Neuroleptics represent a group of drugs with diverse indications and a wide range of toxicity.
- Most overdoses are mild, and CNS depression predominates.
- Dystonic reactions are the most common side effect of neuroleptics. These reactions are dramatic in appearance but easily treatable.
- NMS is a potentially fatal reaction that can be seen in acute or chronic usage of neuroleptics.
- Newer antipsychotics can have delayed onset up to 24 hr.

- Contact the poison control center for further guidance

ADDITIONAL READING

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CODES

ICD9

- 969.1 Poisoning by phenothiazine-based tranquilizers
- 969.2 Poisoning by butyrophenone-based tranquilizers
- 969.3 Poisoning by other antipsychotics, neuroleptics, and major tranquilizers

ICD10

- T43.501A Poisoning by unsp antipsychot/neurolept, accidental, init
- T43.3X1A Poisoning by phenothiaz antipsychot/neurolept, acc, init
- T43.4X1A Poisoning by butyrophen/thiothixen neuroleptc, acc, init

NONCARDIOGENIC PULMONARY EDEMA

Rebecca B. Gilson



BASICS

DESCRIPTION

- Noncardiogenic pulmonary edema (NCPE) occurs secondary to accumulation of excess fluid and protein into the alveoli from factors other than increased pulmonary capillary pressure > 18 mm Hg
- Permeability pulmonary edema:
 - Functional disruption of the capillary–alveolar membrane allows protein and fluid to move freely from the intravascular space into the alveolar space
- Pulmonary parenchymal changes are similar to CHF
- Concomitant CHF may occur in up to 20% of patients with acute respiratory distress syndrome (ARDS)
- Distinction between NCPE and CHF:
 - Pulmonary capillary pressure ≤ 18 mm Hg
 - Often apparent from the clinical circumstances
 - The concentration of protein in the alveolar fluid is identical to that of the intravascular space in patients with NCPE
 - Cephalad redistribution of blood flow, pulmonary effusions, and cardiomegaly are usually not present
- Adult respiratory distress syndrome:
 - Clinical presentation caused by permeability pulmonary edema
 - Associated with severe physiologic impairment
- Typically, onset of the edema is within 1–2 hr of the noxious insult.
- ~ 250,000 cases occur each year in US

ETIOLOGY

- ARDS is the #1 cause:
 - Caused by:
 - Sepsis
 - Pneumonia
 - Nonthoracic trauma
 - Inhaled toxins
 - Disseminated intravascular coagulation (DIC)
 - Radiation pneumonitis
- High-altitude pulmonary edema (HAPE) neurogenic pulmonary edema
- Narcotic overdose
- Pulmonary embolus

- Eclampsia
- Transfusion-related acute lung injury (TRALI)
- Re-expansion of a collapsed lung in patient with a pneumothorax
- Salicylate intoxication
- Inhaled cocaine use
- Near drowning
- HCTZ
- Uremia
- S/p cardiopulmonary bypass; especially if patient taking amiodarone



DIAGNOSIS

SIGNS AND SYMPTOMS

- Shortness of breath
- Fatigue
- Weakness
- Cough
- Malaise

Physical-Exam

- Scattered rhonchi and rales
- Hypoxia
- Dyspnea
- Tachypnea
- Accessory muscle use
- Tachycardia
- Pink, frothy sputum
- You will *not* see the stigmata of left- and right-sided heart failure
 - Lower-extremity swelling
 - Cardiomegaly

ESSENTIAL WORKUP

- History and physical is usually enough to distinguish between cardiogenic and NCPE
- The CXR is essential in confirming the diagnosis and in assessing severity.

DIAGNOSIS TESTS & NTERPRETATION

Lab

General lab abnormalities are not specific to NCPE.

Imaging

CXR:

- Initially can be normal
- Classic butterfly pattern of pulmonary edema
- Lack of cardiomegaly

Diagnostic Procedures/Surgery

Pulmonary artery catheter:

- Pulmonary capillary wedge pressures normal or near-normal in contrast to elevated pressures with cardiogenic pulmonary edema

DIFFERENTIAL DIAGNOSIS

- Cardiogenic pulmonary edema
- Diffuse alveolar hemorrhage
- Diffuse dissemination of cancer such as with lymphoma or leukemia
- Chronic obstructive pulmonary disease exacerbation
- Pulmonary embolus
- Restrictive lung disease
- Pneumonia



TREATMENT

PRE HOSPITAL

- Patent airway
- Adequate oxygenation
- Cautions:
 - _ Patients will typically not respond to usual measures to treat CHF.

INITIAL STABILIZATION/THERAPY

- Supplemental oxygen (nasal cannula or nonrebreather)
- IV catheter
- Continuous cardiac monitor
- Continuous pulse oximetry

ED TREATMENT/PROCEDURES

- The treatment of NCPE is to treat underlying cause and give supportive care.
- Diuretics are *not* used.
- Noninvasive ventilatory support (BiPAP, CPAP) may be used if available and patient not in respiratory distress:
 - _ If oxygenation or ventilation not improving with noninvasive, intubation is required
- Endotracheal intubation is often necessary:
 - _ Improves oxygenation and ventilation

- Decreases work of breathing
- Use low tidal volumes of 6–8mL/kg to reduce barotrauma to the lungs
- Initially place on 100% O₂:
 - o Measure PO₂ and decrease FIO₂ accordingly.
- Positive end-expiratory pressure (PEEP) of 5–10 cm H₂O
- Steroids and cyclooxygenase inhibitors have not been proven effective.
- If at high altitude and concerned for HAPE, have the patient descend in elevation or put them in a hyperbaric chamber.
 - Nifedipine is adjunctive therapy to O₂ and descent.



FOLLOW-UP

DISPOSITION

Admission Criteria

All symptomatic patients should be admitted to ICU:

- Symptoms may worsen at any point for up to 3 days after noxious insult.

Discharge Criteria

Asymptomatic patients (especially narcotic overdose, HAPE, or aspiration):

- Observe in ED for 6–12 hr and then discharge with close follow-up scheduled if no evidence of pulmonary edema is present and adequate oxygenation is demonstrated.

FOLLOW-UP RECOMMENDATIONS

Patients, when discharged from the hospital, should seek medical follow-up within 48 hr.

PEARLS AND PITFALLS

- Utilizing diuretics in the acute setting may worsen patient condition.
- Failure to distinguish between cardiogenic and noncardiogenic etiologies is a pitfall as treatment is different.

ADDITIONAL READING

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- Ware LB, Matthay MA. Clinical practice. Acute pulmonary edema. *N Engl J Med*. 2005;353:2788–2796.



CODES

ICD9

- 506.4 Chronic respiratory conditions due to fumes and vapors
- 508.1 Chronic and other pulmonary manifestations due to radiation
- 508.9 Respiratory conditions due to unspecified external agent

ICD10

- _ J68.1 Pulmonary edema due to chemicals, gases, fumes and vapors
- _ J70.0 Acute pulmonary manifestations due to radiation
- _ J70.9 Respiratory conditions due to unspecified external agent

NONSTEROIDAL ANTI-INFLAMMATORY POISONING

Michele Zell-Kanter

BASICS

DESCRIPTION

- Inhibit cyclooxygenase (COX), thereby blocking the conversion of arachidonic acid to prostaglandin.
- Typically morbidity is low when an NSAID is ingested
- Most literature on nonselective NSAID toxicity involves ibuprofen exposure likely due to its OTC availability.
- Fatalities have been reported with large ingestions.
- Greater potential for toxicity with underlying CHF or renal failure:
 - NSAIDs cause sodium and water retention and decrease renal blood flow.
 - Little overdose experience with the COX-2 inhibitors (celecoxib); treatment should be the same as for the traditional NSAIDs.
 - Patients may ingest rofecoxib and valdecoxib from stored supplies even though both are no longer available in US

ETIOLOGY

- Nonsteroidal medications are available by prescription and over-the-counter.
- NSAIDs include:
 - Diclofenac
 - Diflunisal
 - Etodolac
 - Fenoprofen
 - Ibuprofen
 - Indomethacin
 - Ketoprofen
 - Ketorolac
 - Meclofenamate
 - Meloxicam
 - Nabumetone
 - Naproxen
 - Oxaprozin
 - Piroxicam
 - Sulindac
 - Tolmetin

DIAGNOSIS

SIGNS AND SYMPTOMS

- GI:
 - Nausea
 - Vomiting
 - Epigastric pain
- CNS:
 - Drowsiness
 - Dizziness
 - Lethargy
 - Aseptic meningitis
 - Seizures
- Cardiovascular:
 - Hypotension
 - Tachycardia
- Pulmonary:
 - Eosinophilic pneumonia
 - Apnea
 - Hyperventilation
- Renal:
 - Acute renal failure, hyperkalemia
 - Acute tubular necrosis
 - Acute interstitial nephritis
- Liver:
 - Hepatocellular injury
 - Cholestatic jaundice
- Metabolic:
 - Mild, short-lived metabolic acidosis
- Hypersensitivity:
 - Aseptic meningitis
 - Asthma exacerbation
 - Angioedema, urticaria

ESSENTIAL WORKUP

- Generally, NSAID ingestion results in mild toxicity.
- Exact identification of drug helpful:
 - Subtle toxicologic differences among the NSAIDs
 - Aseptic meningitis more common with ibuprofen exposure
 - Liver toxicity more common with diclofenac and sulindac exposure

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Electrolytes, BUN/creatinine, glucose:

- Baseline renal function
- Check for metabolic acidosis.
- CBC
- Arterial blood gas for large overdoses
- PT/PTT:
 - False-positive bilirubin/ketone dipstick with etodolac ingestion
- Acetaminophen and salicylate level—patients often confuse salicylate, acetaminophen, and NSAID products thinking they are all the same.
- NSAID difficult to detect on toxicology screens and is not beneficial in management

DIFFERENTIAL DIAGNOSIS

Agents causing metabolic acidosis, altered mental status, and GI irritation:

- Salicylates
- Isoniazid
- Ethylene glycol
- Methanol
- Isopropanol



TREATMENT

PRE HOSPITAL

Collect prescription bottles/medications for identification in the ED.

INITIAL STABILIZATION/THERAPY

- ABCs
- Naloxone, thiamine, dextrose (or Accu-Chek) for altered mental status

ED TREATMENT/PROCEDURES

- Supportive care
- Administer activated charcoal.
- Extracorporeal methods to enhance elimination are not beneficial due to high degree of plasma protein binding.

MEDICATION

- Activated charcoal slurry: 1–2 g/kg up to 90 g PO
- Dextrose: D50W 1 amp (50 mL or 25 g; peds: D25W 2–4 mL/kg) IV
- Naloxone (Narcan): 2 mg (peds: 0.1 mg/kg) IV or IM initial dose
- Thiamine (vitamin B₁): 100 mg (peds: 50 mg) IV or IM

Pediatric Considerations

Piroxicam, naproxen, ketoprofen, and mefenamic acid have caused seizures in children.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Protracted vomiting, hematemesis
- CNS depression, seizure activity
- Metabolic acidosis
- CHF, hypotension, hypertension
- Renal failure

Discharge Criteria

Nontoxic ingestion in a patient who is asymptomatic 6–8 hr after ingestion

FOLLOW-UP RECOMMENDATIONS

Psychiatry follow-up/referral for intentional ingestion.

PEARLS AND PITFALLS

- Investigate for coingestions for all NSAID overdoses.
- Obtain acetaminophen and salicylate level on all patients who present with suspected NSAID ingestion.
- NSAID poisoning is generally benign, except with massive overdoses; patients with underlying CHF, coronary artery disease may be at higher risk of toxicity

ADDITIONAL READING

- Dajani EZ, Islam K. Cardiovascular and gastrointestinal toxicity of selective cyclooxygenase-2 inhibitors in man. *J Physiol Pharmacol.* 2008;59(suppl 2):117–133.
- Frei MY, Nielsen S, Dobbin MD, et al. Serious morbidity associated with misuse of over-the-counter codeine-ibuprofen analgesics: A series of 27 cases. *Med J Aust.* 2010;193:294–296.
- Halen PK, Murumkar PR, Giridhar R, et al. Prodrug designing of NSAIDs. *Mini Rev Med Chem.* 2009;9:124–139.



CODES

ICD9

976.0 Poisoning by local anti-infectives and anti-inflammatory drugs

ICD10

- T39.391A Poisoning by other nonsteroidal anti-inflammatory drugs, accidental, init?
- T39.392A Poisoning by other nonsteroidal anti-inflammatory drugs, self-harm, init?
- T39.394A Poisoning by other nonsteroidal anti-inflammatory drugs, undet?, init?

NURSEMAID'S ELBOW

Neha P. Raukar • Daniel L. Savitt

BASICS

DESCRIPTION

The most common elbow injury in children <5 yr old.

ETIOLOGY

- Sudden traction of the distal radius leads to a portion of the annular ligament slipping over the radial head and becoming trapped between the radius and the capitellum. Traction can occur by swinging the child, wrestling, and lifting the child by the arms.
- By the time the child is 5 yr, the annular ligament is thick and strong and resists tearing and/or displacement.

DIAGNOSIS

SIGNS AND SYMPTOMS

- Child refuses to use arm.
- Elbow is slightly flexed, with forearm held close to the trunk.
- Pain with flexion of the elbow
- Pain with forearm supination or pronation
- Absence of point tenderness
- Minimal to no swelling

History

- Child not using affected arm
- 50% report the classic history of pulling the arm.
- Can also be due to a fall, minor trauma to the elbow, or twisting of the forearm
- In children <6 mo, can be due to the child rolling onto the arm.

Physical-Exam

- Affected arm is held close to the body.
- Arm is usually pronated.
- Elbow is either fully extended or slightly flexed.
- Child will not use the elbow.
- Can be mildly tender over anterolateral radial head, but the rest of the elbow is nontender.
- Painless passive range of motion
- Painful with supination

ESSENTIAL WORKUP

Clinical diagnosis:

- Classic history, passive position of arm, and physical exam are sufficient for diagnosis.

DIAGNOSIS TESTS & INTERPRETATION

Imaging

Radiographs:

- Not routinely indicated
- Obtain to exclude or diagnose other injuries if any of the following are present:
 - Point tenderness
 - Soft tissue swelling
 - Deformity
 - Ecchymosis of the elbow
 - Failed reduction
 - Child continues to favor extremity after reduction maneuver.

DIFFERENTIAL DIAGNOSIS

- Humerus, radius, or ulna fracture
- Elbow dislocation
- Joint infection
- Osteomyelitis
- Tumor



TREATMENT

PRE HOSPITAL

Cautions:

- Place ice on the injured elbow to reduce pain and swelling.
- Immobilize in a sling or splint to facilitate transport and prevent further injury.
- Assess distal neurovascular status.

INITIAL STABILIZATION/THERAPY

Assess distal motor, sensory, and vascular function.

ED TREATMENT/PROCEDURES

- 2 common reduction techniques:
 - Supination/flexion:
 - More commonly used
 - Hyperpronation/extension:
 - Nurses and caretakers perceive this method to be less painful
 - More successful

- Supination/flexion technique:
 - Grasp child’s hand in handshake position and apply mild axial traction.
 - Stabilize injured elbow with the other hand with the thumb over the radial head exerting moderate pressure.
 - In 1 smooth, swift motion, fully supinate the forearm and flex the elbow.
- Hyperpronation/extension technique:
 - Grasp child’s hand in handshake position and apply mild axial traction.
 - Stabilize injured elbow with the other hand with the thumb over the radial head exerting moderate pressure.
 - Hyperpronate the arm and extend if arm is not already extended.
- Placing the examiner’s thumb over the radial head may allow palpation of a click.
- Child may cry during the reduction, but is frequently pain free using the arm shortly thereafter. Period of immobility may be some what prolonged if reduction delayed
- Attempt reduction a 2nd time if the child does not use arm 15 min after 1st attempt.
- 1 of the attempts should be the hyperpronation method.
- Consider opposing technique for 2nd reduction attempt.
- Radiographic studies indicated if the 2nd reduction attempt is unsuccessful, evaluate for fractures.
- Perform postreduction neurovascular assessment.

MEDICATION

- Usually unnecessary
- Acetaminophen: 10–15 mg/kg PO q4h; do not exceed 5 doses/24 hr
- Ibuprofen 10 mg/kg PO q6–8h



FOLLOW-UP

DISPOSITION

Admission Criteria

None

Discharge Criteria

- Discharge after child regains full, unrestricted use of the arm.
- Patient instructions:
 - Inform parents not to pull or lift the child by the hand, wrist, or forearm.
 - Recurrence rate of 27–39% until the child reaches 5 yr of age.

Issues for Referral

Unsuccessful reduction:

- If radiologic evaluation is also negative, child should be referred to an orthopedist.
- Place arm in a sling or a posterior splint for outpatient follow-up.
- No long-term sequelae have been reported with short delay in reduction

FOLLOW-UP RECOMMENDATIONS

- None required for successful reduction
- Orthopedics within 24 hr for unsuccessful reduction

PEARLS AND PITFALLS

- Suspect nursemaid's elbow with a classic history.
- Radiographs are not necessary unless the elbow is focally tender or swollen or history does not suggest nursemaid's elbow.
- Reduction attempt should include the hyperpronation method.
- 2 unsuccessful reductions should prompt radiographic evaluation.
- Unsuccessful reductions should be referred to the orthopedist after the arm is placed in a sling or posterior splint.

ADDITIONAL READING

- Bachman D, Santora S. Orthopedic trauma. In: Fleisher GR, Ludwig S, Henretig FM, et al. eds. *Textbook of Pediatric Emergency Medicine*. 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2006:1525.
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- Rudloe TF, Schutzman S, Lee LK, et al. No longer a “nursemaid’s” elbow: Mechanisms, caregivers, and prevention. *Pediatr Emerg Care*. 2012;28(8):771–774.



CODES

ICD9

832.2 Nursemaid's elbow

ICD10

- S53.031A Nursemaid's elbow, right elbow, initial encounter
- S53.032A Nursemaid's elbow, left elbow, initial encounter
- S53.033A Nursemaid's elbow, unspecified elbow, initial encounter

OCULOMOTOR NERVE PALSY

Adam Z. Barkin

BASICS

DESCRIPTION

- Typical presentation of a 3rd cranial nerve (CN) palsy:
 - Eyelid drooping
 - Blurred or double vision
 - Light sensitivity
 - May also have other neurologic signs/symptoms:
 - Hemiplegia
 - Ataxia
 - Tremor
- CN III controls elevation, adduction and depression of the eye. This nerve also raises the lid and mediates pupillary constriction and lens accommodation:
 - Medial rectus:
 - Moves eye medially toward nose (adduction)
 - Superior rectus:
 - Moves eye upward
 - Rotates top of eye toward nose
 - Slight adduction
 - Inferior rectus:
 - Moves eye inferiorly
 - Rotates top of eye away from nose
 - Slight adduction
 - Inferior oblique:
 - Rotates top of eye away from nose
 - Slight elevation and abduction
 - Levator palpebrae superioris:
 - Raises eyelid
- CN IV innervates the superior oblique:
 - Moves eye down when looking medially
 - Rotates eye internally
- CN VI innervates the lateral rectus:
 - Moves eye laterally (abduction)
 - Lesions categorized as:
 - Complete vs. incomplete
 - Pupil involving vs. pupil sparing
- Complete: Total loss of CN III function (“down and out”):
 - Compressive lesions:

- Aneurysms
- Tumors
- Brainstem herniation with compression
- Increased intracranial pressure
- Incomplete: Partial loss of CN III function:
 - Vascular infarction of vasa vasorum
- Pupil involving:
 - 95–97% of compressive lesions (aneurysm, tumor, etc.) involve the pupil
 - Parasympathetic fibers sit peripherally in CN III
- Pupil sparing:
 - Ischemic injury to nerve
 - Diabetics, uncontrolled hypertension

ETIOLOGY

- Intracranial or orbital tumor
- Aneurysm (particularly posterior communicating artery)
- Trauma
- Intracranial hemorrhage
- Diabetes mellitus
- Migraine headache
- Infection, meningitis
- Arteriovenous malformation or fistula
- Cavernous sinus thrombosis
- Neuropathy (e.g., myasthenia gravis, Guillain–Barré)
- Collagen vascular diseases (e.g., sarcoidosis)
- Idiopathic

Pediatric Considerations

Trauma is the most common cause of acquired oculomotor nerve palsies

DIAGNOSIS

SIGNS AND SYMPTOMS

A careful history and physical exam are vital to narrow down the differential diagnosis

History

History is of utmost importance in determining cause:

- Headache
- Pupillary dilation
- Eye pain
- Diplopia
- Blurry vision

- History of long-standing diabetes mellitus
- Head trauma, either recent or distant
- Unintentional weight loss
- Signs and symptoms of infection
- Sudden onset of severe headache, meningeal signs, photophobia
- Proptosis
- Lid swelling

Physical-Exam

- Ophthalmologic exam:
 - _ Extraocular movements
 - _ Fundoscopic exam for papilledema
 - _ Ipsilateral and contralateral pupillary reaction
 - _ Ptosis
 - _ Diplopia
 - _ Chemosis or conjunctival injection
 - _ Tenderness
 - _ Visual acuity
 - _ Exophthalmos
- Pupil sparing lesion:
 - _ Ptosis
 - _ Globe is “down and out”
 - _ No elevation, depression, or adduction
 - _ Normal pupil exam
 - _ CN IV, V, VI intact
 - _ Usually no other neurologic signs/symptoms
 - _ Most commonly caused by ischemia in adults
 - _ Also consider giant cell arteritis and trauma
- Pupil-involving lesion:
 - _ Anisocoria is present with a dilated pupil on affected side
 - _ Need to rule out compressive aneurysm
- Incomplete, 3rd CN palsy:
 - _ May have involvement of 1 or more extraocular muscle and may or may not involve pupil
- Look for associated symptoms:
 - _ Extremity weakness
 - _ Changes in speech
 - _ Dysfunction of other CNs
 - _ Gait or coordination

ESSENTIAL WORKUP

CT/MRI of brain, orbit, sinuses

DIAGNOSIS TESTS & INTERPRETATION

Lab

When indicated based on history and physical exam:

- CBC with differential
- ESR
- Antinuclear antibodies, rheumatoid factor to evaluate for vasculitis
- Lumbar puncture

Imaging

- MRI/MRA of brain and cerebral vessels particularly when pupil is involved
- CT angiogram
- Cerebral arteriogram: Has associated risk of neurologic morbidity and mortality
- Doppler imaging for arteriovenous malformations, dural sinus thrombosis

Diagnostic Procedures/Surgery

- Intraocular pressure to exclude glaucoma
- Slit-lamp exam:
 - _ Observe structural abnormalities of iris or anterior chamber

DIFFERENTIAL DIAGNOSIS

- Intracranial infections
- Malignancy
- Vasculitis
- Aneurysms
- Myasthenia gravis
- Botulism
- Orbital infections
- Trauma
- Lens pathology
- Retinal pathology
- Glaucoma
- MS

Pediatric Considerations

Consider congenital oculomotor nerve palsy



TREATMENT

PRE HOSPITAL

Without associated trauma, no specific pre-hospital care issues exist

INITIAL STABILIZATION/THERAPY

- Initial stabilization of trauma patient should concentrate on underlying injuries
- Any patient with evidence of herniation should have the following measures to control intracranial pressure:
 - Intubation using rapid-sequence induction and controlled ventilation to a PCO₂ level of 35–40 mm Hg
 - Elevate head of bed 30°
 - Mannitol

ED TREATMENT/PROCEDURES

- Differentiation between incomplete and complete oculomotor or pupil-involving vs. pupil-sparing nerve palsy guides focus of ED treatment
- All patients younger than 50 yr with any extent of 3rd nerve palsy should be evaluated for a compressive lesion
- If pupil is involved, neuroimaging is indicated as well as consultation to determine cause
- If pupil is spared and the patient has diabetes or other risk for an ischemic 3rd nerve, discharge is likely reasonable with outpatient follow-up:
 - If partial sparing or patient does not have these risk factors, consultation and neuroimaging is indicated
- Medication regimen determined by cause:
 - Aneurysm:
 - Control severe HTN.
 - Decrease intracranial pressure
 - Controlled ventilation
 - Elevation of head
 - Mannitol
 - Intracranial tumor: Control increasing intracranial pressure
 - Inflammation and edema: Decrease with IV steroids.
 - Meningitis:
 - Rapid administration of IV antibiotics
 - IV steroids may be useful to decrease inflammatory response and edema
 - Vasculitis and collagen vascular diseases: Decrease inflammatory cell infiltration with IV steroids
 - Neuropathy: Myasthenia gravis—edrophonium chloride test
- Neurosurgical consultation as appropriate

Pediatric Considerations

MRI/MRA is indicated for all children with a 3rd nerve palsy

MEDICATION

- Ceftriaxone: 1–2 g (peds: 50–100 mg/kg) IV

- Dexamethasone: 10 mg IV (peds: 0.15–0.5 mg/kg IV single dose in ED)
- Edrophonium chloride: 5–8 mg IV (peds: 0.15 mg/kg IV; 1/10 test dose given 1st)
- Mannitol: 1 g/kg IV (peds: Not routinely recommended)
- Methylprednisolone: Adults/peds: 1–2 mg/kg IV single dose in ED



FOLLOW-UP

DISPOSITION

Admission Criteria

- Complete oculomotor nerve palsy of any cause requires admission and emergency neurosurgical evaluation
- Incomplete oculomotor nerve palsy with abnormal CT or MRI, abnormal lab studies, or other focal neurologic or constitutional symptoms should receive prompt neurologic consultation and imaging

Discharge Criteria

- Incomplete oculomotor nerve palsy with negative CT or MRI, normal lab studies, and no other symptoms can be referred for urgent outpatient neurologic evaluation
- Complete pupil-sparing oculomotor palsy in patients with risk factors for microvascular disease (i.e., diabetic) can receive outpatient neurologic workup

FOLLOW-UP RECOMMENDATIONS

If the patient is being discharged, prompt neurologic follow-up is required

PEARLS AND PITFALLS

- Complete lesions must be assessed rapidly
- Patients < 50 yr old with any extent of CN III palsy should be evaluated for compressive lesions
- If the pupil is involved, compressive lesions are often the cause and immediate MRI/MRA is indicated

ADDITIONAL READING

- Bruce BB, Biousse V, Newman NJ. Third nerve palsies. *Semin Neurol.* 2007;27:257–268.
- Chen CC, Pai YM, Wang RF, et al. Isolated oculomotor nerve palsy from minor head trauma. *Br J Sports Med.* 2005;39:e34.
- Woodruff MM, Edlow JA. Evaluation of third nerve palsy in the emergency department. *J Emerg Med.* 2008;35:239–246.
- Yanovitch T, Buckley E. Diagnosis and management of third nerve palsy. *Curr Opin*



CODES

ICD9

- 378.51 Third or oculomotor nerve palsy, partial
- 378.52 Third or oculomotor nerve palsy, total

ICD10

- H49.00 Third [oculomotor] nerve palsy, unspecified eye
- H49.01 Third [oculomotor] nerve palsy, right eye
- H49.02 Third [oculomotor] nerve palsy, left eye

OPIATE POISONING

Amy V. Kontrick • Mark B. Mycyk

BASICS

DESCRIPTION

- Bind to μ , κ , and δ opiate receptors in the CNS and peripheral nervous system (PNS)
- Physical and psychological dependence occurs.
- Peak plasma levels:
 - PO: 1–2 hr
 - Intramuscular: 0.5–1 hr
 - Intravenous or intranasal: Seconds to minutes

ETIOLOGY

- Overuse or abuse of oral prescription analgesics for moderate to severe pain
- Street preparations of opiate analogs may contain adulterants:
 - Cocaine
 - Clenbuterol
 - Phencyclidine
 - Strychnine
 - Dextromethorphan
 - Quinine
 - Scopolamine

DIAGNOSIS

SIGNS AND SYMPTOMS

- CNS:
 - CNS depression
 - Coma
 - Seizures
- GI:
 - Nausea
 - Vomiting
 - Constipation
- Cardiovascular:
 - Hypotension
 - Bradycardia
 - Palpitations
- Pulmonary:

- Respiratory depression
- Bronchospasm
- Pulmonary edema
- Apnea
- Other:
 - Miosis
 - Hypothermia
- Withdrawal:
 - HTN
 - Tachycardia
 - Tachypnea
 - Abdominal cramps
 - Diarrhea
 - Piloerection
 - Yawning

Pediatric Considerations

- Neonatal withdrawal:
 - Infants born to addicted mothers
 - Onset: 12–72 hr after birth
 - Irritability, tremors, poor feeding, and dehydration
- Diphenoxylate (Lomotil): Toxicity more severe in children than adults and may be fatal

ESSENTIAL WORKUP

Monitor vital signs and pulmonary status with significant exposure:

- Pulse oximetry or arterial blood gases
- CXR if persistent hypoxia or possible aspiration
- Abdominal radiograph if body packing suspected
- Perform a complete exam for occult sticky patches (e.g., fentanyl).

DIAGNOSIS TESTS & NTERPRETATION

Lab

- Plasma opiate levels not clinically useful:
 - Treatment based on clinical presentation, not opiate level
- Urine toxicity screen for opiates may not identify some synthetic opioids (e.g., methadone).
- Acetaminophen level for overuse or abuse of oral prescription analgesic products

DIFFERENTIAL DIAGNOSIS

- Clonidine overdose
- Barbiturate overdose

- Benzodiazepine overdose
- γ -hydroxybutyrate (GHB) overdose
- Neuroleptic overdose
- Occult head injury



TREATMENT

PRE HOSPITAL

- Transport all pills/pill bottles involved in overdose for identification in ED.
- Provide respiratory support.
- Administer naloxone.

INITIAL STABILIZATION/THERAPY

- Check ABCs:
 - _ Airway control is essential.
 - _ Administer supplemental oxygen.
- Administer naloxone:
 - _ Reverses respiratory depression and coma in opiate overdoses
 - _ Intubate if naloxone does not reverse respiratory depression.

ED TREATMENT/PROCEDURES

- Naloxone administration:
 - _ Start with low doses for opiate-habituated patients.
 - _ High doses (10 mg) may be required to reverse the effects of propoxyphene, methadone, and fentanyl.
 - _ Administer repeated doses that reversed symptoms, as needed every 20–60 min.
 - _ For long-acting opioids, consider an hourly infusion of 2/3 of the dose needed to reverse symptoms.
- Decontamination:
 - _ Administer activated charcoal for oral ingestion.
 - _ Administer whole-bowel irrigation with polyethylene glycol for asymptomatic body packers.
- Treat opiate withdrawal with clonidine or methadone.
- Hypotension:
 - _ 0.9% normal saline IV fluid bolus
 - _ Trendelenburg test
 - _ Initiate dopamine for resistant hypotension.
- Seizures:
 - _ Treat initially with diazepam.
 - _ Administer phenobarbital for persistent seizures.

MEDICATION

- Activated charcoal: 1–2 g/kg PO
- Clonidine: 0.1–0.3 mg PO BID for 10 days; 0.1–0.2 mg/kg/d transdermal patch
- Diazepam: 5–10 mg IV (peds: 0.2–0.5 mg/kg IV) q10–15min
- Dopamine: 2–20 µ/kg/min; titrate to effect.
- Methadone: 15–40 mg/d
- Naloxone: 0.4–2 mg (peds: 0.1 mg/kg; neonate dose same as peds except if suspect neonatal withdrawal use 0.001 mg/kg IV) IV, IM, or nebulized
- Phenobarbital: 10–20 mg/kg IV (loading dose); monitor for respiratory depression
- Polyethylene glycol: 2 L/h until clear rectal effluent and/or passage of packets

ALERT

Opioid patches can be abused in various ways (transdermally, orally, smoked, injected). Even used patches still contain a significant dose of drug.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Symptomatic after oral overdose
- Repeated naloxone dosing or infusion needed to reverse symptoms
- Children <5 yr after diphenoxylate ingestion should be observed for 24 hr.
- Opiate body packers
- Persistent symptoms from concomitant toxin exposure (e.g., clenbuterol)

Discharge Criteria

- Asymptomatic 6 hr after oral overdose
- Asymptomatic 4 hr after naloxone administration
- Complete elimination of opiate packets

FOLLOW-UP RECOMMENDATIONS

- Substance abuse referral for patients with oral opiate abuse.
- Patients with unintentional (accidental) poisoning require poison prevention counseling.
- Patients with intentional (e.g., suicide) poisoning require psychiatric evaluation.

PEARLS AND PITFALLS

- Consider occult acetaminophen poisoning in chronic oral opioid-abusing patients.
- Buprenorphine may cause prolonged sedation in pediatric patients.
- Semisynthetic and synthetic opioids will not provide a positive opiate hospital drug screen result.

ADDITIONAL READING

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CODES

ICD9

- 965.00 Poisoning by opium (alkaloids), unspecified
- 965.01 Poisoning by heroin
- 965.02 Poisoning by methadone

ICD10

- T40.1X1A Poisoning by heroin, accidental (unintentional), init encntr
- T40.3X1A Poisoning by methadone, accidental (unintentional), init
- T40.601A Poisoning by unsp narcotics, accidental, init

OPPORTUNISTIC INFECTIONS

Sandra E. Sicular • Colleen M. Rivers

BASICS

DESCRIPTION

Unusual infections that occur when host suffers a decrease in resistance against normally nonpathogenic organisms

ETIOLOGY

- Occurs in HIV patients when the CD4 T-lymphocyte count falls below 200 cells/mm³ or <14% of the total lymphocyte count:
 - *Pneumocystis jiroveci* pneumonia (PCP)
 - Disseminated tuberculosis
 - Cryptosporidiosis
 - Microsporidiosis
 - Isosporiasis
 - Toxoplasmosis
 - Histoplasmosis
 - Cryptococcosis
 - *Mycobacterium avium* complex
 - Tuberculosis pericarditis or meningitis
 - Cytomegalovirus
 - Human herpesvirus-8 (Kaposi sarcoma)
 - JC virus (progressive multifocal leukoencephalopathy)
 - Hepatitis B virus
 - *Penicilliosis marneffeii*
 - Bacterial species
- Cell-mediated deficiency:
 - Hematologic malignancies
 - Lymphoma
 - High-dose glucocorticoid therapy
 - Autoimmune disorders
 - Viral infections
 - Cytotoxic drugs/chemotherapy
 - Radiation therapy
 - Associated with:
 - *Legionella*
 - *Nocardia*
 - *Salmonella*
 - *Mycobacteria*

- Neutrophil impairment/depletion:
 - Cytotoxic drugs
 - Aplastic anemia
 - Drug reactions:
 - Dapsone
 - Neoplastic invasion of bone marrow
 - Arsenic
 - Penicillin
 - Chloramphenicol
 - Procainamide
 - Vitamin deficiencies
 - Associated with:
 - *Staphylococcus* and α -hemolytic *Streptococcus*
 - Enteric organisms and anaerobes
 - Invasive aspergillosis

DIAGNOSIS

SIGNS AND SYMPTOMS

- New or worsening fatigue
- Tachypnea
- Fever
- Chills
- Night sweats
- Pulmonary source of infection:
 - Cough
 - Congestion
 - Rales
- Genitourinary source of infection:
 - Dysuria
 - Increased frequency
 - Urinary retention
- GI source of infection:
 - Abdominal pain
 - Vomiting
 - Diarrhea
 - Bleeding
 - Jaundice
- CNS sources of infection:
 - Confusion
 - Focal neurologic deficits
 - Headache

- Seizure

History

- History for HIV/AIDS (recent CD4 count)
- History of malignancy with active treatment
- History of organ transplant
- History of autoimmune disorder
- Use of cytotoxic drugs
- Use of high-dose glucocorticoid therapy

Physical-Exam

- Complete, detailed physical exam indicated as signs of infection in the immunocompromised patient may be subtle.
- Signs of systemic inflammatory response syndrome:
 - Temperature $> 38^{\circ}\text{C}$ or $< 36^{\circ}\text{C}$
 - Heart rate > 90 bpm
 - Respiratory rate > 20 breaths per minute or $\text{PCO}_2 < 32$ mm Hg
- Septic shock
- Focal neurologic deficits
- New murmur
- Ambulatory hypoxia in PCP pneumonia
- Rales and/or rhonchi in pneumonia
- Skin/mucosa defects as a portal of entry.
- Oropharyngeal candidiasis as an indicator of immune suppression

ESSENTIAL WORKUP

Full workup indicated owing to impaired immunity:

- Signs of infection in the immunocompromised patient may not be present
- Can present with subtle signs with rapid deterioration
- Signs such as fever must lead to a full evaluation of patient
- Thorough physical exam is critical to search for source of infection

DIAGNOSIS TESTS & NTERPRETATION

Lab

- CBC with differential for neutropenia or leukocytosis:
 - WBC $> 12,000$ or $< 4,000$ are criteria for the systemic inflammatory response score
 - Neutropenia:
 - Absolute neutrophil count (ANC) $< 1,500/\mu\text{L}$
 - $\text{ANC} = \text{WBC (cells}/\mu\text{L)} \times \text{percent (PMNS + bands)}/100$
- Cultures (aerobic, anaerobic, fungal, viral as indicated):

- Urine
- Blood
- Wound
- Fecal
- CD4 count:
 - Absolute lymphocyte count (ALC) < 1,000/μL predicts CD4 < 200 if CD4 unknown
 - $ALC = WBC \text{ (cells/}\mu\text{L)} \times \text{percent lymphocytes/100}$
- Urinalysis for presence of WBC, nitrite, leukocyte esterase
- Electrolytes, BUN/creatinine, glucose; anion gap acidosis suggests severe infection
- VBG for acidosis
- Lactate level; elevated value suggests serious infection
- PT/PTT for evidence of disseminated intravascular coagulation
- Lactate dehydrogenase (LDH); elevated in patients with PCP

Imaging

- CXR:
 - Nonspecific for predicting a particular infectious etiology
 - Pneumonia:
 - Segmental or subsegmental infiltrate
 - Air bronchograms
 - Abscess
 - Cavitation
 - Empyema
 - Pleural effusion
 - PCP:
 - Classically reveals bilateral interstitial or central alveolar infiltrates
 - Radiograph normal in up to 25% of patients
- High-resolution chest CT:
 - Early studies show high sensitivity for PCP in HIV-positive patients
 - Reveals patchy ground-glass attenuation
 - Head CT: Contrast-enhancing lesions in *Toxoplasma gondii* encephalitis
- Abdominal and pelvic CT with contrast:
 - Indicated if a GI source of infection is suggested by the clinical exam

Diagnostic Procedures/Surgery

Lumbar puncture:

- CSF analysis if signs of CNS infection
- Diagnostic paracentesis:
- Immunocompromised liver patients for SBP



TREATMENT

INITIAL STABILIZATION/THERAPY

- Check airway, breathing, and circulation
- Initiate 0.9% normal saline IV 500 mL bolus for hypotension
- Oxygen
- Cardiac monitor for unstable vital signs
- Early initiation of antibiotic therapy

ED TREATMENT/PROCEDURES

- Strict isolation
- Antibiotics: Combination of expanded-spectrum penicillin (mezlocillin, ticarcillin, piperacillin) and aminoglycoside (amikacin, tobramycin):
 - Monotherapy with a 3rd-generation cephalosporin (ceftazidime, cefepime), fluoroquinolones (levofloxacin, gatifloxacin), or other broad-spectrum antimicrobials (imipenem/cilastatin) may be considered if aminoglycosides contraindicated
 - Vancomycin if there is a high prevalence of methicillin-resistant organisms in the area
 - Antifungals (amphotericin B, fluconazole) if patient is on adequate antibiotics for 1 wk
 - Trimethoprim/sulfamethoxazole for suspected PCP (alternatives: Pentamidine, clindamycin + primaquine)
- Steroids: Prednisone in PCP with hypoxemia

MEDICATION

- Amphotericin B: 0.25 mg/kg/d IV
- Cefepime: 1–2 q12h IV
- Ceftazidime:
 - Adults: 1–2 g IV q8–12h
 - Pediatric: 100–150 mg/kg/24h IV q8–12h
- Fluconazole: 400 mg 1st dose, then 200–400 mg/d IV (peds: 6–12 mg/kg/24h IV q12h)
- Gatifloxacin: 400 mg/d IV
- Imipenem/cilastatin: 500–1,000 mg IV q6–8h, max. 50 mg/kg/d or 4,000 mg/d
- Levofloxacin: 500 mg/d IV
- Vancomycin: 1–2 g IV q12h (peds: 10–50 mg/kg/24h IV q6h)
- Trimethoprim: 15–20 mg/kg + sulfamethoxazole: 75 mg/kg PO or IV div. q8h
- Prednisone: 40 mg PO BID × 5 days, then 40 mg PO QD × 5 days, then 20 mg PO QD × 11 days

*start within 72 hr of antimicrobials for PCP



FOLLOW-UP

DISPOSITION

Admission Criteria

Suspected or confirmed systemic infection

Discharge Criteria

Systemic infection excluded

Issues for Referral

Consider infectious disease consultation

FOLLOW-UP RECOMMENDATIONS

Patients with systemic opportunistic infections should be admitted to the hospital

PEARLS AND PITFALLS

- Signs of infection in the immunocompromised patient may not be present
- Can present with subtle signs with rapid deterioration

ADDITIONAL READING

- Fishman JA. Infection in solid-organ transplant recipients. *N Engl J Med*. 2007;357(25):2601–2614.
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- Tan IL, Smith BR, von Geldern G, et al. HIV-associated opportunistic infections of the CNS. *Lancet Neurol*. 2012;11(7):605–617.



CODES

ICD9

- 018.90 Miliary tuberculosis, unspecified, unspecified
- 136.3 Pneumocystosis
- 136.9 Unspecified infectious and parasitic diseases

ICD10

- A19.9 Miliary tuberculosis, unspecified
- B59 Pneumocystosis
- B99.9 Unspecified infectious disease

OPTIC NEURITIS

Douglas W. Lowery-North

BASICS

DESCRIPTION

- Optic nerve dysfunction due to an inflammatory process, commonly associated with myelin destruction
- Highly associated with multiple sclerosis (MS); presenting feature in 15–20% of MS patients
- Grouped by site of inflammation:
 - Papillitis: Inflammation of the optic disk
 - Retrobulbar neuritis: Inflammation of the optic nerve proximal to the globe
- 5 yr risk for clinically definite MS following optic neuritis:
 - Normal MRI—16%
 - > 3 lesions on MRI—51%
- Recurrence is seen in 35% of patients.

RISK FACTORS

Genetics

High prevalence of A23, B7, and DR2 HLA alleles in patients with optic neuritis:

- Especially those that progress to clinically definite MS

ETIOLOGY

- Idiopathic:
 - Most common
 - Single isolated events
- MS:
 - 20–50% of patients with optic neuritis
- Viral infections:
 - Chicken pox
 - Measles
 - Mononucleosis
 - HSV and HZV
 - Encephalitis
- Postviral optic neuritis:
 - Usually occurs 4–6 wk after a nonspecific viral illness
- Granulomatous inflammation:
 - TB
 - Syphilis

- Sarcoidosis
- Cryptococcal infection
- SLE
- HIV:
 - Cytomegalovirus
 - Toxoplasmosis
 - Histoplasmosis
 - Cryptococcus
- Lyme disease
- Contiguous inflammation of meninges, orbit, sinuses, and intraocular inflammation
- Drug induced:
 - Amiodarone
 - Ethambutol
 - Tamoxifen



DIAGNOSIS

SIGNS AND SYMPTOMS

- Vision loss and pain most common symptoms
- Visual loss occurring over days (rarely over hours), peaks in 1–2 wk:
 - Adults usually unilateral (70%)
 - Bilateral visual loss more common in children
- Retrobulbar pain: Increased with movement of the affected eye
- Light, color vision, and depth perception loss more pronounced than visual acuity loss
- Afferent pupillary defect almost always occurs in unilateral cases if other eye is healthy.
- Visual field defects:
 - Usually characterized by central scotoma
 - Deficits resolve by 1 yr in 56% of patients, and 73% resolve by 10 yr
- Funduscopic exam usually reveals either swollen (papillitis) or normal disk
- Uhthoff sign:
 - Visual deficit occurring with exercise or increased body temperature
 - Unusual sign seen occasionally

History

- Age (typically women 18–45 yr)
- Pain on eye movement
- Speed of onset of symptoms
- Associated symptoms
- Previous episodes

- Family history of optic neuritis, MS

Physical-Exam

- Check BP.
- Complete ophthalmologic and neurologic exam, especially assessment of:
 - Pupillary function
 - Afferent pupillary defect
 - Visual field defect
 - Color vision (Ishihara color plates)
 - Evaluation of the vitreous body for cells
 - Dilated retinal exam (swollen optic disk)

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC
- ESR
- Rapid plasma reagin, fluorescent treponemal antibody-absorption (FTA-ABS)
- Lyme titer
- Antinuclear antibody
- Purified protein derivative
- HIV

Imaging

- CXR for TB, sarcoid
- CT scan or MRI of brain and orbits:
 - Inflammation of the retrobulbar optic nerve during the acute phase may appear as enlargement, thus falsely raising the issue of an optic nerve mass.
 - Optic nerve inflammation is seen in 95% of gadolinium-enhanced MRIs.
 - Visual field testing (preferably automated testing, such as Octopus or Humphrey)

DIFFERENTIAL DIAGNOSIS

- Acute papilledema
- Ischemic optic neuropathy
- Severe systemic hypertension
- Intracranial tumor compressing the afferent visual pathway
- Orbital mass compressing the optic nerve
- Toxic or metabolic neuropathy:
 - Heavy metal poisoning
 - Anemia
 - Malnutrition
 - Ethanol/Methanol

- Chloroquine
- Ethambutol
- Isoniazid
- Leber hereditary optic atrophy

Pediatric Considerations

In children, infectious and postinfectious causes should be considered.

Geriatric Considerations

In patients >50 yr, ischemic optic neuropathies (e.g., diabetes and giant cell arteritis) are more common, and appropriate workup should be obtained.



TREATMENT

ED TREATMENT/PROCEDURES

- Early ophthalmologic and neurologic consultations
- IV steroid pulse followed by oral steroids:
 - Recommended for those with ≥ 2 demyelinating lesions on MRI without a prior history of MS or optic neuritis, or severe vision loss
 - Decreases recurrence and progression to MS over 2 yr and shortens duration of visual impairment, but does not affect visual outcome at 1 yr nor rate of progression at 5 yr
 - Treatment should be individualized for those with 1 lesion on MRI.
 - Oral steroids used alone increases recurrence and should be avoided.

MEDICATION

Methylprednisolone: 250 mg IV q6h for 3 days, followed by oral prednisone (1 mg/kg/d) for 11 days with subsequent 4 day taper



FOLLOW-UP

DISPOSITION

Admission Criteria

- Bilateral vision loss
- If other sources of acute vision loss cannot be ruled out
- IV steroid pulse treatment needed

Discharge Criteria

- Unilateral visual impairment
- Good home support systems

- Neurology and ophthalmology follow-up arranged

Issues for Referral

Referral for interferon β -1a treatment as outpatient for high-risk patients (those with ≥ 2 demyelinating lesions on MRI):

- Reduces progression to MS

FOLLOW-UP RECOMMENDATIONS

Needs Ophthalmology referral

PEARLS AND PITFALLS

- Rule out space-occupying lesions before making the diagnosis of optic neuritis.
- Acute bilateral loss with a severe headache or diplopia should raise concern for pituitary apoplexy.
- The true benefit of corticosteroids in the treatment of optic neuritis is unclear, and emergency physicians should consult with appropriate specialists to determine the local standard of practice.
- Brain MRI is the most useful predictor of subsequent development of MS.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Visual Loss

The author gratefully acknowledges Vinh D. Ngo's contribution for the previous edition of this chapter.



CODES

ICD9

- 377.30 Optic neuritis, unspecified
- 377.31 Optic papillitis
- 377.32 Retrobulbar neuritis (acute)

ICD10

- H46.00 Optic papillitis, unspecified eye
- H46.9 Unspecified optic neuritis
- H46.10 Retrobulbar neuritis, unspecified eye

ORGANOPHOSPHATE POISONING

Vinodinee L. Dissanayake

BASICS

DESCRIPTION

- Organophosphates (pesticides and nerve agents) irreversibly bind and deactivate cholinesterases, including acetylcholinesterase
- Acetylcholine accumulates at neural synapses, causing central and peripheral cholinergic overdrive
- Predominant effects (muscarinic, nicotinic, CNS) may vary and can overlap.
- Mortality is secondary to respiratory failure:
 - _ Weakness of respiratory muscles
 - _ Bronchorrhea and bronchoconstriction
 - _ Central depression of respiratory drive

Pediatric Considerations

- Symptoms are difficult to differentiate in toddlers
- Common symptoms: Miosis, salivation, and muscle weakness
- Seizure activity in 25% of pediatric cases:
 - _ Only 3% in adults

ETIOLOGY

- Exposure to insecticides (organophosphorus compounds)
- Exposure to chemical nerve agents (sarin, soman, tabun, VX)
- Extremely well absorbed from lung, GI tract, skin, mucosa, eyes

DIAGNOSIS

SIGNS AND SYMPTOMS

- Classic presentation: Cholinergic toxidrome:
 - _ DUMBELS:
 - *Diarrhea/diaphoresis*
 - *Urination*
 - *Miosis/muscle fasciculations*
 - *Bradycardia, bronchorrhea, bronchospasm*
 - *Emesis*
 - *Lacrimation*
 - *Salivation*
 - _ May have garlic odor
- Chronic intermittent exposure, nonspecific symptoms:

- Weakness
- Fatigue
- Malaise
- Anorexia
- Mild exposure:
 - CNS:
 - Headache
 - Dizziness
 - Tremors of tongue and eyelids
 - Weakness
 - GI:
 - Anorexia
- Moderate exposure:
 - CNS:
 - Muscle fasciculation then flaccid paralysis
 - Respiratory muscle weakness
 - Incoordination and ataxia
 - Agitation
 - Tremors
 - Confusion
 - Visual:
 - Pinpoint nonreactive pupils
 - Respiratory:
 - Respiratory muscle weakness
 - Bronchorrhea
 - Cardiovascular:
 - Bradycardia
 - GI:
 - Nausea/vomiting
 - Abdominal cramps
 - Exocrine glands:
 - Salivation
 - Lacrimation
- Severe exposure:
 - CNS:
 - Convulsions
 - Coma
 - Centrally mediated respiratory depression
 - Respiratory:
 - Bronchoconstriction
 - Wheezing
 - Dyspnea
 - Increased bronchial secretions

- Cardiovascular:
 - Bradycardia (tachycardia may follow pulmonary edema and hypoxia)
 - Heart block
 - Cyanosis
- GI:
 - Nausea, vomiting
 - Abdominal pain
 - Diarrhea, fecal incontinence
- Exocrine glands:
 - Diaphoresis
 - Salivation
 - Lacrimation
- Bladder:
 - Frequency
 - Urinary incontinence
 - Nicotinic manifestations

ESSENTIAL WORKUP

Inquire about possible exposure, occupation, recent insecticide at home, mislabeled, or poorly stored insecticides:

- Obtain original container if suicide attempt.
- Look for parasympathetic and CNS signs with muscle weakness or paralysis.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- RBC and plasma cholinesterase levels to confirm diagnosis:
 - RBC (true) cholinesterase level is best for synaptic inhibition (a send-out lab).
 - Plasma (pseudo)cholinesterase level not as reliable but more timely:
 - These are markers for poisoning
 - Depending on the agent and the patient, these levels may vary
 - Cholinesterase levels:
 - Latent exposure: > 50% of normal value
 - Mild exposure: 20–50% of normal value
 - Moderate exposure: 10–20% of normal value
 - Severe exposure: < 10% of normal value
 - Do not wait for cholinesterase results before administering treatment.
- CBC, electrolytes, glucose, BUN, creatinine
- ABG when respiratory symptoms are present

Imaging

- CXR if respiratory difficulty is present or suspect pulmonary edema:

- Pneumonitis from hydrocarbon aspiration
- ECG:
 - Dysrhythmias (atrial fibrillation, ventricular tachycardia, torsades de pointes, QT prolongation)
 - Bradycardia
 - Heart block
 - ST–T-wave abnormalities
- CT scan of head for altered mental status when diagnosis is uncertain

DIFFERENTIAL DIAGNOSIS

- Mild to moderate exposure:
 - Gastroenteritis
 - Asthma
 - Venomous arthropod bite (black widow, scorpion)
 - Progressive peripheral neuropathy (Guillain–Barré syndrome)
 - Carbon monoxide
- Severe exposure:
 - Narcotic overdose
 - Coma and miosis:
 - PCP, meprobamate, phenothiazine, clonidine
 - Muscarinic-containing mushrooms—cholinergic crisis without nicotinic symptoms
 - Nicotine poisoning
 - Metabolic and infectious:
 - Ketoacidosis, sepsis, meningitis, encephalitis
 - Hypoglycemia
 - Reye syndrome
 - Neurologic:
 - Cerebrovascular accident
 - Subdural or epidural hematoma
 - Postictal state



TREATMENT

PRE HOSPITAL

- Decontamination is initial priority:
 - Decontaminate, airway, breathing, circulation (DABC)
 - Remove all clothes and store as toxic waste (double bagged)
- Protection of health care workers of utmost importance:
 - Impenetrable gloves (neoprene, nitrile), gowns, eye protection
- Decontaminate skin with soap and water:
 - Shower or gentle scrubbing ideal if done before entrance into the ED

- Maintain airway and oxygenate.
- IV access and place on cardiac monitor

INITIAL STABILIZATION/THERAPY

- Decontaminate ABCs:
 - _ Decontamination and protection of staff
 - _ Maintain airway and oxygenate.
 - _ For unstable airway, intubate, and ventilate.
 - _ IV access with D₅W 0.9% NS
- Altered mental status: Administer thiamine, glucose, and naloxone (Narcan)

ED TREATMENT/PROCEDURES

- Atropine:
 - _ Blocks acetylcholine at muscarinic receptor sites.
 - _ No effect on nicotinic receptors
 - _ Onset of action is 1–4 min, peaks at 8 min.
 - _ Goal of therapy/end point:
 - Drying secretions of tracheobronchial tree
 - _ Administer test dose 1–2 mg IV/IM:
 - No clinical response: Double dose q5min until muscarinic findings subside
 - _ Dose: 1–4 mg IV q5min (peds: 0.05–0.2 mg/kg)
 - _ Common pitfalls in therapy:
 - Not giving enough atropine
 - Using pupillary findings (mydriasis) as end point of treatment
 - Mistaking dilated pupils or tachycardia as contraindications to atropine
- Pralidoxime (2-PAM):
 - _ Regenerates cholinesterase by reversing the phosphorylation of the enzyme.
 - _ Synergistic with atropine—muscarinic signs/symptoms will start to resolve in 10–40 min.
 - _ Side effects: Neuromuscular blockade with rapid infusion, respiratory arrest, HTN, nausea/vomiting, dizziness, blurred vision.
 - _ End point is resolution of muscle weakness and fasciculations.
 - _ Effective before enzyme aging occurs (permanent inactivation of cholinesterase)
 - _ Onset of aging varies among products
 - _ No restriction to its use even if 24–48 hr have passed
- Supportive care:
 - _ Dermal decontamination: Remove clothes and flush skin with water
 - _ Gastric lavage (early presentation of severe ingestion):
 - Gastric emptying with continuous suction via a nasogastric tube.
 - Handle contents with care—avoid direct contact to prevent personal

exposure.

– Respiratory difficulty:

- Frequent oropharyngeal suction
- Treat bronchospasm with atropine, not bronchodilators.
- Tachycardia may result from hypoxia (pulmonary secretions and bronchospasm).
- Atropine will dry secretions and paradoxically lower the heart rate.
- Intubate and ventilate if necessary.
- Avoid succinylcholine; may have prolonged duration as it is metabolized by cholinesterase.

MEDICATION

- Atropine: 1–2 mg (peds: 0.05–0.2 mg/kg) IV q5min (see the previous section for details)
- Dextrose: D₅₀W, 1 amp (25 g) of 50% dextrose (peds: 2–4 mL/kg D₂₅W) IV push
- Naloxone (Narcan): 2 mg (peds: 0.1 mg/kg) IV/IM
- Pralidoxime: 1–2 g (peds: 25–50 mg/kg) dissolved in 0.9% NS over 30 min IV; repeat in 1 hr if necessary, then q6h as needed:
 - Some propose continuous infusion (500 mg/h) for serum concentration of 4 mg/L.



FOLLOW-UP

DISPOSITION

Admission Criteria

- ICU admission for any exposure confirmed with atropine response.
- Any symptomatic patient should be admitted for monitoring.
- Avoid opioids, phenothiazines, and antihistamines; these may potentiate toxicity of organophosphates.

Discharge Criteria

- Asymptomatic for 6–12 hr after exposure
- Ensure close reliable follow-up and specific instructions when to return for evaluation.

Issues for Referral

Contact toxicologist or poison center for patients with significant exposures requiring repeat atropine administration.

FOLLOW-UP RECOMMENDATIONS

Psychiatry referral for intentional ingestions.

PEARLS AND PITFALLS

- Treatment failure often secondary to inadequate atropine dosing
- Recognize nicotinic manifestations (tachycardia, seizures).

ADDITIONAL READING

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- Yanagisawa N, Morita H, Nakajima T. Sarin experiences in Japan: Acute toxicity and long-term effects. *J Neurol Sci*. 2006;249:76–85.



CODES

ICD9

- 987.9 Toxic effect of unspecified gas, fume, or vapor
- 989.3 Toxic effect of organophosphate and carbamate

ICD10

- T59.94XA Toxic effect of unsp gases, fumes and vapors, undet, init
- T60.0X1A Toxic effect of organophos and carbamate insect, acc, init
- T60.0X2A Toxic effect of organophosphate and carbamate insecticides, intentional self-harm, initial encounter

OSGOOD–SCHLATTER DISEASE

Stephen R. Hayden

BASICS

DESCRIPTION

- Most frequent cause of knee pain in children aged 10–15 yr
- Pain and edema of the tibial tuberosity:
 - Tenderness at insertion site for patellar tendon just below the knee joint
- Extra-articular disease:
 - Pain is worse with activity and improves with rest
 - Caused by repetitive stress and is common in children participating in sports
- Benign, self-limited knee condition

ETIOLOGY

- Etiology is controversial
- Leading theory: Microfractures caused by traction on the apophysis
- Pain occurs during activities that stress the patellar tendon insertion onto tibial tubercle.

DIAGNOSIS

SIGNS AND SYMPTOMS

- Pain and swelling over tibial tuberosity
- Pain exacerbated by running, jumping
- Pain relieved by rest

History

- Risk factors:
 - Age: 10–15 yr of age, associated with growth spurt in puberty
 - More common in boys
 - Sports: Activities with running, jumping, swift changes in direction (i.e., soccer, basketball, figure skating)
- Knee pain is worse with activity and improves with rest.
- Usually unilateral, with 20% occurring bilateral

Physical-Exam

- Prominence and soft tissue swelling over the tibial tuberosity
- Pain reproduced by extending the knee against resistance
- Tenderness over tibial tuberosity at patellar tendon insertion site

- Tight quadriceps and hamstrings compared to unaffected side
- Erythema of tibial tuberosity may be present.
- Knee joint exam is normal.

ESSENTIAL WORKUP

Diagnosis is clinical:

- Pain, swelling, and tenderness localized to the tibial tubercle

DIAGNOSIS TESTS & INTERPRETATION

Imaging

Knee x-ray:

- Irregular ossification and fragmentation at the tibial tuberosity may be seen.
- Ultrasound has the advantage of imaging surrounding soft tissues

DIFFERENTIAL DIAGNOSIS

- Patellar stress fracture
- Patellar or quadriceps tendonitis
- Prepatellar or infrapatellar bursitis
- Osteochondritis dissecans
- Osteomyelitis
- Patellofemoral pain syndrome
- Septic joint
- Inferior patellar pole traction apophysitis (Sinding-Larsen–Johansson disease)
- Fat pad impingement (Hoffa disease)
- Referred pain, especially from the hip



TREATMENT

INITIAL STABILIZATION/THERAPY

- Stabilize lower extremity in position of comfort.
- Apply ice to affected knee.

ED TREATMENT/PROCEDURES

- Rest from painful activities:
 - Limited activity for 6–8 wk
 - Avoid cutting and jumping sports, such as basketball, soccer, volleyball, etc.
- Ice affected area.
- Analgesic medications
- Stretch the quadriceps and hamstrings.
- Apply protective padding to knee during activities.
- Infrapatellar tendon strap may be worn for 6–8 wk.
- Avoid corticosteroid injections.

- Reassurance; it is a benign, self-limited condition.

MEDICATION

First Line

Analgesic medications:

- Ibuprofen: 10 mg/kg PO q6h
- Acetaminophen: 15 mg/kg PO q4h



FOLLOW-UP

DISPOSITION

Admission Criteria

No admission is necessary.

Discharge Criteria

Discharge home.

Issues for Referral

If patient fails nonoperative therapy, then refer to pediatric orthopedic surgery:

- Rarely, surgical excision is required but is delayed until after skeletal maturity.

FOLLOW-UP RECOMMENDATIONS

Rest from painful activities and follow-up with pediatrician in 2–3 wk for repeat exam.

PEARLS AND PITFALLS

- Diagnosis is clinical:
 - Pain, swelling, and tenderness at the tibial tuberosity:
 - Tenderness and pain worse during and after exercise
 - Risk factors:
 - 10–15 yr of age
 - Sports activities with running, jumping
- Treatment is conservative:
 - Treat with rest, ice, and NSAIDs
 - Avoid sports activities until pain resolves.

ADDITIONAL READING

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injuries. *Am Fam Physician*. 2006;73(6):1014–1022.

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CODES

ICD9

732.4 Juvenile osteochondrosis of lower extremity, excluding foot

ICD10

- M92.50 Juvenile osteochondrosis of tibia and fibula, unsp leg
- M92.51 Juvenile osteochondrosis of tibia and fibula, right leg
- M92.52 Juvenile osteochondrosis of tibia and fibula, left leg

OSTEOGENESIS IMPERFECTA

Daniel Davis • Chad M. Valderrama

BASICS

DESCRIPTION

- Inherited abnormality of procollagen amino acid sequence
- Bone hypomineralization and incomplete ossification result in brittle bones.
- Abnormal collagen affects all connective tissue to varying degrees.
- Time course is variable:
 - Most cases involve fractures during childhood followed by quiescence during adolescence and early adulthood.

ETIOLOGY

- Procollagen defects result in abnormalities of bone and connective tissue matrix.
- Defects in different sites on procollagen protein chain result in more severe forms.
- Defects are inherited, either autosomal recessive (generally milder) or autosomal dominant (more severe).
- Lethal cases involve sporadic or new mutations.
- Ehlers–Danlos syndrome involves mutations of the same procollagen protein in different locations.

Pediatric Considerations

- Most cases involve pathologic fractures during childhood.
- Multiple fractures often initiate evaluation for abuse, but the possibility of pathologic fractures also should be considered.

DIAGNOSIS

SIGNS AND SYMPTOMS

- Multiple heritable defects that lead to brittle bones:
 - Often associated with other connective tissue abnormalities
- A suspected fracture with a relatively minor mechanism or a history of multiple fractures in a child suggests the diagnosis.
- Careful social history with consideration for the possibility of nonaccidental trauma
- Bones:
 - Multiple recurrent fractures (especially in long bones) are the hallmark of this disease.
 - Fractures may be present at birth or may recur in the elderly.
 - Shortened or bowed limbs, pectus excavatum, curving of long bones,

vertebral compression fractures, scoliosis, kyphosis, and abnormal skull shape

– All bones are affected to some extent (see Imaging/Special Tests).

- Eyes:

– *Blue sclerae* are another hallmark of this disease.

– No visual changes are reported.

- Ears:

– Hearing loss usually begins in adolescence; >90% of patients have some deficit by age 30 yr.

– Hearing loss is generally sensorineural, although some middle ear abnormalities have been demonstrated.

– Academic difficulties should raise suspicion of possible hearing deficits.

- Other:

– Discolored, fragile, and abnormal shape of teeth

– Shares several features with Ehlers–Danlos syndrome:

- Loose joints

- Valve problems

- Vascular abnormalities

- Thyroid abnormalities may be seen.

- Extreme cases may result in perinatal death.

ESSENTIAL WORKUP

- Diagnosis is usually made as combination of clinical and radiographic findings.

- History of repeated fractures or fractures with unimpressive mechanism

- Thorough search for other tender areas and evaluation of eyes, teeth, and joints is important for diagnosis.

- Careful exam of neurovascular status distal to fracture

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Evaluate for metabolic derangements such as hyperparathyroidism, vitamin C or D deficiencies, and calcium/phosphate abnormalities.

- DNA studies may be indicated for familial analysis, prenatal testing, and genetic counseling.

- Tissue biopsy is controversial but may help differentiate from tumors.

Imaging

- Radiographs of fracture sites:

– May reveal osteopenia (usually mild)

– Crumpled long bones (“accordion femora”)

– Incomplete ossification at physes

- Skeletal survey is mandatory, especially in children.

- Skull films may show wormian appearance of irregular ossification.
- Popcorn-like deposits on long-bone ends are poor prognostic finding.
- Formal audiologic testing as outpatient is required in older patients.

DIFFERENTIAL DIAGNOSIS

- Nonaccidental trauma in children
- Ehlers–Danlos syndrome
- Hypophosphatasia
- Achondroplasia
- Scurvy
- Congenital syphilis
- Celiac disease



TREATMENT

PRE HOSPITAL

Personnel should obtain information about mechanism or social factors that point toward pathologic fracture vs. nonaccidental trauma.

INITIAL STABILIZATION/THERAPY

- Airway management and resuscitation as indicated
- Fracture immobilization/splinting

ED TREATMENT/PROCEDURES

- Specific fracture management dictated by type and location of injury
- Orthopedic consultation regarding need for traction or operative fixation
- No specific treatment for osteogenesis imperfecta exists at present.

MEDICATION

- Pain medications as indicated
- Elderly women may benefit from calcium (1–1.5 g/d) and estrogen replacement (0.625 mg/d).



FOLLOW-UP

DISPOSITION

Admission Criteria

- Admission is determined by multiple trauma or operative needs for fracture repair.
- Pediatric patients may need admission to investigate possibility of nonaccidental trauma.

Discharge Criteria

- Patients may be considered for outpatient management if isolated fracture is present and appropriate home resources are available.
- Most patients should be discharged with orthopedic and primary physician follow-up.

Issues for Referral

- Orthopedic referral is driven by the acute injury.
- The presence of fractures in multiple locations or at different times also suggests nonaccidental trauma, which should prompt acute consultation and/or referral per local protocol.

FOLLOW-UP RECOMMENDATIONS

- Follow-up is generally driven by the acute injuries.
- Follow-up with the primary physician should be instituted to encourage treatment and monitoring of the disease.

PEARLS AND PITFALLS

- The most challenging aspect of caring for these patients is differentiating between pathologic fractures associated with osteogenesis imperfecta and nonaccidental trauma. With any questions, acute consultation and/or referral should be initiated per local protocol.
- It is a myth that children with osteogenesis imperfecta feel less pain than other patients.
- Predisposition to respiratory infections

ADDITIONAL READING

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- Rauch F, Glorieux FH. Osteogenesis imperfecta. *Lancet*. 2004;363(9418):1377–1385.
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See Also (Topic, Algorithm, Electronic Media Element)
Specific Orthopedic Injuries



CODES

ICD9

756.51 Osteogenesis imperfecta

ICD10

Q78.0 Osteogenesis imperfecta

OSTEOMYELITIS

Stephen R. Hayden

BASICS

DESCRIPTION

- Osteomyelitis (OM): Infection of bone with ongoing inflammatory destruction
- Usually bacterial, but fungal OM does occur
- Could be acute or chronic

ETIOLOGY

- Hematogenous OM:
 - _ Primarily in children, elderly, IV drug abuse (IVDA) patients
 - _ Seeding of bacteria to bone from remote site of infection via bloodstream
 - _ Children have acute OM and adults subacute or chronic.
 - _ Hematogenous OM of long bones rarely occurs in adults.
 - _ Most children with acute hematogenous OM have no preceding illness.
 - _ 1/3 have history of trauma to affected area.
 - _ *Staphylococcus aureus* is the most common cause of OM in all ages.
 - _ Neonates: *S. aureus*, *Enterobacteriaceae*, group A and B streptococci, and *Escherichia coli*
 - _ Children: *S. aureus*, group A streptococci, *Haemophilus influenzae*, *Enterobacteriaceae*
 - _ *Salmonella*: Common in sickle cell disease
 - _ Adults: *S. aureus*, *Enterobacteriaceae*, *Pseudomonas*, gram-negative rods, *Staphylococcus epidermidis*, gram-positive anaerobes, especially *Peptostreptococcus*
 - _ Illicit drug users: *Candida*, *Pseudomonas*, *Serratia marcescens*
 - _ Prolonged neutropenia: *Candida*, *Aspergillus*, *Rhizopus*, *Blastomyces*, coccidioidomycosis
- Hematogenous vertebral OM:
 - _ Uncommon
 - _ Most prevalent in adults >45 yr
 - _ Involves the disk and vertebra above and below
 - _ Often in the setting of long-term urinary catheter placement, IVDA, cancer, hemodialysis, or diabetes
 - _ IVDA: OM of pubic symphysis, sternoclavicular, and sacroiliac (SI) joints
 - _ Lumbar vertebrae most common, followed by thoracic, then cervical
 - _ Posterior extension leads to epidural/subdural abscess or meningitis.
 - _ Anterior extension may lead to paravertebral, retropharyngeal, mediastinal, subphrenic, retroperitoneal, or psoas abscess.

- Direct or contiguous OM:
 - Organism(s) directly seeded in bone due to trauma, especially following open fractures:
 - Spread from adjacent site of infection or from surgery
 - More common in adults and adolescents
 - *S. aureus*, *Enterobacteriaceae*, *Pseudomonas*
 - Normal vascularity:
 - *S. aureus* and *S. epidermidis*, gram-negative bacilli, and anaerobic organisms
 - Vascular insufficiency/diabetes:
 - Small bones of feet are common sites.
 - Infection resulting from minor trauma, infected nail beds, cellulitis, or skin ulceration
 - Polymicrobial, including anaerobes
 - Puncture wound through tennis shoe: *S. aureus*, *Pseudomonas*
 - Clavicular OM can occur as complication of subclavian vein catheterization.
- Chronic OM:
 - OM that persists or recurs
 - Distinguishing characteristic is necrotic bone (sequestrum) that must be débrided.
 - *S. epidermidis*, *S. aureus*, *Pseudomonas aeruginosa*, *S. marcescens*, and *E. coli*



DIAGNOSIS

SIGNS AND SYMPTOMS

Vary with duration of disease

History

- Mainly nonspecific symptoms
- Pain: Localized, deep, dull, and throbbing; occurs with and without movement
- Fever and chills; may be absent in chronic OM
- Malaise, nausea, vomiting
- Reluctance to use extremity
- Nonhealing ulcers despite proper therapy
- Consider OM as a cause of fracture nonunion
- Predisposing factors: DM, vasculopathy, IVDA, invasive procedures, trauma

Physical-Exam

- Tenderness to palpation, warmth, erythema, edema, decreased range of motion
- Drainage of sinus tract
- Deep ulcers and palpable bone (+ “probe to bone” test has very high positive predictive value)

- If ulcer size $> 2 \text{ cm}^2$ and $> 3 \text{ mm}$ in depth, bone involvement is likely.

ESSENTIAL WORKUP

- CBC
- ESR and C-reactive protein
- Radiographs
- Blood and wound cultures and sensitivities

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC; WBC may be elevated but often normal
- ESR; elevated in $> 90\%$ of cases
- C-reactive protein (usually elevated)
- Blood cultures (positive in $\sim 50\%$ of cases)

Imaging

- Plays a central role in evaluation
- Start with plain films; other tests often required
- Radiographs:
 - _ May be normal for the 1st 2–3 wk of symptoms
 - _ Earliest finding is periosteal elevation, followed by cortical erosions, then new bone formation.
 - _ 40–50% of focal bone loss needed to detect lucency on radiograph; fewer than 1/3 of cases have diagnostic findings at 10 days
 - _ Obtain CXR if TB suspected
- MRI:
 - _ Best modality to obtain detailed anatomy and extension of soft tissue and bone marrow involvement
 - _ Sensitivity and specificity of $\sim 90\%$
 - _ Reveals bone edema, cortical destruction, periosteal reaction, joint surface damage, and soft tissue involvement before x-rays
 - _ Effective in early detection (3–5 days from onset of infection)
 - _ Test of choice to identify vertebral OM and OM in diabetic foot ulcers
 - _ Occasional false-positive results in trauma, previous surgical procedures, or neuropathic joint disease
 - _ Negative study after 1 wk of symptoms rules out acute OM
- CT:
 - _ Modality of choice when MRI cannot be done
 - _ Reveals bone edema, cortical destruction, periosteal reaction, small foci of gas or foreign bodies, joint surface damage, and soft tissue involvement when plain films not helpful
 - _ Useful in OM of vertebrae, sternum, calcaneus, pelvic bones

- Useful to surgeons in guiding débridement and biopsy
- Bone scan:
 - Technetium 99m methylene diphosphonate ($^{99m}\text{Tc-MDP}$)
 - Measures increase in bone metabolic activity
 - ~95% sensitive but less specific than MRI
 - Bone scan abnormal after 2–3 days of symptoms
 - False-positive may occur in trauma, surgery, chronic soft tissue infection, tumor
 - High radiation burden, useful if suspect multifocal disease
- Leukocyte scintigraphy:
 - Indium 111 -labeled WBCs
 - More specific but less sensitive than bone scan
 - Difficult to distinguish bone inflammation from soft tissue inflammation (i.e., cellulitis, tumors, inflammatory arthritis)
- US:
 - An emerging modality for OM especially in children
 - Periosteal elevation or thickening, fluid collections adjacent to bone often seen
 - May show findings of OM days prior to plain films
 - Useful in guiding biopsy

Diagnostic Procedures/Surgery

- Gold standard for diagnosis is bone biopsy with histology and tissue Gram stains, including culture and sensitivities.
- Needle aspiration has lower sensitivity than open biopsy.
- Culture of sinus or drainage from wound can be misleading; correlates well with *S. aureus*, but not as reliable for other organisms.

Pediatric Considerations

- 70–85% of children have fever higher than 38.5°C.
- Neonates are commonly afebrile.
- Only ~1 in 3 of children will have leukocytosis.
- Blood cultures positive in ~50%
- US

DIFFERENTIAL DIAGNOSIS

- Cellulitis
- Paronychia/felon
- Bursitis, toxic synovitis, septic arthritis
- Extremity fracture
- Bone infarction in sickle cell patients
- Acute leukemia, malignant bone tumors

- Mechanical back pain
- Spinal epidural abscess
- Brucellosis, especially in SI joint
- TB, more common in thoracic spine (Pott disease)



TREATMENT

INITIAL STABILIZATION/THERAPY

Emergent stabilization if septic or if neurologic deficits from spine involvement

ED TREATMENT/PROCEDURES

- Empiric antibiotic treatment in ED
- Cultures should guide subsequent antibiotic regimen.
- Antibiotics: Depend on patient's age and organism (see Medications section)
- Orthopedic and infectious disease consultation
- Surgical intervention may be needed to optimize treatment (e.g., infected fracture or hardware, bone necrosis).
- Parenteral antibiotic treatment for 4–6 wk

MEDICATION

- Newborn–4 mo: Penicillinase-resistant synthetic penicillin (e.g., nafcillin: 37 mg/kg IV q6h) plus a 3rd-generation cephalosporin (e.g., ceftriaxone: 50–75 mg/kg/d IV); if suspect methicillin-resistant *S. aureus* (MRSA) then vancomycin (40–60 mg/kg IV q6h) plus a 3rd-generation cephalosporin. (Note: Doses are based on age > 28 days)
- Children (> 4 mo): Penicillinase-resistant synthetic penicillin (e.g., nafcillin: 37 mg/kg IV q6h to max. 8–12 g/d). If suspect MRSA, then vancomycin (40–60 mg/kg IV q6h to max. 2–4 g/d). Add 3rd-generation cephalosporin if suspicion for gram-negative rods, or presence on Gram stain noted (e.g., ceftriaxone: 50–75 mg/kg IV per day to max. 2–4 g/d)
- Adult: Penicillinase-resistant synthetic penicillin (e.g., nafcillin: 2 g IV q4h); if suspect MRSA, vancomycin (15 mg/kg IV q12h)
- Gram-negative (including pseudomonas) chronic OM: Ciprofloxacin 750 mg PO BID or Levofloxacin 750 mg PO QD
- Sickle cell anemia with OM: Ciprofloxacin 400 mg IV q12h, or levofloxacin 750 mg IV q24h (*not* in children); alternative: 3rd-generation cephalosporin
- Post nail puncture through tennis shoe: Ciprofloxacin 750 mg PO BID or Levofloxacin 750 mg PO q24h; alternative: Ceftazidime 2 g IV q8h
- Involving orthopedic prosthesis or hardware: Add rifampin (10 mg/kg/d PO/IV to max. of 600 mg/d) to regimen for *S. aureus*. Hardware removal generally required.
- Post-traumatic OM: Vancomycin and ceftazidime
- If vancomycin-resistant enterococcus present: Linezolid 600 mg IV q12h × 6 wk

Pediatric Considerations

Children with hematogenous OM may undergo short-course IV antibiotics and then be changed to oral for additional 1–2 mo.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients with acute OM should be admitted.
- Patients with chronic OM usually require admission for surgical procedures, débridement, and obtaining bone cultures and histology.

Discharge Criteria

Subacute or chronic OM patients may be considered for outpatient management if home IV antibiotics arranged, bone specimens obtained, and necrotic bone débrided.

- Cases refractory to débridement and antibiotics benefit from hyperbaric oxygen as an adjunct to standard treatment.
- ~ 2/3 of these cases will demonstrate benefit.

PEARLS AND PITFALLS

- WBC may be normal in many cases.
- Radiographs may be normal in the 1st 2–3 wk of symptoms.
- Wound cultures are low yield in guiding antibiotic therapy.

ADDITIONAL READING

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- Hatzenbuehler J, Pulling TJ. Diagnosis and management of osteomyelitis. *Am Fam Physician*. 2011;84(9):1027–1033.
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- Winters ME, Kluetz P, Zilberstein J. Back pain emergencies. *Med Clin North Am*. 2006;90:505–523.



CODES

ICD9

- 730.00 Acute osteomyelitis, site unspecified
- 730.10 Chronic osteomyelitis, site unspecified
- 730.20 Unspecified osteomyelitis, site unspecified

ICD10

- M86.9 Osteomyelitis, unspecified
- M86.10 Other acute osteomyelitis, unspecified site
- M86.60 Other chronic osteomyelitis, unspecified site

OSTEOPOROSIS

Daniel Davis • Marian Xu

BASICS

DESCRIPTION

- Overall decrease in skeletal mass, generally diffuse
- Trabecular bone (especially vertebrae and femur) affected more commonly and earlier
- Disease begins in adolescence, but fractures do not usually manifest until age ≥ 50
- Females affected much more commonly than males, especially after menopause

ETIOLOGY

- Overall increase in resorption over formation of new bone
- Advanced age is the most important risk factor
- Inadequate dietary calcium an important factor, especially early in life
- Sedentary lifestyle is a risk factor (weight bearing on bone favors new bone formation)
- Decrease in estrogen with menopause key factor in women
- Other risk factors include long-term steroid use, alcoholism, methotrexate, tobacco use, low body weight
- Familial or hereditary factor may coexist

Pediatric Considerations

Although disease appears to start in adolescence, pediatric patients are asymptomatic.

DIAGNOSIS

SIGNS AND SYMPTOMS

- Usually asymptomatic until pathologic fractures occur
- Fractures with insignificant mechanism or recurrent fractures are hallmark
- Vertebral column most commonly involved
- Multiple compression fractures of vertebral column often lead to kyphosis and scoliosis
- Hip fractures (femoral neck and intertrochanteric fractures) also common

History

- A suspected fracture with a relatively minor mechanism or a history of multiple fractures suggests osteoporosis.
- A family history of osteoporosis is an important risk factor

Physical-Exam

Exam findings are related to the acute fracture rather than the disease itself.

ESSENTIAL WORKUP

- Fracture without significant mechanism and identification of risk factors is most important
- Careful neurovascular exam distal to femur or other extremity fracture
- Rectal tone and postvoid residual should be determined in patients with vertebral fractures
- Radiographs of suspected fracture may show osteopenia (late finding in disease)
- Spine films may show old compression fractures
- CT scan should be performed to better evaluate vertebral fractures:
 - Retropulsion, spinal canal compromise is not always apparent on plain films.
 - Make sure CT cuts extend full level above and below injuries on spine radiographs.

DIAGNOSIS TESTS & INTERPRETATION

Lab

Serum chemistries—such as calcium, parathyroid hormone, and alkaline phosphatase—may help differentiate this from other illnesses.

Imaging

- Plain films can identify fractures; however, age of each fracture may be difficult to determine
- Bone scan or CT can help determine age of fractures, especially in spine

Diagnostic Procedures/Surgery

Bone densitometry can provide prognostic information and help guide therapy. Dual-energy x-ray absorptiometry with BMD T-score ≤ -2.5 : Osteoporosis.

DIFFERENTIAL DIAGNOSIS

- Multiple myeloma or other metastatic tumor
- Osteogenesis imperfecta (usually apparent in childhood)
- Hyperparathyroidism
- Other demineralizing bone diseases



TREATMENT

PRE HOSPITAL

Cautions:

- Obtain pre-hospital information on mechanism to help diagnose pathologic fracture
- Avoid aggressive manipulation or movement of patient, as this may exacerbate bony injury

INITIAL STABILIZATION/THERAPY

Immobilize fractures

ED TREATMENT/PROCEDURES

- Fractures are treated with expectation of delayed or incomplete healing
- Prevention is far more effective than treatment
- Long-term therapy is beneficial (see Medication)
- Use of orthotic back braces and vests should be arranged in conjunction with orthopedic spine consultation
- Exercise is also helpful
- Balance must be achieved between osteoporosis risk and steroid or methotrexate therapy

MEDICATION

- Alendronate: 10 mg/d or 70 mg weekly, alternative is risedronate 5 mg/d, 35 mg weekly, or 150 mg monthly
- Zoledronic acid: 5 mg IV yearly
- Raloxifene (selective estrogen receptor modulator): 60 mg PO QD
- Calcium: 1,200 mg daily (total of diet + supplement)
- Vitamin D: 800 IU/d
- Calcitonin: Nasal spray 200 IU/d
- Denosumab (monoclonal antibody): 60 mg SC every 6 mo
- Parathyroid hormone 1–34: 20 µg SC daily
- Estrogen: 0.625 mg/d (with or without medroxyprogesterone)

Pediatric Considerations

Ensure adequate calcium in diet from early age.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Per normal orthopedic protocols, with special considerations for age and social situation
- Compression fractures are generally stable, but possibility of burst fracture with cord compression must be ruled out.

- Any cervical fracture or fracture with neurologic symptoms requires admission with emergent consultation with neurosurgery or orthopedics
- Admission may be necessary for pain control and because of decreased ambulation

Discharge Criteria

- Per normal orthopedic protocols with special considerations for age and social situation
- Patients with minimal injuries, able to care for themselves at home or with appropriate assistance, and adequate postoperative pain control may be discharged with orthopedic follow-up

Issues for Referral

Orthopedic referral is driven by the acute injury.

FOLLOW-UP RECOMMENDATIONS

- Follow-up is generally driven by the acute injuries
- Follow-up with the primary physician should be instituted to encourage treatment and monitoring of the disease to prevent recurrent fractures

PEARLS AND PITFALLS

- A history of recurrent fractures, particularly with a low-energy mechanism, suggests the possibility of osteoporosis
- Reduced bone density on plain radiographs is highly suggestive and warrants referral back to the PCP for further workup and treatment
- Bisphosphonates are 1st-line therapy for treatment

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Specific Orthopedic Injuries.



CODES

ICD9

- 733.00 Osteoporosis, unspecified
- 733.01 Senile osteoporosis
- 733.09 Other osteoporosis

ICD10

- M80.08XA Age-rel osteopor w current path fracture, vertebra(e), init
- M81.0 Age-related osteoporosis w/o current pathological fracture
- M81.8 Other osteoporosis without current pathological fracture

OTITIS EXTERNA

Assaad J. Sayah

BASICS

DESCRIPTION

- Inflammation or infection of the auricle, auditory canal, or external surface of the tympanic membrane (TM):
 - Spares the middle ear
 - Affects 4/1,000 persons in US
- Also called “swimmer’s ear” due to the usual history of recent swimming:
 - Occasional cases after normal bathing
- Necrotizing (malignant) otitis externa:
 - Infection starts at the ear canal and progresses through periauricular tissue toward the base of the skull
 - Occurs in elderly, diabetic, or other immunocompromised patients
 - Caused by *Pseudomonas aeruginosa*
 - Can lead to cellulitis, chondritis, and osteomyelitis
 - Associated with 20% mortality

ETIOLOGY

- Often precipitated by an abrasion of the ear canal or maceration of the skin from persisting water or excessive dryness
- Predisposing factors include:
 - History of ear surgery or TM perforation
 - Narrow or abnormal canal
 - Humidity
 - Allergy
 - Eczema
 - Trauma
 - Abnormal cerumen production
- *P. aeruginosa*, *Staphylococcus aureus*, streptococcal species, and rarely fungi

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Recent swimming or prolonged water exposure
- History of diabetes
- History of chemotherapy, prolonged steroid use, HIV/AIDS, or other processes that

compromises immune system

- Itching of the external ear canal is usually the 1st symptom
- 1–2 day history of progressive pain
- Ear drainage
- Decreased auditory acuity
- Clogged sensation in ear

Physical-Exam

- Pain in ear or with motion of pinna/tragus
- Swollen, erythematous external ear canal
- Ear drainage
- Decreased auditory acuity
- Pain/swelling in preauricular area
- Necrotizing (malignant) otitis externa:
 - _ Pain, tenderness, swelling in periauricular area
 - _ Headache
 - _ Otorrhea
 - _ Cranial nerve palsy:
 - Facial nerve most affected

ESSENTIAL WORKUP

Clinical diagnosis with typical signs/symptoms:

- Pain in ear or with motion of pinna/tragus
- Otoscopic exam
- Swollen, erythematous external ear canal
- Ear drainage
- Cheesy white or gray-green exudate

DIAGNOSIS TESTS & INTERPRETATION

Lab

- None usually indicated, except when possibility of necrotizing otitis externa:
 - _ Signs of systemic toxicity or local spread of infection should be checked
- WBC count
- ESR
- Glucose (check for diabetes)
- Cultures

Imaging

CT/MRI to exclude mastoiditis if the patient has signs of toxicity or bone involvement

Diagnostic Procedures/Surgery

- Remove debris with a soft plastic curette or gentle irrigation with peroxide/water

mix

- Wick placement may be needed to facilitate medication delivery

DIFFERENTIAL DIAGNOSIS

- Necrotizing otitis externa
- Otitis media
- Folliculitis from obstruction of sebaceous glands
- Otic foreign bodies
- Herpes zoster infection of the geniculate ganglion
- Parotitis
- Periauricular adenitis
- Mastoiditis
- Dental abscess
- Sinusitis
- Tonsillitis
- Pharyngitis
- Temporomandibular joint pain
- Viral exanthems

Pediatric Considerations

Consider ear canal foreign bodies in children with purulent drainage from edematous, painful ear canals



TREATMENT

ED TREATMENT/PROCEDURES

- Clean external ear canal:
 - Remove the inflammatory debris by gentle curettage with a cotton-tipped wire applicator
 - Occasional suction with a Frazier suction tip may be necessary
- Insert a cotton or gauze wick 10–12 mm into the canal after cleansing if the ear canal is very edematous
- Management of otitis externa focuses on pain control, eradication of infection, and prevention of reoccurrence

MEDICATION

- Most cases respond well to topical treatment:
 - Antiseptic, anti-inflammatory, and drying otic drops eliminate the pathogenic bacteria and allow for rapid healing of the canal
 - Acetic acid solutions such as Domeboro otic (2% acetic acid): 4–6 drops q4–6h
 - Corticospirin otic (hydrocortisone 1%, polymyxin + neomycin) suspension:

- 4 drops to ear canal QID (use suspensions and not solutions with suspected TM perforation)
- Ofloxacin: 5 drops BID (drug of choice in perforated TM)
- Oral antibiotics:
 - Administer to patients with cellulitis of the face or neck, severe edema of the ear canal, concurrent otitis media, or when the TM cannot be visualized
 - Treat diabetics and other immunocompromised patients with oral ciprofloxacin and follow closely for symptoms of malignant otitis externa
 - Amoxicillin: 500 mg (peds: 40 mg/kg/d) PO TID
 - Ciprofloxacin: 500 mg PO BID
- IV antibiotics for patients with necrotizing otitis externa, severe cellulitis, or septic appearing
- Prophylaxis:
 - Apply rubbing alcohol or acetic acid (2%) to keep the external ear canal dry and prevent recurrence of infection
- Pain management with acetaminophen or NSAID. Consider opioids if severe pain
- Surgical débridement of granulation tissue and bone sequestration or drainage of associated abscess may be necessary in necrotizing otitis externa

COMPLICATIONS

- Mastoiditis
- Chondritis of the auricle
- Necrotizing otitis externa
- Osteomyelitis of the base of the skull
- CNS infections



FOLLOW-UP

DISPOSITION

Admission Criteria

- Necrotizing otitis externa
- Significant involvement of the pinna
- Signs of systemic illness

Discharge Criteria

- Most patients
- Close follow-up for patients at risk of otitis externa
- Patient instructions:
 - Avoid swimming and keep ears completely dry for 3–4 wk
 - Apply medications as directed
 - Return if worse pain, fever, hearing loss develops, or there is any change in

mental or neurologic status

- Follow up if symptoms are not improved within 2–3 days

Issues for Referral

Ear–nose–throat follow-up for:

- Perforated TM
- Worsening of symptoms
- Conductive hearing loss
- Failure of initial management

FOLLOW-UP RECOMMENDATIONS

Follow up with primary care physician or a return ED visit within 2–3 days for removal of the wick or if symptoms are worse.

PEARLS AND PITFALLS

- Concomitant and often erroneous diagnoses of acute otitis externa and otitis media are common because the TM in acute otitis externa is erythematous.
- Avoid ear canal lavage until tympanic integrity is documented.
- Regardless of the topical medications, penetration to the epithelium is key to therapy; any obstruction should be cleared.
- Recurrence can be largely prevented by counseling the patient and explaining how it can be avoided by minimizing ear canal moisture, trauma, or exposure to material that incites local irritation or contact dermatitis.
- Necrotizing otitis externa should be suspected in immunocompromised patients and diabetics who have severe otalgia, purulent otorrhea, and granulation tissue or exposed bone in the external auditory canal.

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- Stone KE. Otitis externa. *Pediatr Rev*. 2007;28:77–78.



See Also (Topic, Algorithm, Electronic Media Element)

- Otitis Media
- Mastoiditis

- Tympanic Membrane Perforation



CODES

ICD9

- 380.10 Infective otitis externa, unspecified
- 380.14 Malignant otitis externa

ICD10

- H60.10 Cellulitis of external ear, unspecified ear
- H60.20 Malignant otitis externa, unspecified ear
- H60.90 Unspecified otitis externa, unspecified ear

OTITIS MEDIA

Assaad J. Sayah

BASICS

DESCRIPTION

- Inflammation of the middle ear
- Most commonly occurs in children 6–36 mo
- Rapid onset of local and/or systemic symptoms
- More than 1/3 of children experience >5 episodes by the age of 7 yr

ETIOLOGY

- Usually associated with (or as a result of) upper respiratory tract infections
- Viral:
 - Parainfluenza
 - Respiratory syncytial virus
 - Influenza
 - Adenovirus
 - Rhinovirus
- Bacterial:
 - *Streptococcus pneumoniae*
 - *Moraxella catarrhalis*
 - *Haemophilus influenzae*
 - *Streptococcus pyogenes*
 - *Mycoplasma pneumoniae*
- Associated with blockage of eustachian tube
- Predisposing factors:
 - Deficient mucus, cilia, or antibodies
 - Intubation, especially nasotracheal
 - American Indians, Eskimos
 - Down syndrome
 - Cleft palate
 - Diabetes
 - Vitamin A deficiency
 - HIV
- Risk factors
 - Family history
 - Daycare
 - Parental smoking
 - Pacifier use
 - Bottle-feeding



DIAGNOSIS

From the American Academy of Pediatrics 2013 Guidelines:

- Diagnose otitis media (OM) when:
 - Moderate to severe bulging of tympanic membrane (TM)
 - Mild bulging of TM *and* recent onset of ear pain (tugging, pulling, rubbing in nonverbal child)
 - New otorrhea not due to acute otitis externa
- Should *not* diagnose if no middle ear effusion (pneumatic otoscopy and/or tympanometry)
- Recurrent OM:
 - 3 episodes in 6 mo or
 - 4 episodes in the last year with 1 in the past 6 mo

SIGNS AND SYMPTOMS

History

- Ear pain (otalgia)
- Irritability
- Rhinitis
- Vomiting, diarrhea
- Poor feeding
- Fever
- Sensation of plugged ear
- Pulling at ear
- Vertigo, tinnitus
- Conjunctivitis

Physical-Exam

- TM inflammation, bulging, and limited mobility
- New onset otorrhea without evidence of otitis externa
- Decreased visibility of the landmarks of the middle ear

ESSENTIAL WORKUP

- Exclude associated conditions
- Consider full septic workup for sick patients with fever
- Otoscopic exam for appearance and mobility of TM:
 - Full visualization essential
 - Increased vascularity, erythema, purulence
 - Obscured landmarks—bony, light reflex
 - Pneumatic otoscopy—bulging, retracted, decreased mobility

DIAGNOSIS TESTS & INTERPRETATION

Lab

Cultures unhelpful unless done by tympanocentesis

Imaging

CT scan if associated mastoiditis is suspected

Diagnostic Procedures/Surgery

- Tympanocentesis—indications:
 - Severe pain or toxicity
 - Failure of antimicrobial therapy
 - Suspicion of suppurative complication
 - Sick neonate
 - Immunocompromised patient
- Tympanometry and acoustic otoscopy may be useful with difficult exams

DIFFERENTIAL DIAGNOSIS

- Infection:
 - Otitis externa
 - Mastoiditis
 - Dental abscess
 - Allergic rhinitis
 - Cholesteatoma
 - Peritonsillar abscess
 - Sinusitis
 - Lymphadenitis
 - Parotitis
 - Meningitis
- Trauma:
 - Perforation of the TM
 - Foreign body in ear
 - Barotrauma
 - Instrumentation
- Serous OM or eustachian tube dysfunction
- Impacted ear cerumen
- Impacted 3rd molar
- Temporomandibular joint dysfunction



TREATMENT

ED TREATMENT/PROCEDURES

- Most mild cases could resolve without antibiotics
- Antibiotics are indicated for:

- All infants < 6 mo
- Children < 2 yr with bilateral OM
- Bilateral OM in kids < 2 yr
- Children > 6 mo with severe infection (otalgia for > 48 hr or temperature 102.2°F or higher)
- Bilateral OM in kids < 2 yr
- Children > 6 mo with ruptured TM with drainage
- For otherwise normal healthy patients ≥ 6 mo with mild symptoms and/or uncertain diagnosis, consider no antibiotics and repeat evaluation in 2–3 days:
 - For reliable parents, may provide a prescription for oral antibiotics, which the family can fill if the child's symptoms get worse or persist after 2 days
- Considerations should include recurrent nature of OM, lack of clinical response, and resistance patterns in community
- Parenteral antibiotics are indicated in febrile toxic children < 1 yr or with immunocompromise
- Antihistamines, decongestants, and steroids have no proven efficacy
- Antipyretics and analgesics are important (avoid local analgesics in perforated TMs)

MEDICATION

- Antibiotics:
 - Amoxicillin: 500–875 mg PO q12h (peds: 80–90 mg/kg/d PO div. q12h) for 10 days
 - Amoxicillin–clavulanic acid: 500–875 mg PO q12h (peds: 90 mg/kg/d PO q12h) for 10 days
 - Azithromycin: 10 mg/kg PO day 1, then 5 mg/kg/d PO days 2–5
 - Cefuroxime: 500 mg PO q12h (peds: 30 mg/kg/d PO div. q12h)
- Analgesia:
 - Acetaminophen: 500 mg PO q6h (peds: 15 mg/kg per dose orally/rectally every 4–6 hr); not to exceed 4 g/24 h
 - Antipyrine/benzocaine (5.4%/1.4% solution): 2–4 drops in ear QID PRN
 - Ibuprofen: 400–600 mg PO q6–8h (peds: 10 mg/kg per dose orally every 6 hr)



FOLLOW-UP

DISPOSITION

Admission Criteria

Febrile toxic children who are:

- < 1 yr, immunocompromised
- Moderately or severely dehydrated

- Unable to tolerate oral fluids or medications
- Suspected or proven associated significant infection
- Suspected abuse
- Unreliable caretaker

Discharge Criteria

Children without any of the aforementioned criteria

FOLLOW-UP RECOMMENDATIONS

- Follow-up in 10–14 days to ensure resolution
- Indications for earlier follow-up:
 - Child does not get better in 24–48 hr
 - Any progression of signs or symptoms
 - New problems develop, including a rash
 - Any concerns arise

COMPLICATIONS

- Recurrent OM:
 - 3 episodes within 6 mo or
 - 4 episodes in 1 yr with the last within 6 mo
- Perforated TM
- Serous OM
- Hearing loss (conductive and sensorineural)
- Facial nerve injury
- Mastoiditis
- Cholesteatoma
- Meningitis
- Subdural empyema
- Labyrinthitis
- Epidural abscess
- Venous sinus thrombosis

PEARLS AND PITFALLS

For otherwise normal healthy patients ≥ 6 mo with mild symptoms and/or uncertain diagnosis, consider no antibiotics and repeat evaluation in 2–3 days.

ADDITIONAL READING

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CODES

ICD9

- 381.4 Nonsuppurative otitis media, not specified as acute or chronic
- 381.60 Obstruction of Eustachian tube, unspecified
- 382.9 Unspecified otitis media

ICD10

- H65.90 Unspecified nonsuppurative otitis media, unspecified ear
- H66.90 Otitis media, unspecified, unspecified ear
- H68.109 Unspecified obstruction of Eustachian tube, unspecified ear

OTOLOGIC TRAUMA

David A. Pearson • Chelsea Kolshak

BASICS

DESCRIPTION

Pinna

- Ear cartilage has no blood supply and is nutritionally dependent on perichondrium
- Hematomas often disrupt perichondrium and cartilage
 - Can lead to:
 - Ischemia
 - Perichondritis
 - Necrosis
 - Cauliflower ear
- Penetrating injuries or bite wounds may lead to infection of cartilage

Middle Ear

- Air-space cavity containing ossicles; susceptible to injuries disrupting pressure (blast, diving)
- Bordered by medial cranial fossa (including temporal and mastoid bones)
- Traumatic fractures can lead to CSF leak (otorrhea/rhinorhea)
 - May disrupt enclosed vestibular system
- Facial nerve passes through cavity—injury to cavity may cause peripheral nerve paralysis

ETIOLOGY

- Blunt trauma:
 - Contact sports such as wrestling
 - Motorcycle helmets
- Penetrating trauma such as tympanic membrane (TM) perforation from cotton swabs
- Human or animal bites
- Blast injury
- Lightning injury:
 - TM and ossicular disruptions occur in 50% of lightning strikes
- Chemical exposure
- Thermal injury
- Diving injuries:
 - Inner ear barotrauma
 - TM rupture

Pediatric Considerations

Consider nonaccidental trauma

DIAGNOSIS

SIGNS AND SYMPTOMS

- Severe ear pain
- Bleeding
- *Signs of auricular deformity:*
 - Edema
 - Hematoma:
 - Bluish, fluctuant, or doughy swelling of auricle
 - Laceration
 - Amputation
 - Loss of contour of the pinna
- *Signs of middle ear trauma:*
 - Decreased hearing:
 - Partial loss suggests TM rupture
 - Complete loss suggests injuries to ossicles or inner ear
 - Tinnitus
 - Middle ear effusion or canal drainage
 - Peripheral facial nerve paralysis
 - Vestibular symptoms, i.e., nystagmus or vertigo:
 - May also result from inner ear injury
- *Signs of basilar skull fracture:*
 - Hemotympanum or serous effusion
 - Retroauricular hematoma (battle sign)
 - CSF otorrhea or rhinorrhea
 - Peripheral facial nerve paralysis

History

- Mechanism
- Associated injuries
- Past otologic history
- Medications and allergies

Physical-Exam

- Head
- Cranial nerves
- Vascular structures
- Pinna
- External ear canal

- TM
- Hearing
- Consider the Weber and the Rinne test to evaluate for conductive hearing loss due to TM rupture or perforation:
 - Rinne test: Place a struck tuning fork to mastoid tip, hold until patient no longer hears ringing, then place fork near external auditory opening:
 - Normal: Patient still hears ringing; air conduction > bone conduction
 - Abnormal: No sound heard; air conduction < bone conduction; implies a conductive hearing loss
 - Weber test: Place a struck tuning fork to center of forehead:
 - Normal: Equal sound perception in both ears
 - Abnormal due to neurosensory loss: Patient will have decreased sound perception in the impaired ear
 - Abnormal due to conductive loss: Increased sound perception in the impaired ear
- Be sure to evaluate for concomitant injuries

DIAGNOSIS TESTS & INTERPRETATION

Lab

Wound culture if signs of infection

Imaging

- Consider head and/or facial CT to evaluate for intracranial injury or bone fracture
- Consider CT temporal bone without contrast if evidence of serious middle ear injury

DIFFERENTIAL DIAGNOSIS

- Infection
- Hemangioma
- Foreign body in ear



TREATMENT

PRE HOSPITAL

If auricle is amputated, wrap in moist gauze and place in plastic bag

INITIAL STABILIZATION/THERAPY

- Check ABCs; full trauma evaluation; resuscitation as appropriate
- Sterile dressing to injured site

ED TREATMENT/PROCEDURES

- All injury types:

- Anesthesia:
 - Local anesthesia via nerve block to auriculotemporal branch of mandibular nerve, lesser occipital nerve, greater auricular nerve, and auricular branch of vagus nerve; use 1% lidocaine or 0.25% marcaine
 - Alternative: Inject ring of anesthetic around base of pinna
- Tetanus prophylaxis if necessary
- Specific injury types:
 - Auricular hematoma: Drainage imperative to reapproximate perichondrium to cartilage to prevent cartilage necrosis, ideally within 72 hr; however, no clearly defined best treatment
 - Antistaphylococcal antibiotics for 7–10 days
 - Aspiration: Preferred alternative if clot not yet formed; use 18G–20G needle for aspiration milk hematoma until totally evacuated; apply pressure dressing
 - Incision and drainage: More effective with larger and/or clotted hematomas; incise along curvature of pinna with no. 15 scalpel, evacuate, and irrigate; apply pressure dressing
 - Vaseline gauze pressure dressing: Place to fill crevices of pinna; place over and behind pinna; wrap soft gauze firmly around head
 - Alternative pressure dressing: Suture dental rolls into place over incised area
 - If patient has 2nd presentation due to reaccumulation, hematoma should be reaspirated and a wick placed for drainage
- Laceration:
 - Prophylactic antibiotics are controversial but for human and animal bites treat with amoxicillin–clavulanate
 - Clean and debride wound, anesthetize as necessary
 - Superficial abrasions: Clean, dress with antibiotic ointment
 - Simple lacerations: 5 or 6 monofilament nylon or polypropylene suture, then pressure dressing; may use absorbable suture to avoid having to bend ear for suture removal
 - Exposed auricular cartilage: Carefully debride jagged edges; completely cover cartilage to prevent perichondritis; can remove small amount of cartilage to allow skin coverage; approximate cartilage 1st with absorbable sutures at major landmarks; include anterior and posterior perichondrium in stitch
 - Avulsions:
 - < 2 cm total avulsions may be used as graft and survive
 - > 2 cm: Consult or urgently refer to otolaryngologist or plastic surgeon

MEDICATION

- Amoxicillin–clavulanate: Adults: 875/125 mg PO BID (peds: 40 mg/kg/d PO BID)

- Dicloxacillin: 250–500 mg PO QID (peds: 30–50 mg/kg/d PO div. q6h)



FOLLOW-UP

DISPOSITION

Admission Criteria

- Concomitant serious traumatic injuries
- Need for IV antibiotics
- Immunosuppressed persons with serious infections, perichondritis, or chondritis

Discharge Criteria

- Able to tolerate oral antibiotics
- Able to arrange close follow-up

FOLLOW-UP RECOMMENDATIONS

- Follow up wound suture repair in 5 days
- Follow up hematomas in 24 hr to evaluate for reaccumulation

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Barotrauma
- Tympanic Membrane Perforation



CODES

ICD9

- 380.00 Perichondritis of pinna, unspecified
- 920 Contusion of face, scalp, and neck except eye(s)
- 959.09 Injury of face and neck

ICD10

- H61.009 Unspecified perichondritis of external ear, unspecified ear
- S00.439A Contusion of unspecified ear, initial encounter
- S09.91XA Unspecified injury of ear, initial encounter

OVARIAN CYST/TORSION

René A. King • Lynne M. Yancey



BASICS

DESCRIPTION

- Ovarian cysts:
 - Generally asymptomatic until complicated by hemorrhage, torsion, rupture, or infection
 - Follicular cysts:
 - Most common
 - Occur from fetal life to menopause
 - Unilocular; diameter 3–8 cm
 - Thin wall predisposes to rupture, which usually causes minimal or no bleeding
 - Rupture during ovulation at midcycle is known as mittelschmerz
 - Corpus luteal cysts:
 - Most significant
 - Diameter 3 cm, but usually < 10 cm
 - Rapid bleeding from intracystic hemorrhage causes rupture
 - Rupture is most common just before menses begins
 - Can cause severe intraperitoneal bleeding
 - Gradual bleeding into cyst or ovary distends capsule and may cause pain without rupture
- Adnexal torsion:
 - 5th most prevalent surgical gynecologic emergency
 - Twisting of vascular pedicle of ovary, fallopian tube, or paratubal cyst
 - Causes adnexal ischemia leading to necrosis
 - Occlusion of lymphatics and venous drainage lead to rapid enlargement of adnexa
 - Greatest risk with cysts 8–12 cm

RISK FACTORS

Adnexal torsion:

- Reproductive-age women
- Ovarian cysts, especially > 5 cm
- Ovarian hyperstimulation
- Tumors: Serous cystadenoma most common; teratomas
- Pelvic surgery: Tubal ligation; hysterectomy
- Pregnancy
- History of pelvic inflammatory disease

Pregnancy Considerations

Torsion in pregnancy usually occurs in the 1st trimester, and in vitro fertilization or ovarian induction are risk factors.

Pediatric Considerations

15% of adnexal torsions occur in children

ALERT

- Anticoagulated patients at increased risk of:
 - _ Hemorrhagic corpus luteal cyst
 - _ Significant bleed from ruptured cyst, including with ovulation

ETIOLOGY

- Ovarian cyst:
 - _ Follicular cysts result from nonrupture of mature follicle or failure of atresia of immature follicle
 - _ Corpus luteal cysts result from unrestrained growth in early pregnancy or from normal intracystic hemorrhage days after ovulation
 - _ Other cysts:
 - Theca lutein
 - Cystic teratoma
 - Endometrioma (chocolate cyst)
- Adnexal torsion:
 - _ Right > left
 - _ Highest frequency in reproductive women

ALERT

Cysts found in postmenopausal women suggest carcinoma

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Ovarian cyst:
 - _ Abdominal pain
 - Sharp, unilateral
 - Intermittent vs. constant
 - Migration
 - Previous episodes
 - May occur with exercise, intercourse, trauma, or pelvic exam
 - _ Fever is rare
 - _ Irregular menses (may suggest polycystic ovary syndrome)

- Infertility
- Pregnancy status
- Previous STDs
- History of breast or GI cancer (may metastasize)
- Adnexal torsion:
 - Variable history
 - Abdominal pain:
 - Sudden, sharp, colicky
 - Localized vs. diffuse
 - Referred pain to groin or flank
 - May be chronic or recurring with torsion/detorsion
 - Fever
 - Nausea/vomiting
 - Vaginal bleeding
 - UTI symptoms

Physical-Exam

- Ovarian cyst:
 - Abdominal tenderness (mild to severe with peritonitis)
 - Adnexal tenderness
 - Pelvic mass
 - Hemorrhagic shock possible:
 - Usually from corpus luteal cyst rupture
 - Orthostasis, hypotension, tachycardia
- Adnexal torsion:
 - Abdominal tenderness (mild to severe)
 - Adnexal tenderness
 - Adnexal mass

ESSENTIAL WORKUP

- Pregnancy test essential to rule out ectopic pregnancy
- Rapid hemoglobin or hematocrit

DIAGNOSIS TESTS & NTERPRETATION

Lab

- Urine or serum human chorionic gonadotropin determination
- CBC
- Urinalysis
- If significant hemorrhage, type and cross packed RBCs
- Cervical cultures to rule out PID

Imaging

- Transvaginal US:
 - Adnexal cysts and masses:
 - Cystic masses <5 cm in premenopausal women generally benign
 - Should be re-evaluated at the end of menstruation
 - Pelvic free fluid
 - Enlarged, edematous ovary (suggests torsion)
- Doppler:
 - May show decreased flow with torsion
 - Important to document normal blood flow on Doppler in ED, even though does not rule out recent torsion of ovary
- MRI:
 - Consider in pregnant patients with right lower quadrant pain and nondiagnostic US and Doppler
- CT:
 - May demonstrate cysts or evidence of torsion or suggest alternative diagnosis
 - May provide enough information to proceed to laparoscopy if abnormal ovary and no other cause of pain identified
 - Uterus may be shifted to side of torted adnexa
 - Ascites may be present

ALERT

US sensitivity for diagnosis of ovarian torsion is not well established; continue workup if high clinical suspicion

Diagnostic Procedures/Surgery

- Culdocentesis:
 - No longer commonly done
 - May yield serosanguinous fluid with ruptured cyst
 - Hematocrit > 15% suggests significant hemoperitoneum
- Laparoscopy is gold standard for torted adnexa and definitive diagnosis

Pediatric Considerations

- Early detorsion of adnexa by laparoscopy is now advocated to preserve ovarian function
- Followed by frequent follow-up visits to monitor for malignancy

DIFFERENTIAL DIAGNOSIS

- Ectopic pregnancy
- PID
- Round ligament pain
- Endometriosis
- Neoplasm

- Torsion of uterus
- Appendicitis

FOLLOW-UP RECOMMENDATIONS

Ovarian cyst

- If pain is resolved and cyst is <4–5 cm, close follow-up is recommended with gynecology for further studies

PEARLS AND PITFALLS

Adnexal torsion:

- Torsion is a clinical diagnosis:
 - US may show flow to an ovary that has detorsed
- Symptoms can be varied and nonspecific
- Always include adnexal torsion in differential of abdominal pain

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See Also (Topic, Algorithm, Electronic Media Element)

- Abdominal Pain
- Ectopic Pregnancy
- Endometriosis
- Pelvic Inflammatory Disease



CODES

ICD9

- 620.0 Follicular cyst of ovary
- 620.2 Other and unspecified ovarian cyst
- 620.5 Torsion of ovary, ovarian pedicle, or fallopian tube

ICD10

- N83.0 Follicular cyst of ovary
- N83.20 Unspecified ovarian cysts
- N83.51 Torsion of ovary and ovarian pedicle

PAGET DISEASE

Anna L. Waterbrook • Matthew C. Kostura



BASICS

DESCRIPTION

- Paget disease involves resorption of normal bone and its replacement with fibrous and sclerotic tissue
- Also known as osteitis deformans
- Usually focal, bones most frequently involved include:
 - Pelvis (70%)
 - Femur (55%)
 - Skull (42%)
 - Tibia (32%)
 - Spine (53%, lumbar spine)
 - Flat bones
- Usually found incidentally and generally asymptomatic
- Occurs in ~1–2% of patients >55 yr old
- Incidence increases with age
- Starts with resorptive or osteolytic phase, during which osteoclasts remove healthy bone
- Hypervascularity begins in resorptive phase:
 - Predisposes to hematoma and fracture
- Resorbed bone is eventually replaced by irregular, dense, disorganized trabecular bone in sclerotic or osteoplastic phase forming “mosaic pattern”
- Malignant transformation is rare:
 - Osteosarcoma is malignancy of concern
 - Usually malignant transformation occurs in 1%
- More common in men
- More common in European descent
- Less common in Asian or Scandinavian descent
- Typically involves 1 bone (monostotic)
- May involve a few bones (polyostotic)

ETIOLOGY

- Unknown
- Genetic component:
 - SQSTM1 mutation seen in many but not all cases
- Environmental influences may also play a role
 - Presence of nucleocapsids from measles, canine distemper, paramyxovirus, or respiratory syncytial virus may implicate viral cause

- Possible association with rural life and close contact with farm animals
- May represent vascular hyperplasia with subsequent inflammation
- Increased nucleoli and intranuclear inclusion bodies seen in osteoclasts on microscopy

Pediatric Considerations

Generally not seen in children

DIAGNOSIS

SIGNS AND SYMPTOMS

- Many patients are asymptomatic, with disease discovered by incidental radiographs or elevated alkaline phosphatase levels
- Deep, aching bone pain occurs late in the clinical course
- Pain with weight bearing if femur or tibia involvement
- Pain worse with rest in nonweight-bearing bones
- Acute (resorptive/osteolytic) phase:
 - Pathologic fractures
 - Pain from acute lysis, fracture, or resultant arthritis
 - Hypercalcemia or renal stones
 - Hypervascularity may result in significant bleeding if affected bone is fractured
 - Widespread disease:
 - Increased vascularity and blood flow may result in high-output cardiac failure
- Secondary (sclerotic/osteoplastic) phase:
 - Long-bone involvement may present with swelling or deformity and gait abnormality
 - Skull involvement may cause headaches or abnormal skull shape (change in hat size)
 - Severe skull or spine involvement may result in CNS compression
 - Hearing loss may result from nerve compression or ossicle involvement

ESSENTIAL WORKUP

- Diagnosis usually suggested by radiographs
- Thorough neurologic exam must be documented, especially with vertebral or pelvis involvement

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Alkaline phosphatase is the most dramatic marker of disease activity
- Calcium and phosphate levels should be checked as well, but are usually normal

- Hypercalcemia seen in immobilization or presence of a fracture, but if elevated in an ambulatory patient, suspect hyperparathyroidism
- EKG if suspect hypercalcemia and CXR with evidence of high-output cardiac failure
- Increased bone formation may lead to elevations in urine hydroxyproline or serum osteocalcin or procollagen fragments
- Alterations in parathyroid hormone (PTH) levels occur as secondary changes during resorptive/osteolytic phase (low PTH) and sclerotic/osteoplastic phase (high PTH)

Imaging

- Plain x-rays:
 - During resorptive phase, lytic lesions are often not seen, except in skull, where lesions are well demarcated (osteoporosis circumscripta)
 - Bowing of long bones may occur with resorption and strength loss
 - New bone initially appears irregular and spotty, and later becomes homogeneous and dense (“ivory pattern”)
 - Excess bone may be deposited along stress lines, leading to cortical irregularities and thickening
- CT or MRI defines margins and helps evaluate for neoplasm or hematoma:
 - Spiral CT to detect renal calculi
- Radionuclide bone scans useful to evaluate extent and activity of disease
- Plain films are usually all that is needed in acute setting to identify/manage fractures

DIFFERENTIAL DIAGNOSIS

- Primary hyperparathyroidism
- Multiple myeloma
- Hodgkin variants
- Acromegaly
- Osteosarcoma



TREATMENT

PRE HOSPITAL

- Pre-hospital personnel should obtain information about mechanism of injury or social factors that suggest pathologic fracture
- Adequate immobilization can limit excessive bleeding around fracture site

INITIAL STABILIZATION/THERAPY

- Airway management and resuscitation, as indicated
- High-output cardiac failure should be treated as outlined in CHF chapter
- Prompt immobilization of fractures will limit excessive bleeding around fracture

site

ED TREATMENT/PROCEDURES

- Analgesia for pain of lytic lesions, fractures, or arthritis includes acetaminophen and narcotics
- Fracture treatment is often more conservative, owing to difficulties with bleeding during operative repair
- Orthopedic consultation for severe arthritis and definitive fracture management
- Hypercalcemia may be treated with IV fluids, calcitonin, and/or bisphosphonates
- CNS compression requires emergent neurosurgical consultation and possible decompression

MEDICATION

Treatment indicated in patients with symptomatic disease or asymptomatic disease located in areas where complications can occur

First Line

- Nitrogen-containing bisphosphonates:
 - Pamidronate: 30 mg IV daily × 3 consecutive days; infuse over 4 hr
 - Alendronate: 40 mg PO daily for 6 mo
 - Risedronate: 30 mg PO daily for 2 mo
 - Zoledronic acid: 5 mg IV × 1; infuse over at least 15 min

Second Line

- Simple bisphosphonates and calcitonin:
 - Etidronate: 5 mg/kg PO daily for 6 mo
 - Tiludronate: 400 mg PO daily for 3 mo
 - Calcitonin: 50–100 U SC as tolerated; not for >6 mo
- Chemotherapy and simple bisphosphonates no longer recommended
- Use of calcitonin and simple bisphosphonates are limited to patients who cannot tolerate or who are allergic to the nitrogen-containing bisphosphonates
- Side effects of bisphosphonates include influenza like syndrome and jaw osteonecrosis
- Often need supplemental vitamin D and Ca to maintain normal Ca levels during treatment



FOLLOW-UP

Admission Criteria

- Admission as indicated for major trauma or injury, or excessive bleeding
- Orthopedic procedures
- Hypercalcemia

- CNS compressive symptoms, nerve entrapment requiring surgery

Discharge Criteria

- No evidence of significant bleeding, neurologic compromise, or hypercalcemia, and adequate pain control
- Appropriate fracture immobilization and orthopedic follow-up

Issues for Referral

- Referral is based upon any acute injuries
- May also consider referral to endocrinologist within 1–2 wk of discharge

FOLLOW-UP RECOMMENDATIONS

- Follow-up is generally driven by the acute injury that led to the radiographs on which the diagnosis of Paget disease was made
- Response to pharmacologic treatment aimed at correction of serum alkaline phosphatase levels
- Consider repeat pharmacologic treatment if rise in serum alkaline phosphatase, return of symptoms, or disease progression seen radiographically

PEARLS AND PITFALLS

- The diagnosis of Paget disease is usually made as an incidental finding on radiographic imaging
- Prompt immobilization of fractures will limit excessive bleeding around fracture site
- Consider Paget disease if elevation of alkaline phosphatase is present without any other explanation

ADDITIONAL READING

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- Whyte MP. Clinical practice. Paget's disease of bone. *N Engl J Med.* 2006;355:593–600.



See Also (Topic, Algorithm, Electronic Media Element)

Specific Orthopedic Injuries



CODES

ICD9

731.0 Osteitis deformans without mention of bone tumor

ICD10

- M88.9 Osteitis deformans of unspecified bone
- M88.88 Osteitis deformans of other bones
- M88.859 Osteitis deformans of unspecified thigh

PAIN (PRINCIPLES/MEDS/COMFORT CARE)

Vincent Bounes

BASICS

DESCRIPTION

Unpleasant sensory and emotional experience that may be secondary to actual or perceived damage to tissue, the somatosensory system, or a psychogenic dysfunction.

- It is an individual, subjective, multifactorial experience influenced by culture, medical history, beliefs, mood and ability to cope.

EPIDEMIOLOGY

Incidence and Prevalence Estimates

- Most common reason for seeking health care
- Up to 78% of visits to the emergency department.
- Pain is severe for 2/3rds of patients presenting with pain.
- Chronic pain is present in up to 35% of the population.
- Prevalence of neuropathic pain is 21.4% in emergency departments.

ETIOLOGY

- Different components of pain can be combined in a same patient.
- Nociceptive pain:
 - Stimulation of peripheral nerve fibers (nociceptors) that arises from actual or threatened damage to non-neural tissue.
 - Visceral pain:
 - Stimulation of visceral nociceptors
 - Diffuse, difficult to locate, and often referred to a distant, usually superficial, structure.
 - Sickening, deep, squeezing, dull.
 - Deep somatic pain:
 - Stimulation of nociceptors in ligaments, tendons, bones, blood vessels, fasciae, and muscles
 - Dull, aching, poorly localized pain.
 - Superficial pain:
 - Stimulation of nociceptors in the skin or other superficial tissue.
 - Sharp, well defined, and clearly located.
- Neuropathic pain:
 - Exacerbation of normally nonpainful stimuli (allodynia).
 - Paroxysmal episodes likened to electric shocks.
 - Continuous sensations include burning or coldness, “pins and needles”

sensations, numbness and itching.

- Psychogenic pain:
 - Pain caused, increased or prolonged by mental, emotional, or behavioral factors.

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- A patient's self-report is the most reliable measure of pain.
- Obtain a detailed description of pain:
 - Onset
 - If caused by an injury, determine the mechanism of injury
 - Localization of pain
 - Severity of pain:
 - Mild pain from >0 to $\leq 3/10$
 - Moderate pain from >3 to $<6/10$
 - Severe pain $\geq 6/10$.
 - Type of pain
 - Duration of pain
 - Variations of pain:
 - Daily/weekly/monthly variations
 - Variations caused by physical activities
 - Effect of previous analgesic drugs taken before the consult.
- Acute vs. chronic pain:
 - Acute pain:
 - Transitory, usually <30 days
 - Lasting only until the noxious stimulus is removed or the underlying damage has healed
 - Resolves quickly
 - Subacute pain:
 - Lasting 1–6 mo
 - Chronic pain:
 - Lasts more than 3–6 mo
 - Pain that extends beyond the expected period of healing
- Numerical Rating Scale (NRS):
 - Patients estimate their pain intensity on a scale from 0 to 10
- Visual Analog Scale (VAS):
 - Patients indicate their pain by a position along a 10 cm continuous line between 2 end points, the left one representing no pain and the right one the worst pain they can imagine.

- Clinically relevant change varies from 13 to 19 mm on a VAS or 1.3–1.9/10 on an NRS.
- Faces Pain Scale:
 - Self-report measure of pain intensity developed for children (4–10 yr old).
- DN4 test:
 - Screening tool for neuropathic pain
 - The score ranges from 0 to 10
 - A score of 4 or more classifies the pain as neuropathic rather than nociceptive.
 - Pain characteristics:
 - Burning? (Yes = 1)
 - Painful cold (Yes = 1)
 - Electric shocks (Yes = 1)
 - Symptoms associated with the pain in the same area:
 - Tingling (Yes = 1)
 - Pins and needles (Yes = 1)
 - Numbness (Yes = 1)
 - Itching (Yes = 1)
 - Decrease in touch sensation (Yes = 1)
 - Decrease in prick sensation (Yes = 1)
 - Can the pain be caused or increased by brushing (Yes = 1)
- Remember to always use the same assessment tool for an individual patient.

Physical-Exam

- Observation needed to determine pain scale in nonverbal patients:
 - Vocalization, e.g., whimpering, groaning, crying, or moaning
 - Facial expression, e.g., looking tense, frowning, grimacing, looking frightened
 - Analgesic attitudes aimed to protect a body zone in rest position (seated or lengthened)
 - Careful movements, spontaneously or when asked.
- All aspects of the physical exam should be gently done.
- Posture, point tenderness, percussion tenderness, passive and active range of motion as well as active resistance.
- It is recommended to move smoothly between the different components of the exam while warning the patient about each phase.
- Always examine uninjured tissues first and avoid sudden movement.
- Repeat physical exam after pain relief.

DIAGNOSIS TESTS & INTERPRETATION

Perform any exam and lab or radiographic studies as indicated by the patient's condition.

ESSENTIAL WORKUP

- Obtain complete history of pain.
- When a person is nonverbal and cannot self-report pain, obtain history from caregivers/other relatives/friends/neighbors.

DIAGNOSIS TESTS & INTERPRETATION

As appropriate for medical condition(s)

Imaging

As appropriate for medical condition(s)

Diagnostic Procedures/Surgery

As appropriate for medical condition(s)

DIFFERENTIAL DIAGNOSIS

- Drug-seeking behavior in opioid dependent patients:
 - Frequent use of emergency facilities, moving from 1 provider to another without coordinated care.
 - Unclear history of illness, only subjective complaints (difficult to objectively verify).
 - Patients tend to be obsessive and impatient, and request repeatedly analgesic medications.
 - Some aspects of the physical exam should be inconsistent.
 - Lab and radiologic studies may remain normal.



TREATMENT

PRE HOSPITAL

- Nonpharmacologic measures are effective in providing pain relief in a pre-hospital setting.
- Nitrous oxide is an effective analgesic agent in pre-hospital situations.
- Morphine, fentanyl, and tramadol can be used in a pre-hospital setting.

INITIAL STABILIZATION/THERAPY

- ABCs
- Treat life-threatening medical/traumatic conditions as appropriate.
- Patients with severe pain should be triaged as a priority and dispatched in a rapid care sector, ensuring rapid pain control.

ED TREATMENT/PROCEDURES

- Nonpharmacologic measures are effective in providing pain relief and should be systematic:
 - Immobilization of injured extremities.

- Elevation of injured extremities.
- Ice.
- Opioids for severe pain:
 - Preferably IV or intraosseous if IV not possible
 - Wide interindividual variability in dose response and the delayed absorption with IM or SC routes
 - Oral opioids associated with acetaminophen represent reasonable alternatives for less severe pain:
 - Oxycodone 5–10 mg
 - Hydrocodone 5–10 mg
 - Codeine 30–60 mg
 - Tramadol 50–100 mg
- Nonsteroidal anti-inflammatory drugs:
 - Mild to moderate trauma pain
 - Musculoskeletal pain
 - Renal and biliary colic
 - Relatively high rate of serious adverse effects including GI bleeding and nephropathy.
- Acetaminophen provides safe and effective analgesia for mild to moderate pain with minimal adverse effects.
- Treat associated anxiety or emotion.
- Regional anesthesia should be considered for acute well-localized problems such as toothache, fractures, hand and foot injuries.

MEDICATION

- Acetaminophen: 500 mg (peds: 10–15 mg/kg, do not exceed 5 doses/24h) PO q4–6h, do not exceed 4 g/24h
- Codeine: 30–60 mg PO q4–6h prn
- Morphine:
 - Initial bolus of 0.05–0.1 mg/kg IV
 - 15–30 mg PO q4–6h
- Hydromorphone:
 - Initial bolus 1 mg IV
 - 2–4 mg PO q4–6h
- Oxycodone: 5–10 mg PO
- Hydrocodone: 5–10 mg PO
- Tramadol: 50–100 mg PO
- Hydrocodone/acetaminophen: 5/500 mg PO q4–6h
- Ibuprofen: 600–800 mg PO q6–8h (peds: 10 mg/kg q6h)
- Naproxen: 250–500 mg PO q12h



FOLLOW-UP

DISPOSITION

Admission Criteria

Disposition determined by medical condition and persistence of pain.

- Medical condition requiring admission.
- Uncontrolled pain.

Discharge Criteria

- Medical condition(s) addressed
- Pain relief defined as a final evaluation of pain $\leq 3/10$, or a decrease of pain $\geq 50\%$ from the baseline, or if the acceptable level of pain is reached for an individual patient.
- Physicians may control pain well in the ED with IV titration, but risk poor pain control after discharge with oral opioids:
 - Be aware of conversion rates between opioids.
 - Be aware of conversion from IV to oral dosing.
 - Opioids should be prescribed at fixed intervals to control pain, with additional as-needed doses as required.

Issues for Referral

Recurrence of pain despite adequate analgesic treatment or new unexpected pain requires a reassessment of the diagnosis and consideration of alternative causes for the pain.

FOLLOW-UP RECOMMENDATIONS

As appropriate for medical condition(s).

PEARLS AND PITFALLS

- In case of severe pain, initiate pain relief simultaneously with the primary assessment.
- Regular assessment of pain leads to improved pain management.
- Nonpharmacologic measures are effective in providing pain relief and should always be considered and used when possible.
- Titrating relatively high doses of opioid provides the best chance of delivering rapid and effective analgesia.

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emergency department: Results of a multicentre prospective study. *Eur J Anaesthesiol.* 2011;28(2):97–105.

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CODES

ICD9

- 338.19 Other acute pain
- 338.29 Other chronic pain
- 780.96 Generalized pain

ICD10

- G89.4 Chronic pain syndrome
- G89.29 Other chronic pain
- R52 Pain, unspecified

PANCREATIC TRAUMA

Stephen R. Hayden



BASICS

DESCRIPTION

- Direct epigastric blow compressing pancreas against vertebral column resulting in blunt trauma
- Injury to pancreas from penetrating object

Pediatric Considerations

- Trauma affects proportionately larger areas, leading to multisystem injuries.
- Children have less protective muscle and SC tissue.
- Malpositioned seat belts and child abuse need to be considered in small children.
- Children will less often present with hypotension.

ETIOLOGY

- Penetrating trauma: Most common mechanism
- Blunt trauma: Deep location of pancreas requires significant force to cause injury:
 - Steering wheel, seat belts, or bicycle handlebars to abdomen
 - In children, evaluate for nonaccidental trauma

COMMONLY ASSOCIATED CONDITIONS

90% of pancreatic injuries associated with injuries to adjacent structures:

- Liver, stomach
- Major arteries and veins
- Spleen, kidney
- Duodenum, colon, small bowel
- Common bile duct, gallbladder
- Spine: Chance fracture



DIAGNOSIS

ALERT

Extent of pancreatic injury may not be apparent on initial evaluation.

SIGNS AND SYMPTOMS

- Abdominal pain:
 - Diffuse or epigastric
 - Often out of proportion to physical exam and vital signs
- Soft-tissue contusion in upper abdomen

- Injury to lower ribs or costal cartilage
- Acute abdomen, often associated with other intra-abdominal injuries
- Concomitant splenic injury can present initially as dull back pain
- Hypotension
- Grey Turner sign:
 - Flank ecchymosis
- Cullen sign:
 - Periumbilical ecchymosis

History

Concise; details of incident especially important for blunt trauma

Physical-Exam

- Inspect for abrasions, contusions, penetrating wounds:
 - Must log roll patient for full inspection.
 - Look for seat belt-related injuries.
- Auscultate for presence or absence of bowel sounds.
- Palpate to determine location and severity of pain, presence of guarding, and rebound tenderness.
- Rectal exam for occult blood, vaginal exam, or penile exam
- Serial physical exams and vital signs for unidentified injuries

ALERT

Vascular injury is the most common cause of mortality related to pancreatic injury. Suspicion necessitates immediate evaluation and possible surgical exploration.

ESSENTIAL WORKUP

- Pace of workup is dictated by patient condition and other injuries.
- Abdominal CT with IV contrast is essential to evaluate for pancreatic trauma.
- MRCP is being used more frequently in trauma centers to better evaluate ductal injury.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Blood type, screen, or cross-match
- Hematocrit, WBC with differential, complete metabolic profile
- Amylase:
 - Not a reliable indicator of pancreatic trauma
 - Serial levels may increase sensitivity, but specificity still poor.
 - Elevated amylase may be early indicator of potential pancreatic injury.
 - Normal amylase does not rule out pancreatic injury.
 - More sensitive and specific if detected in diagnostic peritoneal lavage (DPL)

fluid

- Lipase:
 - No more specific for pancreatic injury
- Urinalysis
- Pregnancy test
- Alcohol and drug screening if indicated
- Prothrombin time/partial thromboplastin time, BUN, and creatinine

Imaging

- Note that all imaging tests may miss pancreatic injury.
- Cervical spine, CXRs, and pelvis films as for all blunt trauma patients
- Bedside US/FAST scan
- CT scan with IV contrast, helical/MDCT if available:
 - Shows better contrast enhancement of pancreatic parenchyma than standard scanning
 - MDCT is particularly useful in pediatric populations
- Magnetic retrograde cholangiopancreatography:
 - Noninvasive evaluation of injury to ductal components
- Endoscopic retrograde cholangiopancreatography:
 - Useful for patients with persistent hyperamylasemia
 - Unexplained abdominal symptoms
 - Some advocating early use to minimize complications
- Operative exploration and intraoperative cholangiogram remains the ideal diagnostic modality, particularly if patient is unstable.

Diagnostic Procedures/Surgery

DPL to identify intraperitoneal injuries:

- Check fluid for amylase level.
- May still miss significant pancreatic injury

DIFFERENTIAL DIAGNOSIS

Other or associated abdominal traumatic injuries



TREATMENT

PRE HOSPITAL

Transport to closest trauma center.

INITIAL STABILIZATION/THERAPY

- Airway management, resuscitation as indicated with crystalloids, colloids, or blood products
- Nasogastric-tube suction may be especially helpful in the setting of pancreatic

trauma.

GENERAL MEASURES

Follow standard trauma treatment for blunt abdominal trauma:

- Penetrating trauma:
 - Tetanus prophylaxis and broad-spectrum antibiotic therapy
- Intra-abdominal injury requiring operative intervention:
 - Broad-spectrum antibiotic therapy
- Must cover for colonic bacteria:
 - Aerobic: *Escherichia coli*, *Enterobacter*, *Klebsiella*, *Enterococcus*
 - Anaerobic: *Bacteroides fragilis*, *Clostridium*, *Peptostreptococcus*

ED TREATMENT/PROCEDURES

Follow ABCDE of trauma and resuscitate unstable patient with emergent surgical consultation or transfer to trauma center as indicated:

- Evaluate for associated abdominal injury.
- Choose imaging modality for rapid evaluation (CT and/or MRCP).
- Early identification of ductal injuries has been shown to reduce morbidity and mortality.
- Surgical: Pancreaticoduodenectomy, distal pancreatectomy, endoscopic stent (controversial), sump/closed suction drainage
- East Trauma Guidelines 2009
- Level III evidence: Grade I and II injuries: Drainage Grade III–V injuries: Resection and drainage

MEDICATION

- Adults:
 - Piperacillin/tazobactam 3.375 g IV OR
 - Cefotetan: 2 g IV + gentamicin 2 mg/kg IV OR
 - Cefoxitin: 2 g IV + gentamicin 2 mg/kg IV OR
 - Ceftriaxone: 1–2 g IV + Flagyl 15 mg/kg IV OR
 - Clindamycin: 600 mg IV + gentamicin 2 mg/kg IV
- Children:
 - Cefotetan: 20 mg/kg IV + gentamicin 2 mg/kg IV OR
 - Cefoxitin: 40 mg/kg IV + gentamicin 2 mg/kg IV OR
 - Ceftriaxone: 50 mg/kg per dose IV + Flagyl 15 mg/kg IV

First Line

Ceftriaxone and Flagyl or piperacillin/tazobactam or carbapenem:

- Goal is to choose broad-spectrum coverage with both aerobic and anaerobic coverage, particularly of enteric gram-negative organisms.

Second Line

Addition of an aminoglycoside, as it has good activity in an alkaline environment:

- Particularly useful if patient is unstable for broader gram-negative coverage

Adjunct Therapy

There is no good evidence to support the use of octreotide as studies still conflict on the benefits and adverse effects.



FOLLOW-UP

DISPOSITION

Admission Criteria

- All patients with suspected pancreatic injuries must be admitted.
- Abdominal pain after blunt trauma requires serial exam and observation for 24–72 hr.
- Intoxicated trauma patient requires admission and serial exams for unidentified injury.

Discharge Criteria

Only for very minor trauma and with no evidence of pancreatic or any other intra-abdominal injury with appropriate follow-up and return precautions

Issues for Referral

- Patient with surgical drains or complications such as fistula formation may need further surgical, GI, and wound care evaluations.
- Most patients need close monitoring and follow-up within 1 wk.

FOLLOW-UP RECOMMENDATIONS

Delayed presentation of pancreatic injury is rare, but complications may arise and should be considered:

- Pancreatitis, pseudocysts, vascular aneurysms (such as splenic artery)
- Rare for exocrine or endocrine dysfunction to occur unless a majority of the pancreas is resected/destroyed:
 - Evaluate for glucose intolerance and digestive abnormalities

PEARLS AND PITFALLS

- Always consider pancreatic injury when evaluating abdominal or back trauma, both blunt and penetrating.
- Beware of nearby vascular injuries.
- Assess for related injuries.
- Choose the best imaging modality and obtain as rapidly as possible.

- Penetrating trauma or unstable patients should be rapidly prepared for surgical exploration.

ADDITIONAL READING

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- Vasquez JC, Coimbra R, Hoyt DB, et al. Management of penetrating pancreatic trauma: An 11-year experience of a level-1 trauma center. *Injury*. 2001;32(10):753–759.
- Wolf A, Bernhardt J, Patrzyk M, et al. The value of endoscopic diagnosis and the treatment of pancreas injuries following blunt abdominal trauma. *Surg Endosc*. 2005;19(5):665–669.



CODES

ICD9

- 863.84 Injury to pancreas, multiple and unspecified sites, without mention of open wound into cavity
- 863.94 Injury to pancreas, multiple and unspecified sites, with open wound into cavity

ICD10

- S36.209A Unspecified injury of unspecified part of pancreas, initial encounter
- S36.229A Contusion of unspecified part of pancreas, initial encounter
- S36.239A Laceration of unspecified part of pancreas, unspecified degree, initial encounter

PANCREATITIS

Trevor Lewis



BASICS

DESCRIPTION

- Inflammation of pancreas due to activation, interstitial liberation, and digestion of gland by its own enzymes
- Acute pancreatitis:
 - Exocrine and endocrine function of gland impaired for weeks to months
 - Glandular function will return to normal.
- Chronic pancreatitis:
 - Exocrine and endocrine function progressively deteriorates with resultant steatorrhea and malabsorption.
 - Dysfunction progressive and irreversible
- Pancreatic pseudocyst:
 - Cystic collection of fluid with high content of pancreatic enzymes surrounded by a wall of fibrous tissue lacking a true epithelial lining
 - Localized in parenchyma of pancreas or adjacent abdominal spaces (lesser peritoneal sac)
 - Requires 4–6 wk to form from onset of acute pancreatitis

ETIOLOGY

- Gallstones and alcohol abuse most common causes of *acute pancreatitis* (75–80%)
- Alcohol abuse accounts for 70–80% of *chronic pancreatitis*.
- Acute:
 - Biliary tract disease
 - Chronic alcoholism
 - Obstruction of pancreatic duct
 - Ischemia
 - Medications
 - Infectious
 - Postoperative
 - Post-ERCP
 - Metabolic diseases
 - Trauma
 - Scorpion venom
 - Penetrating peptic ulcer
 - Hereditary
- Chronic:
 - Chronic alcoholism

- Obstruction pancreatic duct
- Tropical
- Hereditary
- Shwachman disease
- Enzyme deficiency
- Idiopathic
- Hyperlipedemia
- Hypercalcemia
- Pancreatic pseudocyst:
 - Complication in 5–16% of acute pancreatitis; 20–40% of chronic pancreatitis

Pediatric Considerations

Causes mainly viral, trauma, and medications

DIAGNOSIS

SIGNS AND SYMPTOMS

- Frequency:
 - Abdominal pain: 95–100%
 - Epigastric tenderness: 95–100%
 - Nausea and vomiting: 70–90%
 - Low-grade fever: 70–85%
 - Hypotension: 20–40%
 - Jaundice: 30%
 - Grey Turner/Cullen sign: <5%
 - Subcutaneous or SQ
- GI:
 - Severe, persistent epigastric pain radiating to back:
 - Colicky or rebound tenderness suggests nonpancreatic source.
 - Worse when supine
 - Nausea, vomiting, and anorexia
 - Bowel sounds usually decreased or absent
 - Significant GI bleed in patients with acute severe pancreatitis is uncommon.
 - Cullen sign:
 - Bluish discoloration at umbilicus secondary to hemorrhagic pancreatitis
 - Grey Turner sign:
 - Bluish discoloration at flank secondary to hemorrhagic pancreatitis
- Respiratory:
 - Pleuritic chest pain
 - Dyspnea

- Lung exam:
 - o Left pleural effusion (most common)
 - o Atelectasis
 - o Pulmonary edema
- Hypoxemia (30%)
- Cardiac:
 - Tachycardia
 - Hypotension
 - Shock
- Neurologic:
 - Irritability
 - Confusion
 - Coma
 - Chvostek and Trousseau signs are rare despite lab evidence of hypocalcemia.

Ranson Criteria

- Indicators of morbidity and mortality:
 - 0–2 criteria: 2% mortality
 - 3 or 4 criteria: 15% mortality
 - 5 or 6 criteria: 40% mortality
 - 7 or 8 criteria: 100% mortality
- Criteria on admission:
 - Age > 55 yr
 - WBC count > 16,000 mm³
 - Blood glucose > 200 mg/dL
 - Serum lactate dehydrogenase > 350 IU/L
 - AST > 250 IU/L
- Criteria during 1st 48 hr:
 - Hematocrit fall > 10%
 - BUN increase > 5 mg/dL
 - Serum calcium < 8 mg/dL
 - Arterial PO₂ < 60 mm Hg
 - Base deficit > 4 mEq/L
 - Estimated fluid sequestration > 6 L

ESSENTIAL WORKUP

Lab tests to confirm physical diagnosis

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Lipase:
 - Rises within 4–8 hr of pain onset
 - More reliable indicator of pancreatitis than amylase
- Amylase:
 - Rises within 6 hr of pain onset
 - Levels > 3 times limit of normal suggest pancreatitis.
 - Levels > 1,000 IU suggest biliary pancreatitis.
 - May be normal during acute inflammation due to significant pancreatic destruction
 - Secreted from various sources
- Electrolyte, BUN, creatinine, glucose:
 - Hypokalemia occurs with extensive fluid losses.
 - Hyperglycemia
- CBC:
 - Increased hematocrit with fluid losses
 - Hematocrit > 47% at risk for pancreatic necrosis
 - Decreased hematocrit with retroperitoneal hemorrhage
 - WBC count > 12,000 unusual
- Calcium/magnesium:
 - Hypocalcemia indicates significant pancreatic injury.
 - Hypomagnesemia occurs with underlying alcohol abuse.
- Liver function tests:
 - Useful for prognostic indicators if suspected biliary cause
- CRP:
 - Useful to measure severity at 24–48 hr after symptoms onset
- Pregnancy test
- Arterial blood gases:
 - Indicated if hypoxic (assess PO₂) or toxic appearing (assess base deficit)
- ECG:
 - Assess electrolyte imbalances, ischemia

Imaging

- Abdominal series radiograph:
 - Excludes free air
 - May visualize pancreatic calcifications
 - Most common finding is isolated dilated bowel loop (sentinel loop) near pancreas.
- Chest radiograph:
 - Pleural effusion
 - Atelectasis
 - Infiltrate
- US:

- Useful if gallstone pancreatitis suspected
- Abdominal CT indications:
 - High-risk pancreatitis (> 3 Ranson criteria)
 - Hemorrhagic pancreatitis
 - Suspicion for pseudocyst
 - Diagnosis in doubt

Diagnostic Procedures/Surgery

Endoscopic retrograde cholangiopancreatography (ERCP):

- Indicated for severe pancreatitis with cholangitis or biliary obstruction

DIFFERENTIAL DIAGNOSIS

- Mesenteric ischemia/infracton
- Myocardial infarction
- Biliary colic
- Intestinal obstruction
- Perforated ulcer
- Pneumonia
- Ruptured aortic aneurysm
- Ectopic pregnancy



TREATMENT

PRE HOSPITAL

- Initiate IV access in cooperative patients.
- Apply cardiac monitor.

INITIAL STABILIZATION/THERAPY

- ABCs
- Supplemental oxygen
- Cardiac monitor
- IV fluids

ED TREATMENT/PROCEDURES

- Airway management:
 - Pulmonary complaints necessitate supplemental oxygen.
 - Endotracheal intubation for adult respiratory distress syndrome or severe encephalopathy
- Fluid resuscitation:
 - Large fluid volumes (up to 5–6 L in 1st 24 hr) to compensate for fluid losses
 - Continuously assess vitals, urine output, and electrolytes to ensure rapid and adequate replacement of intravascular volume.

- Correct electrolyte abnormalities if present:
 - Hypocalcemia (Calcium gluconate)
 - Hypokalemia occurs with extensive fluid losses.
 - Hypomagnesemia occurs with underlying alcohol abuse.
- Blood products:
 - In hemorrhagic pancreatitis, transfuse to hematocrit level of 30%.
 - Fresh-frozen plasma and platelets if coagulopathic and bleeding
- Analgesia:
 - Opiate analgesia is the drug of choice.
- Nasogastric suction:
 - Not useful in cases of mild pancreatitis
 - Beneficial in severe pancreatitis or intractable vomiting
- Antiemetics
- Antibiotics:
 - Indicated if pancreatic necrosis >30% on abdominal CT

Geriatric Considerations

Consider central venous pressure monitoring when fluid overload is a concern.

MEDICATION

First Line

Analgesics, antiemetics:

- Morphine 2–4 mg IV
- Hydromorphone (Dilaudid) 1 mg IV/IM
- Ondansetron 4 mg IV/IM/PO

Second Line

Electrolyte replacement, antibiotics:

- Potassium chloride: 10 mEq/h IV over 1 hr
- Calcium gluconate 10%: 10 mL IV over 15–20 min
- Magnesium sulfate: 2 g IV piggyback
- Imipenem: 500 mg IV q6h



FOLLOW-UP

DISPOSITION

Admission Criteria

- Acute pancreatitis with significant pain, nausea, vomiting
- ICU admission for hemorrhagic/necrotizing pancreatitis

Discharge Criteria

- Mild acute pancreatitis without evidence of biliary tract disease and able to tolerate oral fluids
- Chronic pancreatitis with minimal abdominal pain and able to tolerate oral fluids

Issues for Referral

- Surgical/GI consultation for ERCP in severe pancreatitis with cholangitis or biliary obstruction
- Emergent surgical consultation mandatory in cases of suspected ruptured pseudocyst or pseudocyst hemorrhage, as definitive treatment is emergent laparotomy

FOLLOW-UP RECOMMENDATIONS

All discharged mild pancreatitis should have scheduled follow-up within 24–28 hr.

PEARLS AND PITFALLS

- Gallstones and alcohol account for etiologies of 75–80% of acute pancreatitis.
- Early aggressive fluid therapy is essential to replace large volume losses.
- Nasogastric suction is not beneficial in routine pancreatitis.
- Consider early CT of abdomen when diagnosis in doubt or patient appears ill by clinical scoring scale (Ranson criteria ≥ 3).

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See Also (Topic, Algorithm, Electronic Media Element)



CODES

ICD9

- 577.0 Acute pancreatitis
- 577.1 Chronic pancreatitis
- 577.2 Cyst and pseudocyst of pancreas

ICD10

- K86.1 Other chronic pancreatitis
- K85.2 Alcohol induced acute pancreatitis
- K85.9 Acute pancreatitis, unspecified

PANIC ATTACK

Juliana H. Chen • Bernie Vaccaro



BASICS

DESCRIPTION

- Characteristic, acute episodes of physical symptoms and intense fear that rapidly peak within 10 min and resolve in ~ 20 min
- There may be a nonfearful variant in medical patients.

Panic Disorder

- Recurrent, unexpected panic attacks with ≥ 1 mo of persistence:
 - _ Concerns about having another attack
 - _ Worry about the implications or consequences of the attacks
 - _ Behavioral change, such as phobic avoidance, related to the attacks
 - _ With or without agoraphobia = anxiety related to fear of escape
- Episodic, recurrent, or chronic attacks
- Frequently comorbid with depression, substance abuse, disability, suicidal tendency

Genetics

- Probably genetic
- Family history of panic or anxiety is common
- Altered serotonin- and benzodiazepine-receptor function

ETIOLOGY

Mechanism

Limbic system, norepinephrine release, other neurotransmitters (e.g., serotonin) implicated, leading to “fight-or-flight” response

RISK FACTORS

- Major life events in the year preceding onset
- Family history of panic or anxiety
- Childhood shyness or separation anxiety
- May develop in the course of predisposing physical illness or cocaine abuse:
 - _ May persist after the illness or substance use has resolved
- Twice as common in women



DIAGNOSIS

SIGNS AND SYMPTOMS

- Multiple systems suggest autonomic arousal
- Cardiac:
 - Palpitations
 - Tachycardia
 - Chest pain or discomfort
- Respiratory:
 - Shortness of breath
 - Smothering
 - Feeling of choking
- Neurologic:
 - Tremor
 - Dizziness
 - Lightheadedness
 - Feeling faint
 - Numbness
 - Tingling
 - Sweating
 - Chills
 - Flushing
 - Feelings of unreality or detachment
- GI:
 - Nausea
 - Cramps
 - Abdominal pain
- Intense fears:
 - Automatic, stereotypic
 - Imminent death
 - Having a heart attack
 - Humiliation
 - Loss of control—”going crazy”

History

- Known medical conditions
- All medications, including over the counter
- Herbal supplements
- Recreational drugs/alcohol use
- Caffeine consumption
- Age at onset
- Initiating life events or stressors
- Childhood antecedents
- Resultant avoidance
- Response to previous medication trials

- Family history of panic, anxiety
- Family history of drugs/alcohol use

Physical-Exam

- Thorough physical and neurologic exam
- Guided by particular symptoms

ESSENTIAL WORKUP

Detailed history, appropriate physical exam:

- Guided by presentation and initial findings
- May be minimal, depending on presentation

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Toxicology screen
- Consider tricyclic antidepressant (TCA) level
- CBC
- Electrolytes, BUN/creatinine, glucose
- Thyroid-stimulating hormone
- Pulse oximetry or arterial blood gases

Diagnostic Procedures/Surgery

- ECG for suspected mitral valve prolapse (MVP), to exclude underlying cardiac disease, or to monitor for QRS widening in patients on TCA:
 - Age > 40 yr
 - Cardiac symptoms
- Holter monitor:
 - If palpitations, near-syncope
- Sleep-deprived EEG if seizure suspected

DIFFERENTIAL DIAGNOSIS

- Consider organic causes if:
 - Panic presents late in life (> 50 yr)
 - No childhood antecedents or family history
 - No initiating or major life events
 - Without avoidance or significant fear
 - With a history of poor response to anxiolytic or antidepressant medication
- Medications:
 - Neuroleptics (akathisia)
 - Bronchodilators
 - Digitalis
 - Anticholinergic agents

- Psychostimulants
 - Diet pills
 - Herbal supplements
- Respiratory:
 - Asthma
 - Hyperventilation
 - Chronic obstructive pulmonary disease
 - Pulmonary embolus
 - Bacterial pneumonia
 - Costochondritis
- Cardiovascular:
 - Angina
 - Myocardial infarction
 - Arrhythmia
 - Anemia
 - MVP
- Substances:
 - Stimulant abuse
 - Withdrawal (alcohol, sedative–hypnotics)
 - Antidepressant discontinuation syndrome (with interruption, dose decrease, or discontinuation of SSRI or SNRI)
 - Excessive caffeine intake
- Endocrine:
 - Hyperthyroidism
 - Hypoglycemia
 - Hypoparathyroidism
 - Pheochromocytoma
- Other metabolic derangements:
 - Hypokalemia
 - Hypomagnesemia
 - Hypophosphatemia
- Neurologic:
 - Complex partial or limbic seizures (fear, physical symptoms, perceptual distortions)
 - Transient ischemic attack
 - Labyrinthitis
 - Benign positional vertigo
- Psychiatric:
 - Obsessive-compulsive disorder
 - Post-traumatic stress disorder
 - Specific phobia or social phobia
 - Somatoform disorder
 - Factitious disorder

- Acute grief
- Domestic violence

Pediatric Considerations

Tachycardia



TREATMENT

PRE HOSPITAL

- If diagnosis is supported by previous events, history and workup:
 - Reassurance and diversion
 - Does not require emergent care
- If 1st episode, treat and transport as appropriate to presentation

INITIAL STABILIZATION/THERAPY

- Be calm and reassuring.
- Most panic attacks resolve within 20–30 min without any treatment.
- Fear may trigger another panic attack.

ED TREATMENT/PROCEDURES

- Patient education, new cognitions:
 - Normal response to abnormal alarm
 - Physiologic explanations for symptoms
- High-potency benzodiazepines (*drugs of choice*):
 - Clonazepam:
 - Slow for emergency use
 - Long-acting without rapid onset/offset phenomena
 - Best choice in this class for maintenance therapy of recurrent panic attacks
 - Alprazolam:
 - Rapid onset
 - Rebound anxiety occurs due to short duration and rapid offset.
 - May lead to escalating doses with continued use
 - Lorazepam:
 - Quick onset
 - Advantage of sublingual (SL) use
 - Longer effect and less abrupt offset than alprazolam
- Avoid low-potency benzodiazepines:
 - Diazepam
 - Chlordiazepoxide
- Treat recurrent panic attacks and panic disorder with selective serotonin reuptake inhibitors (SSRIs) (or TCAs), with or without clonazepam:

- Will not work immediately
- Do not need to be started emergently, especially if there is no clear, established access to follow-up management
- There are a few small studies on the efficacy of atypical antipsychotics (e.g., olanzapine, risperidone) for treatment-resistant panic disorder. However, data to support this use is limited.
- Discharge therapy:
 - Several clonazepam tablets in case of repeated attacks

ALERT

Rapid offset (withdrawal) of alprazolam may trigger further attacks.

MEDICATION

First Line

- Clonazepam: 0.5 mg PO in the ED; 0.25–0.5 mg PO BID for initial outpatient therapy
- SSRI:
 - To be started as an outpatient
 - May require higher doses and longer time to therapeutic response for panic than for depression

Second Line

- Lorazepam: 1 mg PO or SL
- TCA:
 - To be started as an outpatient

Pregnancy Considerations

- Limit use of benzodiazepines.
- Risk/benefit discussion about the relative safety of SSRIs and less anticholinergic TCAs (e.g., nortriptyline, desipramine)
- Physiologic and autonomic effects of pregnancy and postpartum period may trigger attacks in predisposed women.



FOLLOW-UP

DISPOSITION

Admission Criteria

- As medically indicated to rule out organic cause
- Meets criteria for psychiatric admission (suicidal, homicidal)

Discharge Criteria

Most panic attacks do not require inpatient level of care.

Issues for Referral

- Managed care mental health carve-outs
- Psychopharmacologic and cognitive behavioral therapy evaluation for repeated attacks, or interepisode fear or avoidance
- Stigma
- Primary care follow-up may be an acceptable alternative to specialty, mental health/psychiatry referral.

FOLLOW-UP RECOMMENDATIONS

- Appointment with primary care physician or referral to mental health specialty treatment
- Avoid precipitants, e.g., caffeine, stimulants, alcohol.

PEARLS AND PITFALLS

- Panic is “contagious.” Try not to be infected by the patient’s sense of urgency to stop the symptoms; they will resolve spontaneously.
- Be calm so as not to add to the patient’s alarm, but diligent, so patient feels attended to and reassured.
- Cognitive–behavioral therapy (CBT) can start in the ED with brief explanation of the physiologic cause of symptoms.
- Be cautious not to start adolescents and young adults on a lifetime course of benzodiazepines; CBT (\pm SSRI therapy) is associated with good outcomes and fewer deleterious side effects.
- Avoid the use of alprazolam, especially for ongoing treatment.

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See Also (Topic, Algorithm, Electronic Media Element)

- [Psychosis, Medical vs. Psychiatric](#)
- [Withdrawal, Drug](#)



CODES

ICD9

- [300.01 Panic disorder without agoraphobia](#)
- [300.21 Agoraphobia with panic disorder](#)

ICD10

- [F40.01 Agoraphobia with panic disorder](#)
- [F41.0 Panic disorder without agoraphobia](#)

PARAPHIMOSIS

Nicole M. Franks

BASICS

DESCRIPTION

- The entrapment of the retracted foreskin proximal to the glans of the penis
- Leads to lymphatic congestion and venous obstruction, which may result in arterial compromise to the glans
- Paraphimosis is a urologic emergency.

ETIOLOGY

- A number of conditions of the foreskin may predispose to paraphimosis, including:
 - Phimosis
 - Inflammation
 - Trauma
 - Sexually naive may be unaware of the need to reduce foreskin after intercourse
- Commonly iatrogenic, from failure to replace the foreskin after exam, catheterization, or cleaning

DIAGNOSIS

SIGNS AND SYMPTOMS

- Retracted prepuce (foreskin)
- Pain
- Swollen, edematous glans
- Local cellulitis
- Necrosis of glans in untreated cases

Physical-Exam

Exam of the genitalia should include a search for constricting foreign bodies or constricting bands.

ESSENTIAL WORKUP

- Paraphimosis is a clinical diagnosis with the clinical findings described earlier.
- Treatment must not be delayed pending diagnostic lab or radiographic studies.

DIAGNOSIS TESTS & INTERPRETATION

Imaging

If history suggests penile foreign body, radiographs may be obtained once the vascular compromise has been relieved.

DIFFERENTIAL DIAGNOSIS

- Foreign bodies constricting the penile shaft may mimic paraphimosis; these include:
 - Hair tourniquets
 - Wire, string, or other materials used for sexual enhancement or punishment
- Balanoposthitis
- Trauma (zipper injuries)
- Acute idiopathic penile edema



TREATMENT

PRE HOSPITAL

- Patients should be transported promptly; do not attempt reduction in the field.
- Pre-hospital personnel can be advised to apply an ice pack to the glans with adequate protection of the skin.
- Pain control

INITIAL STABILIZATION/THERAPY

- Ice can be applied to the glans while preparing to reduce the prepuce:
 - Use the thumb of a glove as an ice-filled condom to aid in direct application.
- The incarcerated foreskin must be released as soon as possible to prevent ischemia and necrosis of the glans.
- The pain associated with reduction techniques must be managed with some combination of conscious sedation, adequate analgesia, and local anesthesia.

ED TREATMENT/PROCEDURES

- Medical therapy for paraphimosis involves reassuring the patient, reducing the preputial edema, and restoring the prepuce to its original position and condition.
- The following sequence of procedures should be followed:
 - Paraphimosis can most frequently be reduced using a penile block and compressing the glans manually while applying traction on the foreskin.
 - Penile block is performed by infiltrating 5 mL of 1% lidocaine *without* epinephrine in the angle between the inferior rami of the symphysis pubis:
 - Then use another 5 mL to infiltrate a wheel along the sides of the penis.
 - This produces a block after 5 min.
 - Successful reduction requires steady circumferential pressure on the distal edema with simultaneous manual reduction of the foreskin.
 - In children, conscious sedation is usually required.

- If manual reduction is unsuccessful, then the technique of multiple punctures may facilitate reduction:
 - Make ~20 holes in the swollen foreskin with a small sterile needle (26G), allowing expression of edema fluid, then resume manual reduction.
- If this fails to return the foreskin to its original position, it will be necessary to incise the constricting ring of tissue with a dorsal longitudinal slit in the foreskin after sterile preparation:
 - If the incision made is too long, after reduction it may be necessary to suture the incision transversely with 3.0 absorbable sutures.
- If a delay is likely before the paraphimosis can be treated (e.g., NPO status), then applying a gauze swab soaked in 50% dextrose will reduce edema by osmosis and facilitate reduction.
- For patients who want to retain uncircumcised phallus steroid therapy can be attempted to reduce fibrose ring. Consult urology for close follow-up:
 - Triamcinolone cream 0.1% to affected area × 6 wk
 - If unsuccessful, circumcision may still be required.

MEDICATION

- Appropriate analgesics or anesthetics as required
- Antibiotics generally not required unless treating associated cellulitis or balanoposthitis.



FOLLOW-UP

DISPOSITION

Admission Criteria

Necrosis or cellulitis of the penis

Discharge Criteria

- Successful reduction with relief of symptoms
- Close urologic follow-up

Issues for Referral

- Urologic consultation is required.
- Subsequent circumcision to prevent recurrence is an area of clinical debate; historically, it has been common practice.

FOLLOW-UP RECOMMENDATIONS

- Education regarding importance of replacement of the foreskin after retraction for instrumentation or cleaning

- Emphasis on prepuce hygiene

PEARLS AND PITFALLS

- Goal is to reduce penile edema enough to allow the foreskin to return to original position over the glans.
- Generally, noninvasive reduction methods (at least 2 or 3 attempts) are successful and dorsal slit incision is mostly required only in severe cases.

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See Also (Topic, Algorithm, Electronic Media Element)

- Phimosis
- Priapism



CODES

ICD9

605 Redundant prepuce and phimosis

ICD10

N47.2 Paraphimosis

PARKINSON DISEASE

Adam Z. Barkin

BASICS

DESCRIPTION

- Gradual progressive neurologic disorder of middle or late life
- Degeneration of dopaminergic neurons in the substantia nigra
- Development of Lewy bodies in the residual dopaminergic neurons
- Accelerated cortical atrophy
- Can begin unilaterally, but generalizes to symmetric
- Affects 1% of people > 60 yr; 4% > 80 yr
- May have symptoms 20 yr prior to diagnosis
 - _ Nonspecific:
 - Fatigue
 - Constipation
 - Hyposomnia

ETIOLOGY

- Sporadic or idiopathic
- Disorders presenting with parkinsonism:
 - _ Drug induced:
 - Parkinsonism-hyperpyrexia syndrome (dopaminergic drug withdrawal)
 - Amphotericin B
 - Chemotherapeutic drugs
 - Neuroleptic treatment induced
 - _ Toxins:
 - Carbon monoxide
 - Methanol
 - Cyanide
 - Organophosphate poisoning
 - 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine
 - _ Brain lesions:
 - Basal ganglia stroke
 - Midbrain lesions
 - Hydrocephalus
 - _ Infections:
 - *Mycoplasma*
 - Viral encephalitis
 - _ Other:

- Central pontine myelinosis
- Encephalitis lethargica (autoantibodies against basal ganglia antigens)

DIAGNOSIS

SIGNS AND SYMPTOMS

- Nonmotor vs. motor symptoms:
 - _ Nonmotor:
 - Orthostatic hypotension
 - Constipation
 - Delayed gastric emptying
 - Dysphagia
 - Pain sensory dysfunction
 - Depression
 - Hallucinations
 - Dementia
 - Sleep disorders
 - Motor symptoms:
 - _ “Pill-rolling” resting tremor
 - _ “Cog-wheel” rigidity due to increased muscular tone
 - _ Stooped posture and instability of posture
 - _ Bradykinesia: Extreme slowness in movement
 - _ “Masked face” appearance

History

- Sudden change in baseline motor function or mental status:
 - _ May be the only indication of systemic disease such as infection
- Noncompliance (sudden withdrawal) of dopaminergic medications can lead to parkinsonism-hyperpyrexia syndrome:
 - _ Rigidity, pyrexia, reduced consciousness
 - _ Complications:
 - Acute renal failure
 - Venothrombosis
 - Disseminated intravascular coagulation
 - Rhabdomyolysis
 - Autonomic instability

Physical-Exam

- Cog-wheel rigidity:
 - _ Jerking movements when a muscle is passively stretched
- Stooped posture

- Pill-rolling tremor

ESSENTIAL WORKUP

- History is of primary importance:
 - Diagnosis is made based on clinical findings
- Important historical information includes:
 - Onset of symptom, whether gradual or sudden
 - History of potential causes of a Parkinson-like syndrome
 - Patients with established Parkinson disease (PD):
 - Sudden change in baseline motor function
 - Change in mental status
 - Should prompt workup for infectious process

DIAGNOSIS TESTS & INTERPRETATION

Lab

- No specific or recommended lab studies necessary to confirm the diagnosis
- Disorders presenting as PD may require directed lab studies as appropriate for suspected cause
- Directed labs if suspect parkinsonism-hyperpyrexia syndrome

Imaging

- CT and MRI are not required to diagnose PD but are often elements of evaluation for dementia
- CXR may be indicated for any signs of respiratory tract infection

DIFFERENTIAL DIAGNOSIS

- Benign familial tremor
- Major depression
- Wilson disease
- Huntington disease
- Alzheimer disease
- Creutzfeldt–Jakob disease
- Carbon monoxide poisoning
- B₁₂ deficiency
- Hydrocephalus
- Multi-infarct dementia
- Essential tremor disorders
- Hypothyroidism
- Dementia with Lewy bodies



TREATMENT

ED TREATMENT/PROCEDURES

- Treatment with antiparkinsonian medications can be initiated in the ED to alleviate symptoms
- Consultation with neurology for recommended medication regimens and ongoing support and monitoring is prudent
- For patients with mild disease, no medication may be required
- For moderate disease, anticholinergic medications and dopaminergic medications should be used
- Treat underlying infection, if present
- Treat parkinsonism-hyperpyrexia syndrome:
 - Replace levodopa or bromocriptine
 - Supportive
 - Treat complications

MEDICATION

- PD:
 - Amantadine: 100 mg BID
 - Stimulates dopamine release
 - Benztropine: 0.5–1 mg TID
 - Anticholinergic
 - Limited use in tremor-dominant PD
 - Carbidopa/levodopa: 25/100 mg TID
 - Carbidopa lessens peripheral side effects and increased levodopa CNS bioavailability
 - Levodopa is direct precursor to dopamine
 - Entacapone: 200 mg PO BID–QID
 - Adjunct therapy; should be administered concomitantly with carbidopa/levodopa
 - Increases CNS levodopa bioavailability
 - MAO inhibitors
 - May be used in mild disease as first-line therapy
 - Selegiline: 5 mg qam and noon
 - Rasagiline: 1–2 mg QD
 - Dopamine agonists:
 - Pramipexole: 0.5–1.5 mg PO TID
 - Ropinirole: 3–6 mg PO TID
 - Apomorphine: 0.2–0.6 mL SQ PRN
- Parkinsonism-hyperpyrexia syndrome:
 - Levodopa: 50–100 mg IV over 3 hr
 - Bromocriptine: 7.5–15 mg PO TID

First Line

Carbidopa/levodopa



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients with previously diagnosed Parkinson with infections, trauma, cardiovascular emergencies, cerebrovascular emergencies, GI emergencies, electrolyte disturbances, altered mental status, or other medical problems
- Depression with intent to do self-harm
- Confirm diagnosis and levodopa responsiveness
- Medication complications (parkinsonism-hyperpyrexia syndrome)
- Management of motor fluctuations and dyskinesias
- Inability to go home secondary to elder abuse
- Complications from deep brain stimulation devices (e.g., headache, infection, mental status change)
- Failure to thrive

Discharge Criteria

- Mild to moderate disease without medications
- Moderate to severe disease with medications and urgent neurologic outpatient follow-up

FOLLOW-UP RECOMMENDATIONS

Discuss prevention strategies in disease management

PEARLS AND PITFALLS

- Diagnosis is often difficult; keep in mind other conditions commonly misdiagnosed as PD
- Sudden withdrawal of dopaminergic medications can result in parkinsonism-hyperpyrexia syndrome, a medical emergency

ADDITIONAL READING

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CODES

ICD9

- 332.0 Paralysis agitans
- 332.1 Secondary parkinsonism
- 333.0 Other degenerative diseases of the basal ganglia

ICD10

- G20 Parkinson's disease
- G21.9 Secondary parkinsonism, unspecified
- G21.19 Other drug induced secondary parkinsonism

PARONYCHIA

Gene Ma

BASICS

DESCRIPTION

- Disruption of the seal between the nail plate and the nail fold may allow entry of bacteria into the eponychial space.
- Inflammation of the nail folds surrounding the nail plate

ETIOLOGY

- Acute paronychia: Predominantly *Staphylococcus aureus* but also streptococci, *Pseudomonas*, and anaerobes
- Chronic paronychia: Multifactorial due to allergens and irritants in addition to fungal etiologies, predominantly *Candida albicans*, which commonly coexist with *Staphylococcus* species

DIAGNOSIS

SIGNS AND SYMPTOMS

- Pain, warmth, and swelling to the proximal and lateral nail folds, often 2–5 days after trauma
- Symptoms must be present for 6 wk to meet criteria for a chronic paronychia.

History

- Acute paronychia: Nail biting, finger sucking, aggressive manicuring or manipulation, and trauma predispose to development.
- Chronic paronychia: Occupations with persistent moist hands; dish washers, bartenders; also increased in patients with peripheral vascular disease or diabetes

Pediatric Considerations

Frequently anaerobic mouth flora in children from nail biting

Physical-Exam

- Begins as swelling, pain, and erythema in the dorsolateral corner of the nail fold bulging out over the nail plate
- Progresses to subcuticular/subungual abscess
- Green nail coloration suggests *Pseudomonas*
- Nail plate hypertrophy suggests fungal source

ESSENTIAL WORKUP

- History and physical exam with special attention to evaluating for concomitant infections such as felon or cellulitis
- Assess tetanus status.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- No specific tests are useful.
- Cultures are not routinely indicated.
- Tzanck smear or viral culture if herpetic whitlow suspected.

Imaging

Soft tissue radiographs if foreign body is suspected; routine films if osteomyelitis suspected

Diagnostic Procedures/Surgery

Digital pressure test (opposing the thumb and the affected finger) may help identify the margins of an early subungual abscess

DIFFERENTIAL DIAGNOSIS

- Felon
- Herpetic whitlow
- Trauma or foreign body
- Primary squamous cell carcinoma
- Metastatic carcinoma
- Osteomyelitis
- Psoriasis
- Reiter syndrome
- Pyoderma gangrenosum
- Onychomycosis



TREATMENT

ED TREATMENT/PROCEDURES

Acute Paronychia

- Early paronychia without purulence may be managed with warm-water soaks 4 times a day with or without oral antibiotics; may also consider topical antibiotics and corticosteroids.
- Early superficial subcuticular abscess:
 - Elevation of the eponychial fold by sliding the flat edge of a no. 11 blade (18G needle or small clamps may be used) gently between the proximal nail

fold and the nail plate near the point of maximal tenderness

- A digital nerve block or local anesthesia may be necessary.
- Partial nail involvement:
 - If the lesion extends beneath the nail, remove a longitudinal section of the nail.
 - Petroleum jelly or iodoform gauze packing for 24 hr
- Runaround abscess:
 - If the lesion extends beneath the base of the nail to the other side, remove 1/4–1/3 of the proximal nail with 2 small incisions at the dorsolateral edges of the nail fold and pack eponychial fold with petroleum jelly or iodoform gauze to prevent adherence.
- Extensive subungual abscess:
 - Remove entire nail.
- Early paronychia without purulence present may be managed with warm soaks alone; beyond that, antibiotics are recommended if there is any apparent cellulitis, abscess, or systemic sign of infection.
- Trimethoprim–sulfamethoxazole, dicloxacillin, and amoxicillin–clavulanate are appropriate first-line agents, with treatment regimens ranging from 5–10 days, depending on severity.
- Clindamycin or amoxicillin–clavulanate if associated with nail biting or oral contact

Chronic Paronychia

- Avoidance of predisposing exposures and irritants/chemicals
- Topical steroids should be considered first-line therapy, with or without broad-spectrum topical antifungal agent
- Consideration for antistaphylococcal regimen
- For recalcitrant cases:
 - Eponychial marsupialization involving removal of a crescentic piece of skin just proximal to the nail fold, including all thickened tissue down to but not including germinal matrix
 - Oral antifungal therapy

MEDICATION

First Line

- Amoxicillin–clavulanate: 875 mg PO BID for 7 days (peds: 25 mg/kg/d PO q12h)
- Trimethoprim–sulfamethoxazole (Bactrim DS) BID for 7 days
- Dicloxacillin: 500 mg PO QID for 7 days (peds: 12.5–50 mg/kg/d PO q6h)

Second Line

- Clindamycin: 300 mg PO QID for 7 days (peds: 20–40 mg/kg/d div. q6h PO, IV,

IM)

- Topical antibiotics: Polymyxin B/Bacitracin, there is a high incidence of hypersensitivity to neomycin, mucipurin topical (Bactroban), or gentamicin TID for 5–10 days (0.1% ointment)
- Topical antifungal/steroid combination: nystatin–triamcinolone BID–TID until resolution, no longer than 1 mo
- For all topical antibiotics apply a small amount to affected areas TID–QID



FOLLOW-UP

DISPOSITION

Admission Criteria

Admission is not needed for paronychia alone.

Discharge Criteria

- Patients with uncomplicated paronychias may be discharged with appropriate follow-up instructions.
- Patients with packings should be re-evaluated in 24 hr.

Issues for Referral

Chronic paronychias refractory to treatment

PEARLS AND PITFALLS

- Acute paronychias respond well to decompression with or without antibiotics.
- Chronic paronychias are largely a result of chronic exposure to allergens/irritants.
- Reiter syndrome and psoriasis can mimic paronychia.
- Recurrent paronychia should raise suspicion for herpetic whitlow.
- Assess for felons.

ADDITIONAL READING

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1116.



CODES

ICD9

- 112.3 Candidiasis of skin and nails
- 681.02 Onychia and paronychia of finger
- 681.9 Cellulitis and abscess of unspecified digit

ICD10

- B37.2 Candidiasis of skin and nail
- L03.019 Cellulitis of unspecified finger
- L03.039 Cellulitis of unspecified toe

PATELLAR INJURIES

Stacy M. Boore • Stephen R. Hayden

BASICS

DESCRIPTION

Dislocation

- Usually caused by sudden flexion and external rotation of tibia on femur, with simultaneous contraction of quadriceps muscle
- Direct trauma to patella is a less common cause
- Lateral dislocation of the patella is most common, with the patella displaced over the lateral femoral condyle
- Uncommon dislocations include superior, medial, and rare intra-articular dislocation

Fracture

- Direct trauma:
 - Most common mechanism
 - Direct blow or fall on patella
 - Usually results in comminuted or minimally displaced fracture, or open injury
- Indirect forces:
 - The result of excessive tension through the extensor mechanism during deceleration from a fall (can also cause patellar tendon rupture)
 - Avulsion injury from sudden contraction of the quadriceps tendon
 - Usually results in transverse or displaced fracture (often both)
- Types of patellar fractures:
 - Transverse: 50–80% (usually middle or lower 3rd of patella)
 - Comminuted (or stellate): 30–35%
 - Longitudinal: 25%
 - Osteochondral

Patellar Tendon Rupture

- Usually caused by forceful eccentric contraction of quadriceps muscle on a flexed knee during deceleration (e.g., jump landing and weight lifting)
- Often occurs in older athletes
 - Microtrauma from repetitive activity

Patellar Tendinitis

- Overuse syndrome from repeated acceleration and deceleration (jumping, landing)

ETIOLOGY

Dislocation

- Risk factors for patellar dislocation:
 - Genu valgum (knock-knee)
 - Genu recurvatum (hyperextension of knee)
 - Shallow lateral femoral condyle
 - Deficient vastus medialis
 - Lateral insertion of patellar tendon
 - Shallow patellar groove
 - Patella alta (high-riding patella)
 - Deformed patella
 - Pes planus (flatfoot)
- Common injury in adolescent athletes, especially girls
- The younger the patient at the time of initial dislocation, the greater the risk of recurrence

Fracture

- Male:female ratio 2:1
- Highest incidence in those 20–50 yr old

Patellar Tendon Rupture

- Peak incidence in 3rd and 4th decades:
 - Often in athletes
- Risk factors:
 - History of patellar tendinitis
 - History of diabetes mellitus, previous steroid injections, rheumatoid arthritis, gout, systemic lupus erythematosus
 - Previous major knee surgery

Patellar Tendinitis

- Microtears of tendon matrix from overuse
- Seen in high jumpers, volleyball and basketball players, runners

DIAGNOSIS

SIGNS AND SYMPTOMS

Dislocation

- History of feeling knee “go out”; popping, ripping, or tearing sensation
- Pain
- Inability to bear weight

- Obvious lateral deformity of patella
- Mild to moderate swelling
- Often reduces spontaneously before ED evaluation
- Tenderness along patella
- Positive apprehension test or Fairbanks sign:
 - Attempts to push the patella laterally elicits patient apprehension
 - Attempts to push patella medially do not

Fracture

- Pain over anterior knee
- Difficulty ambulating
- Increased pain with movement of patella
- Tenderness and swelling over patella
- Difficulty or inability to extend knee
- Palpable defect, crepitus, or joint effusion/hemarthrosis

Patellar Tendon Rupture

- Abrupt onset of severe pain
- Decreased ability to bear weight
- Occasionally hemarthrosis
- Proximally displaced patella
- Incomplete extensor function
- Inability to maintain knee extension against force

Patellar Tendinitis

- Pain in area of patellar tendon
- Pain worse from sitting to standing or going up stairs
- Point tenderness at distal aspect of patella or proximal patellar tendon

ESSENTIAL WORKUP

Radiographs essential

DIAGNOSIS TESTS & INTERPRETATION

Imaging

- Anteroposterior (AP), lateral, and sunrise views of the knee should be obtained, pre- and postreduction
- Postreduction radiographs help exclude osteochondral fracture (in patellar dislocations)
- Bipartite patella (patella with accessory bony fragment connected to main body by cartilage) may be mistaken for fracture:
 - Comparison view may help differentiate
- For patellar tendon rupture, a high-riding patella (i.e., patella located superior to

level of intercondylar notch) is observed

- For patellar tendinitis, radiographic findings unlikely with symptom duration of < 6 mo

DIFFERENTIAL DIAGNOSIS

- Patellar subluxation
- Femoral or tibial fracture
- Traumatic bursitis
- Quadriceps tendon rupture



TREATMENT

PRE HOSPITAL

Patient should be transported in supine position with knee flexed and supported.

INITIAL STABILIZATION/THERAPY

Appropriate history and physical exam to identify any associated injuries (e.g., femoral fracture, hip fracture, posterior hip dislocation) and assess extensor mechanism

ED TREATMENT/PROCEDURES

Dislocation

- For simple lateral patellar dislocation, reduce dislocation by extending the knee gently to 180°:
 - Occasionally, simultaneous pressure may have to be applied over the lateral aspect of patella in a medial direction
- For other types of patellar dislocation (superior, medial, intra-articular), do not attempt reduction; consult orthopedics
- Aspiration of hemarthrosis with sterile technique is necessary if reduction is difficult
- If osteochondral fracture is present (28–50% of cases), obtain orthopedic consultation
- Although reduction is typically easy to accomplish, procedural sedation or parenteral analgesia may facilitate it
- Conservative (nonoperative) management of dislocations leads to recurrent instability in 60% of patients, but there is no evidence to support operative care in primary dislocations

Fracture

- Orthopedic consultation when patellar fracture is confirmed
- Nondisplaced fractures with intact extensor mechanism are managed nonsurgically
- Initial treatment often consists of long-leg bulky splint and subsequent operative

repair

Patellar Tendon Rupture

- Orthopedic consultation, with surgical repair within 2–6 wk

Patellar Tendinitis

- Rest, avoidance of inciting activity, heat, and NSAIDs

MEDICATION

- Fentanyl citrate: 0.5–1.5 µg/kg (peds: 0.5–1.0 µg/kg) IV
- Midazolam HCl: 1–2.5 mg (peds: 0.05–0.1 mg/kg, max. dose 6 mg) IV
- Morphine sulfate: 2–5 mg per dose (peds: 0.1–0.2 mg/kg per dose) IV
- Meperidine: 50–150 mg (peds: 1.1–1.8 mg/kg) IM q3–4h prn
- Ketorolac: 60 mg IM; 30 mg IV (peds: 0.5–1 mg/kg IV, max. 15 mg dose if <50 kg; max. 30 mg dose if >50 kg, IV)
- Methohexital: 1–1.5 mg/kg (1 mL q5sec) (peds: 0.5–1 mg IV) IV
- Propofol: 1–2 mg/kg IV (20 mg bolus q45sec) push slow IV to avoid dec BP (peds: 1 mg/kg not to exceed 40 mg)



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients with superior, medial, or intra-articular dislocation or in whom a lateral dislocation cannot be reduced require orthopedic consultation in the ED and possible admission
- Patellar dislocation associated with a fracture (osteochondral or lateral femoral condyle) requires orthopedic consultation in the ED
- Indications for operative intervention:
 - _ Fragments displaced > 4 mm
 - _ Unable to raise extended leg off bed
 - _ Articular step-off > 3 mm
- All open fractures require débridement and irrigation; such patients should be admitted.
- For patellar tendon rupture, discuss case with orthopedics.

Discharge Criteria

- Dislocation: Patients with successful reduction of lateral patellar dislocation and normal postreduction radiographs may be discharged with knee immobilization, crutches, and orthopedic follow-up.
- Fracture: If displaced < 3 mm and patient has full active knee extension:

- Knee immobilizer, or bulky long-leg splint, partial to full weight bearing as tolerated with crutches and orthopedic follow-up within a few days

PEARLS AND PITFALLS

- Lateral patella dislocations often reduce spontaneously prior to arrival in ED; do not dismiss patient's history of dislocation.
- In patella tendon ruptures, tendon defect may not be palpable if sufficient time has elapsed and swelling has occurred

ADDITIONAL READING

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CODES

ICD9

- 726.64 Patellar tendinitis
- 836.3 Dislocation of patella, closed
- 836.59 Other dislocation of knee, closed

ICD10

- M76.50 Patellar tendinitis, unspecified knee
- S83.006A Unspecified dislocation of unspecified patella, init encntr
- S83.016A Lateral dislocation of unspecified patella, init encntr

PATENT DUCTUS ARTERIOSUS

Steven Lelyveld

BASICS

DESCRIPTION

- Patent vessel in the fetal heart connects the pulmonary trunk to the descending aorta.
- Shortly after birth, changes normally provoke contraction, closure, and fibrosis:
 - Sudden increase in the partial pressure of oxygen
 - Changes in the synthesis and metabolism of vasoactive eicosanoids
- In the preterm infant, persistent patency of the ductus may be a normal life-saving response.
- The patent ductus usually has a normal structural anatomy.
- Patency results from hypoxia and immaturity.
- In the full-term newborn, patency of the ductus is a congenital malformation.
- Deficiency of both the mucoid endothelial layer and the muscular media of the ductus
- As pulmonary vascular resistance falls, aortic blood is shunted into the pulmonary artery.
- Extent of the shunt reflects the size of the ductus and the ratio of the pulmonary to systemic vascular resistances.
- Up to 70% of the left ventricular output may be shunted through the ductus to the pulmonary circulation.
- Risk factors:
 - Premature birth
 - Coexisting cardiac anomalies
 - Conditions resulting in hypoxia
 - High altitude
 - Maternal rubella infection
 - Female-to-male ratio, 3:1

ETIOLOGY

- Prematurity
- Congenital anomaly
- Hypoxia
- Prostaglandins

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Isolated patent ductus arteriosus (PDA), an unanticipated event
- PDA, as part of a larger congenital cardiac anomaly, may be diagnosed by US during pregnancy.

Physical-Exam

- Asymptomatic when the PDA is small, but otherwise may present with a range of findings.
- Congestive heart failure (CHF), often in 1st day of life
- Wide pulse pressure
- Prominent apical impulse
- Thrill
- Systolic and continuous murmur.
- Sounds like a humming top or rolling thunder
- Begins soon after onset of the 1st sound, reaches maximal intensity at the end of systole, and wanes in late diastole
- Localized to the 2nd left intercostal space or radiates down the left sternal border toward the apex or to the left clavicle
- Recurrent pulmonary infections
- Retardation of physical growth

ESSENTIAL WORKUP

- Establish the diagnosis with imaging studies.
- Rule out complications such as heart failure and endocarditis.

DIAGNOSIS TESTS & NTERPRETATION

Lab

Unhelpful in making the diagnosis

Imaging

- CXR:
 - Usually normal in infants
 - In children and adults:
 - Increased intrapulmonary markings
 - Calcifications
 - Left ventricle and left atrial enlargement
 - Dilated ascending aorta
 - Dilated pulmonary arteries
- EKG:
 - Abnormal if the ductus is large:
 - Left ventricular hypertrophy
 - Right ventricular hypertrophy is a sign of greater severity.

- Echocardiography:
 - Normal if the ductus is small
 - Left atrial enlargement
 - Size of the ductus can be determined by scanning from the suprasternal notch.
 - Doppler studies will determine aortic to pulmonary artery flow during diastole.
- Cardiac catheterization:
 - Normal or increased right-sided pressure
 - Oxygenated blood in the pulmonary artery confirms left-to-right shunting.
 - Injection of contrast into the ascending aorta shows opacification of the pulmonary arteries.

DIFFERENTIAL DIAGNOSIS

- Venous hum:
 - Common insignificant bruit
 - Heard in the neck or anterior portion of the chest
 - Soft humming sound in systole and diastole
 - Decreased by light compression of the jugular venous system
- Total anomalous pulmonary venous connection to the innominate vein:
 - Continuous murmur like venous hum
- Aorticopulmonary septal defect:
 - Murmur is often only systolic.
 - Heard at the right sternal border
- Ruptured sinus of Valsalva
- Coronary arteriovenous fistulas
- Anomalous origin of left coronary artery from the pulmonary artery
- Absence or atresia of pulmonary valve
- Aortic insufficiency with ventricular septal defect
- Peripheral pulmonary stenosis
- Truncus arteriosus



TREATMENT

ALERT

Supplemental oxygen if CHF

PRE HOSPITAL

Monitoring and oxygen

INITIAL STABILIZATION/THERAPY

- Small, asymptomatic shunts may not need closure.
- Pulmonary support

- Supplemental oxygen

ED TREATMENT/PROCEDURES

- Sodium and fluid restriction
- Correction of anemia to hematocrit $> 45\%$
- Antibiotic prophylaxis for endocarditis
- Preterm infants:
 - Usually closes spontaneously
 - Varies with the magnitude of shunting and severity of respiratory distress syndrome
 - Pharmacologic inhibition of prostaglandin synthesis with indomethacin during the 1st 2–7 days of life
- Full-term infants and children:
 - Surgical closure is required, even in asymptomatic patients, as spontaneous closure is rare.
 - Ligation and division
 - Transfemoral catheter technique to occlude PDA with foam plastic plug or double umbrella

MEDICATION

Indomethacin: 0.2–0.25 mg/kg per dose; repeat q12–24h for 3 doses



FOLLOW-UP

DISPOSITION

Admission Criteria

- Heart failure
- Endocarditis
- Pulmonary hypertension

Discharge Criteria

- Asymptomatic
- Prophylactic antibiotics
- Close follow-up with plans for early surgical closure

Issues for Referral

A pediatric cardiologist/neonatologist should be involved in all patients who have any evidence of heart failure, particularly if pharmacologic management is being considered.

PEARLS AND PITFALLS

- CHF may cause decrease in glomerular filtration rate and urinary output.
- Indomethacin may cause GI bleeding.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- <http://www.nhlbi.nih.gov/health/health-topics/topics/pda/>
- <http://www.nlm.nih.gov/medlineplus/ency/article/001560.htm>
- [http://www.heart.org/HEARTORG/Conditions/CongenitalHeartDefects/AboutCongenitalHeartDefects/AboutCongenitalHeartDefects/Ductus-Arteriosis-PDA_UCM_307032_Article.jsp](http://www.heart.org/HEARTORG/Conditions/CongenitalHeartDefects/AboutCongenitalHeartDefects/AboutCongenitalHeartDefects/AboutCongenitalHeartDefects/Ductus-Arteriosis-PDA_UCM_307032_Article.jsp)



CODES

ICD9

747.0 Patent ductus arteriosus

ICD10

Q25.0 Patent ductus arteriosus

PEDIATRIC TRAUMA

Kevin M. Ban



BASICS

DESCRIPTION

- Pathophysiology and anatomy of adolescents and young adults are similar.
- 80% of pediatric trauma is blunt; 80% of multisystem trauma includes head injury.
- Trauma is the leading cause of death and disability in children > 1 yr in US and Europe.
- Most victims of child abuse are < 3 yr. 1/3 of these patients are < 6 mo.

ETIOLOGY

- Most cases of pediatric trauma are single-system, minor, blunt injuries.
- Common mechanisms of injury include motor vehicle collisions and bicycle accidents, struck by a vehicle as a pedestrian, and fall from height.
- Penetrating injuries are rare in younger children.
- Risk factors include inadequate supervision, developmental inadequacy of child to perform task, inadequate attention to task, risk taking, drugs, and alcohol.



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- History is often straightforward and provided by the child, parents, witnesses, or paramedics. If inconsistent with injury, consider child abuse.
- Mechanism(s) of injury relatively poor predictor of injury severity, but may suggest type of injury.
- Variables that increase the likelihood of serious injuries include handlebar injuries, significant passenger space intrusion, and failure to use proper restraint during a motor vehicle collision or helmet when riding a bike or skateboard.
- AMPLE history includes *allergies, medications, past medical history, time of last meal, and events leading up to injury.*

Physical-Exam

- Primary survey:
 - For all children who have sustained a major trauma, a traditional stepwise ABCDE evaluation based on assessing the *airway, breathing, circulation, disability, and exposure* is appropriate.
- Secondary survey:

- General:
 - Mass-to-surface ratio may impact insensible water loss and increase the risk of hypothermia.
 - Compensatory mechanisms may delay signs of hypovolemia. Few findings may be present until loss of 25–30% of blood volume, at which time decompensation abruptly occurs.
 - Smaller total blood volume (80 mL/kg)
- Head:
 - Note bulging fontanel, scalp hematomas, midface instability, auricular and septal hematomas, lacerations, functional or cosmetic deformities to the face, and pupillary abnormalities.
 - Open sutures/fontanelles or multiple skull fractures may delay the onset of other signs and symptoms of increased intracranial pressure.
 - Large head/occiput causes cervical spine flexion when patient is supine on adult backboard.
- Eye/ears, nose, and throat exam:
 - Look for evidence of blood, trauma, hemotympanum, hyphema, and CSF fluid.
 - Large tongue and tonsillar hypertrophy may obstruct the airway.
- Neck:
 - Tracheal deviation and posterior neck step-offs are exceedingly unusual in children.
 - Shorter trachea increases risk of right mainstem intubation.
 - Cricoid cartilage is narrowest portion of airway in children < 8 yr.
 - Children with altered mental status cannot have their cervical spine precautions cleared in the ED. These children should remain in a cervical collar (and be taken off the spinal board) while in the ED.
 - Pseudosubluxation (anterior displacement of C-2 on C-3) occurs in 20% of patients.
 - The term spinal cord injury without radiologic abnormality (SCIWORA) is controversial in the MRI era.
- Chest:
 - Note the overall work of breathing, grunting, asymmetric breath sounds, posterior abrasions, chest wall deformities, and crepitus.
 - Flexible and compliant chest walls make pulmonary contusions more likely than rib fractures in young children. Rib fractures may be a sign of abuse.
 - Diaphragmatic breathing
- Abdomen:
 - Bruising, abrasions, and tenderness
 - Distention is usually caused by gastric air.
 - Liver and spleen relatively large
 - Rib cage covers less of abdomen.

- Bladder is intra-abdominal in children < 2 yr.
- Extremities:
 - Palpation and evaluation of joint stability and tenderness
 - Assess pulses and compartments.
 - Salter–Harris classification of fractures
 - Unique injuries: Greenstick and buckle fractures
- Neurologic exam:
 - Age-appropriate mental status assessment
 - Assess movement of the extremities.
- Skin:
 - Assess for prolonged capillary refill and pallor.
 - Bruising of the ears, dorsa of the feet, or genitalia may suggest nonaccidental trauma.
- Patterns of injury:
 - Car vs. pedestrian: Waddell triad (femur, torso, and head injuries)—uncommon
 - Bicycle handlebar injuries may impale child: Pancreatic or small bowel injury.
 - Lap belt syndrome: Abdominal ecchymoses and intestinal injury with or without lumbar spine fracture (chance fracture)
 - Minor trauma history with major injury: Consider child abuse

ESSENTIAL WORKUP

- History and age-appropriate physical exam are the only essential components to a workup for all children who present for an evaluation following trauma.
- Obtaining standard radiographic and lab “trauma panels” is not evidence based in children.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Lab tests should generally be individualized, reflecting the patient’s clinical presentation.
- A normal initial hemoglobin and hematocrit do not rule out a significant hemorrhage but will provide a baseline value for later comparison; tachycardia may be only sign of fluid/blood loss early in presentation, although it is nonspecific for blood loss.
- Initial electrolyte measurement is unnecessary.
- Routine amylase and lipase are not recommended because of the low incidence of pancreatic injuries; false-positive tests are common.
- Elevated LFTs should not be used as the sole determinant in deciding which children should undergo CT of the abdomen. Patients with AST > 200 IU/L or ALT > 125 IU/L who have sustained abdominal trauma should be considered for CT if

hemodynamically stable. Physical exam should guide imaging decision.

- Gross hematuria (> 50 RBC/HPF) is concerning for urinary tract injuries, but microscopic hematuria is not.
- Blood bank specimen for typing in appropriate patients
- A pregnancy test is indicated for teenage girls.
- Diagnostic peritoneal lavage is rarely indicated with availability of imaging modalities.

Imaging

- The traditional “c-spine, chest, pelvis” set of radiographs is no longer universally obtained; selective approach is more appropriate.
- Forgo cervical spine radiographs in children who are awake, alert, cooperative, neurologically intact without neck pain or midline tenderness on palpation of the neck, are without pain on range of motion testing, and are without distracting injury:
 - An unconscious child will not be able to have the cervical spine cleared in the ED and may later need MRI (less often CT) as an inpatient.
- Chest radiographs indicated for grunting respirations, hypoxia, asymmetric breath sounds, dyspnea, crepitus, endotracheal intubation, and thoracostomy tube or central venous catheter placement in the internal jugular or subclavian veins
- Pelvic radiographs are seldom indicated. Children with clinically significant pelvic pain or instability typically undergo CT of the abdomen and pelvis.
- CT of the head is indicated for abnormal mental status, focal neurologic deficit, prolonged loss of consciousness, bulging fontanel, temporal or parietal scalp hematoma, depressed skull fracture, and uncontrollable persistent vomiting.
- CT of the abdomen and pelvis is typically indicated for children with altered mental status, gross hematuria, abdominal bruising above the ileac crests, handlebar injuries, and abdominal tenderness with hemodynamic effect.
- US has limited utility since the presence of free fluid (i.e., blood) does not always indicate the need for laparotomy. The usefulness of focused abdominal sonography for trauma exam in young children needs further study.

DIFFERENTIAL DIAGNOSIS

Nonaccidental trauma should be considered when the history is inconsistent with the injury.



TREATMENT

PRE HOSPITAL

- Rapid transport to a facility capable of managing the child’s suspected injuries
- Priorities include stabilization of airway (intubation by paramedics in the pre-hospital setting is controversial), breathing, circulation.

- Immobilization of cervical spine and extremity fractures

INITIAL STABILIZATION/THERAPY

- Most traumatized children are stable throughout their ED course.
- Stabilization may require:
 - Cardiorespiratory and pulse oximetry monitoring
 - Early oxygen administration
 - Placement of 2 large-bore IVs and aggressive fluid resuscitation with normal saline
 - Pain control with morphine
 - Labs and radiographs as indicated
 - Administration of packed red blood cells if not responding to 2 crystalloid boluses
 - Endotracheal intubation:
 - Rapid sequence intubation should be performed with etomidate or ketamine and succinylcholine
 - Sedate patient with a benzodiazepine or propofol
 - Cervical spine immobilization
 - Thoracostomy tube as indicated
 - Urinary catheter (look for blood at the meatus)
 - Gastric decompression with a nasogastric or orogastric tube

ED TREATMENT/PROCEDURES

- Risk stratify based on history and physical exam.
- Acknowledge the limitations of using the mechanism of injury to predict its severity.
- Assess priorities; reassess frequently.
- Provide analgesia; sedate as appropriate.
- Clean wounds and splint fractures.
- Tetanus immunization if indicated
- Allow parents at the bedside during resuscitation and treatment.

MEDICATION

- Normal saline/lactated Ringer: 20 mL/kg boluses IV
- Packed red blood cells: 10 mL/kg U IV
- Etomidate: 0.3 mg/kg IV
- Morphine sulfate: 0.1 mg/kg IV
- Succinylcholine: 1.5 mg/kg IV
- Lorazepam: 0.1 mg/kg IV
- Propofol: 2 mg/kg IV
- Ketamine: 2 mg/kg IV (generally thought to raise intraocular and intracranial pressure—usually avoided when head injury is suspected)



FOLLOW-UP

DISPOSITION

Admission Criteria

- Persistent altered mental status, endotracheal intubation, thoracostomy tube placement, intra-abdominal or intracranial injury identified on CT, pulmonary contusion, fractures requiring operative management, nonaccidental trauma
- Hemodynamic instability
- Airway concerns
- CT negative for intra-abdominal injury, but persistent abdominal pain as pancreatic or bowel injury is possible
- Failure to identify an appropriate adult to be responsible for the child (e.g., both parents are admitted to the hospital for their own injuries)

Discharge Criteria

- Most traumatized children with normal mental status and normal radiographic tests (if obtained) can be discharged home to a reliable caregiver.
- Post-traumatic stress syndrome may develop, and parents should be advised to seek appropriate counseling should concerns develop.

FOLLOW-UP RECOMMENDATIONS

- Specialists as indicated by injury
- Psychiatric evaluation may be indicated for evidence of post-traumatic stress.
- Neurologic assessment for evidence of residual from postconcussion syndrome.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Abuse, Pediatric
- Fractures, Pediatric
- Trauma, Multiple



CODES

ICD9

- 920 Contusion of face, scalp, and neck except eye(s)
- 959.01 Head injury, unspecified
- 995.50 Child abuse, unspecified

ICD10

- S00.83XA Contusion of other part of head, initial encounter
- S09.90XA Unspecified injury of head, initial encounter
- T76.92XA Unspecified child maltreatment, suspected, initial encounter

PEDICULOSIS

Andrew B. Ziller

BASICS

DESCRIPTION

- Infestation by organisms that live in close association with host
- Bites are painless
- Signs and symptoms result from host response to saliva and anticoagulant injected during feeding
- Transmitted by direct contact and fomites (inanimate objects)
- Head lice are transmitted by head-to-head contact:
 - Combs
 - Pillows
 - Hats
- Head lice are more common in children and females
- Pubic lice are transmitted by sexual contact
- Obligate human parasites cannot survive away from hosts >7–10 days

ETIOLOGY

Infestation by:

- *Pediculus capitis* (head louse):
 - Most common
 - All socioeconomic groups
- *Pediculus corporis* (body louse):
 - Associated with poverty, poor hygiene, and overcrowding
 - Live in clothing and transfer to human host for feeding
 - Can live up to 30 days off of human
 - Related to bed bugs
- *Phthirus pubis* (pubic or crab louse)

Pediatric Considerations

Pubic lice may also indicate sexual abuse in children

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Head lice:
 - Dandruff

- Pruritus
- Often asymptomatic
- Body lice:
 - Pruritus
 - Excoriation particularly at belt lines or seams of clothing
- Pubic lice:
 - Intense pruritus, worse at night

Physical-Exam

- Examine hair for adult lice and nits:
 - Nits are cemented on hair shafts and are not easily removed
 - Head lice and pubic lice infestation is confirmed by differentiating nits from scales, hair casts, and other easily brushed-off artifacts
 - Empty nits are not diagnostic of active infection
- Scalp and posterior neck erythema, scaling, and excoriated papules:
 - May lead to pyoderma, posterior cervical lymphadenopathy, and bacterial superinfection
- Body lice are observed only in very heavy infestation; infestation is confirmed by finding nits in clothing seams:
 - Linear excoriations of neck and trunk
 - Pus or serum stains on clothing
- Pubic lice:
 - Occasional urticaria with typical flare/wheal formation
 - May infest eyelashes and scalp in children
 - Characteristic bluish macules (maculae ceruleae) appear infrequently on trunk and thighs
 - Prefer the perineum and pubic areas
 - Inguinal adenopathy

ESSENTIAL WORKUP

- Careful history and physical exam
- Universal precautions

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Nits may be visualized under low-power microscopy along hair shafts. They are <1 mm long:
 - Fluorescent under Wood lamp
- Mature lice are 3–4 mm long
- Pubic louse ~ 1 mm long but wider body than head or body louse

Imaging

No imaging indicated

DIFFERENTIAL DIAGNOSIS

- Scabies
- Contact or allergic dermatitis
- Seborrheic dermatitis
- Bed bugs (Cimicidae)



TREATMENT

PRE HOSPITAL

ALERT

Maintain universal precautions

INITIAL STABILIZATION/THERAPY

Not applicable for routine cases

ED TREATMENT/PROCEDURES

- Oral antihistamines and topical steroids may help pruritic symptoms of all lice infestations
- Head lice:
 - Topical pediculicidal agents:
 - Permethrin 1% cream rinse (Nix) is a reasonable agent; it has low toxicity and cost and is ovicidal; however, resistance is becoming more common
 - Pyrethrin (Rid) also has low toxicity but is less effective
 - All agents require reapplication in 7–10 days if further adult lice or nits noted
 - Remove nits with fine-toothed comb
 - Examine all members of household; treat infested individuals
 - Change clothing and machine wash and dry (using hot cycles) all clothing, towels, linens, and headgear:
 - Vacuum floors and furniture
 - Wash combs and brushes in hot water for 10–20 min or coat with pediculicide for 15 min and wash
 - Temperature $> 131^{\circ}\text{F}$ (55°C) for > 5 min kills eggs, nymphs, and mature lice
- Body lice:
 - Wash and dry bedding and clothing using hot cycles
 - Apply topical pediculicide cream or lotions from chin to toes
- Pubic lice:
 - Topical pediculicide applied to hairy areas of chest, axilla, and groin
 - Remove nits with fine-toothed comb

- Treat sexual contacts simultaneously
- Wash and dry bedding and clothing using hot cycles
- Treat eyelash involvement with topical petrolatum twice daily for 9 days

MEDICATION

First Line

- Antipruritics:
 - Diphenhydramine: 25–50 mg PO (peds: 5 mg/kg/d) q6h
 - Hydroxyzine: 25 mg PO q8h (peds: 12.5 mg/dose q6h)
- Pediculicides:
 - Permethrin 1% cream rinse (Nix): Apply to scalp and hair, rinse after 10 min; reapply in 7–10 days if needed
 - Pyrethrin/piperonyl butoxide (Rid): Apply to scalp and hair, wash after 10 min; repeat in 7–10 days; avoid in patients with ragweed allergies
 - Benzyl alcohol lotion 5% (Ulesfia lotion): Apply to scalp and hair, wash off after 10 min; repeat in 7 days
 - Mercuric oxide ophthalmic ointment 1%: Use for louse infestation of eyelids: Apply QID for 14 days

Second Line

- Pediculicides:
 - Ivermectin 0.5% lotion (Sklice): Apply to dry hair and scalp and rinse after 10 min
 - Spinosad 0.9% suspension (Natroba): Apply to dry hair and rinse after 10 min; repeat in 7 days if necessary
 - Ivermectin tablets (Stromectol): 200–400 µg/kg PO once; repeat in 7–10 days later
 - Use if 1st-line agents (Nix, Rid, Ulesfia) are not tolerated or effective
- Antihistamine:
 - Cetirizine (Zyrtec): Age > 12 yr, 5–10 mg PO (peds: 6–11 yr, 5–10 mg PO; 2–5 yr, 2.5 mg PO) daily

Pregnancy Considerations

- Nix is Class B and probably safe in lactation
- Rid is Class C and probably safe in lactation
- Ulesfia is Class B but should read package insert; safety unknown in lactation
- Ivermectin is Class C with safety unknown in lactation
- Spinosad is Class B but should read package insert for specifics; safety unknown in pregnancy

Pediatric Considerations

- Nix can be used in children > 2 mo
- Rid can be used in children > 2 yr
- Ulesfia can be used in children
- Ivermectin can be used in children > 6 mo
- Spinosad can be used in children > 4 yr



FOLLOW-UP

DISPOSITION

Admission Criteria

Extensive bacterial superinfection; systemic hypersensitivity reaction with cardiorespiratory compromise

Discharge Criteria

- Mild-to-moderate infestation with absence of significant superinfection or hypersensitivity reaction
- Children may return to school after initial treatment if repeat therapy is administered in 7–10 days
- Pubic lice are often associated with sexually transmitted diseases; prudent screening is recommended

FOLLOW-UP RECOMMENDATIONS

- Re-evaluation is necessary to observe if treatment has been successful
- Case management and/or social services may be required if concern for child well-being

PEARLS AND PITFALLS

- Diagnosed by direct visualization
- Most of the topical agents need to be reapplied in 7–10 days because unhatched eggs are not killed
- Clothing and bedding must be washed and dried at a high heat to eradicate the infestation
- Lindane is no longer recommended
- Resistance to *Nix* and *Rid* is increasingly more common
 - 2nd-line agents are more expensive

ADDITIONAL READING

- Benzyl alcohol lotion for head lice. *Med Lett Drugs Ther.* 2009;51:57.
- Chosidow O, Giraudeau B. Topical ivermectin – a step toward making head lice dead lice? *N Engl J Med.* 2012;367:1750–1752.

- Frankowski BL, Bocchini JA Jr, et al. Head lice. *Pediatrics*. 2010;126:392–403.
- Gunning K, Pippitt K, Kiraly B, et al. Pediculosis and scabies: Treatment update. *Am Fam Physician*. 2012;86:535–541.
- Ivermectin (Sklice) topical lotion for head lice. *Med Lett Drugs Ther*. 2012;54:61–63.



CODES

ICD9

- 132.0 *Pediculus capitis* [head louse]
- 132.1 *Pediculus corporis* [body louse]
- 132.9 Pediculosis, unspecified

ICD10

- B85.0 Pediculosis due to *Pediculus humanus capitis*
- B85.1 Pediculosis due to *Pediculus humanus corporis*
- B85.2 Pediculosis, unspecified

PELVIC FRACTURE

Andrew T. LaFree • Theodore C. Chan



BASICS

DESCRIPTION

- 3% of all bony fractures
- Pelvis is made up of sacrum and 2 innominate bones:
 - The innominate bones consist of the ilium, ischium, and pubis
- Bony structures are stabilized by a network of ligaments, musculature, and other soft tissues in the pelvic area
- Anterior stability and support are provided by the symphysis pubis and pubic rami
- Posterior stability and support are provided by the sacroiliac (SI) complex and pelvic floor
- Pelvis provides protection for lower urinary tract; GI tract; gynecologic, and vascular, and nervous structures contained in the region:
 - Pelvic fractures have a high associated morbidity and mortality rate and require urgent diagnosis and therapy.
- Unstable pelvic fractures are high risk for associated injuries including:
 - Pelvic hemorrhage and hemorrhagic shock
 - Intra-abdominal and GI tract injuries
 - Genitourinary and urinary tract injuries
 - Uterine and vaginal injuries
 - Neurologic injuries
 - Arterial and venous plexus injuries

ETIOLOGY

- 65% of pelvic fractures are caused by vehicular trauma, including pedestrians struck by automobiles
- 10% caused by falls
- 10% caused by crush injuries
- The remainder caused by athletic, penetrating, or nontraumatic injuries
- Mortality rate from pelvic fractures is 6–19%:
 - Increases with open fractures or evidence of hemorrhagic shock
- Significant hemorrhage can occur in unstable, high-energy pelvic fractures (Tile type B and C fractures):
 - Bleeding most common with posterior injuries involving the vascular plexuses
 - Retroperitoneal hematoma may tamponade in the enclosed pelvic space

Tile Classification System

- Includes stable single bone and avulsion fractures as well as pelvic ring fractures
- Predicts need for operative repair
- Type A: Stable pelvic ring injuries:
 - A1: Avulsion fractures of the innominate bone (ischial tuberosity, iliac crest)
 - A2-1: Iliac wing fractures
 - A2-2: Isolated rami fractures; most common pelvic fracture
 - A2-3: 4-pillar anterior ring injuries
 - A3: Transverse fractures of sacrum or coccyx
- Type B: Partially stable pelvic ring injury (rotationally unstable, but vertically stable):
 - B1: Unilateral open-book fracture
 - B2: Lateral compression injury:
 - B2-1: Ipsilateral double rami fractures and posterior injury
 - B2-2: Contralateral double rami fractures and posterior injury (bucket-handle fracture)
 - B2-3: Bilateral type B injuries
- Type C: Unstable pelvic ring injury—rotationally and vertically unstable, *Malgaigne fracture*:
 - Anterior disruption of symphysis pubis or 2–4 pubic rami with posterior displacement and instability through sacrum, SI joint, or ileum:
 - C1: Unilateral vertical shear fracture
 - C2: Unilateral vertical shear combined with contralateral type B injury
 - C3: Bilateral vertical shear fracture
- Acetabular fractures (posterior lip, central/transverse, anterior column, or posterior column fractures)

Young Classification System

- Based on mechanism of injury
- Only fractures that result in disruption of pelvic ring included; no single bone, avulsion, or acetabular fractures
- Predicts chance of associated injuries and mortality risk:
 - LC: Lateral compression
 - APC: Anteroposterior compression
 - VS: Vertical shear
 - CM: Combination of injury patterns

Pediatric Considerations

- Children can have greater hemorrhage
- Nonaccidental trauma is a concern

Pregnancy Considerations

Gravid uterus may be at risk for injury, including uterine rupture.

DIAGNOSIS

SIGNS AND SYMPTOMS

- Pain, swelling, ecchymosis, tenderness over hips, groin, perineum, and lower back
- Often presents with other traumatic injuries including neurologic, intra-abdominal, genitourinary, perineal, rectal, vaginal, and vascular injury
- Evidence of hemorrhagic shock
- Gross pelvic instability

History

- History of trauma (fall, vehicular trauma, crush injuries, athletic injuries)
- Pain on hip movement, ambulation, sitting, standing, defecation

Physical-Exam

- Ecchymosis, swelling, tenderness over bony prominences, pubis, perineum, pelvic region, lower back
- Lower extremities may be shortened or rotated
- Inability to actively or passively perform range of motion of involved hip
- Tenderness on LC of pelvis, palpation of symphysis pubis or SI joints
- Gross pelvic instability, deformity, asymmetry in lower extremity
- Wounds over pelvis or bleeding from rectum, vagina, or urethra may indicate open fracture
- In hemorrhagic shock:
 - Tachycardia, hypotension, narrowed pulse pressure
 - Altered mental status
 - Cool and pale extremities

ESSENTIAL WORKUP

- Pelvic radiograph is the most common initial test
- A single AP view of the pelvis can confirm diagnosis and should be obtained as early as possible when fracture suspected:
 - Most significant unstable pelvic fractures will be seen on the single AP view
- Other views include:
 - Inlet projection: 30° caudal view; allows visualization of posterior arch
 - Outlet projection: 30° cephalic angulation; allows visualization of sacrum
 - Judet oblique views: Allow evaluation of acetabulum

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Type and cross-match
- Hemoglobin/hematocrit, platelet count, and coagulation studies (prothrombin time, partial thromboplastin time)

Imaging

- CT may further delineate pelvic fracture(s), retroperitoneal hematoma, visceral injuries:
 - CT contrast angiography may delineate source of bleeding (particularly arterial), but should be considered only in hemodynamically stable patients
- Abdominal US focused abdominal sonography for trauma in patients with significant traumatic injury, but differentiation of intraperitoneal from extraperitoneal hemorrhage from pelvic fracture can be difficult
- MRI indicated for neurologic injury

Diagnostic Procedures/Surgery

- Although largely supplanted by US and CT, diagnostic peritoneal lavage (DPL) remains a rapid bedside evaluation for intraperitoneal hemorrhage
- Angiography and selective vessel embolization in the setting of pelvic hemorrhage:
 - Particularly for small-vessel arterial bleeding
- Surgery:
 - As indicated on the basis of clinical findings and orthopedic/surgical consult
 - Surgical stabilization with pelvic packing
 - Direct operative control of pelvic bleeding

DIFFERENTIAL DIAGNOSIS

- Normal variants (i.e., os acetabuli epiphyseal line can mimic type I fracture on radiograph)
- Ligamentous injury
- Spinal injury
- Intra-abdominal injury and hemorrhage



TREATMENT

PRE HOSPITAL

- IV fluid resuscitation as indicated
- Consider stabilization or immobilization measures for pelvis

INITIAL STABILIZATION/THERAPY

- ABCs of trauma care
- IV fluid resuscitation with blood or crystalloid, O-negative or type-specific blood if hemodynamically unstable:
 - Avoid using lower extremity IV sites

- Stabilize and immobilize the pelvis to prevent further injury and decrease bleeding:
 - Compression device: Folded sheet with clamp or commercial compression device wrapped circumferentially around greater trochanters to stabilize and compress pelvis
 - Pneumatic anti-shock garment (PASG): Use in ED is controversial, but allows rapid pelvic immobilization and pelvic compression to slow bleeding
 - External fixator: Requires more time to place than PASG but “splints” pelvis in a similar manner; contraindicated in severely comminuted pelvic fracture
 - Placement of a stabilization device should not interfere with further workup and care (e.g., US, DPL)

ED TREATMENT/PROCEDURES

- Determine which pelvic fractures are stable and which are unstable
- Type A fractures are generally stable
- Type B and C fractures are unstable
- Type A fractures:
 - Treated conservatively with bed rest, analgesics, and comfort measures; management decisions may be made in conjunction with orthopedics
 - For 4-pillar anterior ring injuries, CT should be obtained to evaluate the posterior pelvis
 - Ensure that there are no other breaks in the pelvic ring
- Type B and C fractures:
 - Immediate orthopedics consultation; patient should remain NPO
 - May require ED pelvic stabilization measures
 - Assess for pelvic hemorrhage
- Malgaigne fractures:
 - Anticipate significant hemorrhage and associated injuries
- Acetabular fractures:
 - Immediate orthopedics consultation; patient should remain NPO
- Pelvic hemorrhage:
 - Mechanical stabilization of unstable pelvic fractures (usually by application of external pelvic fixation)
 - Angiography and selective vessel embolization
 - Direct operative control of pelvic bleeding
- Prioritization of studies: CT, angiography, or surgery:
 - In the hemodynamically *unstable* patient:
 - Open B and C fractures: Surgical exploration
 - Closed fractures: DPL or US can help determine management in terms of need for immediate surgical exploration or selective angiography/embolization
- In the hemodynamically *stable* patient, the patient can go to CT for evaluation of the abdomen, pelvis, and retroperitoneum with external fixation as appropriate

MEDICATION

- Crystalloid fluids: 2 L IV bolus of normal saline or lactated Ringer (peds: 20 mL/kg)
- Blood products: 4–6 U cross-matched, type specific, or O-negative (peds: 10 mL/kg)



FOLLOW-UP

DISPOSITION

Admission Criteria

- Hemodynamic instability, and pelvic hemorrhage to the ICU
- Type B or C pelvic fracture
- Acetabular fracture
- Other related injuries (e.g., genitourinary, intra-abdominal, neurologic)
- Intractable pain

Discharge Criteria

Type A pelvic fracture; hemodynamically stable with no evidence of other injuries

Issues for Referral

Close follow-up should be ensured for discharged patients.

FOLLOW-UP RECOMMENDATIONS

Discharged patients should be referred to an orthopedist for follow-up.

PEARLS AND PITFALLS

- Pelvic fractures can be a marker for high-energy traumatic mechanism and injury:
 - Assess for underlying abdominal/pelvic injuries including GI, genitourinary, vascular, and neurologic injuries
- In addition to initial resuscitation, immobilization and stabilization of the pelvis should be considered for unstable or open fractures or where hemorrhage is suspected
- Determination of diagnostic/therapeutic pathways including CT with or without angiography, selective IR angiography, and surgery are dictated by the patient's hemodynamic status, suspected underlying injuries, and type of pelvic fractures
- All patients with Malgaigne fractures should be admitted with consultation by trauma and orthopedic services

ADDITIONAL READING

- American College of Surgeons, Committee on Trauma. *Advanced Trauma Life*

Support for Doctors, 9th ed. Chicago, IL: American College of Surgeons; 2012.

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- Hak DJ, Smith WR, Suzuki T. Management of hemorrhage in life-threatening pelvic fracture. *J Am Acad Orthop Surg*. 2009;17:447–457.
- Rice PL Jr, Rudolph M. Pelvic fractures. *Emerg Med Clin North Am*. 2007;25:795–802.



See Also (Topic, Algorithm, Electronic Media Element)

- Hemorrhagic Shock
- Hip Injury



CODES

ICD9

- 808.8 Closed unspecified fracture of pelvis
- 808.41 Closed fracture of ilium
- 808.42 Closed fracture of ischium

ICD10

- S32.9XXA Fracture of unsp parts of lumbosacral spine and pelvis, init
- S32.309A Unsp fracture of unsp ilium, init encntr for closed fracture
- S32.609A Unsp fracture of unsp ischium, init for clos fx

PELVIC INFLAMMATORY DISEASE

Erich Salvacion



BASICS

DESCRIPTION

- Pelvic inflammatory disease (PID) is an acute, community-acquired, sexually transmitted infection of the upper genital tract, including the uterus, fallopian tubes, ovaries, or adjacent structures
- Most frequent gynecologic cause for ED visits (350,000 per year)
- Represents a spectrum of infection:
 - No single diagnostic gold standard
 - Requires low clinical threshold for considering the diagnosis and starting empiric antibiotic therapy
- Progressive disease can lead to tubo-ovarian abscess (TOA)
- Fitz-Hugh–Curtis syndrome is a capsular inflammation of the liver associated with PID:
 - Sharp right upper quadrant abdominal pain
 - Worse with inspiration, movement, or coughing

ETIOLOGY

- Risk factors:
 - Age < 25 yr
 - Multiple or symptomatic sexual partners
 - Previous episode of PID
 - Nonbarrier contraception
 - Oral contraception
 - African American ethnicity
- Most common causes of PID are *Chlamydia trachomatis* and *Neisseria gonorrhoea*
- Other organisms include groups A and B streptococci, staphylococci, gram-negative rods (commonly *Klebsiella* spp., *Escherichia coli*, and *Proteus* spp.), and anaerobes



DIAGNOSIS

SIGNS AND SYMPTOMS

- Lower abdominal pain, usually bilateral
- Vaginal discharge
- Abnormal uterine bleeding
- Dysmenorrhea
- Dysuria
- Dyspareunia

- Nausea and vomiting
- Fever and chills
- Proctitis
- Lower abdominal tenderness
- Decreased bowel sounds
- Bilateral adnexal tenderness
- Cervical motion tenderness
- Purulent endocervical discharge
- Adnexal mass or fullness
- Right upper quadrant tenderness

History

- Lower abdominal pain is the most common symptom in PID, ranging from subtle to severe pain
- Abdominal pain that worsens during intercourse or onset of pain shortly after or during menses is suggestive of PID
- Abdominal pain is usually bilateral and usually present for ≤ 2 wk
- New vaginal discharge, urethritis, fever, and chills are common symptoms but are neither sensitive nor specific for the diagnosis

Pregnancy Considerations

PID is rare during pregnancy, but if present usually occurs during the 1st trimester before hormonal changes such as mucus plug formation can protect the uterus from ascending bacteria.

Physical-Exam

- Only 50% of patients with PID have fever
- Abdominal exam reveals diffuse tenderness worse in the lower quadrants, usually but not always symmetric
- Rebound tenderness and decreased bowel sounds are commonly found
- Right upper quadrant tenderness is suggestive of perihepatitis (Fitz-Hugh–Curtis syndrome) in the setting of PID
- Pelvic exam can reveal a purulent endocervical discharge, cervical motion tenderness, or adnexal tenderness
- If uterine or adnexal tenderness is not prominent, one must consider other diagnoses

ESSENTIAL WORKUP

- History and physical exam including pelvic exam
- Pregnancy test to rule out ectopic pregnancy or complications of an intrauterine pregnancy
- Cervical culture for *N. gonorrhoea* and *C. trachomatis*

- Minimum criteria for clinical diagnosis:
 - Lower abdominal tenderness *or*
 - Uterine/adnexal tenderness *or*
 - Cervical motion tenderness
- Supportive criteria for diagnosis:
 - Fever > 38.3°C (101°F)
 - Abnormal cervical/vaginal discharge
 - Intracellular gram-negative diplococci on endocervical Gram stain
 - Leukocytosis > 10,000/mm³
 - Elevated erythrocyte sedimentation rate (ESR) or C-reactive protein
 - WBCs or bacteria in peritoneal fluid obtained by culdocentesis or laparoscopy

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC
- Gram stain of endocervix
- Urine polymerase chain reaction tests for *Chlamydia* and *Gonococcus*
- Microscopic exam of vaginal discharge in saline
- Liver enzymes may be elevated in Fitz-Hugh–Curtis syndrome
- Positive urinalysis or occult blood in stool decreases the probability of PID
- ESR or C-reactive protein may be elevated, but not routinely recommended

Imaging

- Patients with adnexal fullness or an adnexal mass on exam should have a transvaginal US to exclude TOA
- Consider obtaining a pelvic US in patients who use an intrauterine device, fail outpatient antibiotic therapy for PID, or who have inadequate pelvic exams due to pain or obesity

Diagnostic Procedures/Surgery

Laparoscopy may be useful in confirming PID in a patient with a high suspicion of competing diagnosis or who failed outpatient treatment for PID

DIFFERENTIAL DIAGNOSIS

- Ectopic pregnancy (must be excluded with a pregnancy test in any woman suspected of having PID)
- Acute appendicitis
- Adnexal torsion
- Endometriosis
- Cystitis

- Urolithiasis
- Ovarian tumor
- Adenomyosis uteri
- Chronic pelvic pain
- Benign ovarian cyst
- Diverticulitis
- Inflammatory bowel disease
- Mesenteric vascular disease
- Irritable bowel syndrome



TREATMENT

PRE HOSPITAL

- No specific pre-hospital considerations
- Appropriate pain management

INITIAL STABILIZATION/THERAPY

- Resuscitation rarely indicated
- Pain control

ED TREATMENT/PROCEDURES

Outpatient

- Ceftriaxone or cefoxitin/probenecid + doxycycline; with metronidazole when anaerobes are a particular concern
- Alternatives include ceftriaxone + azithromycin.
- Must evaluate and treat sex partner as appropriate

Inpatient

- Doxycycline + cefoxitin or cefotetan
- Alternatives include gentamicin + clindamycin; or ampicillin/sulbactam + doxycycline
- Continue parenteral antibiotic administration for 24 hr after clinical improvement, then switch to oral antibiotics to finish 14 day course
- Laparoscopy can be used to lyse adhesions in the acute and chronic stages of Fitz-Hugh–Curtis syndrome
- Add metronidazole when anaerobes are a particular concern

MEDICATION

- Ampicillin/sulbactam: 3 g IV q6h
- Azithromycin: 1 g PO once per week for 2 wk
- Cefotetan: 2 g IV q12h

- Cefoxitin: 2 g IM single dose (outpatient); 2 g IV q6h (inpatient)
- Ceftriaxone: 250 mg IM single dose
- Clindamycin: 450 mg PO QID for 14 days (outpatient); 900 mg IV q8h (inpatient)
- Doxycycline: 100 mg PO BID for 14 days (outpatient); 100 mg IV or PO q12h (inpatient)
 - Oral doxycycline is preferred due to pain of IV infusion
 - IV and oral doxycycline have similar bioavailability
- Gentamicin: 2 mg/kg loading dose followed by 1.5 mg/kg IV q8h. Single daily IV dosing of gentamicin may also be used.
- Metronidazole: 500 mg PO BID for 14 days (outpatient); 500 mg IV q8h (inpatient)
- Probenecid: 1 g PO single dose

First Line

- For outpatient:
 - Ceftriaxone or cefoxitin/probenecid + doxycycline
 - With metronidazole when anaerobes are a particular concern, in suspected *Trichomonas vaginalis* infection
 - Or in women with recent history of pelvic instrumentation
- Of note, oral cephalosporins are no longer a recommended treatment for gonococcal infections (CDC recommends combination therapy with single IM dose of ceftriaxone + oral azithromycin or doxycycline)
- For inpatient:
 - Doxycycline + cefoxitin or cefotetan

Second Line

- For outpatient:
 - Ceftriaxone + azithromycin with or without metronidazole
- For inpatient:
 - Gentamicin + clindamycin; or ampicillin/sulbactam + doxycycline



FOLLOW-UP

DISPOSITION

Admission Criteria

- Uncertain diagnosis and toxic appearance
- Suspected pelvic abscess, including TOA
- Pregnancy
- Immunodeficiency
- Severe illness (e.g., vomiting or severe pain)
- Failure of outpatient therapy

- Probable noncompliance with outpatient therapy (e.g., adolescents)
- Consider admission if appropriate clinical follow-up cannot be arranged

Discharge Criteria

- Patients who do not meet admission criteria may be treated as outpatients
- Recent studies have shown that in women with mild to moderate PID, there was no difference in reproductive outcomes between women randomized to inpatient vs. outpatient treatment

Issues for Referral

TOAs may require drainage or surgical intervention in addition to antibiotics

FOLLOW-UP RECOMMENDATIONS

- If outpatient therapy is selected, it is important to have follow-up in 48–72 hr to assess for clinical improvement
- If the patient has not defervesced by 72 hr, inpatient treatment and further evaluation should be considered

PEARLS AND PITFALLS

- PID represents a spectrum of disease from simple endometritis to fatal intra-abdominal sepsis
- Quinolones and oral cephalosporins are no longer recommended in US for the treatment of gonorrhea or associated conditions such as PID, due to increasing rates of resistance
- Patients with PID should have extensive counseling and testing for other STDs, including HIV
- Male sex partners of women with PID should be treated if they had sexual contact with the patient during the previous 60 days prior to the patient's onset of symptoms

ADDITIONAL READING

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CODES

ICD9

- 079.88 Other specified chlamydial infection
- 098.19 Other gonococcal infection (acute) of upper genitourinary tract
- 614.9 Unspecified inflammatory disease of female pelvic organs and tissues

ICD10

- A54.24 Gonococcal female pelvic inflammatory disease
- A56.11 Chlamydial female pelvic inflammatory disease
- N73.9 Female pelvic inflammatory disease, unspecified

PEMPHIGUS

Dustin G. Leigh • Deepi G. Goyal

BASICS

DESCRIPTION

- Autoantibody (IgG)-mediated blistering disease of the skin and mucous membrane:
 - Characterized by loss of cell-to-cell adhesion called acantholysis
- Median age 71 yr
 - Reports of disease occurring in neonates through elderly
- Rare; worldwide incidence 0.7/100,000
- Females > males, 66 vs. 34%
- *Pemphix* is Greek for bubble or blister
- Pemphigus, specific term for autoantibody disease against some portion of epidermis
- Pemphigoid: A term describing the group of syndromes that cause a separation of the epidermis from the dermis, typically more benign course
- Mortality is highest in those with mucocutaneous involvement
 - If untreated, mortality rates average 60–90%, with treatment this nears 5%
- 3 major subtypes exist:
 - Vulgaris; typically more serious with deeper mucocutaneous involvement:
 - Accounts for 70–80% of all pemphigus
 - Up to 70% with vulgaris present with oral lesions, which is often the presenting complaint
 - Autoantibodies to Dsg 1 and 3
 - Affects most races in middle age and elderly Ashkenazi Jews
 - Follicular; milder and more superficial cutaneous lesions:
 - Oral lesions and better prognosis
 - Autoantibodies to Dsg 1 only
 - Paraneoplastic pemphigus; often with severe mucocutaneous involvement
 - Most commonly seen in lymphoreticular malignancies

Pediatric Considerations

- Pemphigus is rare in neonates and children but may occur in adolescents
- Early diagnosis and treatment significantly impact growth, psychological, social, and cultural development
- Histopathology is identical to adult disease
- Neonates may develop the disease secondary to transplacental transfer of IgG
- Neonatal pemphigus spontaneously resolves in several weeks as the maternal antibodies are catabolized

Pregnancy Considerations

Effective treatment of maternal disease prior to conception lowers the risk of neonatal transmission and gestational complications

ETIOLOGY

- IgG autoantibodies are directed against desmosomal cadherins desmoglein 1 and desmoglein 3 found in all keratinocytes
- Autoantibodies cause histopathologic acantholysis, cytoskeletal derangements, and apoptosis
- Bullae formation is caused by the loss of cell–cell adhesion and separation of the keratinocytes
- Immunogenetic predisposition secondary to higher frequencies of specific human leukocyte antigen HLA haplotypes including DR4 and DRw6
- Drugs such as penicillamine, captopril, rifampin, piroxicam, and phenobarbital can trigger pemphigoid reactions
- Endemic pemphigus foliaceus (fogo selvagem), most common in South America, may be triggered or transmitted by bites from flying insects
- Pemphigoid reactions may occur in association with a neoplasm, usually lymphoma (paraneoplastic pemphigus)



DIAGNOSIS

SIGNS AND SYMPTOMS

- Generalized or focal flaccid bullae (blisters) of the skin and mucosa
- Painful skin erosions with shreds of detached epithelium
- Painful nonhealing oral, vaginal, or mucosal erosions
- Crusting, partially healing skin erosions from ruptured bullae
- Hypertrophic, hyperplastic erosive plaques with pustules in intertriginous areas (pemphigus vegetans)
- Moist, edematous, exfoliative erosions in seborrheic areas (pemphigus foliaceus)
- Erythematous, scaly, crusting skin lesions in a malar distribution (pemphigus erythematosus)
- Lesions usually persist without treatment:
 - May heal with post inflammatory hyperpigmentation

History

- Typically features mucocutaneous blisters followed by erosions
- Often appear 1st in mucous membranes with spread to cutaneous involvement; most commonly to scalp, chest, axillae, and groin
- Skin lesions are painful flaccid blisters that may appear anywhere

Physical-Exam

Nikolsky sign (separation of the epidermis with lateral pressure) is characteristic but not diagnostic:

- Poor sensitivity

ESSENTIAL WORKUP

- Suspected based on clinical presentation
- Biopsy with histologic and immunofluorescence testing is essential for definitive diagnosis (arrange with a dermatologist)

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Serum antibody titers, detected by indirect immunofluorescence, are often used as a marker of disease activity; however, the ED physician usually does not order these titers
- ELISA may be used to identify subtypes

Imaging

No diagnostic imaging test exists

Diagnostic Procedures/Surgery

Deep shave or punch biopsy

DIFFERENTIAL DIAGNOSIS

- Bullous pemphigoid
- Contact dermatitis
- Dermatitis herpetiformis
- Erythema multiforme
- Erysipelas
- Erythroderma
- Toxic epidermal necrolysis
- Epidermolysis bullosa
- Hand, foot, and mouth disease
- Systemic lupus erythematosus
- Systemic vasculitis
- Oral candidiasis
- Herpes simplex gingivostomatitis
- Erosive lichen planus
- Seborrheic dermatitis



TREATMENT

PRE HOSPITAL

- If severe disease:
 - IV access, pulse oximetry monitor, and cardiac monitor

INITIAL STABILIZATION/THERAPY

- If symptoms of hypotension or sepsis are present, IV fluid resuscitation should be guided by the Parkland burn formula
- If signs or symptoms of sepsis are present, initiate broad-spectrum antibiotic coverage
- In steroid-dependent patients, administer stress-dose steroids

ED TREATMENT/PROCEDURES

- Systemic corticosteroids are the mainstay of therapy
- *Mild-to-moderate disease* should receive PO prednisone, and intralesional triamcinolone acetonide may be used
- *Severe disease*: Conventional high-dose corticosteroids:
 - If severe symptoms are unresponsive to high-dose PO corticosteroids, consider pulse IV corticosteroids and admission for plasmapheresis
- Adjuvant immunosuppressive therapy may also be added to decrease the symptoms associated with high-dose systemic corticosteroids or in patients with contraindications to steroid therapy:
 - Dapsone, gold, azathioprine, cyclophosphamide, cyclosporine, methotrexate, mycophenolate, and IV immunoglobulins

MEDICATION

First Line

- Immune suppression:
 - Hydrocortisone: 100–300 mg/d IV stress-dose steroids adjusted based on patients known dosage and use habits
 - Methylprednisolone (pulse IV therapy; adults): 1 g IV over 3 hr daily
 - Prednisone: 1 mg/kg/d PO daily (adults); moderate-to-severe disease PO daily for 5–10 wk, then taper
 - Triamcinolone acetonide for limited intraoral involvement – 10 mg/mL 0.1-mL injection into each superficial lesion
- Pain:
 - Opiates, anti-inflammatory agents, acetaminophen
 - Biobrane synthetic dressing
 - Diphenhydramine and Maalox or Xylocaine oral wash

Second Line

- Usually performed as an inpatient for severe, refractory cases
- Immune suppression:

- IVIG: single cycle 400 mg/kg per day for 5 days
- Rituximab
- Triamcinolone acetonide: 10 mg/mL 0.1-mL injection into each superficial lesion
- Pain:
 - Gabapentin 300 mg daily titrated up to 300 mg TID over a month
- Other considerations: Patients on high-dose steroids should have diets high in vitamin D and calcium and may benefit from a proton-pump inhibitor or bisphosphonates.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Most acute flares are minor and can be managed with PO glucocorticoids and dermatology follow-up
- Admit 1st-time presentations of disease to facilitate treatment and definitive diagnosis with biopsy and rule out of high morbidity blistering skin disease
- Admit patients with extensive mucocutaneous involvement, intractable pain, coexisting bacterial skin infection, or signs of sepsis
- Admit to a floor bed if pulse parenteral steroid therapy or plasmapheresis is indicated
- Admit to the ICU or burn unit if any signs and symptoms of shock or sepsis are present because aggressive fluid resuscitation, wound care, and multiple medications will be required

Discharge Criteria

- Discharge if mild-to-moderate disease will not require aggressive steroid management, plasmapheresis, or aggressive pain control

FOLLOW-UP RECOMMENDATIONS

- A follow-up evaluation with dermatology is essential to monitor the course of the disease and to adjust treatment
- Rheumatology follow-up may be advantageous to assess risk of osteoporosis via bone scan if on high-dose steroids

PEARLS AND PITFALLS

- Mucocutaneous lesions often begin on face, head/scalp, or oral cavity
- Long-term management is the rule; ensure proper dermatology follow-up

- Glucocorticoids are the mainstay of therapy
- Paraneoplastic type often with severe oral mucosal involvement, consider associated lymphoproliferative disorder
- Patients on immunosuppressive treatment including steroids and immunomodulating agents are at very high risk of complications and may present in adrenal crisis, severe sepsis, or hyperosmolar nonketotic acidosis secondary to new-onset type 2 diabetes
- Patients with hypotension require aggressive fluid resuscitation

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See Also (Topic, Algorithm, Electronic Media Element)

- Erythema Multiforme
- Rash
- Toxic Epidermal Necrolysis



CODES

ICD9

- 684 Impetigo
- 694.4 Pemphigus
- 694.6 Benign mucous membrane pemphigoid

ICD10

- L10.0 Pemphigus vulgaris
- L10.2 Pemphigus foliaceus
- L10.9 Pemphigus, unspecified

PENILE SHAFT FRACTURE

Ian R. Grover



BASICS

DESCRIPTION

- Traumatic rupture of the corpus cavernosum and the encompassing tunica albuginea
- May involve the corpus spongiosum and urethra
- Hematoma formation occurs at rupture site.
- Injury is usually unilateral and transverse.
- Most common fracture site is the proximal shaft of the penis.
- During erection, pressure within the corpus cavernosum is maximal, close to arterial pressure, increasing the volume in each corpus to maximum, which thins the tunica albuginea, making it susceptible to rupture.
- Penile erection stretches the spongiosum to the limit, which limits movement vertically while allowing lateral movements; this forms a bend at the base of the penis, making it vulnerable to lateral swing and rupture of corpus cavernosum.
- 25–30% have associated urethral injury, which may be partial or complete.
- Caused by blunt trauma to erect penis during:
 - Sexual intercourse
 - Manipulation
 - Fall on erect penis
 - Entanglement in clothing
 - “*Taghaandan*”—Middle Eastern practice of forcefully bending the erect penis to cause detumescence

ETIOLOGY

- Peyronie disease
- Urethritis in past
- Surgical procedure on corpus cavernosum or trauma to corpus cavernosum resulting in weak scar tissue



DIAGNOSIS

SIGNS AND SYMPTOMS

- Loud popping or crunching sound heard at the time of injury
- Immediate detumescence
- Severe penile pain
- Deviation of the penis away from the side of injury
- Penile swelling and ecchymosis

- There may be blood at the urethral meatus if there is a urethral injury.
- May have dysuria, inability to void, or an increase in the size of the swelling with voiding due to extravasation of urine

History

- Cause of the injury
- Sudden painful sensation in erect penis during sexual intercourse or soon after with loss of erection
- Blood at the urethral meatus after intercourse
- Problems with poor erections after the injury if presentation is delayed
- Penile deviation with erection
- Urinary retention or weak urinary stream

Physical-Exam

- Swelling and blue-black discoloration at base of penis, usually on one side
- Ecchymosis may also involve scrotum.
- Penis flaccid and edematous with angulation away from the side of tear
- Defect in the penile shaft may be palpable at the site of the tear.
- Blood at tip of penis or frank hematuria suggests an associated urethral injury.
- Urethrocavernous or urethrocutaneous fistulas may be present as late complications of a penile fracture.

ESSENTIAL WORKUP

- Urinalysis
- PT/PTT
- Retrograde urethrography if urethral trauma is suspected

DIAGNOSIS TESTS & INTERPRETATION

Lab

Urinalysis to evaluate urethral trauma:

- May have frank blood or microscopic hematuria
- PT/PTT if patient is on Coumadin or has a history of bleeding disorder

Imaging

- Retrograde urethrography—recommended in all cases of suspected urethral trauma:
 - Should be done with low pressure during injection, before urethral catheterization
- Cavernosography and MRI of penis may be needed to confirm diagnosis and site of tear.
- Ultrasonography may also be done to confirm a suspected tear.

Diagnostic Procedures/Surgery

Diagnostic exploration of the penis is recommended when cavernosography is negative but clinical suspicion of a fracture is high.

DIFFERENTIAL DIAGNOSIS

- Cellulitis of penis
- Contusion of penis
- Lymphangitis of penis
- Neoplasm of penis
- Paraphimosis
- Trauma because of constrictive ring or other structure
- Urethral injury
- Vasculature rupture, especially superficial or deep dorsal vein or dorsal artery



TREATMENT

PRE HOSPITAL

- Other injuries take precedence in the setting of multiple trauma.
- Local treatment: Ice packs to the penis; splinting of the penis with tongue blade
- Elevate the area to reduce swelling.

INITIAL STABILIZATION/THERAPY

- Pain control
- Needle suprapubic cystostomy in patients with urethral trauma and a full bladder to relieve patient discomfort

ED TREATMENT/PROCEDURES

- Combined efforts of ED physician and urologist are aimed toward restoration of normal shape of penis and sexual and urinary functions.
- ED treatment is directed to reducing hemorrhage, preventing further complications.
- Prophylactic antibiotic use is unnecessary.
- Urethral catheterization in all cases after excluding urethral trauma
- Urologic evaluation and early surgical treatment are essential to prevent complications such as erectile dysfunction, impotence, penile deformity, urethral stricture.
- All patients with suspected or definite diagnosis *must* have early urologic evaluation.

MEDICATION

- Diazepam: 2–5 mg IV q1–6h PRN anxiety
- Fentanyl: 0.05–0.2 mg IV q1h PRN pain
- Hydromorphone: 0.5–1 mg IV q1–2h PRN pain

- Lorazepam: 0.5–1 mg IV q1–6h PRN anxiety
- Morphine sulfate: 0.1 mg/kg IV q1h PRN pain



FOLLOW-UP

Admission Criteria

ALERT

All patients with penile fracture must be hospitalized for prompt surgery.

Issues for Referral

If immediate urologic consultation and treatment are unavailable, patient may be transferred to a suitable hospital after initial stabilization and transfer criteria have been met.

FOLLOW-UP RECOMMENDATIONS

Follow up with urologist to ensure adequate repair and return to normal sexual and urinary function.

PEARLS AND PITFALLS

- Penile fracture is not a rare occurrence.
- Coitus and penile manipulation are the most common causes.
- Delay in seeking treatment is the major cause of morbidity.
- Mainly a clinical diagnosis:
 - Cavernosography, MRI, and US may be used to confirm the diagnosis.
 - Early surgical repair is important.

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See Also (Topic, Algorithm, Electronic Media Element)

- Urethral Trauma
- Paraphimosis



CODES

ICD9

959.13 Fracture of corpus cavernosum penis

ICD10

S39.840A Fracture of corpus cavernosum penis, initial encounter

PEPTIC ULCER

Yanina Purim-Shem-Tov

BASICS

DESCRIPTION

- Produced by breakdown in gastric or duodenal mucosal defenses
- Imbalance exists between production of acid and ability of mucosa to prevent damage.

ETIOLOGY

- *Helicobacter pylori*:
 - _ Gram-negative spiral bacteria that live in mucous layer
 - _ Responsible for 90–95% of duodenal ulcers and 80% of gastric ulcers
 - _ Increases antral gastrin production and decreases mucosal integrity
- NSAIDs:
 - _ Interfere with prostaglandin synthesis
 - _ Lead to break in mucosa
- Aspirin
- Cigarette smoking
- Alcohol
- Severe physiologic stress
- Hypersecretory states (uncommon)
- Genetics (> 20% have family history)

DIAGNOSIS

SIGNS AND SYMPTOMS

- Epigastric pain or tenderness (80–90%):
 - _ Burning, gnawing, aching pain
 - _ Location: midline, xiphoid, or umbilicus
- Duodenal ulcers:
 - _ Pain occurs 90 min -- 3 hr after meals
 - _ Usually awakens patient at night
 - _ Food and antacids relieve pain
- Gastric ulcers:
 - _ Pain worsens after meals
 - _ Nausea and anorexia
- Difficult to differentiate clinically between gastric and duodenal ulcers
- Relief of pain with antacids
- Heme-positive stools

- Complications of peptic ulcer disease (PUD):
 - Acute perforation:
 - Rigid, boardlike abdomen
 - Generalized rebound tenderness
 - Pain radiation to back or shoulder
 - Obstruction:
 - Pain with vomiting
 - Succussion splash from retained gastric contents and abdominal distention
 - Hemorrhage:
 - Hematemesis
 - Melena
 - Hypotension
 - Tachycardia
 - Skin pallor
 - Orthostatic changes

History

- NSAID, Aspirin
- Smoking
- Previous history of PUD
- Family history of stomach cancer
- Abdominal pain
- Diarrhea
- Weakness

Physical-Exam

- Abdominal pain
- Signs of anemia
- Guaiac-positive stool

ESSENTIAL WORKUP

- Careful physical exam including Hemoccult testing and vital signs with orthostatics
- For stable patients, oral GI cocktail typically relieves pain:
 - Antacid: 30 mL
 - Viscous lidocaine: 10 mL

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Normal lab values in uncomplicated ulcer disease
- CBC:
 - Low hematocrit with bleeding

- Leukocytosis with perforation/penetration
- Amylase/lipase:
 - Elevated with perforation/penetration
 - Pancreatitis in differential diagnosis
- Electrolytes, BUN/creatinine, glucose for critically ill
- Type and cross-match for significant blood loss
- *H. pylori* testing (urea breath test, *H. pylori* antibodies, IgG)

Imaging

- Chest radiograph/abdominal series:
 - Evaluate for perforations/obstructions

Diagnostic Procedures/Surgery

- ECG:
 - For elderly patients
 - Myocardial ischemia in differential diagnosis
- Endoscopy:
 - Procedure of choice
 - Outpatient unless significant hemorrhage
 - Allows for biopsies of gastric/duodenal ulcers for presence of *H. pylori*
 - Detects malignant gastric ulcers
- Upper GI series:
 - Single contrast barium diagnoses 70–80%
 - Double contrast diagnoses 90%
- Gastrin level is elevated in Zollinger–Ellison syndrome

DIFFERENTIAL DIAGNOSIS

- Gastroesophageal reflux
- Biliary colic
- Cholecystitis
- Pancreatitis
- Gastritis
- Abdominal aortic aneurysm
- Aortic dissection
- Myocardial infarction
- Subset with symptoms and no ulcer on endoscopy called *nonulcer dyspepsia*



TREATMENT

PRE HOSPITAL

- ABCs
- IV fluid resuscitation for hypotensive/shock patients

INITIAL STABILIZATION/THERAPY

- ABCs
- Identify ulcer complications (hemorrhage, perforation, obstruction)
- Treat hypotension with lactated Ringer/normal saline fluid bolus via 2 large-bore IVs
- Type and cross early
- Nasogastric tube (NGT) for gastric decompression/check for hemorrhage

ED TREATMENT/PROCEDURES

- Pain control with antacids (GI cocktail) or IV H₂ antagonists
- Avoid narcotics—may mask serious illness.
- Promotion of ulcer healing:
 - Antacids
 - H₂ antagonists (cimetidine, famotidine, ranitidine, nizatidine):
 - May continue for 2–5 yr for ulcer suppression therapy
 - Proton-pump inhibitors (PPIs; omeprazole, lansoprazole, or pantoprazole):
 - If H₂ antagonists have failed
 - Sucralfate
 - Prostaglandin congeners (misoprostol)
 - Sucralfate, H₂-receptor antagonists, and PPIs should not be combined because of lack of documented benefit
- Gastric outlet obstruction:
 - Decompress stomach with NGT
 - IV hydration
- Gastric hemorrhage:
 - IV fluid resuscitation
 - Blood transfusion depending on loss/hematocrit
 - Foley catheter to monitor volume status
 - GI consultation
- Perforation:
 - IV hydration
 - Foley catheter to monitor hydration status
 - Preoperative antibiotics
 - Emergency surgical consultation
- Treatment of *H. pylori* infection:
 - Invasive or noninvasive testing to confirm infection
 - Oral eradication antibiotic therapy options:
 - PPI (omeprazole 20 mg BID or lansoprazole 30 mg PO BID) and 2 antibiotics (clarithromycin 500 mg BID + metronidazole 500 mg BID) for 14 days
 - H₂ blocker, bismuth subsalicylate (Pepto-Bismol) + either amoxicillin

- 1,000 mg BID or tetracycline 500 mg QID in combination with either metronidazole 250 mg QID or clarithromycin 500 mg BID for 14 days
- Most common regimen: Omeprazole 20 mg or lansoprazole 30 mg + clarithromycin 500 mg and amoxicillin 1 g, all taken twice a day for 2 wk
- Stop NSAIDs
- Surgical therapy:
 - Refractory ulcer
 - Complications:
 - Bleeding
 - Perforation
 - Pyloric stenosis

MEDICATION

- Bismuth subsalicylate: 2 525 mg tabs PO
- Maalox Plus: 2–4 tabs PO QID
- Misoprostol: 100–200 mg PO QID
- Mylanta II: 2–4 tabs PO QID
- Sucralfate: 1 g PO QID for 6–8 wk
- Famotidine (H₂ blocker): 40 mg PO nightly at bedtime (peds: 0.5–0.6 mg/kg q12h) for 6–8 wk
- Nizatidine (H₂ blocker): 300 mg PO nightly at bedtime for 6–8 wk; 20 mg PO BID (peds: 0.6–0.7 mg/kg q12–24h) for 2 wk
- Ranitidine (H₂ blocker): 300 mg PO nightly at bedtime (peds: 5–10 mg/kg/24h given q12h) for 6–8 wk
- Cimetidine (H₂ blocker): 400 mg PO BID for 6–8 wk
- Lansoprazole (PPI): 30 mg PO BID for 2 wk
- Pantoprazole (PPI): 40 mg PO daily for 2 wk
- Omeprazole (PPI): 20 mg PO BID for 2 wk
- Rabeprazole (PPI): 20 mg PO daily for 6 wk
- Esomeprazole (PPI): 40 mg daily for 4 wk
- *H. pylori* therapy:
 - PPI (omeprazole 20 mg or lansoprazole 30 mg), clarithromycin 500 mg BID for 2 wk, amoxicillin 1 g BID for 2 wk
 - For penicillin-allergic patients: PPI + clarithromycin 500 mg BID + metronidazole 500 mg BID for 14 days
 - 4-drug therapy: H₂ blocker, bismuth subsalicylate (Pepto-Bismol) + either amoxicillin 1,000 mg BID or tetracycline 500 mg QID in combination with either metronidazole 250 mg QID or clarithromycin 500 mg BID for 14 days

H. pylori eradication regimes:

- PPI (omeprazole 20 mg or lansoprazole 30 mg), clarithromycin 500 mg BID for 2 wk, amoxicillin 1 g BID for 2 wk
- For penicillin-allergic patients: PPI + clarithromycin 500 mg BID + metronidazole 500 mg BID for 14 days
- Sequential 10 day therapy in high prevalence areas:
 - Double therapy for 5 days
 - PPI
 - Amoxicillin
 - Followed by triple therapy for 5 days
 - PPI
 - Clarithromycin
 - Metronidazole
- 4-drug therapy: H₂ blocker, bismuth subsalicylate (Pepto-Bismol) + either amoxicillin 1,000 mg BID or tetracycline 500 mg QID in combination with either metronidazole 250 mg QID or clarithromycin 500 mg BID for 14 days

Second Line

1 wk quadruple therapy:

- Bismuth subsalicylate 120 mg PO QID, tetracycline PO 500 mg QID, metronidazole 400 mg PO QID, esomeprazole 20 mg PO BID
- 80% eradication rate



FOLLOW-UP

DISPOSITION

Admission Criteria

- Gastric obstruction
- Perforation
- Active upper GI bleed
- Melena
- Uncontrolled pain
- Anemia requiring transfusion

Discharge Criteria

- Unremarkable physical exam with normal CBC and heme-negative stools
- If heme-positive stools, discharge if stable vital signs, normal hematocrit, and negative NGT aspiration for upper GI hemorrhage

Issues for Referral

FOLLOW-UP RECOMMENDATIONS

- High-risk patients include those with the following characteristics:
 - Bleeding with hemodynamic instability
 - Repeated hematemesis or any hematochezia
 - Failure to clear with gastric lavage
 - Coagulopathy
 - Comorbid disease
 - Advanced age
 - Patients with ulcer perforation or penetration require operative repair.
- All patients require primary care follow-up in 2–6 wk to evaluate efficacy of treatment.
- Patients > 55 yr and patients with severe symptoms should receive GI referral for endoscopy and testing for *H. pylori*.

PEARLS AND PITFALLS

- *H. pylori* is the most common cause of PUD.
- NSAID-induced PUD is frequently silent.
- Dyspeptic symptoms are nonspecific.
- Endoscopy is diagnostic and should include *H. pylori* screening.
- Treatment should include *H. pylori* eradication and H₂ blockers or PPIs.
- Complications include perforations, hemorrhage, anemia.
- Failure to follow up may result in failure to diagnose gastric cancer.

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- Gastroesophageal Reflux Disease
- Gastritis
- Gastrointestinal Bleeding



CODES

ICD9

- 531.30 Acute gastric ulcer without mention of hemorrhage or perforation, without mention of obstruction
- 532.30 Acute duodenal ulcer without mention of hemorrhage or perforation, without mention of obstruction
- 533.90 Peptic ulcer of unspecified site, unspecified as acute or chronic, without mention of hemorrhage or perforation, without mention of obstruction

ICD10

- K25.3 Acute gastric ulcer without hemorrhage or perforation
- K26.3 Acute duodenal ulcer without hemorrhage or perforation
- K27.9 Peptic ulc, site unsp, unsp as ac or chr, w/o hemor or perf

PERFORATED VISCUS

Rosaura Fernández • Jeffrey J. Schaidler

BASICS

DESCRIPTION

- Perforation/break in the containing walls of an organ with contents spilling into peritoneal cavity
- Inflammation/infection
- Ulceration
- Shearing/crushing or bursting forces in trauma
- Obstruction
- Chemical and/or bacterial peritonitis occurs as result of disruption of gastric or intestinal lining into peritoneal cavity.

ETIOLOGY

- Peptic ulcer disease:
 - Majority of cases caused by NSAIDS and *Helicobacter pylori*
- Esophageal
- Small bowel:
 - Ischemia, foreign body, neoplasms, inflammatory bowel disease
- Large bowel:
 - Diverticular disease, foreign body, neoplasms, inflammatory bowel disease
- Appendicitis
- Penetrating or blunt trauma
- Iatrogenic:
 - Endoscopy, colonoscopy
- Radiation enteritis and proctitis

Pediatric Considerations

- Trauma is the more common cause of rupture:
 - Neonates with difficult birth/child abuse/motor vehicle accidents and falls
- Jejunum is the most common site of rupture.

DIAGNOSIS

SIGNS AND SYMPTOMS

- Sudden severe abdominal pain:
 - Initially local
 - Often rapidly becoming diffuse due to peritonitis
 - Consider persistent local pain due to abscess/phlegmon formation

- Rigidity
- Guarding
- Rebound tenderness
- Absent bowel sounds
- SIRS
- Hypovolemic or septic shock:
 - Hypotension
 - Tachycardia
 - Tachypnea

Geriatric Considerations

- 1/3 without complaints of PUD
- May not have dramatic pain/peritoneal findings on exam:
 - Less rebound and guarding due to less abdominal wall musculature
 - Chronic use of pain meds
- May present with altered mental status
- Hypothermic, suppressed tachycardia

ESSENTIAL WORKUP

Upright chest radiograph:

- Best demonstrates pneumoperitoneum
- When in upright position for 5–10 min, may detect as little as 1–2 mL of free air under diaphragm

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC
- Electrolytes, BUN/creatinine, glucose
- Lipase
- Urinalysis
- Liver function test, coagulation panel
- ABG
- Lactate
- Consider type and cross match for blood

Imaging

- Upright CXR:
 - To detect air under diaphragm
 - Sensitivity ranges from 50% to 85%
- Abdominal radiographs:
 - Left lateral decubitus film more helpful than supine abdomen.
 - Double wall sign of perforated viscus:

- Air in intestinal lumen and peritoneal cavity allows for visualization of both serosal (not normally seen) and mucosal surfaces of intestine.
- Abdominal CT:
 - _ Detects small amounts of free air from perforated viscous
- ECG

DIFFERENTIAL DIAGNOSIS

- Pneumomediastinum with peritoneal extension
- Appendicitis/cholecystitis/pancreatitis
- Pneumonia
- DKA
- Intra-abdominal abscess
- Peptic ulcer disease
- Inferior wall myocardial infarction
- Obstruction

Geriatric Considerations

Atypical symptoms of pain, lack of fever, absence of leukocytosis more likely due to population's suppressed immunity, common comorbidities

- AAA
- Acute mesenteric ischemia
- Atypical presentations of conditions listed in DDx

Pregnancy Considerations

Rule out ectopic pregnancy



TREATMENT

PRE HOSPITAL

Initiate IV fluids for patients with history of vomiting or abnormal vital signs.

INITIAL STABILIZATION/THERAPY

Treat hypotension/tachycardia with 0.9% normal saline:

- Adults: 500 mL–1 L bolus:
 - _ Repeat bolus as necessary permitting patient can tolerate aggressive fluid resuscitation
 - _ Consider vasopressors if fluids not tolerated or not sufficient to maintain physiologic stability
- Pediatric: 20 mL/kg bolus:
 - _ Considerations similar as in adult population

ED TREATMENT/PROCEDURES

- Nasogastric tube
- Foley catheter
- Administer broad-spectrum antibiotics:
 - Cephalosporin/broad-spectrum penicillin +
 - Aminoglycoside/broad-spectrum penicillin/antianaerobe
- Immediate surgical consultation for operative intervention

MEDICATION

Broad coverage antibiotics should be given for enteric gram-negative aerobic and facultative bacilli and enteric gram-positive streptococci

- Metronidazole 500 mg IV (peds: 30–40 mg/kg/d q8h) in addition to 1 of the antibiotics below
- Carbapenem:
 - Meropenem 1 g IV q 8h (peds: 60 mg/kg/d in div. doses q8h)
 - Imipenem–cilastatin 500 mg IV q6h (peds: 60–100 mg/kg/d in div. doses q6h)
 - Doripenem 500 mg IV q8h
- β -lactamase inhibitor combination:
 - Piperacillin–tazobactam 3.375–4 g IV q4–6h (peds: 200–300 mg/kg/d of piperacillin component in div. doses q6–8h)
- Flouroquinolones (used only if hospital surveys indicate > 90% susceptibility of *Escherichia coli* to this class):
 - Ciprofloxacin 400 mg IV q12h
 - Levofloxacin 750 mg IV q24h
- Cephalosporin:
 - Ceftazidime 2 g IV q8h (peds: 150 mg/kg/d in div. doses q8h)
 - Cefepime 2 g IV q8–12h (peds: 100 mg/kg/d in div. doses q12h)
 - Ceftriaxone 1–2 g IV q12–24h (peds: 50–75 mg/kg/d in div. doses q12–24h)
- Morphine sulfate: 2–4 mg (peds: 0.1 mg/kg) IV q2–3h



FOLLOW-UP

DISPOSITION

Admission Criteria

Suspected or confirmed perforation requires admission and immediate surgical consultation.

Discharge Criteria

Discharge not applicable in this situation, as acute perforations are surgical emergencies

Issues for Referral

- General surgery consult for operative intervention
- Consider trauma consult/transfer if applicable

FOLLOW-UP RECOMMENDATIONS

Postoperative surgery follow-up

PEARLS AND PITFALLS

- Obtain upright CXR and abdominal radiographs for patients with suspected perforated viscous.
- CXR without free air does not rule out perforation
- If high clinical suspicion for perforation and plain films normal, obtain CT of abdomen to detect small perforation.
- Obtain immediate surgical consult for operative intervention.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Abdominal Pain



CODES

ICD9

- 533.50 Chronic or unspecified peptic ulcer of unspecified site with perforation, without mention of obstruction
- 562.11 Diverticulitis of colon (without mention of hemorrhage)
- 868.00 Injury to other intra-abdominal organs without mention of open wound into cavity, unspecified intra-abdominal organ

ICD10

- K27.5 Chronic or unsp peptic ulcer, site unsp, with perforation

- K57.20 Diverticulitis of large intestine with perforation and abscess without bleeding
- S36.99XA Other injury of unspecified intra-abdominal organ, initial encounter

PERICARDIAL EFFUSION/TAMPONADE

Louisa S. Canham • Carlo L. Rosen



BASICS

DESCRIPTION

- Pericardial effusion:
 - Pericardial sac usually contains 15–40 cc of fluid
 - Collection of additional fluid = effusion
- Pericardial tamponade:
 - Accumulation of pericardial fluid causes an elevation of pressure in the pericardial space, resulting in impairment of ventricular filling and decreased cardiac output.
 - Depends on size and speed of fluid accumulation
 - Increase of as little as 80–120 cc of fluid may lead to a rise in pericardial pressure.
 - Up to 70% present in “early tamponade” and appear clinically stable
 - Occurs in 2% of patients with penetrating chest trauma

ETIOLOGY

- Medical causes:
 - Pericarditis (20%):
 - 90% idiopathic or viral
 - Bacterial, fungal, parasitic, tuberculosis, HIV
 - Malignancy (13%):
 - Lymphoma, leukemia, melanoma, breast, lung
 - Metastatic disease, primary malignancy, postradiation
 - Postmyocardial infarction (8%):
 - Acute: 1–3 days after acute myocardial infarction (AMI)
 - Subacute (Dressler syndrome): Weeks to months after AMI
 - Incidence reduced with reperfusion therapy
 - End-stage renal disease, uremia (6%)
 - Autoimmune/collagen vascular disease (5%): Rheumatoid arthritis, systemic lupus erythematosus, scleroderma
 - Rheumatic fever
 - Radiation therapy
 - Myxedema
 - Congestive heart failure (CHF), valvular heart disease
 - Drug toxicity (isoniazid, doxorubicin, procainamide, hydralazine, phenytoin)
 - Idiopathic

- Surgical causes:
 - Penetrating chest trauma
 - Thoracic aortic dissection
 - Iatrogenic (cardiac catheterization, postcardiac surgery, central line placement)
 - Blunt trauma rarely causes pericardial effusion.

DIAGNOSIS

SIGNS AND SYMPTOMS

- Beck's triad = classic presentation of cardiac tamponade:
 - Hypotension
 - Muffled heart sounds
 - Jugular venous distention
- Dressler syndrome: Pericarditis seen several weeks after a myocardial infarction:
 - Fever
 - Chest pain
 - Pericardial friction rub

History

- Past medical history is key:
 - History of malignancy?
 - Recent viral illness?
 - Connective tissue disorder?
 - Recent MI?
- History of the present illness:
 - Most are asymptomatic.
 - Pulmonary symptoms: Dyspnea, cough:
 - Dyspnea is the most common symptom seen in tamponade (87–88% sensitivity).
 - Chest pain is the most common symptom:
 - Usually sharp, pleuritic, relieved by sitting forward
 - Can be referred to scapula
 - Can also be dull, aching, constrictive
 - GI symptoms: Nausea or abdominal pain from hepatic and visceral congestion or dysphagia from esophageal compression
 - Generalized symptoms: Fatigue, malaise

Physical-Exam

- Signs of shock or right heart failure:
 - Tachycardia, hypotension
 - Jugular venous distention (may be absent if the patient is also hypovolemic)

- Pericardial friction rub (100% specific):
 - High-pitched “scratchy” sound
 - Best heard at left sternal border
 - Increased by leaning forward
 - Can be transient/intermittent
- Pulsus paradoxus:
 - Fall in systolic BP > 10 mm Hg with inspiration
 - When severe, this can manifest as lack of brachial or radial pulse during inspiration.
 - Sensitive but not specific
- Low-grade fever common; $> 38^{\circ}\text{C}$ is uncommon; if present, consider purulent pericarditis (can also result from autoimmune/connective tissue disease).
- Lungs should be clear; if not, consider CHF or pneumonia.

ESSENTIAL WORKUP

- ECG
- CXR
- US:
 - Echocardiography, including evaluation of aortic root
 - Shock US: Include focused assessment with sonography in trauma, aorta, pleural effusion, and pneumothorax views to rule out other causes of hypotension

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC
- ESR, C-reactive protein:
 - Usually elevated in pericarditis
- Cardiac enzymes:
 - Consider myocarditis if elevated
- Electrolytes:
 - BUN/creatinine in suspected uremic pericarditis
- Coagulation profile:
 - Especially in liver failure, anticoagulation, trauma
- Blood cultures if an infectious source is suspected

Imaging

- Chest radiograph:
 - Cardiomegaly is 89% sensitive for tamponade.
 - Can be normal even with effusion if developed quickly
- Echocardiography:
 - 97–100% sensitive, 90–97% specific

- Effusion: Can detect as little as 20–50 cc of pericardial blood/fluid:
 - Small effusions will only be seen posteriorly.
 - Anterior fat pad may mimic effusion; must also visualize posterior pericardial space for diagnosis of effusion.
- Tamponade:
 - Effusions large enough to cause tamponade should be circumferential.
 - Right atrial or ventricular bowing and eventual collapse
 - “Sniff” test: During inspiration, the inferior vena cava will not collapse in patients with tamponade.
- Chest CT for detecting hemopericardium
- Transesophageal echocardiography
- MRI with gadolinium (for stable patients only)

Diagnostic Procedures/Surgery

- ECG:
 - Low voltage
 - Electrical alternans: Alternating beat-to-beat variation of QRS amplitude (usually only seen with large effusions)
- Pericardiocentesis and fluid analysis:
 - Therapeutic for tamponade or large symptomatic effusion
 - Diagnostic for bacterial effusion (to guide antibiotics) or malignant effusion (for cytology)
- Central venous pressure (CVP) determination:
 - CVP > 15 cm H₂O suggests tamponade, but may be normal in the hypovolemic patient.

DIFFERENTIAL DIAGNOSIS

- Noncardiogenic shock:
 - Hypovolemic, septic, anaphylactic, spinal
- Other cardiac conditions:
 - Myocardial infarction—common misdiagnosis!
 - Pericardial constriction (due to pericardial fibrosis)
 - CHF
- Pulmonary conditions:
 - Pulmonary embolus
 - Tension pneumothorax
 - Hemothorax
- Other causes:
 - Air embolism
 - Aortic dissection
 - Ruptured abdominal aortic aneurysm



TREATMENT

PRE HOSPITAL

- 2 large-bore IV lines
- Start IV fluids.
- Supplemental O₂

INITIAL STABILIZATION/THERAPY

- Continue pre-hospital measures
- Continuous cardiac monitoring
- In tamponade:
 - IV fluid resuscitation with normal saline or blood
 - Pericardiocentesis for unstable patients to decompress the tamponade

ED TREATMENT/PROCEDURES

- Medical causes of tamponade in patients who are unstable:
 - Perform pericardiocentesis with placement of an indwelling catheter for continued drainage:
 - Site of drainage guided by maximum fluid collection
 - Subxiphoid: 2 cm below and 1 cm to the left of the xiphoid process, needle aimed at 30–45° angle toward the patient's left shoulder
 - Left parasternal approach: 5th intercostal space just lateral to sternum, needle inserted perpendicular to the skin
 - Remove fluid as needed to improve clinical condition.
- Traumatic pericardial tamponade:
 - Consult trauma surgeon immediately.
 - Definitive therapy is thoracotomy in the OR.
 - If patient is deteriorating despite resuscitation, ED thoracotomy with pericardotomy is an option.
- Bacterial pericardial effusion:
 - Initiate antibiotic therapy to cover gram-negative and anaerobic organisms and *Staphylococcus aureus*.
 - May ultimately require partial surgical resection of the pericardium
- Uremic pericardial effusion:
 - Arrange urgent dialysis.
- Dressler syndrome and postirradiation pericardial effusion:
 - Initiate aspirin
- Aortic dissection:
 - Immediate cardiothoracic surgical consultation for operative repair

MEDICATION

- Ibuprofen: 800 mg PO q8h

- Indomethacin: 75–150 mg PO daily
- Avoid NSAIDs in patients with CAD
- Steroids:
 - Only for refractory cases (more commonly associated with rebound when tapered)
 - Prednisone: 0.2–0.5 mg/kg, continued for at least 1 mo, slowly tapered



FOLLOW-UP

DISPOSITION

Admission Criteria

- ICU admission for acute, symptomatic pericardial effusion/tamponade
- New pericardial effusion
- Pericarditis with elevated troponin

Discharge Criteria

- Known or incidentally found small pericardial effusion in asymptomatic stable patient
- Pericarditis without evidence of tamponade in a young, healthy person whose pain is controlled with NSAIDs

Issues for Referral

- Trauma surgery:
 - Tamponade in setting of trauma: Will need to go to OR for thoracotomy (or from ED status post ED thoracotomy)
- Cardiothoracic surgery:
 - Tamponade/effusion in the setting of aortic dissection/other primary cardiac problem
 - Patients requiring pericardial window
 - Any patients who have had recent cardiac surgery
- Cardiology/interventional cardiology:
 - Dressler syndrome
 - Recent percutaneous intervention
 - Any patients who need pericardiocentesis

FOLLOW-UP RECOMMENDATIONS

Discharged patients need urgent primary care physician follow-up and repeat echo to evaluate for resolution of effusion.

PEARLS AND PITFALLS

- ECG changes associated with pericarditis include diffuse ST-elevation with PR-depression and eventual T-wave inversion. Should be contrasted with ECG findings of localized ST-elevation with reciprocal ST-depression in AMI.
- Relatively small effusions can cause tamponade if rapidly developing (conversely, large effusions can be relatively benign when they develop slowly).
- Cardiac output can be fluid dependent in tamponade—start fluids early.
- Use bedside US to look for pericardial effusion and other signs of tamponade in the setting of hypotension (including trauma).
- ED thoracotomy should not be employed if there is no OR readily available.

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See Also (Topic, Algorithm, Electronic Media Element)

Cardiogenic Shock



CODES

ICD9

- 423.3 Cardiac tamponade
- 423.9 Unspecified disease of pericardium

ICD10

- I31.3 Pericardial effusion (noninflammatory)
- I31.4 Cardiac tamponade

PERICARDITIS

Terrance T. Lee • Shamai A. Grossman

BASICS

DESCRIPTION

- Inflammation, infection, or infiltration of the pericardial sac surrounding the heart:
 - Pericardial effusion may or may not be present.
- Acute pericarditis:
 - Rapid in onset
 - Potentially complicated by cardiac tamponade from effusion
- Constrictive pericarditis:
 - Results from chronic inflammation causing thickening and adherence of the pericardium to the heart

ETIOLOGY

- Idiopathic (most common)
- Viral:
 - Echovirus
 - Coxsackie
 - Adenovirus
 - Varicella
 - Epstein–Barr virus
 - Cytomegalovirus
 - Hepatitis B
 - Mumps
 - HIV
- Bacterial:
 - Tuberculosis
 - *Staphylococcus*
 - *Streptococcus*
 - *Haemophilus*
 - *Salmonella*
 - *Legionella*
- Fungal:
 - *Candida*
 - *Aspergillus*
 - Histoplasmosis
 - Coccidioidomycosis
 - Blastomycosis

- *Nocardia*
- Parasitic:
 - Amebiasis
 - Toxoplasmosis
 - Echinococcosis
- Neoplastic:
 - Lung
 - Breast
 - Lymphoma
- Uremia
- Myocardial infarction:
 - Dressler syndrome
- Connective tissue disease:
 - Systemic lupus erythematosus
 - Rheumatoid arthritis
 - Scleroderma
- Radiation
- Chest trauma
- Postpericardiotomy
- Aortic dissection
- Myxedema
- Pancreatitis
- Inflammatory bowel disease
- Amyloidosis
- Drugs:
 - Procainamide
 - Cromolyn sodium
 - Hydralazine
 - Dantrolene
 - Isoniazid
 - Penicillins
 - Doxorubicin/daunorubicin



DIAGNOSIS

SIGNS AND SYMPTOMS

- Chest pain
- Fever
- Mild dyspnea
- Cough
- Hoarseness
- Nausea

- Anorexia

History

- Chest pain:
 - _ Pain radiating to the ridge of the trapezius from phrenic irritation
 - _ Central or substernal pain
 - _ Sudden onset
 - _ Sharp
 - _ Pleuritic
 - _ Worse when supine or with cough
 - _ Improved with leaning or sitting forward
- Previous episodes of pericarditis
- History of fever or infection
- History of malignancy or autoimmune disease

Physical-Exam

- Tachypnea
- Tachycardia
- Odynophagia
- Friction rub:
 - _ Heard best at lower left sternal border
 - _ Very specific
 - _ Triphasic rub is classic
 - _ Can have any of these 3 components:
 - Presystolic
 - Systolic
 - Early diastolic
 - _ Intermittent and exacerbated by leaning forward
- Beck triad with the accumulation of pericardial fluid:
 - _ Muffled heart sounds
 - _ Increased venous pressure (distended neck veins)
 - _ Decreased systemic arterial pressure (hypotension)
- Ewart sign:
 - _ Dullness and bronchial breathing between the tip of the left scapula and the vertebral column
- Pulsus paradoxus:
 - _ Exaggerated decrease (> 10 mm Hg) in systolic pressure with inspiration
- Constrictive pericarditis:
 - _ Signs of both right- and left-sided heart failure
 - _ Pulmonary and peripheral edema
 - _ Ascites
 - _ Hepatic congestion

ESSENTIAL WORKUP

- ECG has 4 classic stages
- Stage 1:
 - Concave ST-elevations diffusely except aVR and V1
 - PR segment depressions with elevation in aVR
- Stage 2:
 - Normalization of ST and PR segments
 - T-wave flattening
- Stage 3:
 - Diffuse T-wave inversions
- Stage 4:
 - T-waves normalize, may have some persistent T-wave inversions
- Atypical changes may include localized ST-elevations or T-wave inversions
- Myocardial involvement suggested by intraventricular conduction delay, new bundle branch block, or Q-waves
- Pericardial effusion suggested by electrical alternans

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC:
 - May show leukocytosis
- Erythrocyte sedimentation rate and C-reactive protein:
 - May be elevated, can follow for resolution
- Cardiac enzymes:
 - Helpful in distinguishing pericarditis from myocardial infarction
 - May also be elevated in myopericarditis

Imaging

- CXR:
 - Most often normal
 - May show enlargement of the cardiac silhouette or calcification of pericardium
 - No change in heart size until >250 mL of fluid has accumulated in the pericardial sac
- Echocardiography:
 - Diagnostic method of choice for the detection of pericardial fluid
 - Can detect as little as 15 mL of fluid in the pericardial sac
 - Bedside US good screening tool
- Chest CT:
 - Useful for the detection of calcifications or thickening of the pericardium
 - Can help rule out other etiologies

Diagnostic Procedures/Surgery

Pericardiocentesis:

- Pericardial fluid can help determine underlying etiology.
- Fluid sent for protein, glucose, culture, cytology, Gram and acid-fast stains, and fungal smears

DIFFERENTIAL DIAGNOSIS

- Acute myocardial infarction
- Pulmonary embolism
- Pneumothorax
- Aortic dissection
- Pneumonia
- Empyema
- Cholecystitis
- Pancreatitis



TREATMENT

PRE HOSPITAL

- ABCs, IV access, O₂, monitor
- Consider fluid bolus if no crackles.

INITIAL STABILIZATION/THERAPY

- ABCs
- Emergent pericardiocentesis:
 - For hemodynamic compromise secondary to cardiac tamponade
 - Removal of a small amount of fluid can lead to a dramatic improvement.
 - US guidance if available

ED TREATMENT/PROCEDURES

- Treatment dependent on the underlying etiology
- Idiopathic, viral, rheumatologic, and post-traumatic:
 - NSAID regimens effective
 - Corticosteroids reserved for refractory cases
- Bacterial:
 - Aggressive treatment with IV antibiotics along with drainage of the pericardial space
 - Search for primary focus of infection.
 - Therapy guided by determination of pathogen from pericardial fluid tests
- Neoplastic:
 - Treat underlying malignancy.
- Uremic:

- Intensive 2–6 wk course of dialysis
- Caution should be used if using nonsteroidal medications.
- Expected course/prognosis:
 - Most patients will respond to treatment within 2 wk.
 - Most have complete resolution of symptoms.
- Few progress to recurrent episodes with eventual development of constrictive pericarditis or cardiac tamponade.

MEDICATION

- Ibuprofen 300–800 mg q6–8h for days to weeks depending on severity:
 - Can also be tapered to prevent recurrence
 - Improves coronary blood flow
 - GI prophylaxis with 20 mg omeprazole
- Aspirin 800 mg PO q6–8h × 7–10 days:
 - Taper off over 3–4 wk
 - Omeprazole as with ibuprofen
 - Colchicine 1–2 mg × 1 day, then 0.5–1 mg daily × 3 mo
- Colchicine alone: 1–2 mg × 1 day, then 0.5–1 mg daily × 3 mo:
 - Combination with aspirin decreased recurrence rate
 - Lower doses may also be effective.
- Indomethacin 25–50 mg q6h:
 - May restrict coronary blood flow
- Prednisone 0.2–0.5 mg/kg daily × 2–4 wk with taper:
 - Used for refractory cases
 - For use if aspirin/NSAIDs contraindicated
 - Associated with increased rate of recurrence
 - Also beneficial in uremic and autoimmune pericarditis

Pregnancy Considerations

- NSAIDs and aspirin are not teratogenic in 1st 20 wk of pregnancy
- Glucocorticoids may be used during pregnancy.
- Avoid aspirin and high-dose steroids when breast-feeding.
- Colchicine is generally contraindicated except with familial Mediterranean fever.



FOLLOW-UP

DISPOSITION

Admission Criteria

- ICU:
 - Hemodynamic instability
 - Cardiac tamponade

- Malignant dysrhythmia
- Status postpericardiocentesis
- Telemetry unit:
 - Suspicion of myocardial infarction
 - Severe pain
 - Suspicion of bacterial etiology
 - Any high-risk criteria
- High-risk criteria:
 - Large effusion (> 2 cm total)
 - Anticoagulant use
 - Malignancy
 - Temperature > 38°C
 - Traumatic pericarditis
 - Immunosuppression
 - Pulsus paradoxus
 - Slow onset

Discharge Criteria

- Mild symptoms in patients without any hemodynamic compromise
- Close follow-up
- Able to tolerate a regimen of oral medication
- Debate on need for ECG to evaluate for effusion prior to discharge

Issues for Referral

Follow-up with cardiology:

- Recurrent cases
- Admitted patients

FOLLOW-UP RECOMMENDATIONS

Follow up with primary care physician for re-evaluation and verification of resolution of symptoms and absence of complications in 1–2 wk.

PEARLS AND PITFALLS

- Classic history: Viral illness preceding development of sharp, positional chest pain
- Rub is very specific but not always audible.
- The challenge is distinguishing pericarditis from acute MI and other etiologies of chest pain.
- Mainstay of therapy is NSAIDs.

ADDITIONAL READING

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in acute and recurrent pericarditis. *Heart Fail Rev.* 2013;18(3):355–360.

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- Spodick DH. Risk prediction in pericarditis: Who to keep in hospital? *Heart.* 2008;94:398–399.



See Also (Topic, Algorithm, Electronic Media Element)

Pericardial Effusion/Tamponade



CODES

ICD9

- 420.90 Acute pericarditis, unspecified
- 420.91 Acute idiopathic pericarditis
- 423.2 Constrictive pericarditis

ICD10

- I30.0 Acute nonspecific idiopathic pericarditis
- I30.9 Acute pericarditis, unspecified
- I31.1 Chronic constrictive pericarditis

PERILUNATE DISLOCATION

Judson J. Merritt • Ian R. Grover



BASICS

DESCRIPTION

- Lunate remains located and in line with the radius but the distal carpal bones are displaced dorsally (~95% of the time) or volarly (~5% of the time)
- Early surgical treatment is recommended.
- This injury has a high incidence of post-traumatic arthritis.

ETIOLOGY

- Mechanism of injury is usually wrist hyperextension with ulnar deviation.
- These are high-energy injuries:
 - Falls from a height
 - Motor vehicle accidents
 - Industrial accidents
 - Sporting accidents

ALERT

Scaphoid is frequently fractured with perilunate dislocations.



DIAGNOSIS

SIGNS AND SYMPTOMS

- Severe wrist pain
- Wrist swelling
- Diffuse wrist tenderness
- Paresthesias in the median nerve distribution

History

- History of a high-energy injury
- Any concomitant injuries
- Pain in the wrist
- May complain of paresthesias in the median nerve distribution

Physical-Exam

- Wrist swelling
- Possible deformity of the wrist
- Decreased range of motion of the wrist
- Possible decreased sensation in the median nerve distribution

- Special attention should be paid to skin integrity because open fractures are common.
- Neurovascular status should be monitored closely, including 2-point discrimination.
- Check closely for concomitant injuries, specifically of the upper extremity.

ALERT

Diagnosis is frequently missed on clinical exam.

ESSENTIAL WORKUP

Radiographs of the wrist

DIAGNOSIS TESTS & INTERPRETATION

Imaging

- Radiographic imaging that includes 3 views of the wrist
- Perilunate dislocation visualized best on the true lateral view:
 - Distal carpal row, specifically the capitate, seen dorsally (95% of the time) or volarly (5% of the time) in relation to the lunate
 - Lunate is located and in line with the radius
- CT and MRI are not generally needed for diagnosis, but some orthopedists may request them for preoperative planning.

Pediatric Considerations

- Wrists are rarely sprained in children.
- Wrist radiographs are difficult to interpret in pediatric patients.
- Comparison view of the other wrist may be helpful.

DIFFERENTIAL DIAGNOSIS

- Lunate fracture
- Lunate dislocation:
 - Dislocation occurs between lunate and distal radius.
- Scapholunate dissociation and other similar ligamentous disruptions
- Distal radius fracture

Pediatric Considerations

Consider nonaccidental trauma.



TREATMENT

ALERT

Concern is for concomitant, more serious, injuries.

PRE HOSPITAL

- Assess for other injuries
- Immobilize
- Pain control
- Elevate

INITIAL STABILIZATION/THERAPY

- Identify other, more serious, associated injuries.
- Immobilize
- Elevate
- Ice

ED TREATMENT/PROCEDURES

- Pain control
- Procedural sedation for closed reduction:
 - Etomidate: 0.1–0.15 mg/kg IV
 - Methohexital: 1–1.5 mg/kg IV
 - Propofol: 40 mg IV every 10 sec until induction
- Closed reduction of the dislocation should be done emergently:
 - Arm is hung in traction for 10 min with 10–15 lb of counterweights and the fingers in traps.
 - The fingers are then removed from the traps and manual traction is continued.
 - One of the physician's thumbs is placed volarly over the lunate and then the injury is recreated with wrist extension.
 - Continued traction is applied to the wrist and then slow flexion of the wrist is performed, which usually locates the distal carpal bones.
- Operative fixation to reduce and maintain wrist stability is required.
- Immobilize wrist using a sugar-tong splint in neutral position. Obtain postreduction radiograph.

Pediatric Considerations

Although perilunate dislocation is unusual in pediatric patients, children with wrist pain should be splinted and referred to a pediatric hand surgeon.

MEDICATION

- Diazepam: 2–5 mg IV q2–4h (peds: Max. dose is 0.25 mg/kg q4h) PRN anxiety
- Fentanyl: 0.05–0.2 mg IV q1h PRN pain
- Hydromorphone: 0.5–1 mg IV q4–6h (peds: 0.015 mg/kg/dose q4–6h) PRN pain
- Lorazepam: 0.5–1 mg IV q1–6h (peds: 0.044 mg/kg q4–6h) PRN anxiety
- Morphine sulfate: 0.1 mg/kg IV q1h PRN pain



FOLLOW-UP

DISPOSITION

Admission Criteria

- Open dislocation, presence of multiple trauma, or other, more serious, injuries
- Inability to reduce dislocation or maintain reduction
- Neurovascular compromise

Discharge Criteria

- Closed injuries
- Adequate reduction
- No neurovascular involvement
- Orthopedic follow-up within 2–3 days

Issues for Referral

All patients with perilunate dislocations should be referred to a hand surgeon for surgical stabilization and ligament repair.

FOLLOW-UP RECOMMENDATIONS

- All patients with a perilunate dislocation must follow-up with a hand surgeon for surgical stabilization and ligament repair.
- Follow-up should be within 2–3 days.

PEARLS AND PITFALLS

- Up to 25% of these injuries are missed on initial presentation.
- In a patient with wrist pain, swelling, and limited range of motion, it is important to obtain adequate x-rays of the wrist and make sure that the lunate and capitate are located in their fossa on the lateral wrist x-ray.
- Late presentation of these injuries leads to a very poor outcome and often requires a salvage operation.
- Complications include median nerve injury, tendon problems, complex regional pain syndrome, wrist instability, and post-traumatic arthritis.
- Even with appropriate treatment, there is a high incidence of post-traumatic arthritis and loss of grip strength.

ADDITIONAL READING

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- Stanbury SJ, Elfar JC. Perilunate dislocation and perilunate fracture-dislocation. *J Am Acad Orthop Surg.* 2011;19(9):554–562.



See Also (Topic, Algorithm, Electronic Media Element)

- Carpal Fractures
- Lunate Dislocation
- Scaphoid Fracture



CODES

ICD9

- 814.01 Closed fracture of navicular [scaphoid] bone of wrist
- 833.09 Closed dislocation of wrist, other

ICD10

- S62.009A Unsp fracture of navicular bone of unsp wrist, init
- S63.095A Other dislocation of left wrist and hand, initial encounter
- S63.096A Other dislocation of unspecified wrist and hand, initial encounter

PERIODIC PARALYSIS

Kyle R. Brown • Jeffrey N. Siegelman

BASICS

DESCRIPTION

- Periodic paralysis (PP): Disorder of muscle metabolism usually inherited that leads to flaccid extremity weakness. Exacerbated by hyperkalemia, hypokalemia, thyrotoxicosis
- Primary: Familial AD mutation skeletal muscle calcium, sodium, or potassium channel
- Secondary: Thyrotoxic, hypokalemia, hyperkalemia

EPIDEMIOLOGY

Incidence and Prevalence Estimates

- Hypokalemic PP (HypoPP):
 - MC, 1:100,000 prevalence
 - 1/3 new AD mutations
- Hyperkalemic PP (HyperPP):
 - 1:200,000 prevalence
 - 90% of people with mutation will have clinical symptoms
- Thyrotoxic PP (ThyroPP):
 - Incidence 2% in patients with thyrotoxicosis
 - Higher in Asians
 - Subset of HypoPP, clinically identical
- Andersen–Tawil:
 - Subset of HypoPP
 - Rare
 - Prevalence unknown

ETIOLOGY

- Mutation of skeletal muscle Na channel gene:
 - SCN4A
 - HypoPP, HyperPP:
 - AD inheritance
 - Spontaneous mutation
- Mutation of skeletal muscle calcium channel gene CACN1A5:
 - HypoPP
- Mutation of KCNJ2 gene:
 - Andersen–Tawil:

- AD inheritance
- 50% spontaneous
- M > F
- Age of onset:
 - HypoPP:
 - 1st or 2nd decade
 - HyperPP:
 - 1st decade
 - Andersen–Tawil:
 - 1st or 2nd decade
 - ThyroPP:
 - 2nd–5th decade

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Intermittent weakness:
 - Can be isolated
 - Rapid onset
 - Common for attacks to recur and for weakness to persist between attacks
 - Frequency from single isolated to daily attacks
- Type of attack:
 - Spontaneous
 - At night or early morning
 - Provoked:
 - History of thyroid disease
 - Recent carbohydrate rich meal
 - Rest after strenuous exercise
 - Illness
 - Lack of sleep
 - Medications: Insulin, epinephrine, corticosteroids, β -agonists, diuretics
 - Cold environment
 - Menstruation
 - Reduced sleep
 - Pregnancy
 - Medications that induce thyroid disease
- Length of attack:
 - HypoPP: 1 hr–days
 - HyperPP: 15 min–4 hr

- ThyroPP: Same as HypoPP
- Andersen–Tawil: Variable
- Family history of episodes of weakness

Physical-Exam

- General:
 - ThyroPP:
 - Hyperthermia
- HEENT:
 - HypoPP and HyperPP:
 - Lid lag: Rare
 - Difficulty swallowing: Rare
 - ThyroPP:
 - Exophthalmos
 - Goiter
 - Andersen–Tawil:
 - Dysmorphic features: Short stature, low set ears, broad based nose, micrognathia
- Cardiac:
 - HypoPP and HyperPP:
 - Dysrhythmias possible
 - ThyroPP:
 - Tachycardia, dysrhythmia
 - Andersen–Tawil:
 - Cardiac dysrhythmia
- Pulmonary:
 - HypoPP:
 - Can affect respiratory muscles, rare
 - Severe hypokalemia
- M/S:
 - HypoPP, HyperPP, ThyroPP:
 - Symmetrical muscle weakness in 1 or more extremity
 - Legs > arms
 - Andersen–Tawil:
 - Periodic flaccid muscle weakness <1 hr
 - Proximal > distal
- Neuro:
 - Alert, conscious
 - Sensation intact
 - DTR reduced or absent
 - Skeletal muscle weakness, symmetrical
 - Sphincter normal
- Skin:

- ThyroPP:
 - o Warm, moist

ESSENTIAL WORKUP

Lab tests and EKG

DIAGNOSIS TESTS & NTERPRETATION

- EKG:
 - HypoPP:
 - o Sinus bradycardia
 - o Flattened T-wave
 - o ST-segment depressions
 - HyperPP:
 - o Rarely peaked T-waves
 - ThryoPP:
 - o Tall P-waves, wide QRS, decreased T-wave, AV block, ventricular fibrillation or asystole
 - Andersen–Tawil:
 - o Long QT, ventricular arrhythmias
 - o U-waves, prolonged T-wave downslope
 - o Differentiates Andersen syndrome from other long QT syndromes
- Electrolytes:
 - Potassium:
 - o HyperPP: Normal or increased
 - o HypoPP: Normal or decreased
 - o ThryoPP: Decreased during attacks
 - o Andersen–Tawil: Decreased, normal, or increased
 - Calcium:
 - o ThryoPP: Decreased during attacks
 - Phosphorus:
 - o ThryoPP: Decreased during attacks
- Thyroid Studies:
 - ThryoPP:
 - o TSH: Low
 - o T4: Elevated

Imaging

Not necessary for diagnosis

Diagnostic Procedures/Surgery

None in ED but specialists may consider the following:

- EMG:
 - HypoPP:

- No myotonia
- HyperPP:
 - Myotonia
- Andersen–Tawil
- Muscle biopsy
- Provocative testing:
 - HyperPP:
 - Potassium and epinephrine
 - HypoPP:
 - Insulin and glucose

DIFFERENTIAL DIAGNOSIS

Other causes of hypokalemia or hyperkalemia

- Hyperkalemia:
 - Drugs: Spironolactone, ACE inhibitors, NSAIDs, heparin
 - Hereditary: 21-hydroxylase deficiency, McArdle disease
 - GI:
 - Ileostomy with tight stoma
 - Renal:
 - Chronic renal failure
 - Endocrine:
 - Addison disease
- Hypokalemia:
 - Drugs:
 - Tocolytics, amphotericin B, diuretics, reduced potassium intake, malignant hyperthermia
 - GI:
 - Vomiting
 - Celiac and tropical sprue
 - Short bowel syndrome
 - Renal:
 - Conn syndrome
 - Bartter/Gitelman syndrome
 - Acute tubular necrosis
 - Renal tubular acidosis
 - Neuromuscular:
 - Andersen—Tawil
 - Myasthenia gravis
 - Endocrine:
 - Thyrotoxicosis
 - Hyperaldosteronism
 - DKA



TREATMENT

PRE HOSPITAL

- Supportive:
 - ABC, IV, O₂, monitor

INITIAL STABILIZATION/THERAPY

- Supportive care
- HyperPP:
 - Many attacks brief and do not need treatment
 - IV calcium gluconate may end attack
- HypoPP:
 - Potassium:
 - Preferred: Oral potassium 40 mEq
 - IV potassium 10 mEq 1 or 2 doses only
 - Watch for overcorrection
 - IV hydration can help correct potassium
- Andersen–Tawil:
 - Potassium unpredictable:
 - Could be helpful in hypokalemia
- ThyroPP:
 - Treat thyroid abnormalities:
 - Tachycardia: Nonselective β -blocker
 - Treat underlying abnormalities:
 - Same as in HypoPP
 - See the section on thyrotoxicosis

ALERT

HypoPP should avoid volatile anesthetics and depolarizing muscle relaxants which can cause an attack or malignant hyperthermia



FOLLOW-UP

DISPOSITION

- HypoPP or HyperPP:
 - Lifestyle modifications:
 - Avoid triggers: Ethanol, prolonged exercise, high potassium foods, fasting
- ThyroPP:
 - Depends on severity of underlying disease, if asymptomatic and controlled may consider discharge with consultation with neurologist and endocrinologist.

Admission Criteria

- HypoPP or HyperPP:
 - Consider if severe hypo- or hyperkalemia, still symptomatic, cardiac or respiratory compromise
- Andersen–Tawil:
 - Admit, risk of sudden cardiac death high

Discharge Criteria

- HypoPP, HyperPP, ThyroPP:
 - Resolved symptoms, referral to neurologist, no cardiac or respiratory compromise

Issues for Referral

- Neurology
- Endocrinology for ThyroPP
- Genetic counseling:
 - 50% risk of inheriting primary PP

FOLLOW-UP RECOMMENDATIONS

- Neurology specialist in metabolic myopathies
- Geneticist

PEARLS AND PITFALLS

- Admit Andersen–Tawil patients and all PP patients who remain symptomatic.
- Use caution with volatile anesthetics and depolarizing muscle relaxants in patients with all forms of PP

ADDITIONAL READING

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CODES

ICD9

359.3 Periodic paralysis

ICD10

G72.3 Periodic paralysis

PERIODONTAL ABSCESS

John E. Sullivan

BASICS

DESCRIPTION

- Collection of pus in supporting structures of teeth:
 - Periodontal ligament
 - Alveolar bone
- Periodontal pockets result from progression of periodontal disease and resultant bone loss:
 - Food and debris accumulate in periodontal pockets
 - Coronal epithelial tissues can reattach to tooth while bacteria and food debris remain trapped in pocket, impairing drainage
 - Food and debris become secondarily infected in the setting of impaired drainage
- Complications:
 - Osteomyelitis
 - Dentocutaneous fistula
 - Cavernous sinus thrombosis
 - Ludwig angina
 - Maxillary sinusitis
 - Mediastinitis
 - Tooth loss
 - Sepsis

Pediatric Considerations

- Periodontal abscess is rare in children
- Periapical abscess is more common:
 - Originates in pulp
 - Associated with caries

ETIOLOGY

- Anaerobic gram-negative rods
- Peptostreptococci
- Viridans group streptococci
- Neisseria species
- Usually polymicrobial

DIAGNOSIS

SIGNS AND SYMPTOMS

Periodontal abscess is a clinical diagnosis

History

- Dental pain
- Malaise
- Fever
- Facial swelling

Physical-Exam

- Focal swelling or fluctuance of gums and or face
- Tenderness to palpation
- Increased tooth mobility
- Parulis:
 - _ Pimple-like lesion on gingiva, representing terminal aspect of a sinus tract
 - _ May be seen in chronic abscess
- Expression of pus from sinus tract
- Heat sensitivity
- Lymphadenopathy
- Trismus is generally absent, unless infection has spread to muscles of mastication

ESSENTIAL WORKUP

This is a clinical diagnosis:

- Imaging and lab data are not essential for diagnosis

DIAGNOSIS TESTS & NTERPRETATION

Lab

Anaerobic culture of pus:

- Complicated abscess
- Immunocompromised patients

Imaging

- Panoramic, periapical, or occlusal radiographs
- Bedside US may also aid in confirming diagnosis
- CT may help visualize extension of abscess into adjacent structures
- Imaging can confirm and help define extent of abscess but is not essential to make diagnosis

Diagnostic Procedures/Surgery

Electric pulp testing:

- Performed by dental consultant to verify viability of tooth

- Performed during follow-up visit with dentist

DIFFERENTIAL DIAGNOSIS

- Periapical abscess
- Maxillary sinusitis
- Aphthous ulcers
- Oral herpes
- Salivary gland tumors
- Mumps
- Blocked salivary gland due to sialadenitis or dehydration
- Localized adenopathy due to oral infections
- Facial cellulitis
- Acute otitis media
- Peritonsillar abscess
- Pediatric consideration: Periapical abscess
- For asymptomatic parulis:
 - Fibroma
 - Pyogenic or peripheral ossifying granuloma
 - Kaposi sarcoma



TREATMENT

PRE HOSPITAL

Rarely associated with airway emergencies, but if any signs of airway compromise are present:

- Intubation equipment at bedside
- Transport in sitting position
- Supplemental oxygen
- Suction secretions as needed

INITIAL STABILIZATION/THERAPY

- Assess for airway patency
- Establish definitive airway via endotracheal intubation or cricothyrotomy/tracheostomy in the presence of:
 - Respiratory distress
 - Inability to handle secretions
 - Oropharyngeal tissue swelling that impairs or threatens airway

ED TREATMENT/PROCEDURES

- Analgesia with NSAIDs or opiates may be required
- Incision and drainage:
 - Anesthetize gingiva superficially with 2% lidocaine with 1:100,000 epinephrine until blanching occurs

- Make a 1 cm stab incision using a scalpel blade toward alveolar bone
- Blunt dissection using mosquito hemostat
- Irrigate cavity with saline
- If abscess cavity sufficiently large, place 1/4 in iodoform gauze drain or fenestrated Penrose drain for 24–48 hr:
 - To prevent its aspiration, secure gauze or drain with silk suture
- Antibiotics:
 - Indicated if abscess extensive or if systemic signs present
 - Penicillin considered first-line empiric therapy
 - Erythromycin, azithromycin, clindamycin for penicillin-allergic patients
 - Clindamycin for penicillin-allergic patients or patients not responding to penicillin
 - Ampicillin/sulbactam for severe infections
- Warm salt water rinses hourly while awake for 24–48 hr

MEDICATION

First Line

- Penicillin VK: 250–500 mg PO q6h (peds: 25–50 mg/kg/d PO div. q6h)
- Azithromycin: 500 mg (peds: 10 mg/kg) PO 1st day, then 250 mg (peds: 5 mg/kg) PO per day × 4 days (for penicillin-allergic patients)
- Clindamycin: 150–450 mg PO q6h (peds: 10–25 mg/kg/d div. PO q6h)
- Clindamycin: 300–900 mg IV q8h (peds: 15–25 mg/kg/d IV div. q8h)
- Erythromycin: 250–500 mg PO q6–8h (peds: 30–50 mg/d PO div. q6h)

Second Line

- Ampicillin/sulbactam IV: 1.5–3 g IV q6h (peds > 1 yr, < 40 kg: 300 mg/kg/d IV div. q6h)
- Amoxicillin/clavulanate: 875 mg PO q12h (peds: 25–45 mg/kg/d div. q12h) (oral conversion)
- Moxifloxacin: 400 mg PO or IV QD (not routinely recommended for pediatric use)



FOLLOW-UP

DISPOSITION

Admission Criteria

- Severe infection or complication requiring parenteral antibiotics
- Necrosis or cellulitis involving areas with potential airway compromise
- Cavernous sinus thrombosis
- Osteomyelitis
- Outpatient therapy failure

- Immunocompromised patients:
 - Neutropenia
 - Uncontrolled diabetes
 - Advanced HIV
 - Cancer patients undergoing chemotherapy
- Ludwig angina
- Systemic involvement with significant dehydration
- Patients unable to handle secretions
- Patients unable to manage infection at home because of physical or mental disability or psychosocial factors

Discharge Criteria

- Uncomplicated cases
- Dental follow-up available in 24–48 hr

Issues for Referral

Dental follow-up useful for:

- Viability of affected tooth
- Dental extraction
- Root canal therapy
- Removal of Penrose drain or wic

FOLLOW-UP RECOMMENDATIONS

Dental follow-up in 24–48 hr:

- Lacking dental follow-up, patients should have alternative follow-up in 24–48 hr with provider familiar with disease process (oral surgeon, ED, urgent care, primary care)

PEARLS AND PITFALLS

Maxillary sinusitis may be incorrectly diagnosed without adequate oral exam:

- Dental follow-up is essential for short-term resolution of symptoms and long-term tooth viability and oral hygiene issues

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Toothache



CODES

ICD9

- 522.5 Periapical abscess without sinus
- 522.7 Periapical abscess with sinus
- 523.31 Aggressive periodontitis, localized

ICD10

- K04.6 Periapical abscess with sinus
- K04.7 Periapical abscess without sinus
- K05.21 Aggressive periodontitis, localized

PERIORBITAL AND ORBITAL CELLULITIS

Shari Schabowski

BASICS

DESCRIPTION

Periorbital Cellulitis

- An inflammatory, typically infectious condition affecting the eyelid(s)
- It is anatomically distinguished by its location, isolated to the tissues anterior to the orbital septum:
 - Orbital septum is the connective tissue extension of the orbital periosteum that is reflected into the upper and lower eyelids
 - Extension to the deep tissues is rare because the septum represents a nearly impenetrable barrier but it may be incomplete
- Most commonly presents as a complication of upper respiratory tract infection (URTI) and sinusitis:
 - Swelling is caused by inflammatory edema from vascular and lymphatic congestion
- May occur as a complication of a localized inflammation/infection in the eyelid or adjacent structures:
 - Blepharitis
 - Hordeolum
 - Dacryocystitis
 - Surrounding skin disruptions:
- Insect bites
- Minor trauma
- Impetigo or other dermatologic disorders

Orbital Cellulitis

- Inflammatory process in the structures deep to the orbital septum
- Typically occurs secondary to extension from an adjacent structure:
 - Sinusitis:
 - Most commonly ethmoiditis penetrating through the thin lamina papyracea
 - Dental abscess
 - Retained foreign body in the orbit
 - Puncture wounds
 - Orbital fracture
 - Postoperative infection
 - Hematogenous spread from a remote source due to valveless orbital veins

- Rare cause—direct extension of periorbital cellulitis

ETIOLOGY

Periorbital Cellulitis

- *Streptococcus pneumoniae*
- *Staphylococcus aureus*
- *Streptococcus pyogenes*
- *Moraxella catarrhalis*
- *Haemophilus influenzae*
- *Gonococcus* – rare
- Consider nonbacterial cause

Orbital Cellulitis

- Currently streptococcal and staphylococcal infections are the most common causes:
 - *S. pneumoniae*, *Streptococcus viridans*, *S. pyogenes*, *Streptococcus anginosus*, *S. aureus*
 - Anaerobes, Bacteroides, and gram-negatives may also be seen
- All forms of orbital cellulitis carry a risk of severe morbidity and possible mortality and are therefore a true emergency:
 - Permanent visual loss may occur
 - May extend to subperiosteal space with abscess formation
 - Cavernous sinus thrombosis and CNS infections may be life threatening
- Fungal infections are an uncommon but an even more lethal form particularly in the immunocompromised:
 - Cerebrorhino-orbital phycomycosis (CROP)
 - Rapidly fatal in 75% of cases:
 - 80% of cases occur in patients with a recent episode of diabetic ketoacidosis
 - Predisposing factor: Severe metabolic acidosis and immunocompromise
 - Begins in the paranasal sinuses and proliferates in the blood vessels causing thrombosis and necrosis
 - Bloody nasal discharge is common
 - May present with evidence of necrosis of the palate and/or nasal mucosa

Pediatric Considerations

- Routine vaccinations including Hib and Pneumococcus have dramatically decreased periorbital and orbital cellulitis, but infections may still occur with these organisms particularly in younger children and those without at least 2 Hib vaccines

- Periorbital cellulitis is overall 5 times more common and typically occurs in children <5 yr whereas orbital cellulitis is more common in children over 5 yr



DIAGNOSIS

SIGNS AND SYMPTOMS

Periorbital Cellulitis/Orbital Cellulitis

- Both present with a unilateral, red, swollen eye:
 - Lid swelling may be profound in both
- Differences include:
 - Source of inciting infection
 - Single vs. both lids involved
 - Toxicity, systemic and neurologic symptoms

Orbital Cellulitis

History

- Preceded by sinusitis in 60–90%, dental infection, trauma, puncture wound, or recent operation
- Swelling and redness surrounding eye in addition to eye pain, visual impairment, loss of color vision, restricted eye movements
- Headache, meningismus, and symptoms of systemic illness may occur
- Identify complicating medical problems:
 - Immunocompromise
 - Diabetes

Physical-Exam

- Toxic appearance:
 - Fever >39°C
- Restricted, painful extraocular movements (EOM)
- Afferent pupillary defect
- Conjunctival injection
- Chemosis
- Decreased visual acuity
- Diplopia
- Proptosis
- Meningismus and neurologic findings may be seen.

Periorbital Cellulitis

History

- Preceded by local skin injury, insect bite, URTI, or superficial ocular infection
- Ask about vaccination status in young children
- Low-grade fever
- Subacute presentation

Physical-Exam

- Red, swollen eyelid
- Often single lid involvement but can involve both
- Conjunctival injection common
- Low-grade fever common:
 - Rare systemic symptoms
- Normal visual acuity
 - No symptoms of deep ocular involvement

ESSENTIAL WORKUP

- Complete eye exam:
 - External exam
 - Visual acuity
 - EOM
 - Pupillary exam
 - Fundoscopic exam
 - Intraocular pressure measurement
- Complete neurologic exam

DIAGNOSIS TESTS & INTERPRETATION

Lab

Supportive but not diagnostic:

- CBC:
 - WBC < 15,000 for periorbital cellulitis
 - WBC > 15,000 may suggest bacteremic periorbital cellulitis or orbital cellulitis
- Blood culture
- Gram stain and culture of tissue aspirate or swab of draining purulent material:
 - Chocolate agar plate when gonorrhea suspected

Imaging

CT scan orbits with contrast:

- Indicated if:
 - CNS or systemic signs
 - Visual disturbances
 - Proptosis; restricted or painful EOM
 - Ophthalmoplegia

- Bilateral edema
- No improvement or deterioration at 24 hr
- Demonstrates extent of:
 - Orbital cellulitis
 - Sinusitis
 - Orbital emphysema
 - Subperiosteal abscess
 - Presence of foreign body
 - Cavernous sinus thrombosis

Diagnostic Procedures/Surgery

Lumbar puncture:

- Rule out CNS involvement in patients who appear toxic or manifest meningismus
- Surgery:
 - Evacuate abscess
 - Relieve sinusitis
 - Decompress optic nerve

DIFFERENTIAL DIAGNOSIS

- Allergic reaction
- Dacryoadenitis
- Dacryocystitis
- Graves disease
- Hordeolum
- Inflammatory orbital pseudotumor
- Insect bite
- Orbital rhabdosarcoma
- Periorbital ecchymosis
- Retrobulbar hemorrhage



TREATMENT

INITIAL STABILIZATION/THERAPY

IV fluids for vomiting, dehydration, toxic appearance, clinical need for parenteral antibiotics

ED TREATMENT/PROCEDURES

- Antipyretics
- Pain medication as needed
- Antibiotics

Periorbital Cellulitis

- Typically responds to oral antibiotics unless appears bacteremic or toxic:
 - Augmentin: 500 mg (peds: 45 mg/kg/24 h) PO TID
 - Cephalexin: 500 mg (peds: 100 mg/kg/24 h) PO QID
 - Clindamycin: 300 mg (peds: 20 mg/kg/24 h) PO QID
 - Dicloxacillin: 500 mg (peds: 100 mg/kg/24 h) PO QID
- Parenteral antibiotics:
 - Cefotaxime: 1–2 g (peds: 150 mg/kg/24 h) IV q6–8h
 - Clindamycin: 600 mg (peds: 40 mg/kg/24 h) IV q6h

Orbital Cellulitis

- Early administration of parenteral antibiotics
- Ophthalmologic consultation for any intraocular manifestations
- If sinusitis is the source, consider ENT consultation, and add decongestants to the treatment
- Emergent surgical intervention may be necessary:
 - If *Bacteroides* is suspected organism:
 - Surgical débridement
 - Vancomycin
 - Tetanus toxoid when appropriate
- If proptosis leaves the cornea exposed:
 - Lubricating drops (Lacri-Lube: 2 drops q2–4h PRN)
- If you suspect CROP:
 - Amphotericin B IV at highest tolerated dose
 - Topical amphotericin B (1 mg/mL) irrigation or nasal packing
 - Local debridement

MEDICATION

First Line

- Ceftriaxone: 1–2 g (peds: 100 mg/kg/24 h) IV q12–24h
- Erythromycin ophthalmologic ointment: Applied q4h to lower cul-de-sac

Second Line

Depending on suspected organism:

- Gentamicin: 5 mg/kg/24 h IV
- Metronidazole: 15 mg/kg IV load, then 7.5 mg/kg q6h
- Nafcillin: 1–2 g (peds: 100 mg/kg/24 h) IV q4h
- Vancomycin: 1 g (peds: 40 mg/kg/24 h) q12h



FOLLOW-UP

DISPOSITION

Periorbital Cellulitis

Discharge with oral antibiotics and prompt follow-up unless:

- Evidence of systemic toxicity, neurologic, visual or orbital findings
- Unable to tolerate PO antibiotics
- Progression of infection on oral antibiotics
- Unable to arrange follow up within 24–48 hr
- High-risk *H. influenzae* type B
- Complicating medical problems

Orbital Cellulitis

Admit for:

- IV antibiotics
- Observation for progression
- Specialist consultation
- Surgical incision and drainage

PEARLS AND PITFALLS

- Anytime a patient presents with a red swollen eye, consider the possibility of orbital cellulitis
- Take a careful history for:
 - _ Recent sinusitis
 - _ Recent puncture, history of trauma or surgical procedure
 - _ Recent dental infection—particularly a canine space abscess
 - _ History of immunocompromise or recent or current episode of DKA
 - _ Determine vaccination status in children
- Pay careful attention to exclude:
 - _ Systemic toxicity
 - _ Eye pain or visual impairment
 - _ Restriction of eye movements
 - _ Signs and symptoms of neurologic involvement

ADDITIONAL READING

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- Wald E. Periorbital and orbital infections. *Infect Dis Clin North Am.* 2007;21(2):392–

408.



See Also (Topic, Algorithm, Electronic Media Element)

- [Dacryoadenitis](#)
- [Dacryocystitis](#)
- [Hyperthyroidism](#)
- [Hordeolum and Chalazion](#)
- [Pseudotumor Cerebri](#)



CODES

ICD9

- [373.13 Abscess of eyelid](#)
- [682.0 Cellulitis and abscess of face](#)

ICD10

- [H05.012 Cellulitis of left orbit](#)
- [H05.019 Cellulitis of unspecified orbit](#)
- [H00.039 Abscess of eyelid unspecified eye, unspecified eyelid](#)

PERIPHERAL NEUROPATHY

Minh V. Le

BASICS

DESCRIPTION

Peripheral neuropathy is a general term for peripheral nerve disorders that may affect motor, sensory, or vasomotor nerve fibers and presents with marked muscle weakness, atrophy pain and numbness

ETIOLOGY

Variable, depending on presentation of symptoms; refer to Differential Diagnosis

DIAGNOSIS

SIGNS AND SYMPTOMS

- Sensory nerve dysfunction:
 - Numbness
 - Localized tingling
 - Paresthesias
 - Dysesthesias
 - Vibration and position sensations are decreased with large-fiber neuropathy
 - Pain and temperature sensation are decreased with small-fiber neuropathy
 - Deep tendon reflexes are decreased secondary to decreased sensation of afferent limb
- Motor nerve dysfunction:
 - Weakness:
 - Distal > proximal
 - Occasionally fasciculations
 - Muscle atrophy, diminished tone with long-standing motor nerve involvement
 - Loss of reflexes secondary to slowing of conduction along motor nerve efferent limb
- Autonomic nerve dysfunction:
 - Orthostasis
 - Constipation
 - Urinary retention
 - Impotence

History

- Duration of symptoms

- Symmetric or asymmetric symptoms
- Distal or proximal symptoms
- Motor, sensory, or mixed

Physical-Exam

- Thorough head-to-toe physical exam
- Focus on neurologic exam:
 - Motor weakness
 - Sensory loss typically in stocking-glove distribution

ALERT

Absence of reflexes early in course could represent demyelinating neuropathy such as Guillain–Barré syndrome (acute inflammatory demyelinating syndrome [AIDP]).

ESSENTIAL WORKUP

- Studies based on acuteness, severity of neuropathy, and most likely diagnosis
- Neurologic consult early if acute and severe symptoms

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Basic metabolic panel
- CBC
- Liver function tests
- Urinalysis
- Thyrotropin-stimulating hormone
- HIV or vitamin B₁₂ based on individual presentations
- Electrocardiogram

Imaging

- CXR if indicated
- Head CT if indicated

Diagnostic Procedures/Surgery

- Electromyographic studies, nerve conduction studies, and nerve biopsy per neurologic consult on admission or outpatient follow-up
- Lumbar puncture as appropriate for AIDP

DIFFERENTIAL DIAGNOSIS

- Focal:
 - Entrapment
 - Common sites of compression:
 - Carpal, ulnar tunnel

- Tarsal tunnel
 - Peroneal
- Myxedema
- Rheumatoid arthritis
- Amyloidosis
- Acromegaly
- Trauma
- Ischemic lesions
- Diabetes mellitus (DM)
- Vasculitis
- Leprosy
- Sarcoidosis
- Neoplastic infiltration or compression
- Multifocal (mononeuropathy multiplex):
 - DM
 - Vasculitis:
 - Polyarteritis nodosa
 - Systemic lupus erythematosus
 - Sjögren syndrome
 - Sarcoidosis
 - Leprosy
 - Malignancy related
 - HIV/AIDS
 - Hereditary predisposition to pressure palsies
- Symmetric:
 - Endocrine:
 - Most common is DM
 - Hypothyroidism
 - Medications:
 - Isoniazid
 - Lithium
 - Metronidazole
 - Phenytoin
 - Cimetidine
 - Hydralazine
 - Amitriptyline
 - Amiodarone
 - Nutritional diseases:
 - Alcoholism
 - B₁₂/folate deficiency
 - Thiamine
 - Critical illness neuropathy

- Hypophosphatemia
- Guillain-Barré syndrome (AIDP)
- Toxic neuropathy:
 - Carbon monoxide
 - Acrylamide
 - Carbon disulfide
 - Ethylene oxide
 - Organophosphate esters
 - Lead
- Myelopathy mimicking peripheral neuropathy
- Back pain
- Saddle anesthesia
- Lower extremity weakness



TREATMENT

PRE HOSPITAL

- Pain control as needed
- Airway protection as indicated

INITIAL STABILIZATION/THERAPY

Establish airway protection with severe acute peripheral neuropathy, such as Guillain-Barré syndrome

ED TREATMENT/PROCEDURES

- Variable depending on acuity of symptoms
- Discontinuation offending toxin or agent
- Treatment underlying systemic disease

MEDICATION

- Variable depending on underlying diagnosis
- Opioid analgesics
- Gabapentin 300 mg PO daily then BID on day 2, then TID on day 3 up to 1,800 mg/d div. TID
- Carbamazepine 100 mg PO BID for trigeminal neuralgia
- IV immunoglobulin for Guillain-Barré syndrome (AIDP)



FOLLOW-UP

DISPOSITION

Admission Criteria

- Respiratory distress or acute gait disturbance
- Intractable pain

Discharge Criteria

Stable respiratory and gait status with outpatient follow-up

Issues for Referral

Neurology—based on duration, severity of presentation

FOLLOW-UP RECOMMENDATIONS

Primary care or neurology depending on etiology and severity of symptoms

PEARLS AND PITFALLS

Failure to diagnose Guillain–Barré syndrome (AIDP)

ADDITIONAL READING

- Azhary H, Farooq MU, Bhanushali M, et al. Peripheral neuropathy: Differential diagnosis and management. *Am Fam Physician*. 2010;81:887–892.
- Gilron I, Watson CP, Cahill CM, et al. Neuropathic pain: A practical guide for the clinician. *CMAJ*. 2006;175:265–275.
- Marx JA, Hockberger RS, Walls RM, et al. *Rosen's Emergency Medicine: Concepts and Clinical Practice*. 7th ed. St. Louis, MO: Mosby; 2009.
- Pascuzzi RM. Peripheral neuropathy. *Med Clin North Am*. 2009;93:317–342.



CODES

ICD9

- 356.9 Unspecified hereditary and idiopathic peripheral neuropathy
- 782.0 Disturbance of skin sensation

ICD10

- G62.9 Polyneuropathy, unspecified
- R20.0 Anesthesia of skin
- R20.2 Paresthesia of skin

PERIPHERAL VASCULAR DISEASE

Sally A. Santen • Samantha R. Hauff

BASICS

DESCRIPTION

- Obstruction of ≥ 1 of the peripheral arteries secondary to embolism or thrombus
- Caused by atherosclerosis or embolus
- Patients with PAD may also have coronary artery and cerebrovascular disease.
- Epidemiology:
 - Risks factors (selected):
 - Age
 - Smoking
 - Diabetes
 - Hyperlipidemia
 - HTN
 - Associated with morbidity and mortality from other forms of atherosclerosis (coronary artery disease, stroke)
 - Complications:
 - Aneurysm
 - Thrombosis
 - Ulceration
 - Limb loss
- Chronic arterial insufficiency (CAI):
 - Progressive obstructing atherosclerotic disease causing subacute ischemia and pain (claudication)
 - 10% develop critical leg ischemia.
- Acute arterial insufficiency (AAI):
 - Caused by arterial thrombosis (50%) or embolism
 - Causes acute limb ischemia with signs and symptoms of the 6 Ps (below)
- Atheroembolism:
 - Caused by rupture or partial disruption of an atherosclerotic plaque (aorta, femoral, iliac)
 - Gives rise to cholesterol emboli that shower and obstruct arteriolar networks
 - May be precipitated by invasive arterial procedures such as cardiac catheterization

ETIOLOGY

- Obstruction by atherosclerotic plaques (CAI)
- Arterial thrombosis
- Arterial emboli:

- Cardiac emboli from dysrhythmias, valvular heart disease, or cardiomyopathy (80%)
- Aneurysms
- Infection
- Tumor
- Vasculitis or foreign body
- Thrombosis of plaques from pre-existing CAI
- Atheroembolism

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- CAI:
 - Claudication:
 - Aching pain in the calves (femoropopliteal occlusion) or buttocks and thighs (aortoiliac region)
 - Occurs with activity and slowly relieved by rest or dependent positioning
 - Classic claudication presents in about 1/2 of patients with PVD.
 - Severe disease presents with limb pain at rest:
 - Usually starting in the foot
 - Rapidly progressive claudication or ulceration
- AAI:
 - Extremity pain:
 - Sudden onset
 - Gradual increase in severity
 - Starts distally and moves proximally over time
 - Decrease in intensity once ischemic sensory loss occurs
- Atheroembolism:
 - Complaint of cold and painful fingers or toes
 - Small atherosclerotic emboli may affect both extremities.
 - Usually related to recent arteriography, vascular or cardiac surgery
 - Multiorgan involvement is common (renal, mesentery, skin, others)

Physical-Exam

ALERT

Sudden onset of pain and pallor in extremity is limb and life threatening.

- CAI:
 - Absent or decreased peripheral pulses

- Delayed capillary refill with cool skin
- Increased venous filling time
- Bruits
- Pallor and dependent rubor of the leg
- Muscle and skin atrophy
- Thickened nails and loss of dorsal hair
- Ulcerations (especially toes or heels) or gangrene with severe disease
- AAI:
 - 6 Ps:
 - Pain (1st, sometimes only symptom)
 - Pallor
 - Pulselessness
 - Poikilothermic
 - Paresthesias (late finding)
 - Paralysis (late finding)
 - Identification of a source of a possible embolic process is crucial (atrial fibrillation, cardiomegaly).
- Atheroembolism:
 - Ischemic and painful digits
 - “Blue toe syndrome”
 - Livedo reticularis

ESSENTIAL WORKUP

- CAI:
 - Ankle–brachial index (ankle systolic BP divided by higher arm systolic BP)
 - Bedside test to determine whether CAI is present (see NEJM video reference)
 - Ratio of <0.9 is abnormal and $<0.4-5$ indicates severe disease.
 - Calcific arteries (diabetes) can have false negative ABI or elevated ABI (>1.3).
- AAI:
 - Physical diagnosis using the 6 Ps
 - Those with acute-on-chronic arterial insufficiency tolerate limb ischemia better than those without CAI, due to well-developed collateral circulation.
- Atheroembolism:
 - Clinical diagnosis: Affected areas painful, tender, and may be either dusky or necrotic
 - Workup may investigate source of emboli with duplex US, CT angiogram, EKG.

DIAGNOSIS TESTS & NTERPRETATION

Lab

- CBC and platelets

- Electrolytes, BUN, creatinine, glucose
- Coagulation studies
- Creatine phosphokinase to evaluate for ischemia.
- Special tests for suspected etiologies:
 - Hold blood for hypercoagulable studies
 - Sedimentation rate, CRP for vasculitis
 - Blood cultures for endocarditis

Imaging

- Doppler US:
 - Visualizes both venous and arterial systems
 - Identifies level of arterial occlusion, as well as thrombosis and aneurysm
 - Sensitivity and specificity > 80–90% for occlusion of vessels proximal to the popliteal vessels
- Plethysmography/segmental pressure measurements:
 - Uses measurements of the volume and character of blood flow to detect areas of CAI
 - Less widely available than US, therefore requires an experienced technician
 - Approximates US in sensitivity and specificity
- Angiography:
 - Determines details about the anatomy, including the level of occlusion, stenosis, and collateral flow
 - Useful where the diagnosis of AAI is uncertain or before emergent bypass grafting
 - Advantage is intervention (atherectomy, angioplasty, or intraluminal thrombolytics) can be done at the time of diagnosis.
- CT angiogram:
 - CT is useful for diagnosis of occlusive aortic disease or dissection.
 - Rapidly available and reliable
 - Many centers have moved to CT angiogram as the 1st-line diagnostic tool. The decision for operative or angiographic intervention is based on the CT angiogram.
 - Requires contrast, therefore may not be 1st line for patients with renal insufficiency
- MRI:
 - Sensitive for evaluation of CAI and dissection
 - Disadvantages are that MRI is time consuming and expensive.

DIFFERENTIAL DIAGNOSIS

- Acute thrombosis or emboli
- Arterial dissection
- Deep venous thrombosis
- Venous insufficiency

- Compartment syndrome
- Buerger disease
- Spinal stenosis
- Neuropathy
- Bursitis
- Arthritis
- Reflex sympathetic dystrophy



TREATMENT

PRE HOSPITAL

- Maintain hemodynamic stability with fluids.
- Apply cardiac monitor.
- Place the ischemic limb at rest and in a dependent position.
- Provide oxygen if low oxygen saturation or pulmonary symptoms.

INITIAL STABILIZATION/THERAPY

- IV fluid bolus for hypotension
- EKG, monitor, pulse oximetry
- Supplemental oxygen
- Pain control
- Avoid temperature extremes

ED TREATMENT/PROCEDURES

- CAI:
 - Antiplatelet therapy with 75 or 325 mg of aspirin or clopidogrel (75 mg/day) may be used as 1st-line treatment. Dual therapy has not been shown to improve outcomes, although may be indicated in other forms of atherosclerosis.
 - Other approved drugs include: Cilostazol 100 mg BID, dipyridamole 200 mg BID, pentoxifylline 400 mg TID
 - Revascularization depending on the severity and location of obstruction:
 - Balloon angioplasty
 - Atherectomy
 - Bypass grafting
 - Risk-factor modification:
 - Tobacco cessation
 - Aggressive management of hyperlipidemia, HTN, diabetes
 - Exercise therapy
- AAI:
 - Limit further clot propagation with IV heparin.
 - Do not anticoagulate patients suspected of having an aortic dissection or

symptomatic aneurysm.

- Emergent consultation with vascular surgery or interventional radiology:
 - To determine which diagnostic study is best to make the diagnosis
 - To begin arrangements for possible operative therapy or other intervention
 - Options for operative therapy include thrombectomy, embolectomy, angioplasty, regional arterial thrombolysis, bypass grafting.
 - Blood flow to the affected limb must be re-established within 4–6 hr after onset of ischemic symptoms.
- Complications of AAI include:
 - Compartment syndrome
 - Irreversible ischemia requiring amputation
 - Rhabdomyolysis, renal failure
 - Electrolyte disturbances
- Atheroembolism:
 - Treat conservatively if a limited amount of tissue is involved and renal function is not significantly compromised.
 - No clear therapy for the ischemic digits besides supportive wound care and analgesia
 - Some studies have tried corticosteroids to decrease inflammation, statins to stabilize plaque, aspirin, or dipyridamole
 - Amputation for irreversibly necrotic toes
 - Vascular surgeon referral within 12–24 hr of ED visit
 - Prevent further embolic events by a thorough investigation and correction of the source of atheroemboli.

MEDICATION

- Aspirin: 81–325 mg/d
- Cilostazol: 100 mg BID
- Clopidogrel: 75 mg/d
- Heparin: 80 U/kg bolus IV followed by 18 U/h IV
- Pentoxifylline: 400 mg TID



FOLLOW-UP

DISPOSITION

Admission Criteria

- All patients with AAI are admitted for evaluation and revascularization.
- CAI: Consider admission for rapidly progressive claudication or ischemic pain at rest:
 - To undergo heparinization and angiography to rule out an acute thrombosis

- Atheroembolism admission indicated with large areas involved, significant pain, infection, or renal compromise

Discharge Criteria

- Atheroembolism:
 - If they have small lesions, adequate pain control, no evidence of renal compromise or superinfection, and follow-up within 24 hr
- CAI:
 - No evidence of rapid progression, critical leg ischemia, gangrene, or infection

Issues for Referral

- CAI will need urgent referral to vascular surgery.
- Atheroembolism, depending on the origin of the emboli, may need referral to vascular surgery or to cardiology.

FOLLOW-UP RECOMMENDATIONS

CAI without acute ischemia and atheroembolism with minimal involvement should have close follow-up to evaluate the extent of their disease.

ADDITIONAL READING

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- White C. Clinical practice. Intermittent claudication. *N Engl J Med*. 2007;356:1241–1250.



See Also (Topic, Algorithm, Electronic Media Element)

- Arterial Occlusion

- Venous Insufficiency



CODES

ICD9

- 440.20 Atherosclerosis of native arteries of the extremities, unspecified
- 443.9 Peripheral vascular disease, unspecified
- 444.22 Arterial embolism and thrombosis of lower extremity

ICD10

- I70.209 Unsp athscl native arteries of extremities, unsp extremity
- I73.9 Peripheral vascular disease, unspecified
- I74.4 Embolism and thrombosis of arteries of extremities, unspecified

PERIRECTAL ABSCESS

James A. Nelson • Scott A. Miller

BASICS

DESCRIPTION

Localized infection and accumulation of purulent material adjacent to anus or rectum

ETIOLOGY

- Anal crypt gland infection, with spread to adjacent areas separated by muscle and fascia:
 - Perianal:
 - Most common
 - Usually with red bulge near anus
 - Ischiorectal:
 - Large potential space
 - May become very large before diagnosed
 - Can communicate posteriorly with other side forming “horseshoe” abscess
 - Intersphincteric:
 - Contained at primary site of origin between internal and external sphincters
 - Supralelevator:
 - Very deep above levator ani
 - Needs operative débridement under general anesthesia
 - Often systemic symptoms before diagnosis is made
- Bacterial cause is typically a mix of stool pathogens:
- Associated diseases:
 - Diabetes
 - Inflammatory bowel disease
 - Malignancy
 - Immunocompromised host

DIAGNOSIS

SIGNS AND SYMPTOMS

- Pain: Perianal, rectal, or pelvic
- Swelling, fluctuance, drainage, fever

History

- Perianal pain:

- Aggravated by defecation, sitting, coughing
- Dull deep pelvic or rectal pain:
 - Less pain if arises above dentate line (ischioanal and supralevator)
- Rectal or perirectal drainage
- Fever/chills
- Constipation

Physical-Exam

- Perianal swelling, erythema, induration, fluctuance, tenderness
- Inner cleft buttock abscess = red flag
 - Rectal abscess can track out to buttock
- Rectal exam is the most important diagnostic intervention
 - Rectal swelling or tenderness
 - Fistula can be probed, or palpated as a cord

ESSENTIAL WORKUP

- Careful history and physical exam with rectal exam are paramount in making diagnosis.
- Have high index of suspicion for any constant perirectal pain.

DIAGNOSIS TESTS & INTERPRETATION

No labs or imaging routinely indicated

Lab

- CBC: Leukocytosis with left shift
- Wound culture: Not typically indicated
- Blood cultures: Mainly for sepsis

Imaging

- CT (with IV contrast, +/- PO contrast)
- MRI (helpful with detecting fistulas)
- Endoanal US sometimes used

Diagnostic Procedures/Surgery

Incision and drainage (I&D) is the definitive management.

DIFFERENTIAL DIAGNOSIS

- Anal fissure
- Sentinel pile in the posterior midline or anterior midline
- Thrombosed or inflamed hemorrhoids
- Anal ulcer (i.e., HIV)
- Proctitis (i.e., gonococcal)
- Anorectal carcinoma



TREATMENT

INITIAL STABILIZATION/THERAPY

Pain medication

ED TREATMENT/PROCEDURES

- Delayed drainage may worsen outcome
- Bedside drainage:
 - Only if localized perianal abscess
 - Probe to rule out deeper tract
 - Radial incision close to anal verge
 - Explore cavity, breaking any loculations.
 - Irrigate liberally.
 - Loose packing removed at 48 hr.
- Operative debridement under general anesthesia:
 - If local anesthesia is inadequate, or deeper abscess
- Antibiotics rarely necessary:
 - Extensive cellulitis
 - Immunosuppression
 - Valvular heart disease
 - Systemic infection
 - Prosthetic device
 - PO:
 - Amoxicillin clavulanate or fluoroquinolone
 - Consider MRSA coverage
 - IV:
 - Cefoxitin
 - Ampicillin sulbactam
 - Combination therapy with ampicillin, gentamicin, and clindamycin or metronidazole
- Postoperative care:
 - Sitz baths TID 24 hr after I&D
 - High-fiber diet or bulking agent
 - Analgesic

MEDICATION

- Amoxicillin clavulanate: 875 mg PO q12h or 500 mg PO q8h
- Ampicillin sulbactam: 1.5–3 g IV q6h
- Cefoxitin: 1–2 g IV q6–8h
- Clindamycin: 600–900 mg IV div. q8h
- Gentamicin: 3–6 mg/kg/d IV div. q8h
- Metronidazole: 7.5 mg/kg IV q6h



FOLLOW-UP

DISPOSITION

Admission Criteria

- Need for operative drainage
- Systemic toxicity/signs of sepsis

Discharge Criteria

Adequate I&D with complete drainage

Issues for Referral

All should be referred to surgeon in 24--48 hr

FOLLOW-UP RECOMMENDATIONS

Surgeon referral within 24–48 hr to evaluate for fistula:

- Fistulas develop in 25–50% of anorectal abscesses.

PEARLS AND PITFALLS

- Be certain of extent of abscess:
 - Thorough rectal exam and probing is mandatory.
 - Imaging adds insight into deeper areas not accessible to exam
- Deeper abscesses above dentate line have less pain and can present with isolated fever

ADDITIONAL READING

- Marcus RH, Stine RJ, Cohen, MA. Perirectal abscess. *Ann Emerg Med.* 1995;25(5):597–603.
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- Steele SR, Kumar R, Feingold DL, et al. Practice parameters for the treatment of perianal abscess and fistula-in-ano. *Dis Colon Rectum.* 2011;54:1465–1474.



See Also (Topic, Algorithm, Electronic Media Element)

- Abscess
- Anal Fissure
- Hemorrhoid



CODES

ICD9

- 565.1 Anal fistula
- 566 Abscess of anal and rectal regions

ICD10

- K61.0 Anal abscess
- K61.1 Rectal abscess
- K61.3 Ischiorectal abscess

PERITONSILLAR ABSCESS

Erik Adler • Maria E. Moreira

BASICS

DESCRIPTION

- Suppurative complication of tonsillitis where infection spreads outside the tonsillar capsule between the palatine tonsil and pharyngeal muscles
- Most common deep infection of the head and neck (incidence of 30/100,000 per year)
- In the US, 45,000 cases annually
- Occurs in all ages, more commonly in young adults (mean age 20–40 yr)
- Occurs most commonly Nov–Dec, April–May (coincides with highest incidence rates of streptococcal pharyngitis)
- Complications:
 - Airway compromise (uncommon)
 - Sepsis (uncommon)
 - Recurrence (12–15%)
 - Extension to lateral neck or mediastinum
 - Spontaneous perforation and aspiration pneumonitis
 - Jugular vein thrombosis (Lemierre syndrome)
 - Poststreptococcal sequelae (glomerulonephritis, rheumatic fever)
 - Hemorrhage from extension and erosion into carotid sheath
 - Severe dehydration
 - Intracranial extension (meningitis, cavernous sinus thrombosis, cerebral abscess)
 - Dural sinus thrombosis

ETIOLOGY

- 2 theories explain the development of peritonsillar abscess (PTA):
 - Direct bacterial invasion into deeper tissues in the patient with acute pharyngitis
 - Acute obstruction and bacterial infection of small salivary glands (Weber glands) in the superior tonsil
- Smoking may be a risk factor
- Most common pathogens:
 - *Group-A Streptococcus*
 - Staphylococcal species, including methicillin-resistant *Staphylococcus aureus* (MRSA)
 - Anaerobes (*Prevotella*, *Peptostreptococcus*, *Fusobacterium*)
 - Polymicrobial

SIGNS AND SYMPTOMS

History

- Sore throat
- Fever
- Voice change
- Odynophagia (difficulty swallowing)
- Drooling
- Headache
- Pain radiating to the ear
- Decreased PO intake
- Malaise

Physical-Exam

- Fever
- Trismus
- “Hot potato” voice
- Erythematous tonsils/soft palate
- Inferior and medial displacement of superior pole of tonsil on affected side
- Uvular deviation away from affected side
- Halitosis
- Cervical lymphadenitis
- Tenderness on ipsilateral side of neck at the angle of the jaw

ESSENTIAL WORKUP

- Evaluation for deep space infections beyond the PTA, either with additional imaging or physical exam that may require admission and surgery
- Evaluate and ensure airway patency: Look for stridor, tripod position, or inability to handle secretions
- Definitive management with either needle aspiration or incision and drainage (I&D), followed by a course of antibiotics

DIAGNOSIS TESTS & NTERPRETATION

- Usually a clinical diagnosis made by visually examining oropharynx
- May be difficult with severe trismus

Lab

- Throat culture and monospot (20% incidence of mononucleosis with PTA)
- CBC and culture of the abscess contents may be useful in some cases
- Basic metabolic panel may be useful in patients with decreased oral intake and

clinical signs of dehydration

Imaging

- Bedside intraoral US:
 - Using the high-frequency intracavitary US transducer with a lubricated latex cover can aid in identification and localization of the abscess
 - A cooperative patient can place the transducer at the point of maximum tenderness
- Transcutaneous cervical ultrasound is an option when the patient has too much trismus to use an intracavitary probe
- Soft-tissue lateral neck:
 - If suspicion for epiglottitis or retropharyngeal abscess exists
- Chest radiograph:
 - With severe respiratory symptoms or draining abscess
- CT scan of neck:
 - If suspicion exists for other deep space infection of the neck, CT may be indicated
 - CT also may be indicated if unable to obtain a good exam secondary to trismus
 - CT may locate abscess pocket after failed needle aspiration
- MRI may be useful to evaluate for complications of deep space infections (internal jugular vein thrombosis or erosion into the carotid sheath)

Diagnostic Procedures/Surgery

- Needle aspiration is diagnostic and often curative
- Bedside I&D

DIFFERENTIAL DIAGNOSIS

- Peritonsillar cellulitis
- Epiglottitis
- Retropharyngeal abscess
- Peripharyngeal abscess
- Tracheitis
- Meningitis
- Retropharyngeal hemorrhage
- Cervical osteomyelitis
- Cervical adenitis
- Epidural abscess
- Infectious mononucleosis
- Internal carotid artery aneurysm
- Lymphoma
- Foreign body
- Other deep space infections of the neck



TREATMENT

PRE HOSPITAL

Rarely associated with airway emergencies, but diagnosis is likely to be uncertain in transport, so suction and intubation equipment should be at the bedside:

- Pulse oximetry, supplemental oxygen
- Cardiac monitor
- IV access

Pediatric Considerations

- PTA occurs in children (< 18 yr) in 25–30% of reported cases (14 cases per 100,000 population)
- Young children may need sedation or general anesthesia if I&D or aspiration of the abscess is attempted
- Obtain soft-tissue lateral neck radiograph before oral exam in young children with symptoms of upper airway obstruction

INITIAL STABILIZATION/THERAPY

- Same as for pre-hospital
- Airway management may be necessary
- Equipment for intubation and cricothyroidotomy should be available

ED TREATMENT/PROCEDURES

- Antibiotics should be administered
- IV fluid should be given for dehydration
- Pain control is important
- A single dose of steroids may improve symptoms
- Adequate anesthesia prior to aspiration or I&D procedures is important:
 - Benzocaine spray
 - Lidocaine, 1% with 1:100,000 epinephrine
- No clear benefit for one drainage technique over another:
 - Needle drainage:
 - Successful 87–94%
 - Should be performed by a person experienced in drainage procedure and adept at advanced airway techniques
 - Less painful, less invasive than I&D
 - The internal carotid artery lies ~2.5 cm posterolaterally to the tonsil; sheathing the aspiration needle to prevent introduction of the needle to <0.5 cm is prudent
 - The superior pole of the tonsil is the most common place for maximal fluctuance (followed by the middle pole and then the inferior pole)
 - Repeat aspiration is necessary in 10%

- I&D:
 - Successful 90–92%
 - An 11- or 15-blade scalpel is used to make stab incision to area of fluctuance
 - Guard scalpel with trimmed plastic sheath leaving 1 cm of blade exposed
 - Avoid >0.5 cm depth
 - Medial and superior incisions are safer from the standpoint of potential injury to the carotid artery
 - Incision typically made superior to tonsil in area of soft palate. Incision in the tonsil itself causes excessive bleeding and may miss the abscess, which is located in the peritonsillar soft tissue of the soft palate.
 - Suction should be ready to remove purulent drainage and blood
 - Packing is *not* used
- Tonsillectomy (indications in children):
 - Upper airway obstruction
 - Previous episodes of severe recurrent pharyngitis or PTA
 - Failure of abscess resolution with other drainage techniques
 - Can be performed immediately or after resolution of acute infection

MEDICATION

- Length of antibiotic treatment should be 14 days (<10 day treatment course may be associated with recurrence)
- Adjunct with steroids can improve symptoms

Intravenous Antibiotics

- Ampicillin/Sulbactam (Unasyn), 3 gm q6h
- Penicillin G, 10 million U q6h + Metronidazole (Flagyl), 500 mg q6h
- If allergic to Penicillin, Clindamycin, 900 mg q8h

Oral Antibiotics

- Amoxicillin/Clavulanic acid (Augmentin), 875 mg BID
- Penicillin VK, 500 mg q6h + Metronidazole (Flagyl), 500 mg q6h
- Clindamycin, 600 mg BID or 300 mg q6h

Steroids

- Dexamethasone, 10 mg IV/IM/PO single dose
 - Pediatrics: 0.6 mg/kg; not to exceed 10 mg
- Methylprednisolone, 2 mg/kg; not to exceed 250 mg



FOLLOW-UP

DISPOSITION

Admission Criteria

- Airway compromise
- Sepsis
- Altered mental status
- Dehydration and inadequate PO intake
- Extension of infection beyond the PTA (i.e., deep space neck infections)

Discharge Criteria

- Most patients with PTA can be discharged home on oral antibiotics after abscess drainage
- Must be able to tolerate sufficient oral intake and antibiotics

Issues for Referral

- Referral to an otolaryngologist or surgeon should be provided
- Tonsillectomy is recommended 6–8 wk following treatment of the abscess

FOLLOW-UP RECOMMENDATIONS

Close follow-up recommended in 24–48 hr:

- Treatment failures and recurrences are relatively common

PEARLS AND PITFALLS

- Failure to secure the airway early in a severe infection
- Failure to recognize a more advanced, deep space infection of the neck
- Knowing the anatomy before performing needle aspiration or bedside I&D
- Bedside US is a useful adjunct in differentiating and identifying a PTA vs. peritonsillar cellulitis

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Media Element)

- Epiglottitis
- Retropharyngeal Abscess



CODES

ICD9

475 Peritonsillar abscess

ICD10

J36 Peritonsillar abscess

PERTUSSIS

Adam Z. Barkin

BASICS

DESCRIPTION

- Acute respiratory tract infection spread by small respiratory droplets
- Bacteria (fimbriae) attach to respiratory epithelial cells and proliferate, producing toxins:
 - Ciliary dysfunction, accumulation of cellular debris, increased mucus production, lymphocytic and granulocytic infiltration
- Bronchiolar congestion, obstruction, and necrosis
- Obstruction of the airway due to mucus plug, leading to hypoxia and hypoventilation
- Increased intrathoracic or intracranial pressure
- Secondary bacterial infection may exacerbate respiratory distress/failure.
- CNS injury caused by encephalitis, increased intracranial pressure, and/or hypoxia
- Uncomplicated cases last 6–10 wk; half of the cases last <6 wk.
- Mortality:
 - Mortality greatest in those <1 yr
 - 1.3% for patients <1 mo
 - 0.3% in children 2–11 mo
 - 90% of deaths are secondary to bacterial pneumonia
- Epidemiology:
 - Incubation period is 6–20 days, usually 7–10 days.
 - Mostly young children; 24% in children <6 mo
 - Increasing incidence in adolescents
 - Adults are the primary reservoir
 - Peak incidence is late summer/fall
 - Preventable with diphtheria–tetanus–pertussis (Tdap) vaccine

ETIOLOGY

Bordetella pertussis:

- A fastidious, gram-negative, pleomorphic bacillus

DIAGNOSIS

SIGNS AND SYMPTOMS

- Generally 3 recognized phases with progression:
 - Infants may have indistinct stages
- Catarrhal stage:

- 1–2 wk duration
- Rhinorrhea
- Mild cough
- Minimal fever
- Paroxysmal stage:
 - 1–6 wk duration
 - Classic “whooping” cough, increasing in severity:
 - Coughing spasm that ends with a sudden inflow of air—the whoop; unremitting paroxysms
 - Cyanosis with respiratory distress/failure
 - Apnea (infants < 6 mo)
 - Altered mental status secondary to hypoxia or encephalitis
- Convalescent stage:
 - 2–12 wk duration
 - Waning cough
 - Improving respiratory status
- Atypical presentations:
 - Often atypical in children < 6 mo
 - Partially immunized children have less severe disease
 - Adult manifestations are often only rhinorrhea, sore throat, persistent cough; often in family members

History

- Catarrhal phase:
 - Malaise
 - Low-grade fever
 - Rhinorrhea
 - Sore throat
- Paroxysmal phase:
 - “Whooping” cough
 - Post-tussive cyanosis
 - Post-tussive emesis
- “Whooping” sound during paroxysmal phase
- Catarrhal phase:
 - Persistent cough

Physical-Exam

- Catarrhal phase:
 - Low-grade fever
 - Rhinorrhea
 - Lacrimation
 - Dry cough (late phase)

- Conjunctival inflammation
- Paroxysmal phase:
 - Paroxysmal whooping cough
- Convalescent phase:
 - Occasional paroxysmal cough

ESSENTIAL WORKUP

- The ED diagnosis should be made on clinical grounds
- Attempt to establish a history of a contact
- Observe the paroxysmal cough with the characteristic whoop
- Use ancillary studies to further support the clinical diagnosis and exclude complications

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Polymerase chain reaction:
 - High sensitivity and specificity
 - High sensitivity leads to more false positives
 - Best practices for testing with PCR:
 - Test only those with symptoms
 - Testing after 4 weeks of cough or following antibiotics will increase false negative rate
 - Obtain samples via aspiration or posterior nasopharyngeal swab to maximize DNA recovery
 - Should be used in conjunction with culture
- Direct immunofluorescence assay of nasopharyngeal mucus:
 - High false-positive rate
- Culture of nasopharynx or cough plate on a Bordet–Gengou medium:
 - Takes 7–12 days
 - High specificity
 - Low sensitivity
 - Remains the gold standard test
- Serology:
 - Useful in later diagnosis
 - Perform testing 2–8 weeks after cough onset
- WBC count:
 - Leukocytosis (20,000–50,000 cells/mm³) with marked lymphocytosis
 - Normalizes during convalescent phase
 - Elevation of WBC and lymphocytosis parallels severity of cough
- Immunofluorescent and enzyme immunoassays to exclude respiratory syncytial virus
- Done on either nasal wash or nasopharyngeal swab (Dacron)

Imaging

CXR:

- Most often normal
- Perihilar infiltrates
- Atelectasis
- Occasionally characteristic “shaggy” right heart border
- Secondary bacterial pneumonia

DIFFERENTIAL DIAGNOSIS

- Infection:
 - Parallel whooping cough syndrome caused by *Bordetella parapertussis*, *Chlamydia trachomatis*, *Chlamydia pneumoniae*, *Bordetella bronchiseptica*, or adenovirus
 - Pneumonia:
 - Bacteria
 - *Mycoplasma*
 - *Mycobacterium*
 - Bronchiolitis:
 - Respiratory syncytial virus
 - Influenza
 - Other virus
- Reactive airway disease
- Foreign body
- Cystic fibrosis



TREATMENT

PRE HOSPITAL

- Oxygen
- Monitor airway
- Suction

INITIAL STABILIZATION/THERAPY

- Oxygen and respiratory support
- Suction mucous plugs

ED TREATMENT/PROCEDURES

- Universal precautions:
 - Specifically requires droplet precautions for 5 days after initiation of antimicrobial therapy
- Maintenance of adequate hydration
- Monitor oxygenation during paroxysms; supplement oxygen
- Airway management may be lifesaving in younger children

- **Antibiotics:**
 - Effective in the catarrhal stage
 - Prevent further transmission in the paroxysmal stage
 - Azithromycin is the first-line agent
 - Alternatively, clarithromycin, erythromycin, or trimethoprim–sulfamethoxazole may be used, although the efficacy is unproven; useful if erythromycin is not tolerated
- Corticosteroids and albuterol may reduce paroxysms of coughing, but further studies are required
- With increasing incidence of pertussis among adolescents and adults, emergency physicians can decrease incidence of pertussis by making vaccination routine when also vaccinating against tetanus:
 - Tetanus toxoid, reduced diphtheria toxoid, acellular pertussis (Tdap)

MEDICATION

Bronchodilators and steroids are generally not recommended for pertussis

First Line

- Azithromycin (adult): 500 mg PO day 1, then 250 mg PO QD for 4 days
- Azithromycin <5 mo: 10 mg/kg PO daily for 5 days
- Azithromycin 5 mo–adult: 10 mg/kg PO day 1 (max. 500 mg), then 5 mg/kg PO daily for 4 days (max. 250 mg daily)
- Tetanus toxoid, reduced diphtheria toxoid, Tdap vaccine: 0.5 mL IM:
 - Adacel: Approved for ages 11 and up
 - Boostrix: Approved for ages 10 and up

Pregnancy Considerations

- Advisory Committee on Immunization Practices (ACIP) recommends Tdap for pregnant patients during each pregnancy
- May be given anytime, but preference is between 27--36 weeks gestation

Second Line

- Clarithromycin: 15 mg/kg/d div. BID for 7 days (max. 1 g/d)
- Erythromycin: 40–50 mg/kg/d div. QID for 14 days (max. 2 g/d). Associated with risk of pyloric stenosis when administered in 1st 2 wk of life
- Trimethoprim–sulfamethoxazole: 8/40 mg/kg/d div. BID for 14 days (max. 320/1,600 mg/d):
 - Not for infants <2 mo



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients <1 yr
- Apnea
- Cyanosis during paroxysms of cough
- Significant associated pneumonia
- Encephalitis

Discharge Criteria

- Children without apnea, respiratory compromise, altered mental status, or complications and respiratory distress
- Warm liquids to reduce coughing spasm
- Remove thick secretions with bulb suction in infants
- Good hydration
- Avoid cough triggers: Cigarette smoke, pollutants, perfumes
- Postexposure prophylaxis is recommended to all persons with close contact (within 3 ft of a symptomatic person):
 - Antibiotic recommendations are the same as those with disease
 - Symptomatic children should be excluded from school or work; individuals with pertussis may return after 5 days of full treatment

FOLLOW-UP RECOMMENDATIONS

Children who are discharged need close follow-up to monitor hydration status and for respiratory compromise.

ALERT

Physicians are legally required to report cases of pertussis to state health department.

COMPLICATIONS

- Head, eyes, ears, neck, throat:
 - Epistaxis
 - Subconjunctival hemorrhage
- Respiratory:
 - Acute respiratory arrest
 - Pneumonia caused by secondary infection
 - Pneumothorax
 - SC or mediastinal emphysema with crepitus
 - Bronchiectasis
- GI:
 - Hernia: Inguinal or abdominal
 - Rectal prolapse
- Neurologic:
 - Seizures
 - Encephalitis

- Coma
- Intracranial hemorrhage
- Spinal epidural hemorrhage

ALERT

The child with pertussis may have significant respiratory distress or apnea

PEARLS AND PITFALLS

- Infants ≤ 1 yr need admission for pertussis
- Tdap should be given to eligible patients requiring tetanus prophylaxis
- Droplet precautions should be implemented for 5 days after implementation of effective antimicrobial therapy
- Chemoprophylaxis is recommended for all household contacts irrespective of age and immunization status

ADDITIONAL READING

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CODES

ICD9

- 033.0 Whooping cough due to bordetella pertussis [B. pertussis]
- 033.1 Whooping cough due to bordetella parapertussis [B. parapertussis]
- 033.9 Whooping cough, unspecified organism

ICD10

- A37.00 Whooping cough due to Bordetella pertussis without pneumonia
- A37.10 Whooping cough due to Bordetella parapertussis w/o pneumonia
- A37.90 Whooping cough, unspecified species without pneumonia

PHALANGEAL INJURIES, FOOT

Taylor Y. Cardall

BASICS

DESCRIPTION

- The phalanges of the foot are prone to injury.
- 5th (or small) toe most commonly affected

ETIOLOGY

- Usually the result of direct trauma
- Stubbing the toe, kicking a hard surface, or dropping a heavy object onto toes most common mechanisms of injury

DIAGNOSIS

SIGNS AND SYMPTOMS

History

History may predict the type of injury found and should include:

- Time of injury
- Mechanism
- History of previous trauma
- Status of tetanus immunization if laceration is present

Physical-Exam

- Tenderness, swelling, crepitus, and ecchymosis of affected digit
- Subungual hematomas are often present.
- Lacerations or crush-type wounds
- Document neurovascular status of the affected digit.

ESSENTIAL WORKUP

Radiographs of involved digit

DIAGNOSIS TESTS & INTERPRETATION

Imaging

- Radiographs of involved digit
- Lateral view may be most sensitive.

DIFFERENTIAL DIAGNOSIS

- Fracture
- Contusion
- Abrasion/laceration
- Dislocation



TREATMENT

PRE HOSPITAL

- Ice to affected digit
- Direct pressure and dressing to any wounds

INITIAL STABILIZATION/THERAPY

- Ice to affected digit
- Direct pressure and dressing to any wounds

ED TREATMENT/PROCEDURES

- Fractures involving the proximal phalanx and interphalangeal (IP) joint of the hallux:
 - Nondisplaced, non–intra-articular fractures may be placed in a short-leg walking cast with toe extension for comfort.
 - Displaced, non–intra-articular fractures:
- Closed reduction with digital block anesthesia
- Longitudinal traction
- Placement in short-leg walking cast with toe extension:
 - Intra-articular fractures of the hallux merit orthopedic consult:
 - Frequently treated with open reduction and internal fixation
- Fractures involving the proximal phalanx and IP joint of the lesser toes:
 - Rarely cause long-term disability
- Nondisplaced fractures:
 - Treat with splinting or buddy taping
 - Gauze padding between the taped toes to prevent skin breakdown
- Displaced fractures:
 - Closed reduction by digital block anesthesia
 - Longitudinal traction
 - Buddy taping or splinting
 - Hard-sole shoe, weight bearing as tolerated
 - Oral analgesics for pain
 - Pain usually resolved by 2–3 wk
- IP joint dislocations:
 - Closed reduction by digital block anesthesia
 - Longitudinal traction with gentle downward pressure on distal phalanx
 - Buddy tape to adjacent toe

- Unstable or unsuccessful reductions require orthopedic consultation.
- Oral analgesics for pain
- Distal tuft fractures:
 - Subungual hematomas should be drained.
 - Nail-bed laceration repair may be necessary.
 - Buddy tape digit to adjacent toe.
 - Weight bearing as tolerated
 - Oral analgesics for pain
 - Pain usually resolved in 2–3 wk
- Open fractures:
 - Orthopedic consultation
 - Prophylactic antibiotics

MEDICATION

- NSAIDs are useful in treating acute pain:
 - Ibuprofen 800 mg (peds: 5–10 mg/kg) PO TID
- Narcotic analgesics may be required for severe pain
- Consider antibiotics for open wounds
 - Cefazolin: 1 g IM/IV in ED (peds: 50–100 mg/kg IM/IV in ED) for open fractures
 - Cephalexin 500 mg PO QID (peds 25–50 mg/kg/d in div. doses) for 7 days for dirty wounds.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Unstable or blocked dislocations
- Open fractures require orthopedic consultation in the ED.

Discharge Criteria

All other fractures may be discharged with orthopedic follow-up in 2–3 wk to evaluate healing.

Issues for Referral

Patient copies of any radiographs obtained may facilitate early follow-up.

FOLLOW-UP RECOMMENDATIONS

- Intra-articular fractures involving the proximal phalanx of the great toe require urgent orthopedic or foot and ankle surgery follow-up.
- Simple nondisplaced fractures of the small toes may often be followed by primary

care physicians.

PEARLS AND PITFALLS

Open, displaced, or intra-articular fractures, particularly involving the hallux, merit orthopedic consultation.

ADDITIONAL READING

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CODES

ICD9

- 826.0 Closed fracture of one or more phalanges of foot
- 924.3 Contusion of toe
- 959.7 Knee, leg, ankle, and foot injury

ICD10

- S90.129A Contusion of unspecified lesser toe(s) without damage to nail, initial encounter
- S92.919A Unsp fracture of unsp toe(s), init for clos fx
- S99.929A Unspecified injury of unspecified foot, initial encounter

PHALANGEAL INJURIES, HAND

Asia M.F. Takeuchi • Stephen R. Hayden



BASICS

DESCRIPTION

- 1/3 of all traumatic injuries affect the hand.
- Phalanges account for 1 of the most frequently fractured parts of the skeletal system with the distal phalanx being the most commonly fractured bone in the hand.
- Dorsal displacement of the proximal interphalangeal joint of the finger is the most frequent dislocation.

Pediatric Considerations

Injuries may be more difficult to diagnose in children who are unable to cooperate for a full exam.

ETIOLOGY

- Trauma (commonly work or sports related)
- Infectious sequelae:
 - Skin flora: *Staphylococcus aureus* and *Streptococci*
 - Cat/dog bites: *S. aureus* and *Pasteurella multocida*
 - Human bites: *Eikenella*
 - Thorns or woody plants puncture: Fungal
 - Fresh/salt water exposure: *Mycobacterium marinum* and *Pseudomonas aeruginosa*
- Overuse injury (e.g., “gamekeeper’s thumb”)



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Mechanism of injury:
 - Hyperextension injuries most commonly cause ligamentous injury (e.g., “Jersey finger” which is a rupture of the flexor digitorum profundus tendon from its distal attachment) or chip fractures.
 - Hyperflexion injury to the tip of digits may cause “Mallet finger” injury with avulsion fracture at the insertion of the extensor tendon on the distal phalanx.
 - Crush injuries most commonly cause fractures and diffuse soft-tissue injury.

- Handedness
- Occupation/hobbies
- Other factors may affect healing (e.g., age, diabetes, immune suppression, anticoagulation)

Physical-Exam

- Swelling and/or deformity (e.g., amputation, rotation, shortening, or angulation)
- Skin changes (e.g., ecchymosis, laceration, burn, pallor) or associated nail injury
- Decreased range of motion or weakness
- Pain or change in sensation in the area of injury

ALERT

Kanavel signs (*infectious* flexor tenosynovitis)

- Pain along the tendon with passive extension (early sign)
- Symmetric enlargement of the affected digit
- Slightly flexed finger at rest
- Tenderness along the course of the flexor sheath (later sign)
- Trigger finger (stenosing flexor tenosynovitis):
- *Noninfectious* inflammation of the flexor tendon sheath.
- Painful “snapping” sensation with flexion of the affected digit.
- May awaken with the finger locked in the palm, with gradual “unlocking” as the day progresses.

Pediatric Considerations

In an infant with a painful or swollen digit, it is important to consider a deeply embedded hair tourniquet that may not be readily obvious on superficial exam.

ESSENTIAL WORKUP

- Special attention directed at assessing individual tendon status, neurovascular integrity, and identifying rotational deformity:
 - Isolate and assess each individual joint (PIP, DIP, MCP); range with passive motion and against active resistance
 - Normal 2-point discrimination is ~4–5 mm
 - Malrotation can be evaluated by positioning the fingers with the MCP joints in flexion and the PIP and DIP in extension:
 - Normally, all fingers are directed toward the radius and there should be no overlap or rotation
- Exam conducted 1st to assess function, then under anesthesia, and finally with tourniquet if needed to allow a bloodless field for better exam of lacerated areas.

DIAGNOSIS TESTS & INTERPRETATION

Lab

Consider wound culture if signs of infection present or if there is concern for flexor tenosynovitis.

Imaging

- Plain radiography of involved digits should include AP, true lateral, and oblique views.
- US can help diagnose tendon tears.

Pediatric Considerations

Open epiphyses make radiographic interpretation less sensitive.

DIFFERENTIAL DIAGNOSIS

- Tendon laceration/rupture partial/complete
- Complicated open injuries may include several injuries, and the entire hand should be examined carefully.
- Beware of lacerations over dorsal metacarpal–phalangeal areas, which may be “fight bites” (human bites).

Pediatric Considerations

- Many fractures in children are torus (buckle) fractures of the phalanges.
- The growth plates are typically weaker than the surrounding ligaments, thus dislocations are commonly accompanied by Salter–Harris fractures.



TREATMENT

PRE HOSPITAL

- Reduction of a phalangeal dislocation at the scene SHOULD NOT be considered UNLESS there will be an unusually long transport time or there is vascular or neurologic compromise.
 - Reduction may be successful but prompt the physician to miss significant ligamentous injuries.
- Bleeding should be treated with appropriate direct pressure dressings.

ALERT

- Amputated digits or tissue should be placed in clean moist saline gauze, placed in plastic bag, and then placed in a separate bag with ice. *Do not place digit in direct contact with ice!*
- Indications for reimplantation in amputation:
 - Thumb
 - Single digit between PIP and DIP joints
 - Multiple digits
 - Amputation in a child

INITIAL STABILIZATION/THERAPY

- Remove all rings from injured hand.
- Immobilize the involved areas by proximal-to-distal splinting.
- Intermittent ice pack application with constant elevation for the 1st 24 hr.
- Dislocations or severely deformed fractures producing vascular compromise should be reduced immediately to a neutral position and immobilized.

ED TREATMENT/PROCEDURES

- Interphalangeal reduction:
 - Dorsal dislocation:
 - Provide longitudinal traction and gently hyperextend the joint while pushing the base of the dislocated phalanx into place.
 - Volar dislocation:
 - Provide longitudinal traction and gently hyperflex while pushing the base of the dislocated phalanx into place.
 - Lateral dislocation:
 - Provide longitudinal traction and gently hyperextend the joint while correcting the ulnar or radial deformity.
- Interphalangeal immobilization:
 - DIP dorsal or lateral finger dislocation:
 - Splint the DIP in full extension while allowing full range of motion of the PIP joint.
 - PIP dorsal or lateral finger dislocation:
 - Apply a dorsal splint with the PIP in 20–30° of flexion.
 - Volar finger dislocation:
 - Splint the PIP and DIP in full extension.
- Metacarpophalangeal dislocation:
 - Avoid excessive hyperextension or distraction. Gently distract the affected digit and apply volar pressure to the base of the dislocated proximal phalanx.
- Metacarpophalangeal immobilization:
 - Finger dislocation: Splint the digit in 90° of flexion at the MCP joint.
 - Thumb dislocation: Apply a thumb spica splint with the MCP joint in 20° of flexion.
- Open fracture:
 - Immediate referral to a hand surgeon for treatment within 4–6 hr after trauma.
 - Prophylactic antibiotics directed against gram-positive and gram-negative organisms should be administered parenterally within 6 hr.
- Closed fracture:
 - Distal phalanx:
 - Stable injuries may be splinted with the DIP in flexion and the PIP free; extend tip of splint beyond the end of the digit for added

protection; maintain for 3–4 wk.

– Middle phalanx:

- Nondisplaced stable fractures can be buddy taped to an adjacent digit.
- Displaced/angulated fractures may be reduced (using longitudinal traction with 3-point pressure to align the fragment) and immobilized (buddy tape and ulnar/radial gutter splint).
- Splinting should be done with the wrist in 20–30° of extension, the MCP joints in 70–90° of flexion, and the PIP and DIP joints flexed 5–10°.

– Proximal phalanx:

- A nondisplaced, nonangulated, stable injury can be buddy taped to an adjacent finger; ulnar/radial gutter or Burkhalter splint may be added for comfort.
- A displaced or angulated fracture may be reduced by flexing the MCP and PIP joints to 90°, then using a 3-point reduction technique to reduce the proximal fragment dorsally and the distal fragment volarly. Once reduced, the PIP joint should be extended (to avoid a flexion contracture), the MCP joint should remain in 70–90° of flexion and a radial or ulnar gutter splint should be placed with the fractured finger buddy taped to an adjacent finger.

ALERT

No more than 1 or 2 mm of displacement or shortening is acceptable. Up to 10° of angulation is acceptable but NO amount of rotation is permitted.

● Mallet finger:

- Immobilize the DIP joint in full extension or slight hyperextension (5–15°), while allowing full range of motion of the PIP joint.
- Do **not** attempt to reduce any displaced fractures before splinting because any reduction is unlikely to be maintained without surgery; refer for urgent orthopedic consult.

● Jersey finger:

- Apply an aluminum splint with the PIP joint and the DIP joint slightly flexed.
- DIP extension should be avoided until the digit can be evaluated by a hand specialist (definitive treatment of complete tendon rupture is surgery).

● Trigger finger:

- Immobilize by buddy taping to the adjacent finger for 4–6 wk.
- A metal or thermoplastic finger splint can be used if buddy taping is unsuccessful.

● Gamekeeper's thumb:

- Apply ice to the MP joint acutely.
- Immobilize with a thumb spica splint (MP joint is flexed to 20°) for 3 wk.

● Subungual hematoma:

- Nail trephination using a heated paper clip, electric cautery, or an 18G needle.
- This injury does not have to be treated as an open injury *unless* there is an underlying tuft fracture.
- Nail avulsions:
 - Clean and repair using fine (e.g., 6-0) absorbable suture.
 - Splint the eponychium and germinal matrix with the avulsed nail or small piece of gauze or foil to avoid adhesions.
- Open distal and volar directed fingertip wounds with no protruding bone and smaller than 1 cm may be allowed to heal by secondary intention.

MEDICATION

- Evaluate tetanus status and vaccinate per immunization schedule.
- Digital nerve block should be done with an anesthetic that does NOT contain epinephrine.
- Antibiotics:
 - Not indicated for simple clean wounds
 - For grossly contaminated injury, puncture wounds, or infectious tenosynovitis therapy should be tailored to specific pathogen exposure (e.g., skin flora, fresh water, bites)



FOLLOW-UP

DISPOSITION

- Patients with a stable injury, in an appropriate splint, may be discharged for orthopedic follow-up and possible repeat imaging in 1 wk time.

ALERT

Emergent orthopedic consult is required for:

- Amputation
- Open joint injuries or fractures
- Digital neurovascular compromise
- Signs of joint infection or infectious tenosynovitis
- High-pressure injection injury
- Urgent orthopedic consult:
 - Unstable fractures (rotational deformity, oblique or angulated fractures, joint involvement, epiphyseal injuries)
 - Any joint dislocation with tendon rupture
 - Digit dislocation that is irreducible
 - Unstable joint after attempted dislocation reduction

PEARLS AND PITFALLS

- Rotational deformity may not be apparent if finger is straight, exam under flexion is required.
- Jersey finger (FDP tendon rupture) is often misdiagnosed as a “jammed” or sprained finger, but requires more urgent management than these minor injuries.
- Always check for stability postreduction by having patient perform active range of motion and checking a postreduction x-ray.

ADDITIONAL READING

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- Tuttle HG, Olvey SP, Stern PJ. Tendon avulsion injuries of the distal phalanx. *Clin Orthop Relat Res.* 2006;445:157–168.



CODES

ICD9

- 816.00 Closed fracture of phalanx or phalanges of hand, unspecified
- 834.00 Closed dislocation of finger, unspecified part
- 959.5 Finger injury

ICD10

- S62.609A Fracture of unsp phalanx of unsp finger, init for clos fx
- S63.259A Unspecified dislocation of unspecified finger, init encntr
- S69.90XA Unsp injury of unsp wrist, hand and finger(s), init encntr

PHARYNGITIS

John C. Greenwood • Brian J. Browne



BASICS

DESCRIPTION

- Inflammation/infection of the pharynx
- 3rd most common complaint for physician visits
- 30 million cases diagnosed annually
- Group A β -hemolytic streptococcus (GAS):
 - *Streptococcus pyogenes*
 - Unusual in children < 3 yr old
 - Cause of 20–30% of childhood pharyngitis
 - Bimodal incidence, highest in ages 5–7 and 12–13 yr
 - Cause of 5–15% of adult pharyngitis
 - Peak months: January–May; also at the start of the school year

ETIOLOGY

- Viral (most common infectious cause):
 - Rhinovirus (20%)
 - Coronavirus (> 5%)
 - Adenovirus (5%)
 - Herpes simplex virus (4%)
 - Parainfluenza virus (2%)
 - Influenza virus (2%)
 - Coxsackievirus (< 1%)
 - Epstein–Barr virus (< 1%)
 - Acute human immunodeficiency virus (HIV)
- Bacterial:
 - GAS (*S. pyogenes* [15–30%])
 - *Fusobacterium necrophorum* (10%)
 - Group C & G β -hemolytic streptococcus (5%)
 - *Neisseria gonorrhoea* (< 1%)
 - *Corynebacterium diphtheriae* (< 1%)
 - *Arcanobacterium haemolyticum* (< 1%)
 - *Chlamydia pneumoniae*
 - *Mycoplasma pneumoniae* (< 1%)
 - Syphilis
 - Tuberculosis
- Fungal:
 - *Candida* (thrush)

- Chemical burns
- Foreign bodies
- Inhalants
- Postnasal drip
- Malignancy
- GERD



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Viral:
 - Cough
 - Rhinorrhea
 - Sore throat usually follows
 - Have a high suspicion for acute HIV in at-risk patients presenting with persistent pharyngitis despite treatment
- Bacterial:
 - Sudden-onset sore throat that usually precedes other symptoms
 - Odynophagia
 - Fever
 - Headache
 - Abdominal pain
 - Nausea and vomiting
 - Uncharacteristic symptoms:
 - Coryza
 - Hoarseness
 - Diarrhea

Physical-Exam

- High-risk features for a serious complication of pharyngitis:
 - Stridor, respiratory distress
 - Drooling
 - Dysphonia
 - Marked neck swelling
 - Neurologic dysfunction
- Viral:
 - Cough
 - Coryza
 - Rhinorrhea
 - Pharyngeal erythema

- Gingivostomatitis
- GAS:
 - Tonsillopharyngeal erythema/exudates
 - Soft palatal petechiae
 - Beefy red, swollen uvula
 - Anterior cervical lymphadenopathy
 - Scarletiform rash
 - Uncharacteristic signs:
 - Conjunctivitis
 - Anterior stomatitis
 - Discrete ulcerative lesions
- Mononucleosis:
 - Mistaken for GAS due to similar presentation:
 - Exudative pharyngitis
 - Tender cervical lymphadenopathy
 - Fever
 - Rash
 - Other possible exam findings:
 - Hepatosplenomegaly
 - Jaundice
- Diphtheria:
 - Consider in nonimmunized patients
 - Airway-threatening gray pharyngeal membrane
 - Myocarditis (2/3 of patients); clinically evident cardiac dysfunction (10–25%)
 - Cranial and peripheral neuropathies (5%)
- Gonococcal pharyngitis:
 - Can be asymptomatic
 - Always evaluate children for sexual abuse
 - Recurrent episodes of pharyngitis

ESSENTIAL WORKUP

Modified Center criteria for the diagnosis of GAS pharyngitis (most widely used decision rule):

- Criteria (points):
 - Absence of cough (+1)
 - Tonsillar exudates or swelling (+1)
 - Swollen and tender anterior cervical nodes (+1)
 - Temperature > 38°C (+1)
 - Age in years:
 - 3–14 (+1)
 - 15–44 (0)
 - > 45 (–1)

- Scoring:
 - < 1 should not be tested or treated
 - 3 is associated with a risk of 28–35%
 - > 4 is associated with a risk of 51–53%
- Patients with 3 criteria should receive a rapid antigen detection test (RADT)
- Presumptive treatment without testing has led to inappropriate use of antibiotics in about 50% of cases
- Some suggest that patients with a score > 4 should be treated empirically without a RADT

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Throat culture:
 - Gold standard
 - 24–48 hr for results, will delay treatment
 - Necessitates contacting patient/family
 - Obtain when *Gonococcus* is suspected
- GAS RADT:
 - Results are available within 30 min
 - Treat all patients with (+) RADT results
 - Technique: Performed by swabbing the tonsils or posterior pharynx:
 - Avoid contact with the tongue, buccal mucosa, and lips
 - Sensitivity 85–95%
 - Specificity 96–99%:
 - Confirm with conventional throat culture in children/adolescents with negative RADT
 - Optical immunoassay is extremely accurate; negative results do not require confirmatory culture
- Monospot:
 - Detects heterophil antibody:
 - Sensitivity:
 - < 2 yr old: < 30%
 - 2–4 yr old: 75%
 - > 5 yr old: 90%
 - CBC with peripheral smear: 50% lymphocytes, 10% atypical lymphocytes
 - Obtain rapid viral loads if HIV is suspected

Imaging

- Lateral neck radiograph for suspected epiglottitis, retropharyngeal abscess, or foreign body
- Contrast-enhanced CT of the neck is useful to identify complications such as peritonsillar abscess and retropharyngeal abscess

DIFFERENTIAL DIAGNOSIS

- Epiglottitis
- Peritonsillar/retropharyngeal abscess
- Diphtheria
- Mononucleosis
- Lemierre disease
- Ludwig angina
- *Candida* infection
- Gonorrhea
- Acute HIV infection
- Acute leukemia/lymphoma
- Oropharyngeal cancer
- Foreign body
- Inhalants and chemical burns
- Postnasal drip
- GERD



TREATMENT

PRE HOSPITAL

- Observe/manage airway for respiratory distress
- Normal saline (NS) hydration for hypotension/dehydration

INITIAL STABILIZATION/THERAPY

- ABCs
- Fluid resuscitation: 1 L (peds: 20 mL/kg) NS bolus for signs of volume depletion or if patient is unable to tolerate oral solutions

ED TREATMENT/PROCEDURES

- Antipyretics/analgesics:
 - Acetaminophen
 - Ibuprofen
 - Topical analgesics (e.g., Chloraseptic spray)
- GAS infection:
 - Often mild and self-limited:
 - Antibiotic therapy accelerates symptom relief (fever and pain) by 1–2 days
 - Goal of antibiotic treatment is to reduce the incidence of acute rheumatic fever, symptoms, and suppurative complications
- Antibiotics:
 - Penicillin V: Antibiotic of choice for GAS pharyngitis
 - Cephalosporins or macrolides are an acceptable alternative treatment for

nonresponders and penicillin-allergic patients

- Corticosteroids:
 - In conjunction with antibiotics, corticosteroids have a 3-fold increase in the likelihood of symptom resolution at 24 hr
 - Number needed to treat: 3.3–3.7
 - Avoid in diabetics and immunocompromised patients
- Potential complications of streptococcal infection:
 - Suppurative complications:
 - Peritonsillar/retropharyngeal abscess
 - Lemierre disease
 - Otitis media/mastoiditis
 - Nonsuppurative complications:
 - Acute rheumatic fever:
 - Rare in industrialized countries, but still the leading cause of cardiac death within 1st 5 decades of life
 - Sequelae of GAS; not proven in association with group C or G
 - Acute poststreptococcal glomerulonephritis
 - Sydenham chorea
 - Reactive arthritis
 - PANDAS: Pediatric autoimmune neuropsychiatric disorder associated with streptococcal infection:
 - Sudden onset of symptoms similar to obsessive–compulsive disorder
 - Caused by an autoimmune reaction affecting the basal ganglia
 - Uncommon and controversial
- Diphtheria:
 - Goals of therapy:
 - Prevent airway obstruction
 - Treat infection
 - Penicillin or macrolide antibiotic
 - Complications:
 - Exotoxin-mediated myocarditis and neuritis (cranial neuropathies)
- Gonococcal pharyngitis:
 - 3rd-generation cephalosporin plus macrolide for possible *Chlamydia* coinfection

MEDICATION

First Line

- Penicillin G:
 - < 27 kg: Benzathine penicillin G (Bicillin LA): 0.6 million U IM × 1
 - > 27 kg: Benzathine penicillin G (Bicillin LA): 1.2 million U IM × 1
- Penicillin V:

- <12 yr: 25–50 mg/kg/d PO div. q6–8h × 10 days
- >12 yr: 250–500 mg PO q6–8h × 10 days
- Amoxicillin:
 - 50 mg/kg PO QD, (max. 1 g) × 10 days

Second Line

- Macrolides:
 - Azithromycin: 20 mg/kg/d × 3 days (max. 500 mg per dose)
 - Erythromycin: 40–50 mg/kg PO div. q6h × 10 days (max. 500 mg per dose)
- Oral cephalosporins:
 - Cephalexin: 20 mg/kg/dose PO BID × 5 days (max. 500 mg per dose)
- Steroids:
 - Dexamethasone: 0.6 mg/kg IM/PO × 1 (max. 10 mg)
 - Prednisone: 40–60 mg PO × 1
- Special conditions:
 - Suspected gonococcal pharyngitis:
 - Ceftriaxone: 125–250 mg IM × 1



FOLLOW-UP

DISPOSITION

Admission Criteria

- Airway compromise
- Severe dehydration
- Suspected child abuse

Discharge Criteria

Able to tolerate oral intake

FOLLOW-UP RECOMMENDATIONS

- If symptoms do not improve within 72 hr
- Patients are no longer contagious after 24 hr of antibiotic treatment
- Mononucleosis patients should avoid contact sports

PEARLS AND PITFALLS

- Use the modified Centor criteria to make the decision to test for GAS pharyngitis
- Children with negative RADT need follow-up throat culture
- Acute rheumatic fever is a more common complication of GAS pharyngitis in nonindustrialized nations
- Evaluate for high-risk complications of bacterial pharyngitis (e.g., peritonsillar

abscess, retropharyngeal abscess, Lemierre disease)

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Epiglottitis
- Mononucleosis
- Peritonsillar Abscess
- Retropharyngeal Abscess
- Rheumatic Fever



CODES

ICD9

- 034.0 Streptococcal sore throat
- 054.79 Herpes simplex with other specified complications
- 462 Acute pharyngitis

ICD10

- J02.0 Streptococcal pharyngitis
- J02.8 Acute pharyngitis due to other specified organisms
- J02.9 Acute pharyngitis, unspecified

PHENCYCLIDINE POISONING

Steven E. Aks



BASICS

DESCRIPTION

- Phencyclidine (PCP) is a dissociative anesthetic structurally related to ketamine:
 - Causes decreased perception of pain and agitation
- Half-life of 21–24 hr, but may be longer in overdose
- Enterohepatic recirculation—recirculated into the stomach

ETIOLOGY

- Drug of abuse:
 - Frequently encountered as an adulterant of marijuana
- Street names for PCP include:
 - Angel dust
 - Wicky stick
 - Wicky weed
 - Wacky weed
 - Wet
 - Illy
 - Embalming fluid
 - Sherman

Pediatric Considerations

Exposure in toddlers reported via passive exposure



DIAGNOSIS

SIGNS AND SYMPTOMS

- CNS:
 - Altered mental status
 - Agitation
 - Bizarre/violent behavior
 - Belligerence
 - Coma
 - Seizures
 - Nystagmus (vertical, horizontal, or rotatory)
- Cardiovascular:
 - HTN
 - Tachycardia

- Musculoskeletal:
 - Traumatic injury (decreased pain perception)
 - Rhabdomyolysis (due to vigorous muscular contraction)
- Vital signs:
 - Hyperthermia

History

How was the PCP consumed?

- Smoked with marijuana
- Ingested

Physical-Exam

- Agitation
- Coma
- Hypertension
- Tachycardia
- Diaphoresis
- Nystagmus (vertical, horizontal, or rotatory)
- Hyperthermia
- Vigorous muscular contraction

ESSENTIAL WORKUP

- Clinical diagnosis based on presentation supported by urine toxicology screen:
 - Dextromethorphan and ketamine may give false positive.
- Careful physical exam for occult trauma
- Exclude other causes of altered mental status.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC
- Electrolytes, BUN/creatinine, glucose
- Urinalysis:
 - Dip for myoglobin (rhabdomyolysis)
- Creatine phosphokinase:
 - If urine dip for blood is positive
- Ethanol level

Imaging

- Chest radiograph for aspiration pneumonia
- Extremity/spine radiographs when there is associated trauma
- CT of the head when there is head trauma/altered mental status

DIFFERENTIAL DIAGNOSIS

- Drugs of abuse:
 - Cocaine
 - Amphetamines
 - Designer drugs:
 - Methcathinone (“Cat”)
 - “Ecstasy”
 - “Ice” (methamphetamine)
 - Alcohols
 - Ketamine
 - Sympathomimetics
- Drugs that cause nystagmus:
 - Lithium
 - Carbamazepine
 - Sedative–hypnotics
 - Alcohols
 - Phenothiazines
 - Dextromethorphan



TREATMENT

PRE HOSPITAL

ALERT

Use restraints/additional personnel to control combative patient.

INITIAL STABILIZATION/THERAPY

- ABCs
- IV
- Cardiac monitor
- Naloxone, thiamine, glucose (or Accu-Chek) if altered mental status
- Protect patient and staff from injury.

ED TREATMENT/PROCEDURES

- Maintain patient in a quiet place; avoid stimulation.
- Physical restraints for violent patient
- Sedation:
 - Benzodiazepines
 - Butyrophenones (haloperidol) theoretically can lower the seizure threshold.
- Activated charcoal if oral coingestants
- IV 0.9% normal saline for hydration, sodium bicarbonate/mannitol for rhabdomyolysis

MEDICATION

First Line

- Ativan (lorazepam): 2 mg IV increments
- Diazepam: 5 mg IV increments

Second Line

- Activated charcoal slurry: 1–2 g/kg up to 90 g PO
- Dextrose: D50W 1 amp: 50 mL or 25 g (peds: D25W 2–4 mL/kg) IV
- Mannitol: 25–50 g IV
- Naloxone (Narcan): 2 mg (peds: 0.1 mg/kg) IV or IM initial dose
- Sodium bicarbonate: 2 amps (50 mEq per amp) diluted in 1 L of D5W, given at 125–250 mL/h (for rhabdomyolysis) to urine pH of 7
- Thiamine (vitamin B₁): 100 mg (peds: 50 mg) IV or IM



FOLLOW-UP

DISPOSITION

Admission Criteria

- Prolonged altered mental status
- Significant traumatic injuries
- Rhabdomyolysis
- Hyperthermia

Discharge Criteria

Becomes lucid after a period of observation (6 hr)

FOLLOW-UP RECOMMENDATIONS

Psychiatry or social work referral for suicidal ideation or chronic drug use

PEARLS AND PITFALLS

- PCP poisoning can lead to traumatic injuries that can become life threatening.
- Adequate chemical restraints with benzodiazepines are needed to prevent excessive muscular activity leading to rhabdomyolysis.
- Dextromethorphan is a common cause for a false-positive PCP urine toxicology screen.
- Tramadol has been reported to cause a false-positive screen for PCP
- Ketamine abuse presents with similar signs and symptoms of PCP abuse.

ADDITIONAL READING

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- Wills B, Erickson T. Drug- and toxin-associated seizures. *Med Clin North Am*. 2005;89:1297–1321.



CODES

ICD9

968.3 Poisoning by intravenous anesthetics

ICD10

T40.991A Poisoning by oth psychodyslept, accidental, init

PHENYTOIN POISONING

Michele Zell-Kanter



BASICS

DESCRIPTION

- Phenytoin follows zero-order pharmacokinetics:
 - Small incremental increase in dose can result in a large increase in plasma concentration.
- Half-life in overdose prolonged; may be up to 70 hr
- Cardiovascular toxicity from IV administration likely due to the diluent propylene glycol
- Fosphenytoin, a prodrug for parenteral administration, is metabolized to phenytoin, its active moiety.

ETIOLOGY

- Phenytoin intoxication results from acute, chronic, or acute-on-chronic administration.
- If the cause of the intoxication is unclear in a patient receiving chronic phenytoin therapy, consider that there may have been a:
 - Change in the brand of phenytoin
 - Change in dosage form
 - Drug interaction
 - Change in serum albumin



DIAGNOSIS

SIGNS AND SYMPTOMS

- Level 20–40 $\mu\text{g/mL}$ (or mg/L):
 - Nystagmus
 - Dizziness
 - Ataxia
 - Drowsiness
 - Nausea/vomiting
 - Diplopia
 - Slurred speech
- Level 40–90 $\mu\text{g/mL}$:
 - Confusion
 - Disorientation
- Level >90 mg/mL :
 - Coma

- Respiratory depression
- Paradoxical seizures
- Hypotension/bradycardia with rapid IV administration:
 - Fosphenytoin injection does not contain propylene glycol
 - Hypotension/dysrhythmia unlikely with fosphenytoin
- Hypersensitivity reaction following chronic use:
 - Rash
 - Fever
 - Neutropenia
 - Agranulocytosis
 - Hepatitis
 - Cholangitis

ESSENTIAL WORKUP

- Determine the time, route, and amount of ingestion.
- Phenytoin level:
 - After oral overdose, the peak plasma concentration may not be reached until 24 hr or more post acute ingestion.
 - Absorption differs with various oral preparations and manufacturers
 - Repeat levels every 4 hr until levels have peaked and continue to steadily decline.
 - Once levels begin declining, check every 24 hr until $< 30 \mu\text{g/mL}$.
 - Free phenytoin level may be required in patients who are hypoalbuminemic or patients who are poor metabolizers.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Fosphenytoin level:
 - Measured as phenytoin
 - Measure fosphenytoin after conversion to phenytoin is complete (2 hr post IV infusion or 4 hr post IM injection).
 - Prior to complete conversion to phenytoin, immunoanalytic techniques may overestimate plasma phenytoin concentrations due to cross-reactivity with fosphenytoin.
- Electrolytes, BUN, creatinine, glucose:
 - Check for anion gap metabolic acidosis due to coingestant, seizure activity, from propylene glycol in the IV formulation
 - Determine glucose with altered mental status.

DIFFERENTIAL DIAGNOSIS

- Intoxication with other CNS depressants
- Guillain–Barré syndrome

- Botulism
- Posterior fossa tumor
- Acute cerebellitis



TREATMENT

PRE HOSPITAL

- Differentiate phenytoin-induced altered mental status from other potentially serious causes:
 - _ Head trauma common in seizure population
- Collect/transport prescription bottles and medications to aid in identification and quantification of ingestion

INITIAL STABILIZATION/THERAPY

- ABCs:
 - _ IV access
 - _ Cardiac monitor (with IV overdose)
- For altered mental status:
 - _ Accu-Chek.
 - _ Administer naloxone, dextrose, and thiamine as indicated.
- Treat hypotension with IV fluids and Trendelenburg position:
 - _ Dopamine for refractory hypotension
- Treat paradoxical seizures with diazepam.

ED TREATMENT/PROCEDURES

- Provide supportive care
- Activated charcoal
 - _ Administer single dose.
 - _ Multiple-dose activated charcoal may increase the clearance of phenytoin; does not correlate with clinical improvement in patients with phenytoin toxicity.

MEDICATION

- Activated charcoal slurry: 1–2 g/kg up to 90 g PO
- Dextrose: D50W 1 amp: 50 mL or 25 g (peds: D25W 2–4 mL/kg) IV
- Dopamine: 2–20 µg/kg/min IV titrated to desired BP
- Naloxone (Narcan): 2 mg (peds: 0.1 mg/kg) IV or IM initial dose
- Thiamine (vitamin B₁): 100 mg (peds: 50 mg) IV or IM



FOLLOW-UP

DISPOSITION

Admission Criteria

- Altered mental status, severe ataxia, increasing phenytoin level
- Level $> 25 \mu\text{g/mL}$
- ICU admission with intoxication from IV phenytoin
- Fall precautions

Discharge Criteria

- Level $\leq 25 \mu\text{g/mL}$
- Ambulatory without ataxia

FOLLOW-UP RECOMMENDATIONS

- Psychiatric referral for intentional ingestions/suicide attempts.
- Close primary care follow-up to check phenytoin levels.
- Anticipate altered pharmacokinetics and phenytoin levels with any change in manufacturer or dosage formulation

PEARLS AND PITFALLS

- Small incremental increases in dose of phenytoin can result in toxicity since phenytoin follows zero-order kinetics.
- Repeat phenytoin levels every 4 hr until declining.

ADDITIONAL READING

- McCluggage LK, Voils SA, Bullock MR. Phenytoin toxicity due to genetic polymorphism. *Neurocrit Care*. 2009;10:222–224.
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- Von Winckelmann SL, Spriet I, Willems L. Therapeutic drug monitoring of phenytoin in critically ill patients. *Pharmacotherapy*. 2008;28:1391–1400.



CODES

ICD9

966.1 Poisoning by hydantoin derivatives

ICD10

T42.0X1A Poisoning by hydantoin derivatives, accidental, init

PHEOCHROMOCYTOMA

David N. Zull



BASICS

DESCRIPTION

- Pheochromocytoma (pheo) is a catecholamine-producing tumor arising from the chromaffin tissues of the sympathetic nervous system.
- Origin from the adrenal medulla or sympathetic ganglia:
 - 80% solitary adrenal (usually the right side)
 - 10% bilateral (usually inherited form)
 - 10% extra-adrenal in location:
 - Abdominal, within mesenteric ganglia (86%)
 - Thorax (10%), neck (3%), bladder (1%)
 - 10% malignant (usually inherited form)
- Incidence:
 - 0.2–0.4% of hypertensive patients, but higher proportion of patients with severe hypertension
 - 2–8/million population per year
 - Peaks in decades 3–5, 10% in children
 - Male = female
 - In about 1/2 of the cases, the diagnosis is made postmortem.
 - 10% asymptomatic, incidental on CT
- Genetics:
 - Inherited form 25%, autosomal dominant
 - Usually associated with multiple endocrine neoplasia (MEN) 2A, less so with MEN 2B or von Hippel–Lindau (VHL) disease:
 - MEN 2A (medullary thyroid carcinoma [CA], pheo, and hyperparathyroidism)
 - MEN 2B (medullary thyroid CA, pheo, oral mucosal neuromas, skeletal and bony abnormalities)
 - VHL (hemangioblastomas of retina and CNS, pancreas and renal cysts, and pheo)
 - Other associated diseases: Neurofibromatosis, tuberous sclerosis, Sturge–Weber syndrome, paragangliomas of the neck

ETIOLOGY

- The tumor synthesizes and stores catecholamines in the same manner as the normal adrenal medulla.
- Tumors predominantly secrete norepinephrine, and to a lesser extent epinephrine (some tumors are epinephrine predominant, in which hypotensive episodes are

characteristic)

- Paroxysmal release of catecholamines:
 - Spontaneously due to changes in blood flow or tumor necrosis
 - Direct pressure on the gland from external forces (trauma, exercise)
 - Precipitation of release (opiates, glucagon, metoclopramide, steroids, foods with tyramine, iodinated contrast media)
 - Augmentation of catecholamine effect (tricyclic antidepressants [TCAs], β -blockers, sympathomimetics)

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Hypertension, moderate to severe, refractory to treatment:
 - 40%: Paroxysms with normal BP between episodes
 - 30%: Sustained hypertension with paroxysms
 - 30%: Sustained hypertension without paroxysms
 - Sometimes normotensive in familial forms and small tumors: < 5%
- Paroxysmal symptoms
 - Sudden onset, gradual resolution
 - Duration: Minutes to hours (average 20 min)
 - Intervals: Hours to months (average weekly)
 - Increasing frequency, duration, and severity with time
- Clinical characteristics of paroxysms
 - Hypertensive crisis or urgency
 - Headache – abrupt, throbbing, bilateral
 - Tachycardia/palpitations
 - Profuse diaphoresis/pallor
 - Apprehension/anxiety/tremulous
 - Shock associated with trauma, surgery, parturition, anesthesia
- Acute crisis
 - Prolonged (> 24hr) severe paroxysm
 - Severe HTN or shock, hyperpyrexia
 - Multiorgan failure/lactic acidosis
 - Pulmonary edema due to cardiomyopathy (dilated, hypertrophic or Takotsubo)
 - Stroke (SAH, PRES, RCVS, embolic)
 - Severe headache/encephalopathy
 - Chest pain (MI/dissection)
 - Acute abdomen
 - Hemorrhagic tumor necrosis

- Mesenteric infarction

- Chronic symptoms
 - Chest pains/palpitations
 - Orthostasis (decreased plasma volume and blunted sympathetic reflexes)
 - Constipation can be severe, leading to ileus or pseudo-obstruction (catecholamines inhibit peristalsis)
 - Weight loss/fevers (increased metabolism)
 - Lethargy, fatigue (catecholamine withdrawal))
 - Polydipsia, polyuria (glucose intolerance)
 - Anxiety, tremors, heat intolerance

Physical-Exam

- Moderate to severe hypertension, often with orthostatic changes
- Tachycardic, diaphoretic, evidence of weight loss, low-grade fever
- Pallor, cold hands and feet (flushing not seen, except rarely after a paroxysm)
- Tremor, anxiety
- Mydriasis, hypertensive retinopathy
- Café au lait spots, neurofibromas, thyroid nodule
- No palpable masses (tumors tend to be small)

ESSENTIAL WORKUP

- Accurate BP determination with orthostatic BPs
- ECG to exclude ischemia or dysrhythmias

DIAGNOSIS TESTS & INTERPRETATION

- Overdiagnosis in > 20% from misinterpretation of borderline biochemical tests and overzealous imaging
- Underdiagnosis is common from failure to consider the diagnosis or ignoring adrenal masses on CT.

Lab

- CBC:
 - Elevated hemoglobin due to diminished plasma volume
 - Elevated WBC from demargination
- Electrolytes, BUN, creatinine, glucose:
 - Lactic acidosis
 - Renal failure secondary to hypertensive nephropathy
 - Hyperglycemia due to impaired response to insulin and effect of catecholamines
 - Hypercalcemia due to excess parathyroid hormone
- Urinalysis: Proteinuria and hematuria
- Plasma-free metanephrine (fractionated):
 - 96% sensitive, 85% specific—best screening test. Normal level excludes

- diagnosis, but many false positives
- Least likely to be interfered by medications or stress and no special prep for venipuncture
- 24 hr urine collection for free catecholamines and metanephrine (total and fractionated):
 - 99.7% combined specificity and 87.5% sensitivity (best test for confirmation)
 - Must include creatinine to verify adequate collection
 - Medications that interfere: Levodopa, methyldopa, monoamine oxidase inhibitors (MAOIs), labetalol, propranolol, radiographic contrast media, sympathomimetics, benzodiazepines, TCAs, caffeine, nicotine

Imaging

- CT sensitive for adrenal masses > 1 cm (IV contrast may pose a slight risk):
 - 5% of incidental adrenal tumors seen on CT are pheos.
- MRI or positron emission tomography more sensitive in identifying adrenal pheos as well as identifying extra-adrenal tumors
- Metaiodobenzylguanidine (radionuclear scintiscan: High specificity for localization, but not sensitive enough to exclude pheo)
- Chest radiograph for pulmonary edema
- CT head for CVA/intracranial bleed

Diagnostic Procedures/Surgery

- Clonidine suppression test if diagnosis uncertain (levels not suppressed if pheo)
- Provocative testing with glucagon is not recommended.
- Fine-needle aspiration is contraindicated.
- Laparoscopic resection is feasible in many cases.

DIFFERENTIAL DIAGNOSIS

- Alcohol withdrawal syndrome
- Autonomic hyperreflexia
- Cerebral vascular accident
- Cocaine or amphetamine intoxication
- Hypertensive crisis
- Migraines/subarachnoid hemorrhage
- Panic attack
- Postural tachycardia syndrome
- Paroxysmal supraventricular tachycardia
- Posterior reversible encephalopathy syndrome
- Serotonin syndrome
- Thyrotoxicosis
- Toxemia



TREATMENT

PRE HOSPITAL

- IV access, oxygen
- Continuous cardiac/BP monitoring
- Nitroglycerin 0.4 mg SL for chest pain and HTN

ED TREATMENT/PROCEDURES

Management of Hypertensive Paroxysm

- Phentolamine: α -blockade:
 - 1 mg IV test dose
 - 2.5–5 mg IV bolus given at 1 mg/min repeat bolus every 5–15 min to BP control. Follow by infusion
 - Infusion starting at 0.1 mg/min titrated up to 1 mg/min
 - Vigorous fluid resuscitation required as vasoconstriction is relieved
 - Traditional approach, but Nicardipine or Nitroprusside drip may be more practical
- β -blockade:
 - Add to α -blockade for further BP control
 - If tachycardia develops during induction of α -blockade
 - NEVER USE ALONE: Institution of β -blockade without prior α -adrenergic blockade may exacerbate hypertension by antagonizing β -mediated vasodilation in smooth muscle.
 - Esmolol: Load 500 μ g/kg over 1 min, followed by 50 μ g/kg/min for 4 min; if adequate therapeutic effect not achieved within 5 min, repeat loading dose and increase infusion to 100 μ g/kg/min; repeat loading dose and titrate infusion rate upward at 50 μ g/kg/min q4–q5min as needed; omit further loading doses once nearing therapeutic target.
 - Labetalol: Begin with 10–20 mg IV; BP falls within 5 min, maximum effect at 10 min; can double IV dose q15–q30min until target reached (α -blockade inadequate to be relied on as a single agent).
 - Metoprolol: 5 mg IV q15min until response
- Resistance to α - and β -blockade or 1st-line option if unfamiliar with Phentolamine:
 - Nitroprusside:
 - Start at 0.5 μ g/kg/min
 - Titrate by 0.5 μ g/kg/min increments
 - Maximum dose 10, average needed 3–4
 - Nicardipine:
 - Start infusion at 5 mg/hr
 - Titrate up by 2.5 mg/hr every 15 min
 - 15 mg/hr maximum dose

- Add β -blockade to vasodilator if needed
- Ventricular tachydysrhythmias:
 - Lidocaine:
 - 50–100 mg bolus
 - Repeat bolus q5min (5 mg/kg max.)
 - Esmolol 50–200 μ g/kg/min infusion

MEDICATION

First Line

- Phenoxybenzamine: Start at 10 mg BID orally, titrate up 10 mg every other day until desired effect (start at least 7 days preop).
- Other α -blockers (1st dose effect):
 - Doxazosin: 1–8 mg/d (start at 1 mg)
 - Terazosin: 1–10 mg/d (start at 1 mg)
- β -blocker added to control reflex tachycardia:
 - Metoprolol or atenolol: 25–100 mg/d

Second Line

- Calcium-channel blockers:
 - Amlodipine, nifedipine, or nifedipine
- Inhibition of catecholamine synthesis:
 - Metyrosine: 250–500 mg q6h

ALERT

The following medications can precipitate hypertensive crisis in pheo:

- β -blockers (if not pretreated with α -blocker)
- Glucagon
- Glucocorticoids
- Iodinated contrast media (ionic)
- Ketamine
- Metoclopramide
- Opiates
- Sympathomimetics, including over-the-counter decongestants

Pregnancy Considerations

- May be confused with toxemia, but proteinuria is usually absent
- MRI is the preferred imaging modality.
- Nitroprusside should not be used for hypertensive crisis, but all other BP medications are acceptable.
- Spontaneous vaginal delivery will likely precipitate hypertensive crisis, such that C-section should be planned.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Suspicion of pheo in an ill or toxic patient with labile swings in BP
- Hypertensive urgency or crisis
- Cardiac arrhythmias
- End organ compromise: Congestive heart failure, myocardial infarction, renal insufficiency, CVA, abdominal pain

Discharge Criteria

Stable patient with mild hypertension.

FOLLOW-UP RECOMMENDATIONS

- Obtain plasma-free metanephrine during a hypertensive episode.
- Consider initiating doxazosin or terazosin or a calcium-channel blocker for BP control.
- Arrange close follow-up

PEARLS AND PITFALLS

- Paroxysms of severe hypertension, headache, intense diaphoresis, and palpitations comprise a tetrad very suggestive of pheo.
- Pallor and sweating, not flushing, is typical of pheo crisis.
- Orthostasis is common in pheo and it is further aggravated by α -blockade, unless volume repletion is not done concomitantly.
- Consider pheo in unexplained shock, multisystem organ failure, cardiomyopathy, new glucose intolerance with weight loss.
- Never administer β -blockers (even labetalol) before α -blockade in patients with pheo
- Plasma-free metanephrine during an attack is very sensitive but not specific in the diagnosis

ADDITIONAL READING

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- Scholten A, Cisco RM, Vriens MR, et al. Pheochromocytoma is not a surgical emergency. *J Clin Endocrinol Metab*. 2013;98(2):581–591.
- Yu R, Nissen NN, Chopra P, et al. Diagnosis and treatment of pheochromocytoma in an academic hospital from 1997 to 2007. *Am J Med*. 2009;122:85–95.



CODES

ICD9

- 194.0 Malignant neoplasm of adrenal gland
- 227.0 Benign neoplasm of adrenal gland

ICD10

- C74.10 Malignant neoplasm of medulla of unspecified adrenal gland
- C74.12 Malignant neoplasm of medulla of left adrenal gland
- D35.00 Benign neoplasm of unspecified adrenal gland

PHIMOSIS

Nicole M. Franks



BASICS

DESCRIPTION

- True phimosis is the pathologic inability to retract the foreskin over the glans of the penis as a result of scarring.
- The inability to retract a normal, supple foreskin is not true phimosis.
- The foreskin is rarely retractable at birth due to normal adhesions between the glans and the inner prepuce.
- ~90% are retractable by 3 yr of age, and 99% are retractable by 17 yr, as the epithelial cells that comprise smegma are shed.
- Parents should be instructed not to forcibly retract the foreskin.

ETIOLOGY

Possible causes of true phimosis include:

- Trauma from forcible retraction of the foreskin
- Repetitive bouts of diaper dermatitis
- Recurrent balanoposthitis
- Poor hygiene
- Poorly performed circumcision
- Congenital anomalies



DIAGNOSIS

SIGNS AND SYMPTOMS

- Dysuria, hematuria
- Poor urinary stream
- Whitish, narrowed preputial opening of the foreskin
- Edema, erythema, and tenderness of prepuce
- *Balanoposthitis* (inflammation of the glans and foreskin)
- Ballooning of foreskin on urination in severe cases

Physical-Exam

Exam should include an evaluation for potential complications:

- Obstruction and vascular compromise of glans
- Occur only in the most extreme cases

ESSENTIAL WORKUP

- In the majority of cases, no workup is necessary.

- In patients with severe stenosis, the complication of an *obstructive uropathy* may occur. This should be investigated by:
 - Evaluation of kidney function:
 - BUN and creatinine
 - Renal sonogram
- Phimosis secondary to recurrent balanoposthitis should prompt a workup for *diabetes mellitus*:
 - Urinalysis, serum glucose, or glycosylated hemoglobin (Hgb A1C)

DIFFERENTIAL DIAGNOSIS

- Preputial adhesions are normal in young children.
- Balanoposthitis without phimosis



TREATMENT

PRE HOSPITAL

- Pre-hospital personnel and family members should be instructed not to attempt retraction of the foreskin prior to medical evaluation.
- Unwarranted attempts may traumatize a normal, nonretractable prepuce or convert the situation to a more emergent *paraphimosis*.

INITIAL STABILIZATION/THERAPY

None required in most cases

ED TREATMENT/PROCEDURES

- Relieve obstructive uropathy, if present, with urethral catheterization or suprapubic aspiration.
- If vascular flow to the glans is compromised, a dorsal slit must be made in the foreskin:
 - Performed after achieving adequate penile block (see Paraphimosis for more detailed description of procedure)
 - This is rarely necessary in phimosis.
- Potent topical steroids for a multiweek course have been reported to successfully reduce phimosis:
 - Betamethasone dipropionate 0.05–0.1%: Apply to preputial orifice twice daily for 4–6 wk.

Pediatric Considerations

For foreskin incision, procedural sedation will likely be needed in place of penile block.

MEDICATION

Pain control as required



FOLLOW-UP

DISPOSITION

Admission Criteria

- Obstructive uropathy
- Severe balanoposthitis with ischemia or necrosis

Discharge Criteria

- Ability to urinate
- Adequate urologic follow-up

Issues for Referral

Urologic follow-up for response to steroid therapy, dilation of the preputial opening, operative repair, or elective circumcision as necessary

FOLLOW-UP RECOMMENDATIONS

Physiologic phimosis requires waiting for age-appropriate development and continued preputial hygiene.

PEARLS AND PITFALLS

- Foreskin is normally nonretractable from the neonatal period to age 3 yr.
- Do not forcibly retract foreskin especially in children 3–17 yr, as phimosis may still be physiologically normal.
- Vascular compromise of the glans penis requires a dorsal slit to the foreskin to prevent necrosis.

ADDITIONAL READING

- Donohoe JM, Burnette JO, Brown JA. Paraphimosis treatment. *eMedicine*. Available at <http://www.emedicine.medscape.com/article/442883>. Updated October 7, 2009.
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- Marx JA, Hockberger RS, Walls RM. *Rosen's Emergency Medicine: Concepts and Clinical Practice*. 7th ed. St. Louis, MO: Mosby; 2009:2201–2202.
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See Also (Topic, Algorithm, Electronic Media Element)

- [Paraphimosis](#)
- [Priapism](#)



CODES

ICD9

605 Redundant prepuce and phimosis

ICD10

[N47.1](#) [Phimosis](#)

PITYRIASIS ROSEA

Benjamin S. Heavrin



BASICS

DESCRIPTION

- A self-limited skin exanthem of unknown origin primarily affecting children and young adults
- Skin findings often begin with an isolated “herald patch,” an ovoid erythematous raised lesion seen along the trunk and extremities
- A secondary eruption usually follows, where multiple smaller exanthems appear along the Langer lines of the trunk and proximal extremities in a symmetric “Christmas tree pattern”
- Nearly 80% of symptoms resolve within 2 mo

ETIOLOGY

- Unknown, although there is weak evidence for a viral etiology such as herpes 6 and 7
- Many medications have been associated with a pityriasis-like reaction:
 - Barbiturates
 - Captopril
 - Clonidine
 - Gold
 - Isotretinoin
 - Metronidazole
 - Bismuth
 - Hepatitis B vaccine
 - Gleevec
 - Interferon
- Eczema, asthma, and underlying malignancies may be weakly associated



DIAGNOSIS

SIGNS AND SYMPTOMS

Prodromal symptoms and characteristic skin findings are discussed below

History

Prodromal symptoms occur in 60–70% of patients:

- Malaise
- GI symptoms
- Respiratory symptoms

Physical-Exam

Dermatologic findings

- Herald patch:
 - Solitary, erythematous, slightly raised papule 2–10 cm in diameter
 - Seen in 50–90% of cases
- Secondary eruption:
 - Widespread salmon-colored, elliptic, finely scaling papules
 - Usually appear symmetrically along Langer lines in a “Christmas tree” pattern
 - Generally follows herald patch by 7–14 days
 - Lesions are concentrated on the trunk and proximal extremities
 - Pruritus is common
- Lesions concentrated on the face and distal extremities with minimal trunk involvement characterize *inverse pityriasis*

Pediatric Considerations

- Inverse pityriasis, lesions on the face and distal extremities characterize *inverse pityriasis* and may be seen more often in pediatric populations
- Rarely, pediatric presentations may have oral lesions, usually punctate hemorrhage and ulceration

ESSENTIAL WORKUP

Exclude other diagnoses, especially when a herald patch is not seen:

- Secondary syphilis can have similar skin findings. Consider RPR in a patient with STI risk factors
- KOH prep may diagnose tinea

DIAGNOSIS TESTS & NTERPRETATION

Lab

None required:

- KOH and RPR if other diagnoses are considered

DIFFERENTIAL DIAGNOSIS

- Herald patch:
 - Nummular eczema
 - Tinea corporis
- Secondary eruption:
 - Secondary syphilis
 - Drug eruption
 - Guttate psoriasis
 - Kaposi sarcoma
 - Lichen planus

- Occult malignancy
- Scabies
- Seborrheic dermatitis
- Tinea versicolor
- Dermatomyositis
- Cutaneous lymphoma
- Lupus



TREATMENT

INITIAL STABILIZATION/THERAPY

None required

ED TREATMENT/PROCEDURES

- Pityriasis is self-limiting
- Pruritus may improve after treatment with steroids, antihistamines, and, interestingly, erythromycin

MEDICATION

- Diphenhydramine: Adult: 25–50 mg PO QID (peds: 5 mg/kg/d div. QID)
- Erythromycin: 400 mg (peds: 10 mg/kg) PO QID
- Hydrocortisone: 1% cream TID
- Prednisone: 15–40 mg (peds 0.25–0.5 mg/kg) daily

First Line

- Diphenhydramine: Adult: 25–50 mg PO QID (peds: 5 mg/kg/d div. QID)
- Hydrocortisone: 1% cream TID

Second Line

- Prednisone: 15–40 mg (peds 0.25–0.5 mg/kg) daily
- Erythromycin: 400 mg (peds: 10 mg/kg) PO QID



FOLLOW-UP

DISPOSITION

Admission Criteria

Pityriasis rosea is a self-limited disease; admission is not required

Discharge Criteria

Patients with a clear diagnosis of pityriasis rosea may be discharged

Issues for Referral

Severe refractory pruritus may require dermatology follow-up

FOLLOW-UP RECOMMENDATIONS

- With primary care provider as needed
- Symptoms usually resolve over 1–2 mo

PEARLS AND PITFALLS

- Pityriasis is usually limited to the proximal extremities and trunk. Consider alternative diagnoses beyond *inverse pityriasis* in a patient with mucous membrane or distal extremity involvement.
- Consider alternative diagnoses in those patients who appear toxic or have atypical presentations.

ADDITIONAL READING

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- Stulberg DL, Wolfrey J. Pityriasis rosea. *Am Fam Physician.* 2004;69:87–91.



CODES

ICD9

696.3 Pityriasis rosea

ICD10

L42 Pityriasis rosea

PLACENTAL ABRUPTION

Rebecah W. Schwartz



BASICS

DESCRIPTION

- Hemorrhage at the decidual–placental interface leading to complete or partial separation of the normally implanted placenta before delivery of the fetus
- Incidence/prevalence:
 - ~ 1% of all pregnancies
 - 30% of bleeding episodes in the 2nd half of pregnancy
 - 15% of all fetal deaths
 - Neonatal death in 10–30% of cases
 - 6% of all maternal mortality
- Synonym(s): Abruptio placentae, accidental hemorrhage (in UK)

ETIOLOGY

- Primary cause unknown
- Vascular injury with dissection of blood into the decidua basalis or mechanical shearing between the placenta and uterus leading to bleeding and clot formation
- More severe cases lead to:
 - Development of disseminated intravascular coagulation (DIC)
 - Maternal–fetal compromise
- Research suggests that the majority of abruptions are due to chronic processes:
 - Inflammatory changes in the placenta
 - Manifestation of ischemic placental disease
- Acute abruption can occur due to:
 - Trauma
 - Rapid uterine decompression
 - Placenta implantation over a uterine anomaly or fibroid
- Multiple known risk factors:
 - Previous abruption (10–20% recurrence risk)
 - Maternal hypertension (> 140/90) and preeclampsia
 - Increased parity and maternal age
 - Multiple gestation
 - Fibroids or other uterine/placental abnormalities
 - Tobacco use
 - Cocaine abuse
 - Trauma
 - Premature rupture of membranes, particularly if associated with chorioamnionitis or oligohydramnios

- Rapid uterine decompression:
 - Polyhydramnios with membrane rupture
 - Rapid delivery of 1st twin
- Elevated 2nd trimester maternal serum α -fetoprotein
- Thrombophilias
- Maternal race:
 - More common among African American and Caucasian women
 - Incidence increasing more rapidly among African American women

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- 20+ wk of pregnancy
- Vaginal bleeding (> 80%, *usually painful*)
- Abdominal or back pain (> 50%)
- Uterine cramps, tenderness, frequent contractions, or tetany
- Nausea, vomiting
- Otherwise unexplained preterm labor
- History of recent trauma should be elicited
- Recent drug use, particularly cocaine or other sympathomimetics
- Prior abruption or other risk factors
- Estimated gestational age
- Prenatal care history

Physical-Exam

- Signs of *hypotensive shock* may be present
- Uterine tenderness frequently present
- Vaginal bleeding (absent in 20–25%)
- Petechiae, bleeding, and other signs of DIC
- Decreased fetal heart tones and movement
- Fetal bradycardia or nonreassuring fetal heart rate tracings

ALERT

- Sterile vaginal exam must be performed with caution to avoid tissue injury, especially if placenta previa suspected:
 - Assess for presence of amniotic fluid (nitrazine paper turns blue; ferning of fluid on glass slide)
 - Evaluate for vaginal or cervical lacerations

ESSENTIAL WORKUP

- Large-bore IV access

- Blood type, Rh, and cross-match
- Rapid hemoglobin determination
- Determine fetal heart tones by Doppler
- Fetal monitoring to detect signs of early fetal distress
- Uterine tocographic monitoring

DIAGNOSIS TESTS & INTERPRETATION

Diagnosis is primarily clinical, supportive tests include

Lab

- Blood type and Rh
- CBC
- PT/PTT
- Fibrinogen levels (normally 450 in latter half of pregnancy) and fibrin split products
- Fibrinogen < 200 mg/dL and platelets < 100,000/ μ L highly suggestive of abruption
- Kleihauer–Betke if mother Rh-negative (significant fetal-to-maternal hemorrhage more likely in traumatic abruption)

Imaging

- US demonstrates evidence of abruption in only 50% of cases (false-negative common)
- MRI sensitive but impractical
- If abdomen/pelvis CT scan done as part of maternal trauma evaluation, evidence of abruption may be visible (must ask the radiologist to evaluate specifically)

DIFFERENTIAL DIAGNOSIS

- Placenta previa
- Bleeding during labor
- Vaginal or cervical lacerations
- Uterine rupture
- Preterm labor
- Ovarian torsion
- Pyelonephritis
- Cholelithiasis/cholecystitis
- Appendicitis
- Other blunt intra-abdominal or pelvic injuries



TREATMENT

PRE HOSPITAL

- Patients with abruption may be in shock and need full resuscitative measures
- Transport in the left lateral recumbent position

INITIAL STABILIZATION/THERAPY

- Airway, breathing, circulation (ABCs), oxygen
- Cardiac monitor
- Placement of large-bore IVs
- IV crystalloid resuscitation

ED TREATMENT/PROCEDURES

- Maternal cardiac and tocographic monitoring
- Continuous fetal monitoring
- Transfuse PRBCs, fresh frozen plasma (FFP), cryoprecipitate, and platelets as indicated (may require massive transfusion protocol)
- Immediate OB/GYN consultation
- Foley catheter for close monitoring of urine output
- Tocolysis is generally contraindicated
- If abruption is suspected in the setting of trauma, maternal stabilization is of primary importance:
 - All indicated radiographs should be performed as needed

MEDICATION

First Line

- Rh-immunoglobulin in Rh-negative women:
 - 300 µg IM in women at ≥ 12 wk gestation
 - Higher doses if indicated by results of Kleihauer–Betke test
- Blood products as indicated

Second Line

Consider with obstetrician recommendation:

- Magnesium sulfate if tocolysis is indicated
- Steroids for fetal lung maturation if gestational age between 24 and 34 wk



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients with placental abruption must be admitted for maternal and fetal monitoring
- Admit to ICU if DIC, amniotic fluid embolism, or significant hemorrhage (known

or suspected)

- Victims of multiple trauma with abruption should be admitted and managed in accordance with trauma protocols
- Transportation to higher trauma or obstetric level of care is appropriate if the patient is stable for transfer or appropriate care unavailable at existing facility

Discharge Criteria

- Trauma patients with no evidence of abruption or other significant injury may be discharged after 4–6 hr of normal maternal and fetal monitoring
- Discharge instructions include pelvic rest, no intercourse, no heavy lifting, no prolonged standing
- Discharge decision should be made in consultation with OB/GYN and include close follow-up

Issues for Referral

All cases of confirmed or suspected abruption require immediate obstetric consultation

PEARLS AND PITFALLS

- Primarily a clinical diagnosis: No single test reliably confirms or rules out placental abruption
- Hypotension typically occurs late in the course of hypovolemic shock in pregnancy
- Anticipate a consumptive coagulopathy and consider the need for blood products early in presentation
- Abruption may be associated with severe preeclampsia, causing a hypovolemic patient to be normotensive:
 - Maintain a high index of suspicion for preeclampsia in patients with severe abruption and no obvious cause

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Placenta Previa
- Trauma in Pregnancy
- Vaginal Bleeding in Pregnancy
- DIC



CODES

ICD9

- 641.20 Premature separation of placenta, unspecified as to episode of care or not applicable
- 641.21 Premature separation of placenta, delivered, with or without mention of antepartum condition
- 641.23 Premature separation of placenta, antepartum condition or complication

ICD10

- O45.90 Premature separation of placenta, unsp, unsp trimester
- O45.91 Premature separation of placenta, unsp, first trimester
- O45.92 Premature separation of placenta, unsp, second trimester

PLACENTA PREVIA

Roneet Lev



BASICS

DESCRIPTION

- Placental tissue overlying or proximate to the internal cervical os
- Uterine enlargement and cervical dilation cause placental vessels near the cervix to tear, resulting in vaginal bleeding
- >90% of placenta previa diagnosed before 20 weeks will migrate and have normal placental location at term
- If placenta covers the internal os by >20 mm, then previa is expected at birth
- Increased amount of placental overlap (>15–23 mm) predicts placenta previa present at birth
- Causes 20% of all antepartum hemorrhage
- Classifications:
 - Complete placenta previa: Cervical os is completely covered by placenta
 - Partial placenta previa: Cervical os is partially covered by placenta
 - Marginal placenta previa: Edge of placenta is at margin of cervical os
 - Low-lying placenta: Placenta edge is within 2 cm to cervical os

ETIOLOGY

- Unknown etiology
- Incidence: 4/1,000 births = 0.4% of pregnancies at term
- Maternal mortality: 0.03%
- Perinatal morbidity and mortality: Triple, due to preterm delivery
- Factors affecting location of implantation:
 - Increased number of curettages from spontaneous or induced abortions
 - Abnormal endometrial vascularization
 - Delayed ovulation
- Risk factors:
 - Multiparity (5% grand multiparous patients vs. 0.2% nulliparous)
 - Multiple gestation
 - Prior C-section (up to 3× increase, increases with number or prior C-sections)
 - Increased maternal age (0.7% age <19 yr, 1% age ≥35 yr)
 - Previous placenta previa (4–8% recurrence)
 - Smoking (2–4 times increase)
 - Male fetus (14% increase)
 - Assisted fertilization
 - Residence at higher altitude

- Asian maternal race
- Unexplained elevated maternal serum alpha fetal protein (MSAFP)
- Associated conditions:
 - Congenital anomalies
 - Abnormal fetal presentation
 - Preterm premature rupture of the membranes
 - Amniotic fluid embolism; associated with pathologies of the placenta
 - Vasa previa: Fetal vessels course through membranes and cover os
 - Placenta accreta, increta, percreta (growth of placenta into uterine wall) occur in 5–10% of patients with placenta previa; sustained bleeding may require C-section hysterectomy

DIAGNOSIS

SIGNS AND SYMPTOMS

Painless vaginal bleeding in pregnancy after 20 wk is placenta previa until proven otherwise

History

- Painless bright red vaginal bleeding in 70%
- Uterine contraction in 20%
- Common incidental finding on US in 2nd trimester (6% at 16–18 wk)
- 1st episode of bleeding typically occurs at 27–32 wk
- Bleeding may range from minor to massive; number of bleeding episodes does not correlate with degree of placenta previa
- Inciting factors—usually no cause; recent intercourse or heavy exercise may contribute
- Initial bleeding is often self-limited and not lethal, but often recurs

Physical-Exam

- Never do a digital exam or instrument probe of the cervix in 2nd-trimester vaginal bleeding until placenta previa is ruled out
- Sterile speculum exam can be safely performed prior to US to identify if blood is from the os, a vaginal lesion, or hemorrhoids
- Blood seen at patient's feet is a sign of heavy bleeding
- Hypotension and tachycardia may indicate hemorrhagic shock
- Fetal heart tones should be monitored along with other vital signs

ESSENTIAL WORKUP

Vaginal ultrasonography is the diagnostic procedure of choice

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC, platelets
- Type and screen; upgrade to cross-match if transfusion is indicated
- Kleihauer–Betke (KB)—detects > 5 mL of fetal cells in maternal circulation (it takes only 0.1 mL to sensitize mother if Rh negative)
- If coagulopathy suspected (rare): Prothrombin time/partial thromboplastin time, fibrin-split products, fibrinogen (< 300 mg/dL is abnormal)
- Rh status

Imaging

- Transabdominal US: 93–98% accurate:
 - _ False negative: Obesity, posterior or lateral placenta, fetal head over cervical os
 - _ False positive: Overdistended bladder
 - _ No sufficient accuracy for placenta previa position, need to obtain transvaginal US if placenta previa is detected or uncertain findings
- Transvaginal US: 100% accurate:
 - _ Vaginal probe does not exacerbate bleeding
- Color flow Doppler US: Used to determine placenta accreta
- MRI: May be useful in evaluating placental abnormalities such as accreta and percreta

DIFFERENTIAL DIAGNOSIS

- Placenta abruption (may occur concurrently)
- Uterine rupture
- Fetal vessel rupture
- Cervical/vaginal trauma
- Cervical/vaginal lesions
- Bleeding disorder
- Spontaneous abortion
- “Bloody show” of labor



TREATMENT

PRE HOSPITAL

- Patient with vaginal bleeding at > 24 wk should be transported to a facility that can handle high risk and premature delivery
- Place patient in left lateral recumbent position if hypotensive in 2nd half of pregnancy
- O₂ and IV as with other patients

INITIAL STABILIZATION/THERAPY

- Resuscitation for hemorrhagic shock as with any source with monitoring of fetus and higher cut off of blood transfusion
- ABCs
- 2 large-bore IVs with normal saline (NS) or lactated Ringer (LR) for resuscitation
- Left lateral recumbent position if hypotensive in 2nd half of pregnancy
- Fluid resuscitation
- Blood transfusion for hematocrit (Hct) < 30 or hypotension not responding to fluids
- Fresh-frozen plasma if coagulopathy
- Fetal monitoring (heart rate < 120 or > 160 bpm is abnormal)
- Immediate OB consultation for symptomatic patients

ED TREATMENT/PROCEDURES

- Emergent OB consultation for patients with active bleeding
- Volume resuscitation with 2 large-bore IVs with NS or LR
- Blood transfusion to keep Hct 30–35%
- RhoGAM if mother is Rh negative
- Fetal monitoring
- Keep NPO and on bed rest until considered stable by OB
- Magnesium sulfate only for contractions of preterm labor when delivery is not recommended
- Antenatal steroids (betamethasone) at 24–34 wk to stimulate prenatal lung maturity
- Emergency C-section or delivery for continued bleeding or fetal compromise

MEDICATION

- RhoGAM: 1 vial (300 µg) IM if not already given at 28 wk; may need > 1 vial if KB indicates > 15 mL of fetal RBS
- Magnesium sulfate: 6 g IV over 20 min, then 2–4 g/h; adjust to contractions
- Betamethasone: 12 mg IM q24h × 2 doses



FOLLOW-UP

DISPOSITION

Admission Criteria

- Active bleeding placental previa is a potential obstetric emergency, and all patients should be admitted
- Select patients may be managed on outpatient basis if bleeding is resolved. In consultation with OB

Discharge Criteria

- Asymptomatic patients
 - Bed rest is not necessary. Avoid strenuous physical activity. Report bleeding or contractions
 - <20 wk and placenta not over the os: No special follow up necessary
 - <20 wk and placenta 0–20 mm: Repeat US at 28 wk
 - Placenta >20 mm over os is unlikely to resolve. C-section at 36–37 wk
 - Pelvic rest (no intercourse or tampons in vagina) if placenta previa found after 28 wk or at any time if associated with bleeding
- 70% of patients will have a 2nd episode of bleeding

FOLLOW-UP RECOMMENDATIONS

Patients with incidental finding of placenta previa found at <20 wk will need outpatient US to determine migration of placenta

PEARLS AND PITFALLS

- Do not perform digital vaginal exam if suspect vaginal bleeding after 2nd trimester. Do US first
- Sterile speculum exam and transvaginal US are safe and do not increase bleeding
- Painless vaginal bleeding after 20 wk is placenta previa until proven otherwise
- Painful vaginal bleeding after 20 wk is placental abruption until proven otherwise
- The 2 above conditions can occur simultaneously

ADDITIONAL READING

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- Marx JA, Hockberger RS, Walls RM, et al. *Rosen's Emergency Medicine: Concepts and Clinical Practice*. 7th ed. St. Louis, MO: Mosby; 2009.
- Scott JR, Gibbs RS, Karlan BY, et al. *Danforth's Obstetrics and Gynecology*. 10th ed. Philadelphia, PA: Lippincot Williams & Wilkins; 2008.



See Also (Topic, Algorithm, Electronic Media Element)

Placental Abruption



CODES

ICD9

- 641.00 Placenta previa without hemorrhage, unspecified as to episode of care or not applicable
- 641.01 Placenta previa without hemorrhage, delivered, with or without mention of antepartum condition
- 641.10 Hemorrhage from placenta previa, unspecified as to episode of care or not applicable

ICD10

- O44.00 Placenta previa specified as w/o hemorrhage, unsp trimester
- O44.03 Placenta previa specified as w/o hemorrhage, third trimester
- O44.10 Placenta previa with hemorrhage, unspecified trimester

PLANT POISONING

Patrick M. Lank

BASICS

DESCRIPTION

- Plant exposure is 1 of the most common reasons to contact the poison center
- Majority of cases involve unintentional ingestion in children <6 yr old.

ETIOLOGY

Identification of ingested plant species should be attempted whenever possible.

Plants with Anticholinergic Properties

- Genera include *Atropa*, *Datura*, *Hyoscyamus*, *Solandra*, and *Solanum*.
- Common names of anticholinergic plants include Jimson weed, deadly nightshade, henbane, and angel trumpet.
- Competitive antagonists of acetylcholine at the muscarinic acetylcholine receptor

Plants with Cardiac Glycosides

- Genera include *Digitalis*, *Nerium*, *Thevetia*, and *Helleborus*.
- Common names of cardiac glycoside plants include foxglove, oleander, yellow oleander, lily of the valley, and hellebore/Christmas rose
- Inhibit Na^+/K^+ -ATPase:
 - See separate chapter on Digoxin Poisoning

Plants with Nicotine-like Alkaloids

- Genera include *Nicotiana*, *Caulophyllum*, and *Conium*
- Common names of plants containing nicotine include tobacco, blue cohosh, and poison hemlock.
- Direct-acting agonists at the nicotinic acetylcholine receptor

Plants with Cyanogenic Compounds

- Genera include *Hydrangea*, *Malus*, *Prunus*, *Sambucus*
- Common names of plants containing cyanogenic compounds (in some part of the plant) include apricot, cherry, peach, plum, apple, cassava, and elderberry
- Metabolized to cyanide, which interferes with electron transport chain and leads to cellular poisoning

Plants with Calcium Oxalate Crystals

- Genera include *Alocasia*, *Dieffenbachia*, and *Philodendron* as well as many others
- Common names for calcium oxalate-containing plants include elephant's ear,

dumb cane, and mother-in-law's tongue

- Clinical manifestations occur after release of intracellular calcium oxalate crystals:
 - Local tissue exposure to calcium oxalate crystals leads to inflammatory response.

Plants with Pyrrolizidine Alkaloids

- Genera include Crotalaria, Heliotropium, Senecio, and Sesbania
- Common names of plants with pyrrolizidine alkaloids include scorpion's tail, ragwort, groundsel, and rattlebox
- Metabolized to highly reactive pyrroles, which are directly hepatotoxic acutely and lead to hepatic vascular proliferation and veno-occlusive disease chronically.

Plants with Sodium Channel Activity

- Genera include Aconitum, Delphinium, Leucothoe, Lyonia, Pernettya, Pieris, Rhododendron, Schoenocaulon, Veratrum, and Zigadenus
- Common names of plants with aconitine, veratrum alkaloids, and zygacine, all sodium channel openers, include: Aconite (monkshood, wolfsbane, delphinium), veratrum (false hellebore), and zygacine (death camas)
- Common names of plants with grayanotoxins, which have variable effects on sodium channels, include sweet bells, rhododendron, azalea, and lily-of-the-valley bush
- Variable sodium channel effects depending on toxin, although most lead to prolonged sodium channel influx.

Plants with Toxalbumins

- Genera include Abrus, Jatropha, Phoradendron, Ricinus, Robinia, Wisteria
- Common names of plants with toxalbumins include rosary pea, mistletoe, and castor bean
- Cause direct cellular toxicity by interfering with ribosomal function

DIAGNOSIS

SIGNS AND SYMPTOMS

Anticholinergic

- Dry, warm, and flushed skin
- Absent bowel sounds
- Urinary retention
- Agitated delirium

Cardioactive Steroids

- Digoxin-like toxicity

- Abdominal pain, nausea, vomiting
- Multiple cardiac effects, ranging from junctional bradycardia to v-tach/v-fib
- \pm hyperkalemia

Nicotine-like Alkaloids

- Hypertension
- Tachycardia \rightarrow bradycardia (late)
- Diaphoresis
- Salivation
- Vomiting
- Fasciculations
- Muscle weakness

Cyanogenic Compounds

- Potentially delayed (unlike cyanide gas)
- Initial symptoms:
 - Abdominal pain, nausea, vomiting
 - Lethargy
 - Diaphoresis
- Followed by:
 - Altered mental status
 - Lactic acidosis
 - Seizures
 - Cardiovascular collapse
 - Multiorgan system failure

Calcium Oxalate Crystals

- Oropharyngeal burning pain and swelling (after biting or chewing)
- Ocular exposure results in keratoconjunctivitis
- Dermal irritation after dermal contact

Pyrrolizidine Alkaloids

- Acutely causes hepatitis
- Chronic exposure may lead to hepatic veno-occlusive disease

Sodium Channel Activators

- Abdominal pain, nausea, vomiting
- CNS: Progress from paresthesias to depressed mental status, respiratory depression, coma, and seizures.
- CV: Bradycardia with atrioventricular blocks, progressing to tachydysrhythmias

Toxalbumins

- Effects depend on plant (e.g., mistletoe is rarely toxic, whereas ricin can be deadly) and route of exposure
- Gastroenteritis, diarrhea, or abdominal pain if ingested
- Localized pulmonary effects if inhaled
- Diffuse organ dysfunction if injected

Pediatric Considerations

- Often present with lip, tongue, and oropharyngeal irritation and swelling from oxalate crystal-containing plants:
 - Potential for airway compromise
- Usually consume leaves, seeds, and berries
- Nicotine group: 1 or 2 cigarettes potentially lethal
- Jimson weed:
 - Seeds highly concentrated
 - 100 seeds equal 6 mg of atropine
 - Lethal at 4–5 g of leaf
- Yellow oleander:
 - 2 leaves lethal in 12.5 kg child

ESSENTIAL WORKUP

- Identification of ingested material
- Workup depends on plant ingested

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Electrolytes, BUN, creatinine, glucose, liver function tests
- ABG/VBG for blood pH determination
- Digoxin level for cardiac glycoside plants
- Lactic acid for cyanogenic plants

Imaging

- ECG
- CXR

DIFFERENTIAL DIAGNOSIS

- Altered mental status:
 - Drug use/alcohol
 - Seizures
 - Trauma
 - Cerebrovascular accident
 - Hypoglycemia
- Digoxin toxicity

- Gastroenteritis
- Agents causing metabolic acidosis (see Acidosis chapter)
- Cardiotoxic drugs



TREATMENT

PRE HOSPITAL

- Examples of common nontoxic houseplants:
 - African violet
 - Bird of paradise
 - Ficus
 - Gardenia
 - Grape ivy
 - Orchids
 - Poinsettia (despite reputation)
 - Rubber tree
 - Spider plant
 - Wandering Jew
 - Wax plant
 - Zebra plant
- Collect seeds, leaves, spores in paper bag.
- Contact local botanist.
- Syrup of ipecac is not recommended

INITIAL STABILIZATION/THERAPY

- Airway, breathing, and circulation management (ABCs)
- 0.9% normal saline IV:
 - Aggressive volume replacement for dehydration/hypotension
 - Initiate pressors for hypotension unresponsive to fluids
- Cardiac monitoring
- Supportive care for most ingestions

ED TREATMENT/PROCEDURES

- Supportive care
- Rare plants necessitate focused/antidotal therapy

Anticholinergic

- Benzodiazepines for agitation
- Consider physostigmine for severe agitated delirium

Cardioactive Steroids

Digoxin-specific Fab indicated in:

- Significant bradycardia
- Tachydysrhythmia
- Hyperkalemia

Nicotine-like Alkaloids

- Parenteral short-acting antihypertensives such as nitroprusside for hypertensive crisis
- Treat seizures with benzodiazepines.

Cyanogenic Compounds

- Correction of electrolyte abnormalities
- Hydroxocobalamin or prepackaged cyanide antidote kit if severe lactic acidosis or hemodynamic compromise

Calcium Oxalate Crystals

- For mild symptoms, popsicles may decrease burning
- Viscous lidocaine and analgesics for more severe oral exposure
- Copious irrigation for ocular, oropharyngeal, and dermal exposure

Pyrrolizidine Alkaloids

- Supportive care
- Removal from source. Pyrrolizidine alkaloids have been found in herbal medication products as well as food contaminants
- Liver-specific management in conjunction with hepatologist

Sodium Channel Activators

- Atropine for bradycardia and atrioventricular blocks
- Normal saline bolus for hypotension, or vasopressor therapy if normal saline fails

Toxalbumins

Supportive care based on clinical symptoms: Replace GI losses with intravenous fluids, replete electrolytes.

MEDICATION

- Atropine: 0.5 mg (peds: 0.02 mg/kg) IV, repeat 0.5–1 mg IV
- Cyanide antidote kit:
 - Inhale amyl nitrite ampule for 30 sec every minute until sodium nitrite given.
 - Sodium nitrite: 10 mL of 3% solution or 300 mg IV over 3–5 min (peds: 0.15–0.33 mL/kg):
 - Monitor methemoglobin levels to keep < 30%.
 - Sodium thiosulfate: 50 mL IV of 25% solution or 12.5 g (peds: 1.65 mL/kg)

- Digoxin Fab fragments: Empiric dose 5–10 vials
- Hydroxocobalamin: 5 g IV for adults; 70 mg/kg not to exceed 5 g IV for pediatrics
- Magnesium: 2–4 g IV
- Physostigmine: 0.5–2 mg IV
- Sodium bicarbonate 8.4%: 1 amp IV push until narrowing of QRS complex



FOLLOW-UP

DISPOSITION

Admission Criteria

- Dysrhythmias for cardiac monitoring
- Intractable vomiting
- Refractory hypotension
- Evidence of end-organ damage (e.g., hepatic dysfunction, acidosis) or concern for potential for end-organ damage
- Altered mental status

Discharge Criteria

- Baseline mental status
- Tolerating oral fluids
- Normal cardiac activity
- Delayed sequelae not anticipated

Pediatric Considerations

Lower threshold to admit children:

- Tend to eat more concentrated parts of plants
- Lower lethal dose
- Symptoms less specific

FOLLOW-UP RECOMMENDATIONS

Follow-up with medical toxicologist or primary care physician

PEARLS AND PITFALLS

- Death from unintentional plant exposures is rare.
- Intentional exposures from herbal remedies, attempted abuse or therapeutic misadventures can be deadly.
- Contact your regional poison center if concerned about a patient ingesting a potentially poisonous plant: 1-800-222-1222

A special thanks to Dr. Harry Karydes, who contributed to the previous edition.

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See Also (Topic, Algorithm, Electronic Media Element)

- Acidosis
- Cyanide Poisoning
- Digoxin Poisoning



CODES

ICD9
988.2 Toxic effect of berries and other plants eaten as food

ICD10

- T62.1X1A Toxic effect of ingested berries, accidental, init
- T62.2X1A Toxic effect of ingested (parts of) plant(s), acc, init

PLEURAL EFFUSION

Sierra Beck • Steven M. Lindsey



BASICS

DESCRIPTION

- Normal conditions:
 - Pleural space contains 0.1–0.2 mL/kg (30 mL in an adult) of clear, low-protein fluid that facilitates movement of the pulmonary parenchyma within the thoracic space.
 - Fluid formation and reabsorption are governed by hydrostatic and oncotic forces.
 - Normally, the sum of these forces results in movement of fluid into the pleural space from the parietal surface and reabsorption at the visceral surface.
 - Lymphatics help remove any excess fluid.
- Alteration of any of the above factors results in abnormal fluid accumulation.
- Classification:
 - Transudative effusion:
 - An ultrafiltrate of serum, containing low protein and cells
 - Results from increase in hydrostatic pressure and/or decrease in oncotic pressure
 - Pleural surface is not involved in the primary pathologic process.
 - Exudative effusion:
 - Contains high protein and cells
 - Results from pathologic disease of the pleural surface leading to membrane permeability and/or disruption of lymphatic reabsorption

ETIOLOGY

- Transudative effusions:
 - Congestive heart failure (CHF)
 - Peritoneal dialysis
 - Cirrhosis with ascites
 - Pulmonary embolism
 - Acute atelectasis
 - Nephrotic syndrome
 - Myxedema
 - Hypoproteinemia
 - Superior vena cava syndrome
 - Meigs syndrome:
 - Triad of ascites, benign ovarian tumor, and pleural effusion

- Exudative effusions:
 - Pulmonary or pleural infection:
 - Bacterial, viral, fungal, tuberculosis (TB), parasitic
 - Primary lung cancer
 - Mesothelioma
 - Metastasis (often from breast cancer, ovarian cancer, or lymphoma)
 - Pericarditis
 - Pulmonary embolism
- Intra-abdominal disorders:
 - Pancreatitis, hepatitis, cholecystitis
 - Subdiaphragmatic abscess
 - Esophageal rupture
 - Peritonitis
 - Meigs syndrome
- Rheumatologic disease:
 - Systemic lupus erythematosus
 - Rheumatoid arthritis
 - Sarcoidosis
- Trauma:
 - Hemothorax
 - Chylothorax
- Drugs:
 - Drug-induced lupus
 - Nitrofurantoin, methysergide, dantrolene, amiodarone, bromocriptine
 - Crack cocaine

DIAGNOSIS

SIGNS AND SYMPTOMS

- Small effusions are often asymptomatic.
- Dyspnea, pleuritic chest pain, and/or cough
- Tachypnea, hypoxia, decreased breath sounds, and/or dullness to percussion

History

- Underlying primary pathologic process (CHF, pneumonia, pulmonary embolus, pancreatitis) is often the source of complaints.
- Dyspnea on exertion or at rest
- Cough with large effusion
- Pleuritic chest pain with inflammation of pleura
- Empyema: Fever, fatigue, weight loss

Physical-Exam

- Decreased breath sounds
- Decreased tactile fremitus
- Increased egophony for large effusions
- Dullness to chest percussion
- Pleural friction rub
- Examine for the primary cause of pleural effusion.

ESSENTIAL WORKUP

- Cardiac monitor and pulse oximetry
- CBC, comprehensive metabolic panel, coagulation panel
- Chest radiography
- Search for underlying cause

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC
- Electrolytes, BUN/creatinine, glucose, serum lactate dehydrogenase (LDH), serum protein
- Pulse oximetry or arterial blood gas
- Coagulation panel
- Pleural fluid analysis to determine if transudative or exudative effusion:
 - _ Check pleural protein and LDH levels.
 - _ Light criteria: Fluid is likely exudative if 1 or more of the following criteria are met:
 - Pleural fluid protein/serum protein > 0.5
 - Pleural fluid LDH/serum LDH > 0.6
 - Pleural fluid LDH > 2/3 upper limit of normal serum LDH
- If effusion is transudative, no further fluid analysis is usually necessary.
- Determining etiology of exudative effusion:
 - _ Initial testing: Cell count with differential, Gram stain and culture, acid fast bacilli stain, pH, glucose, and cytology
 - _ Based on clinical scenario consider: Triglycerides, amylase, albumin, creatinine, adenosine deaminase, and tumor markers.
 - _ RBC and Hct:
 - 5,000–100,000/mm³ nonspecific
 - > 100,000/mm³ suggestive of malignancy, trauma, or pulmonary embolus
 - Pleural fluid Hct > 0.5 serum Hct is by definition a hemothorax.
 - Other causes: Malignancy, TB, aortic rupture
 - Heparinize and chill hemorrhagic samples to be sent for cytology.
 - _ WBC:

- 1,000–10,000/mm³ nonspecific
- >10,000/mm³ suggestive of parapneumonic effusion, empyema, pancreatitis, rheumatologic, malignancy, or TB
- Glucose:
 - Glucose < 60 mg/dL suggestive of complicated parapneumonic effusion/empyema, malignancy, esophageal rupture, or rheumatologic disease
- Triglyceride:
 - Triglycerides > 100 mg/dL suggestive of chylothorax, disruption of thoracic duct
- Amylase:
 - Amylase > 200 IU/L suggestive of pancreatitis, esophageal rupture, malignancy, TB, or empyema
- pH:
 - Send in a chilled heparinized arterial blood gas syringe.
 - pH < 7 suggests complicated parapneumonic effusion or empyema
- Cytology identifies malignant cells.

Imaging

- Chest radiograph:
 - Upright chest film:
 - Blunting of the costophrenic angle
 - Requires at least 200–250 mL of fluid
 - Presence of subpulmonic effusions may be indicated by loss of supradiaphragmatic vascular markings or an increased space between the gastric bubble and pulmonary parenchyma.
 - Lateral decubitus film:
 - Can identify as little as 5–10 mL of fluid.
 - Suspect a loculated effusion or alternative diagnosis if effusion fails to layer.
- US:
 - Has similar sensitivity to lateral decubitus film and can detect as little as 5–10 mL of fluid.
 - Can differentiate simple effusions from loculated fluid collections.
 - Improves patient safety and decreases risk of pneumothorax for thoracentesis
- CT chest with IV contrast:
 - Most sensitive study for detecting pleural fluid collections and identifying loculated effusions.
 - Useful for determination of underlying lung process such as masses and pleural thickening

ALERT

- Consider pulmonary embolism as a cause of unexplained pleural effusion
- Obtain lateral decubitus films, or bedside US prior to performing thoracentesis to avoid misdiagnosis and procedural complications.

Diagnostic Procedures/Surgery

Diagnostic/therapeutic ED thoracentesis:

- **Indication:**
 - _ Diagnose new effusion in a toxic patient.
 - _ Relieve symptomatic dyspnea caused by large effusions.
 - _ Diagnostic thoracentesis in a stable patient can be deferred until after the patient has been admitted.
- No absolute contraindications.
- Relative contraindications:
 - _ Platelets $< 50,000/\text{mm}^3$
 - _ Prothrombin and partial thromboplastin time $> 2 \times$ normal level
 - _ Serum creatinine > 6
- Correct coagulopathy if present.
- Position patient upright with arms crossed in front to elevate scapula.
- Identify superior border of effusion with US, percussion, or egophony.
- Mark area 1 interspace below this in the posterior axillary line or the midscapular line.
- Prepare area with Betadine, dry, and drape for sterile field.
- Anesthetize with 2% lidocaine.
- Attach 3-way stopcock between needle and syringe. Enter superior border of rib with needle bevel down, aspirating while advancing.
- Use 20G needle for diagnostic aspiration.
- Use 16G–18G needle/catheter (commercial kit) for therapeutic aspiration.
- Advance catheter once pleural space entered.
- Minimum of 100 cc required for basic studies (protein, LDH, cell count, Gram stain and culture)—more for cytology/additional studies.
- Avoid withdrawing $> 1,500$ cc to prevent re-expansion pulmonary edema.
- Intraprocedural chest pain may indicate trapped lung or pneumothorax; stop procedure and obtain chest radiograph.
- After obtaining fluid, withdraw needle, apply pressure, dress, and obtain post procedural chest radiograph for pneumothorax.
- Indications for tube thoracostomy:
 - _ Loculated effusion
 - _ Aspiration of pus
 - _ Complicated parapneumonic effusion with pH < 7 , or pleural glucose < 60 mg/dL, or positive pleural Gram stain or culture
 - _ Hemothorax

DIFFERENTIAL DIAGNOSIS

- Intraparenchymal densities:
 - Lobar collapse
 - Mass, tumor, infiltrative disease
 - Pneumonia
- Pleural densities:
 - Pleural scarring
 - Mesothelioma, metastatic disease
- Other:
 - Herniated abdominal contents
 - Paralyzed diaphragm



TREATMENT

PRE HOSPITAL

IV access, high-flow oxygen, cardiac monitor, and pulse oximeter.

INITIAL STABILIZATION/THERAPY

- ABCs
- High-flow oxygen for shortness of breath
- Emergent thoracentesis for significant respiratory compromise.

ED TREATMENT/PROCEDURES

- Identify and treat underlying pathologic process
- Surgical consult for tube thoracostomy if empyema found.
- Consult interventional radiology or pulmonology for loculated effusions.

MEDICATION

- CHF: Diuresis
- Parapneumonic effusion: Antibiotics
- Pulmonary embolism: Anticoagulation:
 - Bloody effusion is not a contraindication to anticoagulation.
- Rheumatologic disease: NSAIDs and steroids
- Loculated effusion: Injection of streptokinase or urokinase into pleural space by thoracic surgeon or pulmonologist



FOLLOW-UP

DISPOSITION

Admission Criteria

- Respiratory compromise

- Unknown cause of the effusion
- Primary process requires hospitalization
- Presence or suspected parapneumonic effusion or empyema
- Observation for 6 hr or admission for potential complications of thoracentesis:
 - Pneumothorax
 - Re-expansion pulmonary edema
- ICU admission for severe hemodynamic and respiratory compromise

Discharge Criteria

- Source of the pleural effusion is known.
- No evidence of respiratory compromise exists.
- Majority of effusions will resolve if the primary process is treated appropriately.
- Patient must be reliable and have access to a telephone, a supportive social environment, and adequate follow-up.

Issues for Referral

Arrange appropriate follow-up with oncologist or pulmonologist prior to discharge.

FOLLOW-UP RECOMMENDATIONS

Patients should be instructed to return to the ED for worsening dyspnea, fever/chills, or other symptoms of respiratory distress.

PEARLS AND PITFALLS

- The most common causes of pleural effusion are CHF, pneumonia, and malignancy.
- Identify and treat the underlying cause of the pleural effusion.
- Bedside US can help characterize the effusion and reduce the risk of pneumothorax with thoracentesis.
- Failure to identify fatal causes of pleural effusion such as pulmonary embolism, esophageal rupture, or hemothorax
- Failure to drain large effusions that are causing respiratory or circulatory compromise

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Congestive Heart Failure
- Hemothorax
- Pancreatitis
- Pneumonia, Adult
- Pneumonia, Pediatric
- Pulmonary Embolism
- Systemic Lupus Erythematosus
- Tube Thoracostomy

Acknowledgment

The authors gratefully acknowledge the contributions of Scott Murray, Edward Ullman, and Jeremy Chou for their previous editions of this chapter.



CODES

ICD9

- 511.1 Pleurisy with effusion, with mention of a bacterial cause other than tuberculosis
- 511.9 Unspecified pleural effusion
- 511.89 Other specified forms of effusion, except tuberculous

ICD10

- J90 Pleural effusion, not elsewhere classified
- J91.0 Malignant pleural effusion
- J94.0 Chylous effusion

PNEUMOCYSTIS PNEUMONIA

Alan M. Kumar

BASICS

DESCRIPTION

- Originally called *Pneumocystis carinii* pneumonia, then renamed *Pneumocystis jirovecii* but still referred to as PCP
- Most common opportunistic infection in patients with HIV, even with PCP prophylaxis and antiretroviral therapy
- Believed to be transmitted by respiratory-aerosol route:
 - Cysts colonize respiratory tract.
 - Cysts rupture and multiple trophozoites release and form foamy exudate in alveoli.
- Most cases are believed to represent reactivation of latent disease, although person-to-person transmission suggested.
- Actual mode of transmission is unclear.

ETIOLOGY

- *Pneumocystis* is classified as a fungus.
- *Pneumocystis* occurs in hosts with altered cellular immunity:
 - HIV infection (most common, especially when CD4 count < 200 cells/mm³)
 - Cancer
 - Corticosteroid treatment
 - Organ transplantation
 - Malnutrition

Pediatric Considerations

PCP in children is typically more severe.

DIAGNOSIS

SIGNS AND SYMPTOMS

- Subacute presentation
- Up to 7% of patients can be asymptomatic.
- Patients on inhaled pentamidine prophylaxis may have milder symptoms:
 - Increased incidence of pneumothorax
 - Increased incidence of extrapulmonary disease

History

- Fever

- Cough with none or minimal amount of white sputum
- Dyspnea on exertion or at rest:
 - Progressive over days (most common in non-HIV-immunocompromised hosts)
 - Indolent, developing over weeks to months (more common in HIV-positive hosts)
 - Oxygen desaturation with exercise
- Chills
- Fatigue
- Weight loss
- Chest pain

Physical-Exam

- Tachypnea
- Tachycardia
- Crackles and rhonchi on lung exam

ESSENTIAL WORKUP

- CBC
- Electrolytes
- Arterial blood gas (ABG)
- Lactate dehydrogenase (LDH)
- Blood cultures
- Chest x-ray

DIAGNOSIS TESTS & NTERPRETATION

Lab

- ABG:
 - Obtain in all cases of PCP.
 - Calculate the alveolar-arterial (A-a) gradient (usually increased).
 - Adjunctive corticosteroid therapy for A-a gradient > 35 mm Hg or $\text{PaO}_2 < 70$ mm Hg
- LDH:
 - Elevated in HIV-positive patients with PCP compared to non-PCP pneumonia
 - Higher levels correlate with poorer prognosis.

Imaging

- Chest radiograph:
 - Classically reveals bilateral interstitial or central alveolar infiltrates
 - Radiograph normal in up to 25% of patients with PCP

- Early or mild infection associated with decreased sensitivity
- Atypical presentations include:
 - Lobar infiltrates
 - Cysts
 - Pneumothoraces
 - Pleural effusions
 - Nodular infiltrates
- Prophylaxis with aerosolized pentamidine is a risk factor for developing predominantly upper lobe.
- Chest radiograph abnormalities can persist for months after treatment.
- High-resolution chest CT:
 - High sensitivity for PCP in HIV-positive patients.
 - Reveals patchy ground-glass attenuation

Diagnostic Procedures/Surgery

- Induced sputum:
 - Definitive diagnosis requires presence of *Pneumocystis* organisms in an appropriately stained respiratory specimen.
 - Specificity approaches 100%, but sensitivity depends on quality of induced sputum and lab expertise.
 - Less sensitive in patients on inhaled pentamidine prophylaxis and non-HIV-positive patients
- Bronchoalveolar lavage:
 - Perform if the induced sputum is nondiagnostic and the suspicion for PCP is still high.
 - Sensitivity 80–100%

DIFFERENTIAL DIAGNOSIS

Constellation of dyspnea, fever, diffuse radiographic infiltrates, minimal or nonproductive cough, and slow progressive course suggests atypical cause of the pneumonia:

- *Chlamydia pneumoniae*
- *Legionella*
- *Mycoplasma*
- Tuberculosis
- Viral pneumonia (especially cytomegalovirus)



TREATMENT

PRE HOSPITAL

Provide supplemental oxygen for symptomatic patients.

INITIAL STABILIZATION/THERAPY

- ABCs
- Provide adequate oxygenation with nasal cannula up to 100% nonrebreather.
- Perform endotracheal intubation in those with refractory hypoxemia despite maximal oxygenation or hypercarbic respiratory failure.
- At least 500–1,000 cc 0.9% normal saline IV bolus for hypotension, sepsis, dehydration

ED TREATMENT/PROCEDURES

- Initiate antibiotics:
 - _ IV Bactrim is the first-line agent.
 - _ IV pentamidine for those who cannot tolerate Bactrim
 - _ Oral therapy is an option for well-appearing patients.
 - _ Alternative regimens include trimethoprim–dapson, clindamycin–primaquine, and atovaquone.
 - _ Continue antibiotics for 21 days.
- Adjunctive corticosteroids in patients with A–a gradient > 35 mm Hg or $\text{PaO}_2 < 70$ mm Hg:
 - _ Must start within 1st 72 hr of treatment
- Isolate suspected PCP patients from others who are immunocompromised.

MEDICATION

- Atovaquone: 750 mg (peds: Dosing not established) PO q12h
- Clindamycin/primaquine: Clindamycin 900 mg (peds: Dosing not established) IV q8h or 300–450 mg PO q6h and primaquine 15–30 mg (peds: Dosing not established) PO per day
- Pentamidine: 4 mg/kg/24h IV over 1 hr (peds: 3–4 mg/kg IM or IV once/day for 21 days)
- Prednisone: 40 mg (peds: Dosing not established) PO q12h for 5 days, 40 mg PO per day for 5 days, then 20 mg PO per day for 11 days (IV methylprednisolone at 75% of the prednisone dose may be substituted)
- Trimethoprim/dapsone: Trimethoprim 15–20 mg/kg/d IV div. q8h + dapsone 100 mg PO per day (peds: Dosing not established)
- Trimethoprim/sulfamethoxazole (Bactrim): Trimethoprim 15–20 mg/kg/d IV div. q6h and sulfamethoxazole 100 mg/kg/d IV div. q6h (peds: Dosing same)

Pediatric Considerations

- Treatment of choice is IV trimethoprim/sulfamethoxazole, followed by IV pentamidine.
- Dosing for alternative medications not yet established (consult pediatric infectious disease specialist).



FOLLOW-UP

DISPOSITION

Admission Criteria

- Moderate to severe disease ($\text{PaO}_2 < 70$ mm Hg or A–a gradient > 35 mm Hg)
- Inability to digest medications
- Inability to return for careful follow-up

Discharge Criteria

- Nontoxic clinical appearance
- Mild disease state (no hypoxemia or A–a gradient)
- Ability to tolerate medications
- Close follow-up arranged
- If results of induced sputum are not available, add macrolide to empirical regimen.

FOLLOW-UP RECOMMENDATIONS

Close follow-up must be arranged with infectious disease specialist to allow for outpatient management.

PEARLS AND PITFALLS

- Include PCP in differential diagnosis in any patient presenting with shortness of breath who is immunocompromised or is suspected of having undiagnosed HIV.
- Patients considered for PCP are also more likely to have TB or atypical bacterial pneumonia.
- Well-appearing patients with low oxygen saturations are at higher risk for complications.

ADDITIONAL READING

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- Huang L, Quartin A, Jones D, et al. Intensive care of patients with HIV infection. *N Engl J Med*. 2006;355:173–181.
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See Also (Topic, Algorithm, Electronic Media Element)

- HIV/AIDS
- Pneumonia, Adult
- Pneumonia, Pediatric

- Tuberculosis



CODES

ICD9

136.3 Pneumocystosis

ICD10

B59 Pneumocystosis

PNEUMOMEDIASTINUM

Matthew D. Bitner



BASICS

DESCRIPTION

- Presence of free air or gas within the mediastinum (mediastinal emphysema)
- May originate from esophagus, lungs, or bronchial tree (*aerodigestive process*)
- May occur spontaneously (primary pneumomediastinum) or as result of trauma, surgery, or other pathologic processes (secondary pneumomediastinum)
- Spontaneous pneumomediastinum:
 - Caused by extrapleural tracheobronchial injury:
 - Increased intra-alveolar pressure, low perivascular pressures, or both
 - Terminal alveolar rupture into the lung interstitium and bronchovascular tissue sheath
 - Dissection of air into the hilum and subsequently the mediastinum along a pressure gradient
 - Mediastinal air then dissects into the fascial planes, most commonly into the tissues of the neck.
 - Often in setting of a Valsalva maneuver, forceful vomiting, in association with bronchospasm or inhalational drug use
 - Men > women (2:1 in some series)
 - Young > old (most common in 2nd/3rd decades of life in most series)
 - Pediatric patients have a bimodal age distribution of peak incidence (<7 and 13–17 yr)
- Relatively rare, 1/30,000–50,000 hospital admissions

ETIOLOGY

- Primary or spontaneous pneumomediastinum:
 - Associated with forced Valsalva maneuvers:
 - Forceful vomiting
 - Forceful straining during exercise
 - Straining during defecation
 - Coughing/sneezing
 - Intense screaming
 - Labor and delivery
 - Playing wind instruments
 - Pulmonary function testing
 - Anorexia nervosa
 - Obesity
 - Pre-existing lung disorders (interstitial lung disease, pulmonary

- fibrosis, pneumonitis)
 - Illicit inhalation drug use (marijuana, cocaine, methamphetamine)
 - Tobacco abuse
- A majority of cases will have no identified precipitating event/cause
- Has been rarely described after dental extraction/procedures.
- Secondary pneumomediastinum:
 - Secondary to thoracic barotrauma
 - Common traumatic mechanisms:
 - Motor vehicle collision
 - Fall
 - Blows to chest or neck
 - Recent esophageal/tracheobronchial instrumentation
 - Positive-pressure ventilation
 - Esophageal rupture (Boerhaave syndrome)
 - In association with mediastinal infection caused by gas-forming organisms
- Tension pneumomediastinum:
 - Rare but life-threatening event
 - Usually in patients on positive-pressure ventilation
- May be associated with pneumopericardium and/or extension of a pneumothorax/tension pneumothorax

DIAGNOSIS

SIGNS AND SYMPTOMS

- Chest pain (most common symptom in multiple series):
 - Sharp
 - Pleuritic
 - Retrosternal
 - Radiating to back and arms
 - Often positional
- Dyspnea
- Neck pain:
 - Occurs in association with dissection of air into soft tissues of neck
 - Often described as “neck swelling,” “neck pain,” “throat pain,” or “difficulty swallowing”
- SC emphysema:
 - Most commonly located at the supraclavicular area and anterior neck
- Dysphagia/odynophagia
- Dysphonia/hoarseness
- Hamman crunch: Presence of a precordial crinkling or crepitation during systole:
 - Uncommon but pathognomonic
 - Best heard with patient in left lateral decubitus position

- Meckler triad (esophageal rupture): Vomiting, lower chest pain, and cervical SC emphysema following overindulgence of food or alcohol

History

- Inhalational drug use
- Asthma exacerbation
- Pre-existing lung disorders
- Forceful vomiting (such as in diabetic ketoacidosis [DKA], or hyperemesis)
- Preceding strenuous athletic activity

Physical-Exam

- SC emphysema
- Hamman crunch

ESSENTIAL WORKUP

- Exclude secondary causes, notably esophageal rupture.
- Chest radiography
- Chest CT (if high index of suspicion)

DIAGNOSIS TESTS & INTERPRETATION

Lab

CBC if there is suspicion of mediastinitis (the most concerning consequence of esophageal rupture, with high morbidity and mortality)

Imaging

- CXR:
 - Most valuable initial test
 - Important to include lateral view because mediastinal air is often missed on posterior–anterior view
 - Aids in excluding pneumothorax, pneumopericardium
 - Identification of a pleural effusion or parenchymal infiltrate may suggest an esophageal rupture.
 - Negative in up to 30–35% of cases
 - Spinnaker sail sign or “angel wing” sign (produced by air lifting the thymus off the heart and major vessels)
 - Continuous diaphragm sign (air collecting between the diaphragm and the pericardium)
 - SC or superior mediastinal emphysema
- Chest CT:
 - Imaging test of choice if suspicion is high but CXR is negative (CXR has high false-negative rate)
- Esophagram with water-soluble contrast material:

- Study of choice to exclude diagnosis of esophageal rupture

Diagnostic Procedures/Surgery

- Esophagoscopy:
 - Limited usefulness (overutilized)
 - May be used to further delineate injuries identified with CT and/or esophagram
- Laryngoscopy/bronchoscopy:
 - Limited usefulness (overutilized)
 - May be used to exclude diagnosis of laryngeal/tracheobronchial injury
- Pericardiocentesis:
 - Only in the setting of tension pneumopericardium in the crashing patient
- Tube thoracostomy:
 - Only in the setting of concomitant pneumothorax of sufficient size, or one that is rapidly progressing

DIFFERENTIAL DIAGNOSIS

- Aortic dissection
- Coronary ischemia
- Esophageal diverticula
- Esophageal webs
- Mediastinitis
- Myocarditis
- Pericarditis
- Pneumonia
- Pneumopericardium
- Pneumothorax/tension pneumothorax
- Pulmonary embolus
- Schatzki rings



TREATMENT

PRE HOSPITAL

- Resuscitation of the acutely ill patient (as in the patient with septic mediastinitis)
- In the appropriate setting, standard care of the trauma patient
- Withhold PO intake
- Rapid patient evolution and transport to an appropriate facility

INITIAL STABILIZATION/THERAPY

- IV access
- Oxygen
- Cardiac monitoring

- Pulse oximetry

ED TREATMENT/PROCEDURES

- Spontaneous pneumomediastinum:
 - Usually a benign, self-limiting condition
 - Does not require specific treatment
 - Efforts should focus on pain relief and reassurance once diagnosis is confirmed.
 - High-flow oxygen may facilitate the reabsorption of nitrogen and provide comfort.
 - Withhold PO intake if suspected esophageal source (pending diagnostic studies)
 - Condition is self-limiting and may be expected to resolve over 2–5 days.
- Secondary pneumomediastinum:
 - Once diagnosis is made, direct invasive diagnostic modalities toward the most likely underlying cause (esophagoscopy, laryngoscopy, bronchoscopy).
 - Direct therapy toward underlying cause.

MEDICATION

- Treat underlying cause aggressively (e.g., asthma exacerbation or DKA).
- Oxygen 15 L via nonrebreather mask
- Analgesia (non-narcotic and narcotic as necessary)
- Antibiotics have limited use, but in the setting of concern for mediastinitis use broad-spectrum coverage to include GI flora, resistant organisms, and *Pseudomonas*:
 - Vancomycin 10–15 mg/kg IV q12h *and*
 - Piperacillin/tazobactam 3.375–4.5 g IV q6h *and*
 - Clindamycin 600–900 mg IV q8h *or* Metronidazole 500 mg IV q8h



FOLLOW-UP

DISPOSITION

Admission Criteria

- Secondary pneumomediastinum
- Associated pneumothorax
- Possibility of esophageal rupture has not been excluded
- Abnormal vital signs
- Ill/toxic-appearing patient
- Intractable pain
- Underlying disorder requires admission (asthma exacerbation, exacerbation of lung disorder, DKA).

- Social situation prevents compliance or follow-up
- Extremes of age (pediatric and elderly)
- Immunosuppression
- Failure of outpatient management

Discharge Criteria

- Spontaneous pneumomediastinum
- Normal vital signs
- No pneumothorax
- No significant comorbidities
- Period of observation in the ED with resolution of symptoms
- Close outpatient follow-up

FOLLOW-UP RECOMMENDATIONS

- Patients should be followed up for re-evaluation of clinical symptoms and imaging for resolution of the process.
- Recurrent spontaneous pneumomediastinum may warrant cardiothoracic consultation for further diagnostic evaluation (invasive studies).

PEARLS AND PITFALLS

- Ensure that underlying causes are excluded.
- Be aware of typical presenting features (chest pain, dyspnea, and neck swelling), pre-existing conditions, and precipitating factors associated with pneumomediastinum.
- Hamman crunch is pathognomonic but not commonly seen.
- Remember Meckler triad:
 - Vomiting
 - Lower chest pain
 - Cervical SC emphysema

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- [Pneumothorax](#)
- [Vomiting, Adult](#)

Acknowledgment

The author gratefully acknowledges the contributions of Jennifer De la Pena and Leon D. Sanchez for previous editions of this chapter.



CODES

ICD9

518.1 Interstitial emphysema

ICD10

[J98.2 Interstitial emphysema](#)

PNEUMONIA, ADULT

Jason C. Imperato



BASICS

DESCRIPTION

- Epidemiology:
 - 7th leading cause of death and leading cause from infectious disease in US
- Highest mortality in elderly and patients with the following coexisting conditions:
 - Chronic heart, lung, liver, and kidney disease
 - Diabetes mellitus
 - Alcoholism
 - Malignancy
 - Asplenia
 - Immunosuppression
 - Use of antimicrobials within last 3 mo
- Classifications:
 - Source based:
 - Community acquired (CAP)
 - Health care associated (HCAP)
 - Hospital acquired (HAP)
 - Ventilator associated (VAP)
 - Symptom based:
 - Typical
 - Atypical
- Complications:
 - Bacteremia
 - Sepsis
 - Abscess
 - Empyema
 - Respiratory failure

ETIOLOGY

- CAP (typicals):
 - *Streptococcus pneumoniae*
 - *Haemophilus influenzae*
 - *Klebsiella pneumoniae*
 - *Moraxella catarrhalis*
 - *Streptococcus pyogenes*
 - *Staphylococcus aureus*
- CAP (atypicals):

- *Mycoplasma pneumoniae*
- *Chlamydophila pneumoniae*
- *Legionella pneumophila*
- Viral
- HCAP/HAP/VAP:
 - Gram negatives (*Pseudomonas*, *Stenotrophomonas*)
 - Methicillin-resistant *S. aureus* (MRSA)
- Immunosuppressed:
 - *Mycobacterium tuberculosis*
 - *Pneumocystis jirovecii*
- Aspiration:
 - Chemical pneumonitis ± oral and gastric anaerobes

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Typical:
 - Acute onset
 - Fever
 - Chills
 - Rigors
 - Cough
 - Purulent sputum
 - Shortness of breath
 - Pleuritic chest pain
- Atypical:
 - Subacute onset
 - Viral prodrome
 - Nonproductive cough
 - Low-grade fever
 - Headache
 - Myalgias
 - Malaise
 - Absence of pleurisy and rigors

Physical-Exam

- Vital signs:
 - Tachypnea
 - Tachycardia
 - Hypoxia

- Fever
- Pulmonary exam:
 - Dullness to percussion
 - Tactile fremitus
 - Egophony
 - Rales
 - Rhonchi
 - Decreased breath sounds
- Note that pneumonia may be present in the absence of the above signs of consolidation.

Geriatric Considerations

- Elderly patients have higher morbidity and mortality from pneumonia.
- Atypical presentations are more common.

ESSENTIAL WORKUP

Combination of clinical and radiographic diagnosis

DIAGNOSIS TESTS & INTERPRETATION

Lab

- General:
 - CBC with differential
 - Serum chemistry
- Others:
 - Blood cultures (ICU only)
 - Sputum cultures and Gram stain (ICU only)
 - Urine antigen tests for *S. pneumoniae* & *Legionella*
 - C-reactive protein possibly helpful
 - Lactate may be helpful
 - Influenza viral test

Imaging

Chest radiograph:

- General:
 - Findings are nonspecific for particular infectious etiologies.
 - May be deferred in young, healthy patients receiving empiric outpatient management.
 - Negative imaging should not preclude antimicrobial therapy in patients with clinical diagnosis.
- Suggestive findings:
 - Silhouette sign (R. heart border = RML, L. heart border = lingula, R. hemidiaphragm = RLL, L. hemidiaphragm = LLL)

- Air bronchograms
- Segmental or subsegmental consolidation
- Diffuse interstitial opacities
- Pleural effusion
- Empyema
- Abscess
- Cavitation

Diagnostic Procedures/Surgery

Thoracentesis:

- For large effusions, enigmatic pneumonia, and patients who fail to respond to standard therapy

DIFFERENTIAL DIAGNOSIS

- Asthma
- Bronchitis
- CHF
- COPD
- Foreign-body aspiration
- Occupational or environmental exposure
- Pneumothorax
- Pulmonary embolism
- Tumor



TREATMENT

PRE HOSPITAL

- IV access
- Supplemental oxygen
- Cardiac monitor
- Consider inhaled bronchodilators.
- Consider endotracheal intubation in cases of severe respiratory distress.

INITIAL STABILIZATION/THERAPY

- IV access and fluid resuscitation as needed
- Supplemental oxygen
- Cardiac monitor
- Inhaled bronchodilators
- Endotracheal intubation in cases of severe respiratory distress as indicated

ED TREATMENT/PROCEDURES

- American Thoracic Society guidelines for empiric therapy:

- Outpatient:
 - Previously healthy, no coexisting conditions:
 - Macrolide (azithromycin) OR doxycycline
 - Significant coexisting conditions (see above):
 - Combination β -lactam (ceftriaxone, cefuroxime, cefpodoxime, high-dose amoxicillin, Augmentin) PLUS macrolide (azithromycin) OR
 - Respiratory fluoroquinolone (levofloxacin, moxifloxacin) alone
- Inpatient:
 - Noncritical care:
 - Combination β -lactam PLUS macrolide OR
 - Respiratory fluoroquinolone alone
 - Critical care:
 - Combination β -lactam PLUS macrolide OR respiratory fluoroquinolone
 - For *Pseudomonas*, consider adding antipseudomonal agent (piperacillin/tazobactam, imipenem, meropenem, cefepime) PLUS antipseudomonal fluoroquinolone (high-dose levofloxacin) OR antipseudomonal agent (see above) PLUS aminoglycoside (gentamicin) PLUS macrolide (azithromycin).
 - For MRSA, consider adding vancomycin OR linezolid.
 - For aspiration, consider adding clindamycin OR metronidazole.
 - For drug-resistant *S. pneumoniae*, consider adding vancomycin.

MEDICATION

- Amoxicillin–clavulanate (Augmentin): 500 mg PO q12h
- Ampicillin–sulbactam (Unasyn): 1.5–3 g IV q6h
- Azithromycin: 500 mg PO on day 1 and 250 mg PO on days 2–5 OR 500 mg PO daily for 3 days OR 500 mg IV daily
- Aztreonam: 1–2 g IV q12h
- Cefepime: 2 g IV q12h
- Cefotaxime: 1–2 g IV q8h
- Cefpodoxime: 200 mg PO q12h
- Ceftazidime: 2 g IV q12h
- Ceftriaxone: 1–2 g IV daily
- Cefuroxime: 0.75 and 1.5 g IV q8h
- Doxycycline: 100 mg PO/IV q12h
- Ertapenem: 1 g IV daily
- Levofloxacin: 500–750 mg PO/IV daily
- Linezolid: 600 mg PO/IV daily
- Imipenem: 500 mg IV q6h
- Meropenem: 1 g IV q8h
- Moxifloxacin: 400 mg IV daily
- Piperacillin–tazobactam (Zosyn): 3.375–4.5 g IV q6h
- Vancomycin: 1 g IV q12h

First Line

- Outpatient:
 - Healthy:
 - Azithromycin 500 mg PO day 1, 250 mg PO days 2–5 OR 500 mg PO daily for 3 days
 - Comorbidities:
 - Levofloxacin 750 mg PO daily for 5 days
- Inpatient:
 - Non-ICU:
 - Levofloxacin 750 mg IV daily
 - ICU:
 - Ceftriaxone 1 g IV daily AND levofloxacin 750 mg IV daily ± piperacillin–tazobactam 4.5 g IV q6h ± vancomycin 1g IV q12h

Second Line

Aztreonam may be substituted for β -lactams in confirmed penicillin-allergic patients for the above ICU regimens.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Based on severity of illness, coexisting conditions, ability of home care, and follow-up
- Clinical decision-making rules may aid in stratifying patients but should not supersede clinical judgment.
- CURB-65 rule:
 - Criteria:
 - Confusion (Abbreviated Mental Test ≤ 8)
 - Urea >7 mmol/L OR BUN >19
 - Respiratory rate ≥ 30 /min
 - BP with SBP <90 mm Hg, DBP <60 mm Hg
 - Age ≥ 65 yr
 - Interpretation:
 - 0–1: Outpatient treatment
 - 2: Close outpatient vs. brief inpatient
 - 3–5: Inpatient with ICU consideration
- Pneumonia Severity Index:
 - Demographics:
 - If Male: + age (yr)
 - If Female: + age (yr) – 10

- If nursing home resident: +10
- Comorbid illness:
 - Neoplastic disease: +30
 - Liver disease: +20
 - Congestive heart failure: +10
 - Cerebrovascular disease: +10
 - Renal disease: +10
- Physical exam findings:
 - Altered mental status: +20
 - Pulse ≥ 125 /min: +20
 - Respiratory rate > 30 /min: +20
 - SBP < 90 mm Hg: +15
 - Temperature $< 35^\circ\text{C}$ or $\geq 40^\circ\text{C}$: +10
- Lab and radiographic findings:
 - Arterial pH < 7.35 : +30
 - BUN ≥ 30 mg/dL: +20
 - Sodium < 130 mmol/L: +20
 - Glucose ≥ 250 mg/dL: +10
 - Hematocrit $< 30\%$: +10
 - PaO₂ < 60 mm Hg: +10
 - Pleural effusion: +10
- Interpretation:
 - 0: Class I (outpatient)
 - < 70 : Class II (outpatient vs. short observation)
 - 71–90: Class III (home with IV antibiotics vs. short observation)
 - 91–130: Class IV (inpatient)
 - > 130 : Class V (inpatient)
- Additional considerations:
 - Previous hospitalization within last year for pneumonia
 - Failed outpatient therapy
 - Social conditions preventing safe outpatient disposition

Discharge Criteria

- Age < 65 yr
- No comorbid illnesses
- Nontoxic appearance
- Normal vital signs
- Normal lab studies
- Primary care follow-up within 72 hr

Issues for Referral

Follow-up with primary care within 72 hr

FOLLOW-UP RECOMMENDATIONS

Primary care follow-up within 72 hr

PEARLS AND PITFALLS

- Delayed initiation of antibiotics in ill-appearing patients
- Failure to recognize pneumonia in patients assumed to have exacerbations of underlying lung conditions
- Failure to question patients regarding TB and HIV risk factors
- Elderly and immunocompromised patients may not exhibit any classic symptoms of pneumonia when ill.

ADDITIONAL READING

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- Nazarian DJ, Eddy OL, Lukens TW, et al. Clinical policy: Critical issues in the management of adult patients presenting to the emergency department with community-acquired pneumonia. *Ann Emerg Med*. 2009;54:704–731.



See Also (Topic, Algorithm, Electronic Media Element)

- Pneumonia, Pediatric
- *Pneumocystis carinii* Pneumonia



CODES

ICD9

- 481 Pneumococcal pneumonia [Streptococcus pneumoniae pneumonia]
- 486 Pneumonia, organism unspecified
- 507.0 Pneumonitis due to inhalation of food or vomitus

ICD10

- J13 Pneumonia due to Streptococcus pneumoniae
- J18.9 Pneumonia, unspecified organism
- J69.0 Pneumonitis due to inhalation of food and vomit

PNEUMONIA, PEDIATRIC

Gary D. Zimmer • Karen P. Zimmer



BASICS

DESCRIPTION

- Mechanism is often unknown.
- Source is oropharyngeal aspiration (most common) or hematogenous.
- Distribution depends on the organism: Interstitial (*Mycoplasma pneumoniae*, virus), lobar (*Streptococcus pneumoniae*), abscesses (*Staphylococcus aureus*), or diffuse (*Pneumocystis carinii*)

ETIOLOGY

- < 2 wk:
 - Group B *Streptococcus* species
 - Enteric gram-negative organisms
 - Respiratory syncytial virus (RSV)
 - Herpes simplex virus
 - *S. aureus*
- 2 wk–3 mo:
 - *Chlamydia trachomatis*
 - Parainfluenza virus
 - RSV
 - *S. pneumoniae*
 - *S. aureus*
 - *H. influenza*
 - *Bordetella pertussis*
- 3 mo–8 yr:
 - Viral (predominate):
 - RSV
 - Parainfluenza virus
 - Influenza virus
 - Adenovirus
 - *S. pneumoniae*
 - *H. influenza* in unimmunized children
 - Group A streptococcus
 - *S. aureus*
 - *B. pertussis*
- > 8 yr:
 - *M. pneumoniae* most common
 - Viral

- *S. pneumoniae*
- Recent immigrants from developing countries:
 - *Mycoplasma tuberculosis*
 - *H. influenza*
 - *B. pertussis*
- Immunocompromised (e.g., HIV, cancer):
 - *P. carinii*
 - *Mycoplasma avium* complex
 - *M. tuberculosis*
 - *Klebsiella pneumoniae*
 - *Pseudomonas aeruginosa*
- Less common:
 - Fungal (coccidioidomycosis, histoplasmosis)
 - *Rickettsia* (Q fever)



DIAGNOSIS

SIGNS AND SYMPTOMS

- General (in all ages):
 - Cough
 - Rales
 - Fever
 - Hypoxia
 - Tachycardia
 - Tachypnea, retractions, grunting
 - Rash (up to 10% of cases); usually maculopapular
 - Nonspecific symptoms of toxicity
 - Pulmonary exam:
 - Decreased breath sounds, ventilation
 - Dullness to percussion
 - Wheezing, ronchi, rales
- Infants < 6 mo:
 - Altered behavior: Listless, irritable
 - Apnea (esp. RSV in premature infants)
 - Conjunctivitis (*Chlamydia* < 1 mo old)
 - Cyanosis
 - Grunting
 - Poor feeding
 - Temperature instability (hypothermia/hyperthermia)
 - Vomiting, often with coughing
 - Cough
 - Nasal congestion

- Nasal flaring
- Wheezing
- Staccato cough (*Chlamydia*)
- Children >5 yr:
 - Pleuritic chest pain
 - Productive cough
 - Rigors, chills

History

- Immunization history
- Past medical history include immune status
- Exposures
- Progression of signs and symptoms

Physical-Exam

- Pulmonary exam may be helpful, particularly in children >5 yr.
- Peripheral and central cyanosis should be assessed.
- Evidence of respiratory compromise, distress, failure

ESSENTIAL WORKUP

- Pulse oximetry
- Chest radiograph:
 - Gold standard for diagnosis
 - Should be ordered for patients with signs of lower respiratory tract infection and patients <36 mo old with marked leukocytosis or neutrophilia (WBC >15,000 or absolute neutrophil count [ANC] >9,000).
 - Much overlap between viral and bacterial findings
 - Viral and *M. pneumoniae* tend to show interstitial infiltrates, often perihilar and peribronchial.
 - Bacterial pneumonias may show focal lobar consolidation, focal alveolar infiltrates, and possibly effusion or pneumatocele.
 - Round pneumonia pathognomonic of *S. pneumonia*
 - Lateral decubitus films may aid in demonstrating effusion.

DIAGNOSIS TESTS & NTERPRETATION

Lab

- CBC with differential:
 - Patients with bacteremia tend to have leukocytosis with left shift.
 - Sensitivity and specificity are poor.
 - Patients with WBC $\geq 20,000$ or ANC >9,000 are at increased risk of pneumococcal bacteremia.
 - *B. pertussis* usually has elevated WBC with lymphocytosis.

- Blood culture:
 - Low yield (< 10–20%)
 - Recommended in children < 36 mo
 - Probably worthwhile in toxic patients requiring hospitalization
- Arterial blood gas may be useful in determining degree of respiratory insufficiency in critically ill patients.
- Electrolytes to exclude syndrome of inappropriate antidiuretic hormone secretion and in hypotensive children
- Sputum for Gram stain and culture may be obtained in older children with suspected bacterial infection.
- *Mycoplasma* IgM or cold agglutinin titers:
 - Useful if suspecting this organism
 - More likely positive with severe illness
- Nasopharyngeal washes for direct fluorescent antibody and culture:
 - Identify RSV, *C. trachomatis*, and *B. pertussis* infections

Imaging

Chest radiographs are still the imaging modality of choice:

- Posteroanterior and lateral films should be obtained whenever possible.
- CT provides additional detail and better identification of underlying lung pathology but adds little as an initial testing modality.

Diagnostic Procedures/Surgery

Pleural fluid (if present) for culture, Gram stain, protein, glucose, and cell counts

DIFFERENTIAL DIAGNOSIS

- Reactive airway disease (asthma, bronchiolitis [age < 2 yr])
- Aspiration:
 - Gastroesophageal reflux
 - Vascular ring
 - H-type tracheoesophageal fistula
 - Foreign body
 - Hydrocarbon
- Congestive heart failure
- Congenital:
 - Cystic fibrosis
 - Sequestered lobe
 - Congenital lobe absence
 - Hemangioma
- Neoplasm



TREATMENT

PRE HOSPITAL

- Pulse oximetry
- Administer high-flow oxygen for respiratory distress.
- IV fluids (0.9% normal saline [NS] 20 mL/kg initial bolus) for volume depletion, hypotension
- Support and intubation for respiratory failure

INITIAL STABILIZATION/THERAPY

- If moderately or severely ill:
 - Secure airway, as appropriate; intubate for clinical respiratory failure. Children with severe sepsis or septic shock benefit from aggressive airway management.
 - High-flow oxygen
 - IV hydration (0.9% NS 20 mL/kg initial bolus) and resuscitation if in shock or hypovolemia
- Monitor
- Ongoing pulse oximetry
- Arterial blood gas if inadequate ventilation
- Check bedside glucose in severely ill-appearing infants and toddlers:
 - If hypoglycemic, administer glucose D25 at 2 mL/kg IV for toddlers or D10 at 5 mL/kg IV for neonates.

ED TREATMENT/PROCEDURES

- Continue pre-hospital and initial stabilization therapy.
- Early antibiotic therapy should be broad enough to address local resistance patterns in your area.
- Often have concurrent reactive airway disease that needs specific treatment with bronchodilator (albuterol or levalbuterol)
- Perform thoracentesis if pleural effusion is compromising respiratory function or for diagnostic tests.

MEDICATION

- Empiric therapy with oral antibiotics for most well-appearing children ≥ 6 mo:
 - Infants < 2 mo:
 - Outpatient treatment generally not recommended unless child has no respiratory distress or associated conditions or issues.
 - Children 3 mo–5 yr:
 - Amoxicillin
 - Amoxicillin—clavulanate
 - Trimethoprim—sulfamethoxazole
 - Erythromycin—sulfisoxazole
 - Macrolide (azithromycin or clarithromycin)
 - Children 5–18 yr:

- Macrolide (azithromycin or clarithromycin)
- Initiate IV antibiotic therapy for moderate to severely ill children who require admission:
 - _ Neonate:
 - Ampicillin, and cefotaxime or gentamicin
 - Azithromycin for suspected *C. trachomatis* or *B. pertussis* pneumonia
 - _ Infants 1–2 mo:
 - Ampicillin and cefotaxime
 - Azithromycin or erythromycin for suspected *C. trachomatis* or *B. pertussis*
 - _ Children ≥ 3 mo:
 - Cefotaxime, cefuroxime, or ceftriaxone
 - Vancomycin for suspected or confirmed penicillin-resistant *S. pneumoniae*
 - Macrolide (i.e., azithromycin) for suspected *M. pneumoniae*
 - Clindamycin if group A strep suspected in patient with severe disease
- Unusual organisms require specific therapy in coordination with infectious disease consultation.
- Albuterol (0.5% solution or 5 mg/mL): Nebulizer 0.015 mg (0.03 mL)/kg per dose up to 5 mg per dose q10–20min as needed; metered dose inhaler (with spacer; 90 mg per puff) 2 puffs q10–20min up to total of 10 puffs
- Amoxicillin: 80 mg/kg/24 h q12h PO
- Amoxicillin–clavulanate: 30 mg/kg/24 h q12h PO
- Ampicillin: 100–150 mg/kg/24 h q6h IV
- Azithromycin: 10 mg/kg/24 h daily for 1 day, then 5 mg/kg/24 h daily for 4 days
- Cefotaxime: 50–75 mg/kg/24 h q8h IV, max. 2 g q8h
- Ceftriaxone: 100 mg/kg/24 h q12–24 h IV, max. 2 g q12h
- Cefuroxime: 100 mg/kg/24 h q8h IV, max. 2 g q8h
- Clarithromycin: 15 mg/kg/24 h q12h PO, max. 500 g q12h
- Clindamycin 30–40 mg/kg/24 h q6–8h IV
- Erythromycin–sulfisoxazole: 40 mg/kg/24 h as erythromycin q8h PO, max. 2 g/d
- Gentamicin: 5–7.5 mg/kg/24 h q8–12h IV
- Trimethoprim–sulfamethoxazole: 8–10 mg/kg/24 h as TMP q12h PO
- Vancomycin: 10–15 mg/kg/24 h q8–12h IV; max. 1,000 mg



FOLLOW-UP

DISPOSITION

Admission Criteria

- Toxic appearance
- Respiratory distress or failure

- Dehydration/vomiting
- Apnea
- Infants < 2 mo
- Infants < 6 mo with lobar pneumonia
- Hypoxia (O₂ saturation < 92% on room air [sea level])
- Pleural effusion
- Poor response to outpatient oral therapy
- Immunocompromised children
- Concern about noncompliant parents

Discharge Criteria

- Most cases are mild and can be discharged home if no evidence of hypoxia, significant work-of-breathing, dehydration, vomiting, or noncompliance.
- Ensured follow-up within 1–2 days

Issues for Referral

Respiratory failure, effusion, toxicity

FOLLOW-UP RECOMMENDATIONS

Clinical resolution should be ensured through follow-up.

PEARLS AND PITFALLS

- Early, aggressive airway management for patients with severe sepsis and septic shock
- Delays to antibiotic therapy should be avoided.
- Discharged patients should have clear evidence of good support, follow-up, and lack of toxicity.
- Local patterns of drug resistance should be known and empiric therapy should take these resistance patterns into consideration.

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- Shah SS, Dugan MH, Bell LM, et al. Blood cultures in the emergency department evaluation of childhood pneumonia. *Pediatr Infect Dis J.* 2011;30:475–479.



See Also (Topic, Algorithm, Electronic Media Element)

Asthma



CODES

ICD9

- 483.0 Pneumonia due to mycoplasma pneumoniae
- 486 Pneumonia, organism unspecified
- 507.0 Pneumonitis due to inhalation of food or vomitus

ICD10

- J15.7 Pneumonia due to Mycoplasma pneumoniae
- J18.9 Pneumonia, unspecified organism
- J69.0 Pneumonitis due to inhalation of food and vomit

PNEUMOTHORAX

William Porcaro

BASICS

DESCRIPTION

- Presence of free air in the intrapleural space
- Spontaneous pneumothorax is due to atraumatic rupture of alveolus, bronchiole, or bleb.
- Primary spontaneous pneumothorax (2/3 of incidences):
 - No underlying pulmonary pathology present
 - Rupture of small subpleural cyst or bleb
 - Primarily young, healthy patients (20–40 yr old) with tall, thin body habitus
 - Risk factors: Smoking, family history, Marfan syndrome, homocystinuria, thoracic endometriosis
- Secondary spontaneous pneumothorax from underlying pulmonary pathology (see Etiology)
- Tension pneumothorax:
 - Air continues to enter pleural space through bronchoalveolar disruption and becomes trapped via “ball-valve” mechanism.
 - Intrapleural pressure increases.
 - Venous return to right heart decreases, resulting in decrease in cardiac output.
 - Mediastinum shifts toward uninvolved side, mechanically interfering with right atrial filling.
 - Ventilation compromise and ventilation/perfusion mismatch result in hypoxemia

ETIOLOGY

- Idiopathic
- Airway disease:
 - Chronic obstructive pulmonary disease (COPD)
 - Asthma
 - Cystic fibrosis
- Infections:
 - Necrotizing bacterial pneumonia
 - TB
 - Fungal pneumonia
 - *Pneumocystis carinii*
- Neoplasm
- Interstitial lung disease:

- Sarcoidosis
- Idiopathic pulmonary fibrosis
- Lymphangiomyomatosis
- Tuberosus sclerosis
- Pneumoconioses
- Connective tissue diseases
- Pulmonary infarction
- Endometriosis
- Blunt chest trauma
- Penetrating trauma to neck or trunk
- Iatrogenic:
 - Central line placement
 - Other vascular access procedures

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Severity of symptoms is generally proportional to size of the pneumothorax.
- Chest pain on the ipsilateral side:
 - Sharp, pleuritic pain
 - Sudden onset
 - Dull ache in delayed presentations
- Shortness of breath
- Rarely cough, asymptomatic, or generalized malaise

Physical-Exam

- Tachypnea
- Heart rate < 120 bpm generally seen in simple spontaneous pneumothoraces
- Jugular venous distention and tracheal deviation to the contralateral side may be evident in tension pneumothorax.
- Cardiac and pulmonary exam:
 - Asymmetric decreased breath sounds
 - Hyperresonance to percussion of ipsilateral side
- Tension pneumothorax:
 - Hypotension
 - Tachycardia, heart rate > 120 bpm
 - Diaphoresis
 - Cyanosis
 - Cardiovascular collapse
 - Tracheal deviation

ESSENTIAL WORKUP

- Imaging is mainstay of the workup
- DO NOT delay chest decompression if the patient is hemodynamically unstable and there is sufficient clinical evidence of pneumothorax.

DIAGNOSIS TESTS & INTERPRETATION

Lab

Arterial blood gas offers little over oxygen saturation.

Imaging

- Chest radiograph:
 - Upright chest radiograph
- Patients unable to tolerate upright chest radiograph can be taken in decubitus position with the suspected side up:
 - Absence of lung markings distal or peripheral to the visceral pleural white line
 - Displacement of mediastinum or anterior junction line
 - Deep sulcus sign
- On frontal view, larger lateral costodiaphragmatic recess than on opposite side
- Diaphragm may be inverted on the side with deep sulcus:
 - A rough estimate of pneumothorax size is sufficient to make clinical decisions.
- Expiratory film:
 - May demonstrate small pneumothorax but has not been shown to increase yield of detection
- Chest CT:
 - Very sensitive for small pneumothorax but has little practical advantage over chest radiograph
- US:
 - User experience required
 - Rapid at bedside
 - Lack of lung sliding and comet-tail artifact signifies pneumothorax.
 - M-mode confirms pneumothorax with smooth lines above and below pleural line.
 - With experience, sensitivity surpasses chest radiograph

Diagnostic Procedures/Surgery

ECG:

- Often necessary to rule out cardiac etiologies of chest pain
- Nonspecific changes include T-wave inversion, left axis deviation, and decreased R-wave amplitude.

DIFFERENTIAL DIAGNOSIS

- Acute abdominal processes
- Aortic aneurysm or dissection
- Asthma exacerbation
- Chest wall pain
- COPD exacerbation
- Myocardial infarction
- Pericarditis
- Pleuritis
- Pneumomediastinum
- Pulmonary embolus



TREATMENT

PRE HOSPITAL

ALERT

Unstable patients with a suspected tension pneumothorax require immediate needle thoracostomy.

INITIAL STABILIZATION/THERAPY

- Cardiac monitor
- Pulse oximetry
- Oxygen 100% via nonrebreather face mask
- IV access
- Suspected tension pneumothorax requires either immediate needle thoracostomy or tube thoracostomy.
- Needle thoracostomy:
 - _ Immediate placement indicated in unstable patients with a tension pneumothorax
 - _ 14G–18G angiocatheter in the 2nd intercostal space at midclavicular line or 4th or 5th intercostal space at anterior axillary line
 - _ NOTE: The length of most standard angiocatheters is too short to penetrate the pleural cavity in moderate to large framed patients – longer, purpose-specific catheters may be required

ED TREATMENT/PROCEDURES

- Nontraumatic pneumothorax estimated at < 15% collapse and no cardiovascular or respiratory compromise:
 - _ Observe with 100% oxygen support for 4–6 hr.
 - _ Repeat chest radiograph and discharge if unchanged.
- Simple aspiration:
 - _ Indications:

- Simple pneumothorax with only 15–30% collapse
 - Increase in size of a small pneumothorax during observation
- Placement of aspiration catheter (typically 8F) with 3-way stopcock
- Aspirate air until resistance or 3 L of air aspirated.
 - If the pneumothorax is no longer visible on 2 subsequent chest radiographs at 4 hr intervals, remove catheter.
 - If a final chest radiograph is normal 2 hr after the catheter is removed, the patient may be discharged.
 - A 2nd aspiration may be attempted if the pneumothorax does not resolve.
- Heimlich valve:
 - Indicated when < 30% collapse after failure of aspiration
 - Attach Heimlich valve to aspiration catheter or chest tube.
- Suction:
 - Indicated when the Heimlich valve fails
 - Attach aspiration catheter to suction at 20 cm H₂O.
 - Observe in ED for 1 hr.
- Tube thoracostomy:
 - Indications:
 - Suspicion of a tension pneumothorax
 - Gunshot wound to the chest
 - Clinical evidence of a pneumothorax following blunt chest trauma or penetrating chest trauma
 - Presence of a pneumothorax of any size in patient receiving positive-pressure ventilation
 - Pneumothorax with > 30% collapse
 - Most cases of secondary pneumothorax
 - Definitive therapy after needle thoracostomy
 - Tube size:
 - Small-caliber (7–14F) tube for primary spontaneous pneumothoraces
 - 20–28F for secondary spontaneous pneumothorax
 - 28F when there is detectable pleural fluid or an anticipated need for mechanical ventilation
 - Check for tube kinks by fully rotating the inserted tube.
 - All side holes in the tube must be within the chest wall to avoid leak.
 - Following insertion, the tube should be connected to a water-seal device.
 - A Heimlich valve may be used instead of a water-seal device in stable patients without a pleural effusion.
 - Re-expansion edema is a rare complication requiring supportive care.
- Possible complications:
 - Intercostal vessel bleeding
 - Inadequate drainage:
 - Kinked tube

- Clogged tube
- Communication outside of pleural cavity with leak
- Re-expansion pulmonary edema:
 - Treatment with fluid resuscitation

MEDICATION

- Local anesthetic:
 - 1% lidocaine with epinephrine 1:100,000
 - Max. dose: 7 mg/kg–500 mg
- Consider procedural sedation in stable awake patients
- No indication for antibiotics in a clean procedure



FOLLOW-UP

DISPOSITION

Admission Criteria

- Tension pneumothorax
- Chest tube required

Discharge Criteria

- <15% collapse, no expansion while in the ED or successful aspiration with catheter removed:
 - Discharge with follow-up in 24 hr and 1 wk for chest radiograph to assure re-expansion.
- Reliable patients with the thoracic vent and successful aspiration or secured catheter and Heimlich valve:
 - Discharge with 24 and 48 hr follow-up.
 - At 48 hr follow-up:
 - Clamp catheter, observe for 2 hr, and repeat chest radiograph.
 - Remove thoracic vent or catheter if no re-expansion.
 - Observe for 2 hr and repeat chest radiograph.
 - If no re-expansion, discharge with 24 hr and 1 wk follow-up.
- Discharge instruction should include prompt return for new onset of chest pain or dyspnea.
- Patients without re-expansion at 1 wk require a cardiothoracic surgery consult.

FOLLOW-UP RECOMMENDATIONS

Pulmonary medicine and/or chest surgery

PEARLS AND PITFALLS

- Delay in chest decompression in the unstable patient leading to rapid

hemodynamic compromise

- Avoid poor tube placement involving kinks or improper depth, which may necessitate repeating the procedure.
- Avoid placement of catheter or tube too low on the lateral chest wall, which may lead to iatrogenic abdominal injuries.
- Failure to detect associated mediastinal or lower neck injuries
- If pneumomediastinum is detected, evaluate for esophageal pathology

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Chest Pain
- Dyspnea



CODES

ICD9

- 512.0 Spontaneous tension pneumothorax
- 512.81 Primary spontaneous pneumothorax
- 512.89 Other pneumothorax

ICD10

- J93.0 Spontaneous tension pneumothorax
- J93.9 Pneumothorax, unspecified
- J93.11 Primary spontaneous pneumothorax

POISONING

Mark B. Mycyk

BASICS

DESCRIPTION

- Poisoning may be intentional or unintentional.
- Patients with change in mental status without clear cause should have poisoning (intoxication, overdose) considered in differential diagnosis.

ETIOLOGY

- Intentional:
 - Depression
 - Suicide
 - Homicide
 - Recreational drug abuse
- Unintentional (accidental):
 - Common cause in children
 - Therapeutic error (e.g., double dose)
 - Recreational drug experimentation

Pediatric Considerations

- Accidental ingestions—typically young children (1–5 yr)
- Consider child abuse if inconsistent or suspicious history.

DIAGNOSIS

SIGNS AND SYMPTOMS

- Neurologic:
 - Lethargy
 - Agitation
 - Coma
 - Hallucinations
 - Seizures
- Respiratory:
 - Tachypnea, bradypnea, apnea
 - Inability to protect airway
- Cardiovascular:
 - Dysrhythmias
 - Conduction blocks
- Vital signs:

- Varies depending on toxic substance
- Hyperthermia, hypothermia
- Tachycardia, bradycardia
- Hypertension, hypotension

Selected Toxidromes (see Poisoning, Toxidromes)

- **Anticholinergic:**
 - Altered mental status (confusion, delirium, lethargy)
 - Dry skin and mucous membranes
 - Fixed dilated pupils
 - Tachycardia
 - Hyperthermia
 - Flushing
 - Urinary retention
- **Cholinergic:**
 - Secretory overdrive (salivation, lacrimation, urination, diaphoresis)
 - Miosis
 - Bronchospasm, wheezing
- **Opiate:**
 - CNS and respiratory depression
 - Miosis
- **Sympathomimetic:**
 - CNS excitation
 - Seizures
 - Tachycardia
 - Hypertension
 - Diaphoresis

ESSENTIAL WORKUP

- A complete set of vital signs, including core temperature
- A complete physical exam, including eyes, skin, odors

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Electrolytes, BUN/creatinine, glucose
- Calculate anion gap: $Na + (Cl + HCO_3)$:
 - Normal anion gap: 8–12
 - Use mnemonic *A CAT MUD PILES* for elevated anion gap acidosis:
 - Alcoholic ketoacidosis
 - Cyanide, carbon monoxide
 - Aspirin, other salicylates
 - Toluene
 - Methanol, metformin

- Uremia
- Diabetic ketoacidosis
- Paraldehyde, phenformin
- Iron, isoniazid
- Lactic acidosis from other causes
- Ethylene glycol
- Starvation ketosis
- Serum osmol gap:
 - Calculate osmol gap if elevated anion gap acidosis from potential toxic alcohol.
 - Most sensitive *early* in poisoning
 - Normal osmol gap does not completely rule out toxic alcohol ingestion.
 - Calculated osmolality = $2(\text{Na}^+) + \text{glucose}/18 + \text{BUN}/2.8 + \text{ethanol (in mg/dL)}/4.6$.
 - Osmol gap = measured osmolality – calculated osmolality.
 - Use mnemonic *ME DIE A* when osmol gap > 10:
 - Methanol
 - Ethanol
 - Diuretics (mannitol, glycerin, sorbitol)
 - Isopropyl alcohol
 - Ethylene glycol
 - Acetone
- Pregnancy test
- Acetaminophen level for suicidal ingestions
- Toxicology screen

Imaging

- ECG for dysrhythmias or QRS/QT changes
- CT of head for altered mental status not clearly due to toxin
- Chest radiograph if suspected aspiration or pneumonia

DIFFERENTIAL DIAGNOSIS

- Causes of altered mental status
- Intracranial mass, bleeding
- Infection, sepsis
- Endocrine abnormalities
- Hypothermia
- Hypoxia
- Metabolic abnormalities
- Psychogenic



TREATMENT

PRE HOSPITAL

- Search for clues at scene:
 - Pills/pill bottles
 - Drug paraphernalia
 - Witnesses
 - Transport all drugs and pill bottles for identification.
- Restrain uncooperative patients for patient and health care giver protection.
- Consider comorbid conditions:
 - Trauma
 - Medical illness
 - Environmental exposure
- Pre-hospital administration of activated charcoal may optimize decontamination if prolonged transport time.

INITIAL STABILIZATION/THERAPY

- ABCs:
 - Endotracheal intubation as needed for airway protection, oxygenation, ventilation, and orogastric lavage
 - Supplemental oxygen for hypoxia
 - Pulse oximetry
 - Cardiac monitor
 - IV access
- Hypotension:
 - Administer 0.9% normal saline IV fluid bolus.
 - Trendelenburg
 - Vasopressors for persistent hypotension
- Bradycardia:
 - Atropine
 - Cardiac pacing
- If altered mental status, administer coma cocktail: Thiamine, D50W (or Accu-Chek), naloxone

ED TREATMENT/PROCEDURES

- Decontamination:
 - See Poisoning, Gastric Decontamination.
 - Prevents systemic absorption of ingested toxin
- Orogastric lavage:
 - Consider in potentially lethal ingestions without known antidote within 1 hr of ingestion.
 - Protected airway *essential* prior to lavage
- Activated charcoal:
 - Most effective within a few hours of most toxic ingestions
 - Contraindicated if caustic ingestion, unprotected airway, or bowel

obstruction

- Drugs not effectively bound to charcoal: Metals (borates, bromide, iron, lithium), alcohols, potassium
- Whole-bowel irrigation:
 - Polyethylene glycol (Colyte, GoLyteLy) evacuates bowel without causing electrolyte disturbances.
 - Consider in toxins not well adsorbed by charcoal (e.g., iron and lithium), body packers/stuffers, sustained-release ingestions.
 - Contraindicated if bowel obstruction, perforation, or hypotension
- Enhanced elimination:
 - Enhances removal of systemically absorbed toxin
- Multiple-dose activated charcoal:
 - Theophylline
 - Carbamazepine
 - Phenobarbital
- Urinary alkalization:
 - Salicylates
 - Phenobarbital
- Hemodialysis/hemoperfusion:
 - Lithium
 - Salicylates
 - Theophylline
 - Toxic alcohols
 - Valproate
- Seizures
 - Treat initially with diazepam or lorazepam.
 - For persistent seizures, consider phenobarbital.
 - Phenytoin *not* indicated in toxicologic seizures:
 - Indicated only if seizures secondary to idiopathic epilepsy, post-traumatic, or status epilepticus
- Antidotes:
 - Acetaminophen: N-acetylcysteine
 - Anticholinergic: Physostigmine
 - Benzodiazepines: Flumazenil
 - β -blockers: Glucagon
 - Calcium-channel blockers: Calcium chloride/gluconate, insulin
 - Carbon monoxide: Oxygen, hyperbaric oxygen
 - Coumadin: Vitamin K₁
 - Cyanide: Cyanide antidote kit, hydroxocobalamin
 - Digoxin: Digibind
 - Ethylene glycol: Ethanol, 4-methylpyrazole
 - Iron: Deferoxamine

- Isoniazid: Pyridoxine (vitamin B₆)
- Methanol: Ethanol, 4-methylpyrazole
- Methemoglobinemia: Methylene blue
- Opiates: Naloxone
- Organophosphates: Atropine, pralidoxime
- Tricyclic antidepressants: NaHCO₃

MEDICATION

- Activated charcoal slurry: 1–2 g/kg PO
- Dextrose: D50W 1 amp: 50 mL or 25 g (peds: D25W 2–4 mL/kg) IV
- Diazepam: 5–10 mg (peds: 0.2–0.5 mg/kg) IV every 10–15 min
- Lorazepam: 2–6 mg (peds: 0.05–0.1 mg/kg) IV every 10–15 min
- Naloxone (Narcan): 0.4–2 mg (peds: 0.1 mg/kg) IV or IM initial dose
- Thiamine (vitamin B₁): 100 mg (peds: 50 mg) IV or IM



FOLLOW-UP

DISPOSITION

Admission Criteria

- Altered mental status
- Cardiopulmonary instability
- Suicidal
- Lab abnormalities
- Potential for decompensation from delayed acting substance

Discharge Criteria

- Psychiatrically clear
- Detoxified
- Hemodynamically stable

Issues for Referral

- Patients with unintentional (accidental) poisoning require poison prevention counseling.
- Patients with intentional (e.g., suicide) poisoning require psychiatric evaluation.
- Consider substance abuse referral for patients.

Pregnancy Considerations

In general, treating the mother is also the best treatment strategy for the fetus.

FOLLOW-UP RECOMMENDATIONS

- Consider substance abuse referral for patients with recreational drug abuse.
- Patients with unintentional (accidental) poisoning require poison prevention counseling.
- Patients with intentional (e.g., suicide) poisoning require psychiatric evaluation.

PEARLS AND PITFALLS

- Do not forget to consider nontoxicologic etiologies for altered mental status.
- Do not rely on the urine drug screen to make a diagnosis: It only provides screening tests for a limited number of drugs.
- Call a toxicologist or a poison center for help: 800-222-1222.

ADDITIONAL READING

- Erickson TB, Thompson TM, Lu JJ. The approach to the patient with an unknown overdose. *Emerg Med Clin North Am.* 2007;25(2):249–281.
- Levine M, Brooks DE, Truitt CA, et al. Toxicology in the ICU: Part 1: General overview and approach to treatment. *Chest.* 2011;140(3):795–806.
- Mycyk MB. Poisoning and drug overdose. In: Longo D, Fauci A, Kasper D, et al., eds. *Harrison's Principles of Internal Medicine.* 18th ed. New York, NY: McGraw Hill; 2012:e50.1–e50.16.
- Wills B, Erickson T. Drug- and toxin-associated seizures. *Med Clin North Am.* 2005;89:1297–1321.



See Also (Topic, Algorithm, Electronic Media Element)

- Poisoning, Antidotes
- Poisoning, Gastric Decontamination
- Poisoning, Toxidromes



CODES

ICD9

- 971.1 Poisoning by parasympatholytics (anticholinergics and antimuscarinics) and spasmolytics
- 977.9 Poisoning by unspecified drug or medicinal substance
- 977.9 Poisoning by unspecified drug or medicinal substance

ICD10

- T44.3X1A Poisoning by oth parasympath and spasmolytics, acc, init
- T65.91XA Toxic effect of unspecified substance, accidental (unintentional), initial encounter
- T65.91XA Toxic effect of unspecified substance, accidental (unintentional), initial

POISONING, ANTIDOTES

Suzan S. Mazor



TREATMENT

N-ACETYLCYSTEINE (NAC)

- Indications: Acetaminophen overdose
- Warnings:
 - _ Unpleasant odor, nausea, vomiting
 - _ Most effective if given in 1st 8 hr postingestion
- Dose:
 - _ PO: 140 mg/kg, then 70 mg/kg q4h for 17 doses
 - _ IV (consult poison center): 150 mg/kg in 200 mL D₅W over 60 min, then 50 mg/kg in 500 mL D₅W over 4 hr, then 100 mg/kg in 1,000 mL D₅W over 16 hr

Pediatric Considerations

This volume of D₅W will need to be reduced in dosing pediatric patients to avoid fluid overload/hyponatremia. Discuss with pharmacy.

ATROPINE

- Indications:
 - _ Bradycardia owing to drugs
 - _ Organophosphate insecticides
- Warnings:
 - _ Myasthenia gravis, narrow-angle glaucoma, HTN, coronary ischemia, and urinary obstruction
- Dose:
 - _ Adult: 1–2 mg IV
 - _ Pediatric: 0.02 mg/kg (min. 0.1 mg) IV
 - _ Large repeated doses needed in organophosphate poisoning

BENZTROPINE (COGENTIN)

- Indications: Acute dystonic reactions
- Warnings: Carbamates, myasthenia gravis, narrow-angle glaucoma, HTN, coronary ischemia, and urinary obstruction
- Dose:
 - _ Adult: 1–2 mg IV (for acute reaction) or PO (to prevent reaction)
 - _ Pediatric: 0.02 mg/kg IV (for acute reaction) or PO (to prevent reaction)

BENZODIAZEPINE

- Indications: Agitation, stimulant drugs, seizures
- Warnings: Respiratory/CNS depression
- Dose:
 - Midazolam:
 - Adult: 1 mg IV/IM every 2–3 min PRN
 - Pediatric: 0.1 mg/kg IV/IM
 - Diazepam:
 - Adult: 2–5 mg IV/IM, repeat in 10–15 min
 - Pediatrics: 0.1 mg/kg IV/IM

BICARBONATE, SODIUM

- Indications: Cyclic antidepressant poisoning, metabolic acidosis, urinary alkalinization
- Warnings: May cause CHF, excessive alkalosis, hypokalemia
- Dose:
 - Serum alkalinization:
 - 1 mEq/kg IVP
 - Urine alkalinization:
 - 100–150 mEq in 1 L DW at 2–3 mL/kg/h IV, goal urine pH 7–8

BLACK WIDOW SPIDER ANTIVENIN (LACTRODECTUS MACTANS)

- Indications: Severe HTN, muscle spasms not alleviated by analgesics and muscle relaxants; consider in extremes of age (<5 or >65 years), pregnant women with threatened abortion
- Warnings:
 - Equine serum derived: Immediate hypersensitivity, serum sickness 10–14 days
 - Premedicate for anaphylaxis if know equine serum hypersensitivity.
- Dose: 1–2 vials IM or IV slowly over 15–30 min; dilute 1 vial in 50 mL saline for IV

BOTULIN ANTITOXIN TRIVALENT A, B, E

- Indications: Clinical botulism, prior to onset of paralysis
- Warnings:
 - Binds only free toxins
 - Not for infant botulism
 - Equine serum derived: Immediate hypersensitivity, serum sickness 10–14 days
 - Premedicate for anaphylaxis if know equine serum hypersensitivity.
 - Administer slow IV push.
- Dose: 1–2 vials IV q4h for 4 or 5 doses; reconstitute 1 vial with 2 mL sterile water. Administer 0.5 mL/kg/h IV. Double rate after 15 min if no ill effects.

CALCIUM

- **Indications:**
 - Hyperkalemia with cardiac toxicity
 - Hydrofluoric acid burn
 - Calcium channel blocker overdose
 - Citrate, oxalate, phosphate poisoning
- **Warnings:**
 - Avoid in digoxin toxicity, hypercalcemia
 - Calcium chloride (CaCl) corrosive to skin, SC tissue
 - Incompatible with certain IV solutions
 - Administer slow IV push.
- **Dose:**
 - Adult: 5–10 mL of 10% CaCl, or 10–20 mL of 10% Ca gluconate
 - Pediatric: 0.1–0.2 mL/kg of 10% CaCl, or 0.2–0.3 mL/kg of 10% Ca gluconate

CALCIUM EDTA (EDETATE DISODIUM)

- **Indications:** Lead, chromium, nickel, manganese, zinc toxicity
- **Warnings:** Nausea, vomiting, chill, nephrotoxicity, hypercalcemia
- **Dose:** 1 g/m²/day IV over 8–12 hr for 5 days, skip 2–4 days, then repeat. Follow lead (Pb) level

CORAL SNAKE ANTIVENIN (MICRURUS FULVIUS)

- **Indications:** Eastern or Texas coral snake
- **Warnings:**
 - Equine serum derived: Immediate hypersensitivity, serum sickness 10–14 days
 - Premedicate for anaphylaxis if we know the equine serum hypersensitivity.
- **Dose:** 4–10 vials slow IV push over 15–30 min

CYANIDE ANTIDOTE KIT

- **Indications:** Cyanide poisoning
- **Warnings:** Hypotension, methemoglobinemia
- **Dose:**
 - Amyl nitrite: 1–2 amp crushed, inhaled
 - Use amyl nitrite only until IV access is established
 - Sodium nitrite:
 - Adult: 300 mg in 10 mL IV over 5 min
 - Pediatric: 0.3 mL/kg of 3% solution IV
 - Sodium thiosulfate:
 - Adult: 12.5 g IV, may repeat in 1 hr
 - Pediatric: 50 mg/kg IV

HYDROXOCOBALAMIN

- Indications: Cyanide poisoning
- Warnings: Erythema, HTN
- Dose:
 - Adult: 5 g IV over 15 min; may repeat a 2nd 5 g dose depending on severity of poisoning and clinical response. Max. 10 g. Reconstitute each 2.5 g vial with 100 mL 0.9% NaCl
 - Pediatric: Safety and efficacy have not been established in children. Suggested initial dose: 70 mg/kg IV.

DANTROLENE

- Indications:
 - Malignant hyperthermia
 - Neuroleptic malignant syndrome
 - Serotonin syndrome
 - Muscle rigidity
- Warnings: Muscle weakness, respiratory depression, hepatitis
- Dose: 1–2 mg/kg IV bolus, repeat q10–15 min PRN, max. 10 mg/kg

DEFEROXAMINE (DESFERAL)

- Indications: Iron toxicity
- Warnings:
 - Do not treat for > 24 hr, risk for delayed adult respiratory distress syndrome (ARDS).
 - Hypotension if > 15 mg/kg/h, flushing, urticaria
- Dose: 10–15 mg/kg/h IV, may increase in severe iron (Fe) poisoning

DIGOXIN ANTIBODY (DIGIBIND)

- Indications: Digoxin, digitoxin toxicity
- Warnings:
 - Falsely elevated digoxin levels after use
 - Development of CHF/atrial fibrillation in patients requiring digoxin
- Dose:
 - 1 vial (40 mg) binds 0.6 mg digoxin.
 - Number of vials = digoxin level (ng/mL) × weight (kg)/100
 - Dose estimate: Acute overdose 10–20 vials, chronic overdose 4–6 vials

DIMERCAPROL (BAL)

- Indications: Arsenic, gold, mercury, lead-induced encephalopathy
- Warnings: Renal toxicity, fever, nausea, vomiting, urticaria, cholinergic symptoms
- Dose:
 - 3 mg/kg deep IM q4h for 2 days, then q12h for 7 days; follow metal levels
 - For Pb level > 100 µg/dL: 4–5 mg/kg IM q4h until Pb < 50 µg/dL, in

conjunction with EDTA

DIPHENHYDRAMINE (BENADRYL)

- Indications: Antihistamine, acute dystonic reaction
- Warnings: Sedation, excitation in children, anticholinergic symptoms
- Dose:
 - Adult: 25–50 mg IV/IM/PO q4–6h
 - Pediatric: 0.5–1 mg/kg IV/IM/PO q4–6h

SUCCIMER, CHEMET

- Indications: Pediatric lead poisoning
- Warnings:
 - Caution in renal impairment—urinary elimination
 - Nausea, vomiting diarrhea
- Dose: 10 mg/kg PO q8h for 5 days, then q12h for 14 days, then reassess blood lead levels

EPINEPHRINE

- Indications: Angioedema, anaphylaxis, acute asthma, spinal shock, β -blocker overdose
- Warnings: Dysrhythmias, HTN, tremor, anxiety
- Dose:
 - Hypotension/shock:
 - Adult: 1–4 μ g/min IV infusion
 - Pediatric: Start IV infusion at 0.1 μ g/kg/min.
 - Anaphylaxis
 - Adult: 0.3–0.5 mg IM/SC
 - Pediatric: 0.01 mg/kg IM/SC

ETHANOL

- Indications: Methanol or ethylene glycol toxicity
- Warnings:
 - Disulfiram reaction, CNS sedation
 - Hypoglycemia in pediatric population
 - Increase dose during dialysis, for chronic alcoholics.
- Dose:
 - IV: 10 mL/kg load as 10% solution over 1 hr, then 1 mL/kg/h maintenance
 - PO: 1.5 mL/kg as 100-proof solution, then 0.3 mL/kg/h maintenance
 - Goal: Ethanol level of 100–150 mg/dL

FLUMAZENIL (ROMAZICON)

- Indications: Benzodiazepine overdose
- Warnings:

- Contraindicated in tricyclic antidepressant (TCA) overdose
- Lowers seizure threshold
- Induces benzodiazepine withdrawal
- Dose:
 - Adult: 0.2 mg IV slow, repeat q2–3min to 1 mg max.
 - Pediatric: 0.01–0.05 mg/kg IV over 30 min–1 hr

FOMEPIZOLE (4-MP, ANTIZOL)

- Indications: Methanol or ethylene glycol toxicity
- Warnings: Nausea, dizziness, headache
- Dose: 15 mg/kg load IV, then 10 mg/kg q12h for 4 doses, then 15 mg/kg q12h

GLUCAGON

- Indications:
 - β -blocker or calcium channel blocker overdose with bradycardia/hypotension
 - Hypoglycemia
- Warnings:
 - Nausea, vomiting, hyperglycemia
 - Hypotension from diluent (phenol containing)
- Dose:
 - β -blocker or calcium channel blocker overdose:
 - Adult: 5–10 mg IV over 1 min
 - Pediatric: 0.15 mg/kg IV over 1 min
 - Hypoglycemia:
 - Adult: 0.5–1 mg IM/IV/SC
 - Pediatric: 0.025–0.1 mg/kg IM/IV/SC (max. 1 mg per dose)

INSULIN/GLUCOSE

- Indications:
 - Calcium channel blocker overdose with severe hypotension/symptomatic bradycardia refractory to other therapies
 - Hyperkalemia
- Warnings:
 - Experimental therapy: Consult a poison control center/medical toxicologist.
 - Follow serum glucose q15min for 1 hr after the 1st bolus or after any increase in dose, then q1h
- Dose:
 - Bolus:
 - 0.5–1 IU/kg regular insulin, followed by 25 g glucose (1 amp D₅₀)
 - Maintenance:
 - Insulin 0.5 IU regular insulin per kg/hr, titrate to 1 IU regular insulin per kg/hr

- Glucose D₁₀ start at 100 mL/h (10 g/h) and titrate to keep glucose ≥ 100 mg/dL

INTRALIPIDS

- Indications:
 - Cardiac arrest due to local anesthetic toxicity, most commonly bupivacaine, however may be useful for other lipid-soluble drugs
- Warnings:
 - Experimental therapy: Consult a poison control center/medical toxicologist.
- Dose:
 - Intralipid 20% bolus; 1.5 mL/Kg over 1 min followed by infusion 0.25 mL/kg/min.
 - Repeat bolus in 3–5 min if circulation not restored.

METHYLENE BLUE

- Indications: Methemoglobinemia with dyspnea or > 25%
- Warnings: G6-PD deficiency
- Dose: 1–2 mg/kg slow IV as 1% solution, repeat in 1 hr

NARCAN

- Indications:
 - Opiate poisoning, empiric treatment of coma
- Warnings:
 - Acute opiate withdrawal, severe agitation
- Dose:
 - Adult: 0.4–2 mg IV or IM, repeat to 10 mg
 - Pediatric: 0.1 mg/kg IV or IM

OCTREOTIDE

- Indications: Sulfonylurea overdose with hypoglycemia
- Warnings: Use with caution in diabetic patients.
- Dose:
 - Adult: 50 µg SC q6h
 - Pediatric: 4–5 µg/kg/d SC div. q6h

OXYGEN, HYPERBARIC

- Indications: Carbon monoxide (CO) poisoning
- Warnings:
 - Tympanic membrane (TM) perforation, seizures owing to oxygen toxicity
 - Difficulty monitoring patient
- Dose: 100% oxygen at 2–3 atm

PENICILLAMINE

- Indications: Arsenic, copper, lead, mercury with/following BAL or EDTA
- Warnings: Contraindicated in penicillin allergy, renal insufficiency
- Dose:
 - _ Lead:
 - Adult: 250–500 mg per dose PO q8–12h
 - Pediatric: 25–40 mg/kg/d PO in 3 div. doses
 - _ Arsenic: 100 mg/kg/d PO div. in 4 doses for 5 days (max. 1 g/d)
 - _ Mercury:
 - Adult: 250 mg PO QID
 - Pediatric: 20–30 mg/kg/d PO in 4 div. doses

PHENTOLAMINE

- Indications:
 - _ Hypertensive crisis: Stimulants, sympathomimetics, MAO–tyramine reaction, and extravasated pressors
 - _ Reversal of cocaine-mediated vasospasm
- Warnings: HTN, tachycardia, dysrhythmias
- Dose:
 - _ HTN (HTN):
 - Adult: 1–5 mg IV bolus
 - Pediatric: 0.02–0.1 mg/kg bolus
 - _ Extravasation:
 - Adult: 5 mg diluted in 10–15 mL saline SC
 - Pediatric: 0.1 mg/kg diluted in 10–15 mL saline SC

PHYSOSTIGMINE

- Indications: Severe anticholinergic syndrome
- Warnings: Contraindicated in TCA overdose
- Dose:
 - _ Adult: 0.5–1 mg IV, repeat in 10 min PRN
 - _ Pediatric: 0.02 mg/kg IV, repeat in 10 min PRN

PRALIDOXIME (2-PAM, PROTOPAM)

- Indications:
 - _ Organophosphate toxicity
 - _ Reversal of nicotinic effects
 - _ Reactivates enzyme
 - _ Use in conjunction with atropine
- Warnings:
 - _ Myasthenic crisis if myasthenia gravis
 - _ Nausea, headache, dizziness, laryngospasm, muscle rigidity
- Dose:
 - _ Adult: 1–2 g IV in 100 mL NaCl over 15 min, repeat in 1 hr PRN, repeat in 6

hr if nicotinic symptoms return

- Pediatrics: 25–50 mg/kg over 15 min, repeat in 1 hr PRN, repeat in 6 hr if nicotinic symptoms return

PROTAMINE

- Indications: Reversal of heparin anticoagulation
- Warnings:
 - Hypersensitivity in patients with fish allergy
 - Avoid benzyl alcohol diluent in neonates.
- Dose: 1 mg for each 100 IU heparin, 1/2 dose if 30–60 min; 1/4 dose if 2 hr after heparin bolus;
 - 25–50 mg slow IV over 15 min. Initial dose should not be > 50 mg

PYRIDOXINE (VITAMIN B₆)

- Indications:
 - Isoniazid-induced seizures
 - *Gyromitra* mushroom
- Warnings: None, nontoxic
- Dose:
 - Isonicotinic acid hydrazide (INH)–induced seizures:
 - Unknown ingested amount: 5 g for adult or 1 g for pediatrics
 - Dose (mg) = amount INH ingested (mg)
 - *Gyromitra*: 25 mg/kg IV over 30 min–1 hr

RATTLESNAKE ANTIVENIN (CROTALINE)

- Indications: Significant envenomation by *Crotaline* species: Rattlesnake, cottonmouth, water moccasin, pit viper
- Warnings:
 - Ovine-derived products: Immediate hypersensitivity, serum sickness 10–14 days
 - Premedicate for anaphylaxis if we know the equine/ovine serum hypersensitivity.
- Dose:
- Ovine derived (CroFab):
 - 4–6 vials slowly; may repeat dose of 4–6 vials if control of envenomation not achieved, then 2 vials q6h for 3 doses
 - Reconstitute each vial with 25 mL sterile water. Dilute in 250 mL 0.9% NaCl and infuse over 1 hr.

VITAMIN K (PHYTONADIONE, AQUA MEPHYTON)

- Indications: Reversal of Coumadin anticoagulation
- Warnings: Hypersensitivity from IV administration
- Dose:

- 2–10 mg SC/slow IV, may repeat in 8 hr
- 2–10 mg PO, may repeat in 12–48 hr



CODES

ICD9
977.9 Poisoning by unspecified drug or medicinal substance

ICD10
T65.91XA Toxic effect of unspecified substance, accidental (unintentional), initial encounter

POISONING, GASTRIC DECONTAMINATION

Frank LoVecchio

BASICS

DESCRIPTION

Modalities to decontaminate the GI tract of poisons

TREATMENT

ALERT

- Ipecac is contraindicated in ambulance setting.
- Controversies:
 - _ Home use of ipecac in general is not recommended.
 - _ In extremely rare cases (e.g., very prolonged transit times, protecting airway), consider ipecac administration only after consultation with regional poison control center.
 - _ Decreased time to activated charcoal administration when given in pre-hospital setting

INITIAL STABILIZATION/THERAPY

- Airway, breathing, and circulation management (ABCs):
 - _ Secure airway for decreased mental status/inability to protect airway.
 - _ IV access
 - _ Cardiac monitor
- With altered mental status from overdose:
 - _ Naloxone
 - _ Thiamine
 - _ Dextrose (or Accu-Chek)

ED TREATMENT/PROCEDURES

- Activated charcoal:
 - _ General:
 - Prepared by treating heated wood pulp, which creates a large surface area to bind toxins
 - Mainstay of gastric decontamination
 - Effective when contents have reached small intestine
 - _ Dose:
 - 1–2 g/kg of body weight or an activated charcoal-to-drug ratio of 10:1; often mixed with sorbitol (see below)
 - Oral or nasogastric tube administration

- Indications:
 - Administer in every toxic ingestion (see below for exceptions).
 - Optimal for toxic ingestions presenting within 1 hr of ingesting a drug that is absorbed by charcoal in a patient with a patent airway
- Adverse effects:
 - Vomiting and constipation
 - Charcoal aspiration and subsequent charcoal pneumonitis
- Contraindications:
 - Caustic ingestions
 - Unprotected airway
 - Bowel obstruction or ileus
- Drugs not effectively bound to charcoal:
 - Metals (borates, bromide, iron, lithium)
 - Alcohols
 - Potassium
 - Potassium cyanide (poorly absorbed)
 - Hydrocarbons
 - Caustics
- Pediatric considerations:
 - Mix with palatable substance (cola or juice) to facilitate intake or administer via gastric tube.
- Controversies:
 - Randomized, controlled trials have shown a slightly worse outcome and higher complication rate when *asymptomatic* patients received charcoal vs. nothing.
 - An extremely small minority of patients are likely to benefit from gastric lavage.
- **Multiple-dose activated charcoal:**
 - General:
 - Used in toxic ingestions that are well absorbed by charcoal and undergo enterohepatic circulation
 - Dose:
 - 1 g/kg followed by 0.5 g/kg q2–6h
 - Never use cathartics in conjunction with multiple-dose activated charcoal.
 - Indications:
 - Salicylates
 - Theophylline
 - Multiple-dose activated charcoal may decrease area under the curve for drugs such as phenobarbital, phenytoin, and carbamazepine but has not been proven to improve outcome.
- **Cathartics:**
 - General:

- Used in combination with activated charcoal to prevent constipation and to enhance GI transit time
- Limited data available to demonstrate any decreased absorption when a cathartic (sorbitol) is added to activated charcoal
- Cathartics alone are of no proven benefit and should be avoided.
- *Never* use cathartics in conjunction with multiple-dose activated charcoal.

– Dose:

- Magnesium citrate: 10% solution: 250 mL (peds: 4 mL/kg)
- Magnesium sulfate: 15–20 g (peds: 250 mg/kg)
- Sorbitol: 0.5–1 g/kg to a max. 100 g of 70% solution (peds: > 1 yr old: 0.5–1 g/kg as a 35% solution to a max. 50 g) PO mixed in the activated charcoal slurry—use only in 1st dose.

– Adverse effects:

- Dehydration
- Hypermagnesemia
- Diarrhea
- Abdominal discomfort

– Contraindications:

- Pre-existing dehydration
- Renal disease (cathartics containing magnesium)
- Avoid in children

– Controversies:

- No proven benefit and some cases of harm reported

● **Whole-bowel irrigation:**

– General: Cleansing of bowel

– Indications:

- Toxins not well absorbed by charcoal, such as toxic iron and lithium ingestions
- Toxins in sealed containers (body packers) without signs of GI perforation
- Toxic, sustained-release product ingestions

– Dose:

- Polyethylene glycol (Colyte, GoLytely)
- Solution at 2 L/hr in adults (0.5 L/hr in children) until rectal excretions clear
- Administer via nasogastric tube with activated charcoal via continuous or bolus method as indicated.

– Adverse effects:

- Bloating
- Rectal irritation
- Frequent bowel movements

– Contraindications:

- Mechanical or pharmacologic ileus
- Bowel obstruction
- Hypotension
- Intestinal perforation
- Unprotected airway
- **Orogastric lavage:**
 - General:
 - Placement of large-bore tube (32F–36F) in stomach for removal of ingested toxins
 - Effectiveness of orogastric lavage depends on time since ingestion, timing of last meal, and toxin ingested.
 - Protected airway is essential prior to any attempts at orogastric lavage.
 - Indications:
 - Currently, rarely performed
 - Presentation within 1 hr of taking a potentially lethal ingestion with no known antidote
 - Poisoned intubated patient arriving within ~ 1 hr
 - Adverse effects:
 - Intubation of respiratory tree
 - Esophageal or gastric perforation
 - Charcoal aspiration
 - Patient discomfort
 - Contraindications:
 - Large pills (limited by lavage-tube port size) ingestion
 - Caustics (acids and alkali) ingestion
 - Hydrocarbon ingestion
 - Ingestion of agents that rapidly depress mental status
 - Unprotected airway
 - Pediatric considerations:
 - Avoid in children
 - Unlikely to result in any clinically significant pill extraction secondary to smaller-bore orogastric tube (i.e., 18F)
 - Risk of aspiration increased in children
 - Controversies: Several randomized, controlled trials have documented no benefit when lavage plus activated charcoal is compared with activated charcoal alone.
- **Ipecac:**
 - General:
 - Rarely used
 - Derived from the roots of the plant *Cephaelis acuminata*
 - Exerts emetic action by direct gastric irritation and centrally mediated chemoreceptive trigger-zone stimulation

- Delays administration of activated charcoal
- Offers no advantage over activated charcoal alone when both treatments are potentially effective
- Dosage:
 - >12 yr: 30 mL
 - 1–12 yr: 15 mL
 - 6 mo–1 yr: 5–10 mL + 15 mL clear fluid
- Indications:
 - No utility in ED
- Adverse effects:
 - Vomiting may complicate and worsen clinical presentation.
 - Delay to administration of activated charcoal or oral antidotes
- Contraindications:
 - Caustics (acids and alkali) ingestion
 - Hydrocarbon ingestion
 - Ingestion of agents that rapidly depress mental status
 - Patient actively vomiting

PEARLS AND PITFALLS

- Ipecac has no utility in the ED.
- Administer activated charcoal in almost every toxic ingestion that presents within 1 hr with a patent airway
- Never use multiple doses of cathartic in conjunction with multiple-dose activated charcoal.

ADDITIONAL READING

- Albertson TE, Owen KP, Sutter ME, et al. Gastrointestinal decontamination in the acutely poisoned patient. *Int J Emerg Med.* 2011;4:65.
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See Also (Topic, Algorithm, Electronic Media Element)

- Poisoning
- Poisoning, Antidotes
- Poisoning, Toxidromes



CODES

977.9 Poisoning by unspecified drug or medicinal substance

ICD10

T65.91XA Toxic effect of unspecified substance, accidental (unintentional), initial encounter

POISONING, TOXIDROMES

Michael S. Wahl



BASICS

DESCRIPTION

- A toxidrome is the constellation of signs and symptoms that result from the effects of a particular toxin (toxic syndrome)
- Mechanism of action varies with each class of toxin to which the patient may be exposed and the target receptors.



DIAGNOSIS

SIGNS AND SYMPTOMS

Toxidromes

- There are multiple toxidromes:
 - Anticholinergic
 - Cholinergic
 - Sympathomimetic
 - Hallucinogenic
 - Opiate
 - Sedative–hypnotic
 - Withdrawal syndromes
 - Serotonin syndrome
 - Malignant neuroleptic syndrome
- **Anticholinergic:** Mnemonic: *“Blind as a bat, mad as a hatter, red as a beet, hot as a hare, dry as a bone, the bowel and bladder lose their tone, and the heart runs alone”*:
 - Hyperthermia (“hot as a hare”)
 - Dry, flushed skin (“dry as a bone” and “red as a beet”)
 - Dilated pupils (“blind as a bat”)
 - Delirium (“mad as a hatter”)
 - Tachycardia (“the heart runs alone”)
 - Hypertension
 - Hyperthermia
 - Urgency retention (“bowel and bladder lose their tone”)
 - Decreased bowel sounds (“bowel and bladder lose their tone”)
 - Seizures
 - Mental status changes
 - Somnolence
- **Cholinergic:** Mnemonic: *DUMBELS for the muscarinic component*:
 - Muscarinic signs:

- Diarrhea, diaphoresis
- Urination
- Miosis
- Bradycardia, bronchorrhea, bronchospasm (the killer Bs)
- Emesis
- Lacrimation
- Salivation
- Nicotinic signs:
 - Mydriasis
 - Tachycardia
 - Weakness
 - Hypertension
 - Fasciculations
- **Sympathomimetic:** Similar to anticholinergic presentation except for skin and bowel differences (diaphoresis and increased bowel sounds may be present in sympathomimetic presentations):
 - Diaphoresis
 - Mydriasis
 - Tachycardia
 - Hypertension
 - Hyperthermia
 - Seizures
 - Increased peristalsis
- **Hallucinogenic:** May have significant overlap with sympathomimetic toxidrome as many sympathomimetic drugs have hallucinogenic properties (e.g., MDMA/ecstasy, cathinones, hallucinogenic amines). Other hallucinogens include LSD, psilocybin, peyote, mescaline:
 - Disorientation
 - Hallucinations
 - Anxiety
 - Panic
 - Seizures
- **Opiate:**
 - Classic triad:
 - Miosis
 - Hypoventilation
 - Coma
 - May also present with:
 - Bradycardia
 - Hypotension
 - Hypothermia
 - Decreased bowel sounds
- **Sedative–hypnotics and alcohol:**

- Sedation
- Mental status changes (confusion, delirium, hallucinations)
- Vision changes (blurred vision, diplopia)
- Slurred speech
- Ataxia
- Nystagmus
- **Withdrawal** (alcohol, benzodiazepine, barbiturates):
 - Mydriasis
 - Tachycardia
 - Hypertension
 - Hyperthermia
 - Increased respiratory rate
 - Diaphoresis
 - Increased bowel sounds
 - Tremor
 - Agitation
 - Anxiety
 - Hallucinations
 - Confusion
 - Seizures
- **Withdrawal** (opioid):
 - Nausea
 - Vomiting
 - Diarrhea
 - Abdominal cramps
 - Increased bowel sounds
 - Mydriasis
 - Piloerection
 - Tachycardia
 - Lacrimation
 - Salivation
 - Hypertension
 - Yawning
- **Neuroleptic malignant syndrome:**
 - Recent treatment with typical and atypical antipsychotic medications:
 - Generally occurs from hours to several weeks of starting or increasing the dose of a medication, but can occur at any time.
 - Hyperthermia
 - Muscular rigidity
 - Diaphoresis
 - Mental status changes
 - Hypertension or hypotension may be seen
 - Sialorrhea

- Tremor
- Incontinence
- Increased creatinine phosphokinase
- Leukocytosis
- Metabolic acidosis
- **Serotonin syndrome:**
 - Occurs soon after the increase in dose or addition of serotonergic medications.
 - Syndrome with variable presentation
 - Following are most common, seen 25–57% of the time:
 - Mental status changes (confusion, agitation, hypomania, lethargy)
 - Seizures
 - Myoclonus
 - Hyperreflexia
 - Muscle rigidity
 - Tremor
 - Nystagmus
 - Hyperthermia
 - Diaphoresis
 - Tachycardia
 - Hypertension
 - Mydriasis

Physical-Exam

- **Bradycardia:**
 - α_2 -adrenergic agonists (e.g., clonidine)
 - β -blockers
 - Calcium-channel blockers
 - Digoxin and related substances
 - Cholinergics
 - Opioids
- **Tachycardia:**
 - Sympathomimetics
 - Anticholinergics
 - Methylxanthines
 - Tricyclic antidepressant
 - Withdrawal
 - Phenothiazines
 - Atypical antipsychotics
 - α_1 -blockade with reflex tachycardia
 - Phosphodiesterase type 5 inhibitor (e.g., Sildenafil)
- **Hyperthermia:**

- Anticholinergics
- Sympathomimetics
- Serotonin syndrome
- Neuroleptic malignant syndrome
- Malignant hyperthermia
- Dinitrophenol
- Salicylates
- Withdrawal
- **Hypothermia:**
 - Carbon monoxide
 - Oral hypoglycemics
 - Opiates
 - Ethanol
 - Sedative–hypnotics
 - α_2 -adrenergic agonists
- **Hypertension:**
 - Sympathomimetics
 - Anticholinergics
 - Nicotine
 - Phencyclidine (PCP)
 - Ergot alkaloids
- **Hypotension:**
 - α_2 -agonists
 - α_1 -antagonists
 - β -blockers
 - Calcium-channel blockers
 - Angiotensin converting–enzyme inhibitors
 - Methylxanthines
 - Nitrates
 - Opioids
 - Phenothiazines
 - Phosphodiesterase type 5 inhibitors
 - Sedative–hypnotics
 - Ethanol
 - Tricyclic antidepressants
 - Atypical antipsychotic medications
- **Miosis:**
 - Cholinergics
 - Clonidine
 - Reserpine
 - Phenothiazines
 - Atypical antipsychotics

- **Mydriasis:**
 - Anticholinergics
 - Sympathomimetics
 - Withdrawal (esp. opioids)
 - Botulism
- **Seizures:** *Mnemonic with a limited list of causes for toxic seizures OTIS CAMPBELL:*
 - Organophosphates
 - Tricyclic antidepressants
 - Isoniazid, insulin
 - Sympathomimetics, salicylates
 - Camphor, cocaine, citalopram
 - Amphetamines, anticholinergic agents
 - Methylxanthines (theophylline, caffeine), mushrooms (*Gyromitra*: monomethyl hydrazine group), meperidine
 - PCP, propoxyphene, plants (nicotine, water hemlock)
 - Benzodiazepine withdrawal, bupropion
 - Ethanol withdrawal
 - Lithium, lidocaine
 - Lead, lindane
- **Diaphoresis:**
 - Sympathomimetics
 - Cholinergics
 - Salicylates
 - Withdrawal
 - Serotonin syndrome
- **Bradypnea:**
 - Opiates
 - Sedative–hypnotics
 - Ethanol
 - γ -hydroxybutyric acid and congeners
 - Botulism
 - Muscular receptor blockade
- **Tachypnea:**
 - Paraquat (and other drugs that cause pneumonitis)
 - Salicylates
 - Sympathomimetics
 - Dinitrophenol
 - Methylxanthines
 - Drugs that cause acidosis

Dermatologic

- **Mees lines:**
 - Arsenic
 - Thallium

- Chemotherapy agents
- Radiation
- Bullae:
 - Barbiturates
 - Carbon monoxide
 - Captopril
- Flushed or red appearance:
 - Anticholinergics
 - Disulfiram reactions
 - Niacin
 - Boric acid
 - Scombroid poisoning
 - Monosodium glutamate
 - Carbon monoxide (frequently postmortem)
 - Cyanide (rare)
 - Vancomycin
- Blue skin:
 - Ergotamines
 - Methemoglobinemia from:
 - Nitrite
 - Nitrate
 - Dapsone
 - Aniline dye
 - Phenazopyridine
 - Benzocaine
 - Chloroquine
 - Pseudocyanosis from:
 - Chlorpromazine
 - Amiodarone
 - Minocycline
 - Silver (argyria)
 - Gold (chrysiasis)

ESSENTIAL WORKUP

Depends on ingested substance:

- CBC
- Electrolytes, BUN, creatinine, glucose
- Urinalysis
- Arterial blood gas, venous blood gas
- Carboxyhemoglobin, methemoglobin levels
- Toxicology screen
- Aspirin and Acetaminophen level
- Prothrombin time

- Liver function tests

DIAGNOSIS TESTS & INTERPRETATION

- **Anion gap acidosis:** Mnemonic: *A CAT MUD PILES* (encompasses a limited number of common causes):
 - Alcohol ketoacidosis
 - CO/cyanide
 - Acetaminophen in fulminant hepatic failure
 - Toluene
 - Methanol
 - Uremia
 - Diabetic ketoacidosis
 - Paraldehyde, phenformin/metformin
 - Iron, isoniazid
 - Lactic acidosis
 - Ethylene glycol
 - Salicylates, sodium azide, hydrogen sulfide
- **Increased osmolar gap:**
 - Methanol
 - Ethylene glycol
 - Isopropyl alcohol
 - Ethanol
 - Acetone
 - Glycerol
 - Mannitol
 - Glycine



TREATMENT

INITIAL STABILIZATION/THERAPY

ABCs

ED TREATMENT/PROCEDURES

Depends on ingested substance (see Poisoning; Poisoning, Gastric Decontamination)

PEARLS AND PITFALLS

- Obtain appropriate lab tests.
- Recognize signs and symptoms and lab clues to the toxidromes.

ADDITIONAL READING

- Boyer EW, Shannon M. The serotonin syndrome. *N Engl J Med.* 2005;352:1112–

1120.

- Nelson L, Lewin N, Howland MA, et al. *Goldfrank's Toxicologic Emergencies*. 9th ed. New York, NY: McGraw-Hill; 2010.
- Weatherald J, Marrie TJ. Pseudocyanosis: Drug-induced skin hyperpigmentation can mimic cyanosis. *Am J Med*. 2008;121(5):385–386.



See Also (Topic, Algorithm, Electronic Media Element)

- [Poisoning](#)
- [Poisoning, Gastric Decontamination](#)



CODES

ICD9

- [971.0 Poisoning by parasympathomimetics \(cholinergics\)](#)
- [971.1 Poisoning by parasympatholytics \(anticholinergics and antimuscarinics\) and spasmolytics](#)
- [971.2 Poisoning by sympathomimetics \[adrenergics\]](#)

ICD10

- [T44.1X1A Poisoning by oth parasympath, accidental, init](#)
- [T44.3X1A Poisoning by oth parasympath and spasmolytics, acc, init](#)
- [T44.901A Poiso by unsp drugs aff the autonm nervous sys, acc, init](#)

POLIO

Philip Shayne • Marie Carmelle Tabuteau

BASICS

DESCRIPTION

- Caused by poliovirus infection
- Incubation period 7–14 days
- Duration < 1 wk
- Clinical manifestations are defined as follows:
 - Subclinical (i.e., not apparent) 90–95%
 - Abortive poliomyelitis 4–8%:
 - Clinically indistinct from many other viral infections (fever, myalgias, malaise)
 - Only suspected to be polio during an epidemic
 - Nonparalytic poliomyelitis 1–2%:
 - Differs from abortive poliomyelitis by the presence of meningeal irritation
 - Course similar to any aseptic meningitis
 - Paralytic poliomyelitis 0.1%, which is further subdivided:
 - Spinal paralytic poliomyelitis (frank polio)
 - Bulbar paralytic poliomyelitis (10% of paralytic polio): Paralysis of muscle groups innervated by cranial nerves; involves the circulatory and respiratory centers of the medulla with high mortality
 - Mixed bulbospinal poliomyelitis
 - Postpoliomyelitis syndrome:
 - New onset of increased muscle weakness, pain, and focal or generalized atrophy
 - Occurs 8–70 yr after the active illness, usually in the previously affected limb
 - Risk factors include age at time of infection, extent of recovery and female sex (increased risk with better recovery)
 - Gradual progression

ETIOLOGY

- Polioviruses:
 - Picornaviruses
 - Small, nonenveloped RNA viruses of the enterovirus genera
 - 3 subtypes: 1, 2, 3
- Fecal–oral route transmission
 - Enters through oral cavity

- Replicates in pharynx, GI tract, and lymphatics
- Humans are the only natural host and reservoir
- Poliovirus selectively destroys motor and autonomic neurons
- Natural (wild) virus has been completely eliminated in US since 1979
- Oral poliovirus vaccine (OPV):
 - Accounts for only poliomyelitis seen in US
 - 8–10 cases/yr of vaccine-associated paralytic poliomyelitis (VAP): Neurovirulent conversion of vaccine virus; decreased since widespread use of inactivated poliovirus vaccine (IPV)
 - VAP occurs in poorly immunized regions by acquiring properties of wild-type virus.
 - There has been a recent increase in some third world countries



DIAGNOSIS

SIGNS AND SYMPTOMS

- Primarily asymptomatic
- Viral symptoms: Fever, headache, malaise. Respiratory symptoms: Sore throat, fatigue GI symptoms: Nausea, vomiting
- Nonparalytic aseptic meningitis: Stiff neck, and or back
- Muscle pain and weakness
- Progressive weakness for < 1 wk:
- Dysphagia and dysarthria with bulbar involvement

History

- Vaccination history
- History of prior polio infection
- Recent exposure to individual vaccinated with OPV
- Recent travel to endemic countries (Nigeria, Pakistan, India, Afghanistan)
- Comorbid conditions affecting immunocompetence especially B-lymphocyte disorders (e.g., hypogammaglobulinemia and agammaglobulinemia)

Physical-Exam

- Fever (37°C –39°C)
- Headache, photophobia
- Nuchal rigidity
- Neurologic changes:
 - Muscle soreness that becomes severe muscle spasm, progressing rapidly to spotty flaccid weakness and paralysis
 - Asymmetric paralysis more prominent in the lower than the upper extremities
 - Urinary retention (50% of paralytic cases)

- Reflexes initially hyperactive, then absent
- Apprehensive and irritable, occasionally drowsy
- No sensory loss associated with the motor deficit

Pediatric Considerations

More likely to have a biphasic acute course:

- Viral-type syndrome for 1–2 days
- Symptom-free period of 2–5 days
- Then an abrupt onset of the major illness

ESSENTIAL WORKUP

- Clinical diagnosis
- Differentiate from other causes of acute paralysis.
- Notify public health officials when diagnosis suspected.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC:
 - WBC normal or mildly elevated
- Diagnosis confirmed by:
 - Comparing acute with convalescent sera for antigen titers
 - Isolation of virus from blood, CSF, stool, throat secretions (within week 1 of infection)

Diagnostic Procedures/Surgery

- Lumbar puncture/CSF analysis:
 - Abnormalities typical of aseptic meningitis (increased lymphocytes and elevated protein)
 - Poliovirus rarely isolated from the CSF
- Electrodiagnostics:
 - Normal to slow motor function
 - Sensory function intact

DIFFERENTIAL DIAGNOSIS

- Abortive poliomyelitis is similar to many viral illnesses.
- Nonparalytic poliomyelitis is indistinguishable from any viral, aseptic meningitis.
- Paralytic poliomyelitis:
 - Amyotrophic lateral sclerosis
 - Guillain–Barré (not febrile, symmetric, not ill appearing)
 - Acute transverse myelitis
 - Spinal cord compression/infarction
 - Multiple sclerosis

- Rhabdomyolysis
- Acute intermittent porphyria
- West Nile virus
- Diphtheria
- Botulism
- Tick paralysis
- Encephalitis



TREATMENT

ALERT

Rare fatal cases come from respiratory insufficiency, which requires prompt ventilatory support.

INITIAL STABILIZATION/THERAPY

Aggressive pulmonary toilet and early intubation mandated for respiratory insufficiency

ED TREATMENT/PROCEDURES

- Supportive and symptomatic management
- Analgesics for severe muscle pain and spasm
- Bed rest to prevent augmentation or extension of paralysis
- Paralytic poliomyelitis tends to localize to a limb that has been the site of intramuscular injection or injury within 2–4 wk prior to the onset of infection:
 - Avoid any unnecessary tissue damage in suspected cases
 - No antiviral agents available
 - Prevention
- Prevention
- IPV:
 - Costly
 - Painful
 - No conferred immunity
 - No VAP, which previously accounted for all poliomyelitis cases in US
- OPV:
 - Accounted for only poliomyelitis seen in US (8–10 cases/yr)
 - Incidence of VAP: 1/900,000 (immunocompromised: 1/1,000):
 - Most at risk are the underimmunized young and their caretakers.
 - Confers immunity to unvaccinated contacts by fecal–oral spread.
 - Inexpensive
 - No longer available in US
 - Still remains the vaccine recommended by WHO Expanded Program on Immunization



FOLLOW-UP

DISPOSITION

Admission Criteria

All acute-phase paralytic poliomyelitis for strict bed rest and observation for respiratory symptoms:

- Isolate from nonvaccinated personnel.

Discharge Criteria

No evidence of nervous system involvement and no danger of contact with nonvaccinated population:

- Deterioration of muscle strength usually ends after 3–5 days

FOLLOW-UP RECOMMENDATIONS

Physical therapy:

- Only 1/3 of the people with acute flaccid paralysis regain full strength
- Lamotrigine may decrease pain, improve symptoms and quality of life.
- IV immunoglobulin (IVIg) may improve muscle strength, has not been proven to decrease pain or improve quality of life.

PEARLS AND PITFALLS

- Most cases are asymptomatic, with symptoms ranging from viral illness to acute flaccid paralysis.
- IPV is the only vaccine available in US; however OPV is still the vaccine of choice for global eradication.
- Diagnosis is primarily clinical and is confirmed by virus isolation from blood, CSF, stool, or throat secretions.
- Treatment is supportive; all acute-phase paralytic poliomyelitis patients should be admitted for observation with close monitoring of the respiratory system.
- If the patient survives the acute stage, paralysis of respiration and deglutition usually recovers completely.
- Paralytic poliomyelitis may occur decades after initial infection and manifests with neurologic and non-neurologic symptoms.

ADDITIONAL READING

- Alexander L, Birkhead G, Guerra F, et al. Ensuring preparedness for potential poliomyelitis outbreaks: Recommendations for the US poliovirus vaccine stockpile from the National Vaccine Advisory Committee (NVAC) and the Advisory Committee on Immunization Practices (ACIP). *Arch Pediatr Adolesc Med.* 2004;158:1106–1112.

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- Centers for Disease Control and Prevention (CDC). Updated recommendations of the Advisory Committee on Immunization Practices (ACIP) regarding routine poliovirus vaccination. *MMWR Morb Mortal Wkly Rep*. 2009;58(30):829–830.
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- Shahzad A, Köhler G. Inactivated polio vaccine (IPV): A strong candidate vaccine for achieving global polio eradication program. *Vaccine*. 2009;27(39):5293–5294.



See Also (Topic, Algorithm, Electronic Media Element)

- Amyotrophic lateral sclerosis
- Botulism
- Encephalitis
- Guillain–Barré Syndrome
- Multiple Sclerosis
- Rhabdomyolysis
- Spinal Cord Syndromes
- Tick Bite
- West Nile Virus



CODES

ICD9

- 045.00 Acute paralytic poliomyelitis specified as bulbar, poliovirus, unspecified type
- 045.20 Acute nonparalytic poliomyelitis, poliovirus, unspecified type
- 045.90 Acute poliomyelitis, unspecified, poliovirus, unspecified type

ICD10

- A80.30 Acute paralytic poliomyelitis, unspecified
- A80.4 Acute nonparalytic poliomyelitis
- A80.39 Other acute paralytic poliomyelitis

POLYCYTHEMIA

David N. Zull



BASICS

DESCRIPTION

- Increase in hemoglobin (Hgb) above the normal range:
 - _ Men: Hgb > 17.5 g/dL, hematocrit (Hct) > 52%
 - _ Women: Hgb > 16 g/dL, Hct > 48%
- Symptoms are related to blood viscosity, which increases exponentially at Hct > 60%.

ETIOLOGY

- Relative (apparent) polycythemia:
 - _ Resulting from decrease in plasma volume
 - _ Acute: Dehydration
 - _ Chronic: Gaisbock syndrome (stress polycythemia): Obese, hypertensive, middle-aged smokers
- Primary erythrocytosis:
 - _ Polycythemia vera (PV): A stem cell disorder characterized by panhyperplasia of all bone marrow elements leading to increased production of RBCs, WBCs, and platelets. Erythrocytosis is the most prominent feature:
 - Mutation in the tyrosine kinase (JAK2), which acts in signaling pathways of the EPO-receptor, rendering those cells hypersensitive to erythropoietin
 - Median age 60, 5% < 40, peak in 70s
 - Higher in Ashkenazi Jews and lower in Asians and African Americans.
 - May progress to myelofibrosis or acute leukemia
- Secondary polycythemia:
 - _ Central hypoxia increasing erythropoietin:
 - Chronic pulmonary disease
 - Sleep apnea (5–10% have high Hgb)
 - Obesity hypoventilation syndrome (Pickwickian syndrome)
 - Congenital heart disease (right-to-left shunt)
 - High altitude (chronic)
 - Smoker's erythrocytosis
 - Carbon monoxide poisoning (chronic)
 - Chronic methemoglobinemia
 - _ Renal-mediated causes of increased erythropoietin production:
 - Renal cell carcinoma

- Renal artery atherosclerotic narrowing
- Focal glomerulonephritis
- Postrenal transplant with or without rejection
- Chronic hydronephrosis
- Polycystic kidney disease and renal cysts
- Inappropriate autonomous erythropoietin production:
 - Hepatomas
 - Cerebellar hemangioblastoma
 - Wilms tumor
 - Parathyroid carcinoma and adenoma
 - Ovarian tumors
 - Adrenal adenomas and carcinomas (pheochromocytoma, Cushing)
 - Uterine leiomyomata
- Blood doping:
 - Recombinant erythropoietin abuse
 - Autologous transfusions
- Drug abuse:
 - Chronic cocaine abuse
 - Androgenic steroids
- Genetic disorders with polycythemia:
 - High-affinity Hgb variants
 - Bisphosphoglycerate deficiency
 - von Hippel–Lindau syndrome
 - Chuvash polycythemia
 - Erythropoietin-receptor mutations
 - Congenital methemoglobinemia
- Infections:
 - Viral hepatitis, AIDS

Diagnostic Criteria for Polycythemia Vera

- Major criteria:
 - Hgb > 18.5 g/dL in men, > 16.5 g/dL in women
 - Presence of JAK2 mutation by polymerase chain reaction (PCR) – clinches the dx
 - Oxygen saturation > 92% and no other cause for secondary erythrocytosis
- Minor criteria:
 - Low serum erythropoietin level
 - Bone marrow aspirate and biopsy revealing panhyperplasia
- Adjuncts to diagnosis:
 - Platelets > 400,000/mm³
 - ANC > 10,000 (WBC > 12,000/mm³)
 - Splenomegaly on exam or by CT

- Leukocyte alkaline phosphatase elevation
- B₁₂ >900 pg/mL; unbound vitamin B₁₂-binding capacity >2,200 pg/mL

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- General:
 - Dyspnea
 - Weakness/fatigue
 - Excessive sweating
 - Epistaxis/gingival bleeding
 - Pruritus (40% of PV):
 - Generalized
 - Exacerbated by warm bath or shower
 - Excoriations common in PV
 - Gouty arthritis and tophi
- Neurologic (hyperviscosity):
 - Headache
 - Vertigo/dizziness/tinnitus
 - Lethargy/confusion
 - Paresthesias
 - Cerebrovascular accident/TIAs
- Visual (hyperviscosity):
 - Amaurosis fugax
 - Scotoma/blurred vision
 - Ophthalmic migraine
- Cardiovascular:
 - CHF
 - Angina/myocardial infarction
 - Deep vein thrombosis (DVT)
 - Hypertension
- Extremities:
 - Erythromelalgia:
 - Secondary to capillary sludging
 - Burning pain in the feet or hands
 - Warmth, erythema/cyanosis and puffiness of hand and feet
 - Acral paresthesias
 - Worse at night
 - Relief with cooling and aspirin
 - Pulses intact

- Painful ulcers of fingers and toes (digital ischemia)
- GI (unique to PV):
 - Hepatomegaly/splenomegaly
 - Sudden spleen enlargement in known PV suggests development of myelofibrosis
 - Epigastric discomfort/early satiety
 - Peptic ulcer disease/GI bleed
 - Budd–Chiari syndrome (hepatic vein thrombosis): Ascites and peripheral edema

Physical-Exam

- Hypertension
- Conjunctival suffusion
- Fundus: Venous engorgement
- Ruddy complexion/plethora
- Erythema/rubor of hands, feet, nail beds
- Skin excoriations from severe pruritus
- Splenomegaly (75% in PV)
- Hepatomegaly (30% in PV)
- Thrombotic complications:
 - 2/3 arterial, 1/3 venous
 - Cumulative risk of 2–5% per year
 - TIAs, stroke, MI, digital infarcts
 - Unusual venous thrombosis:
 - Splenic or mesenteric veins
 - Hepatic vein and IVC clot with subsequent cirrhosis/ascites (Budd–Chiari syndrome)
 - Cerebral sinus thrombosis
 - DVT, PE
- Complications of hyperviscosity:
 - Lethargy/confusion
 - Headaches, dizziness, vision changes
 - Crackles/findings of CHF
- Hemorrhagic complications:
 - Ecchymosis
 - Epistaxis
 - Gingival bleeding

ESSENTIAL WORKUP

CBC with platelets

DIAGNOSIS TESTS & NTERPRETATION

Lab

- 1st priority: Distinguish relative from true erythrocytosis:
 - Volume repletion IV or PO, then repeat CBC
- 2nd priority: Evaluate for secondary causes:
 - Pulse oximetry with $pO_2 < 92\%$
 - Carboxyhemoglobin level
 - Erythropoietin level (normal or elevated if secondary)
 - CXR, chest CT, pulmonary function tests
 - Sleep study
 - Hgb electrophoresis
- RBC mass:
 - Cr-51–labeled RBCs by nuclear medicine
 - Concomitant plasma volume with I-131–labeled albumin
 - Not necessary if Hgb > 18.5 in men, or > 16.5 in women
 - Red blood cell mass < 35 mg/kg (males) or < 31 mg/kg (females) is normal.
 - Decreased plasma volume with normal RBC mass verifies relative erythrocytosis.
 - Elevated RBC mass suggests PV or secondary polycythemia.
 - Falsely low if iron deficient or obese
- PV suspected if:
 - Hgb > 18.5 g/dL (men), 16.5 g/dL (women)
 - Absolute neutrophil count $> 10,000$
 - Platelet count $> 400,000$
 - Pulse oximetry $> 92\%$
 - Low erythropoietin level – a major clue
 - Vitamin B₁₂ level elevated in 30% (unbound vitamin B₁₂-binding capacity elevated in 75%)
 - Uric acid elevated in 40%
 - Leukocyte alkaline phosphatase elevated in 70%
 - PCR for JAK2 gene mutation diagnostic of PV (seen in 97%)

Imaging

Abdominal US or CT can detect a splenomegaly

DIFFERENTIAL DIAGNOSIS

See Etiology.



TREATMENT

INITIAL STABILIZATION/THERAPY

ABCs with emphasis on fluid resuscitation if no evidence of CHF

ED TREATMENT/PROCEDURES

Emergency Management of Hyperviscosity Syndrome or Hct > 60%

- Fluid resuscitation to achieve hemodilution:
 - Withhold if evidence of CHF
- Emergency phlebotomy of 250–500 mL of blood over 1–2 hr replacing with an equal amount of 0.9% normal saline (NS)
- Removal of 1,000–1,500 mL of blood over 24 hr with a goal of Hct < 60 or relief of symptoms:
 - Keep Hct > 45.
 - Replace with an equal amount of 0.9% NS.
- Phlebotomize the elderly and those with cardiovascular disease more slowly:
 - Every-other-day phlebotomy
- Emergent surgery with polycythemia:
 - Phlebotomize to Hct of 45 to avoid thrombotic complications postoperatively.
- Thrombocytosis therapy:
 - Administer aspirin if platelet count is 500,000–1,500,000/mm³ and there are no hemorrhagic complications.
- Treat pruritus with diphenhydramine.

Long Term Management

- Phlebotomy: Maintain Hct at 45% for men and 42% for women.
- Aspirin 81 mg daily if thrombocytosis
- Interferon- α (normalizes CBC in 80%):
 - Especially helpful for refractory pruritus and painful splenomegaly
 - Suggested in symptomatic patients < 60 yr
- Anagrelide:
 - Specific for thrombocytosis
 - No risk of leukemia, ideal for younger patients with postphlebotomy thrombocytosis
 - Effective alone and can decrease need for or frequency of chemotherapy
- Hydroxyurea:
 - Mainstay of therapy, especially for patients > 60 yr, with frequent phlebotomy requirements, thrombotic episodes, or refractory thrombocytosis
- Aldylating agents: Busulfan:
 - Severe refractory disease in the elderly
 - High risk of leukemic transformation

Pregnancy Considerations

Temporary remission during pregnancy, no treatment usually needed

Pediatric Considerations

- In the neonate, defined as a peripheral venous Hct > 65%, Hgb > 22 g/dL:
 - Sample must be obtained > 6 hr post delivery.
 - Capillary Hgb and Hct are 10% higher than venous (always rely on venous)
 - 1–5% of neonates
 - Up to 50% of neonates with intrauterine growth retardation
- Etiology:
 - Maternal–fetal hypoxemia secondary to maternal heart or lung disease, diabetes, preeclampsia, hypertension, or smoking
 - Delayed clamping of the umbilical cord with increase cord transfusion
- Symptoms and signs (most asymptomatic):
 - Acrocyanosis/plethoric
 - Tachypnea/respiratory distress
 - Irritable, lethargic, jittery, poor feeding
- Hypoglycemia and hyperbilirubinemia common
- Treatment:
 - Observation and serial CBCs
 - 0.9 NS 100 mL/kg per day (symptomatic)
 - Partial exchange transfusion: Remove 20 mL/kg blood and infuse equal amount of saline (persistent or severe symptoms)
 - Dextrose solutions if hypoglycemia

Geriatric Considerations

Caution with speed of phlebotomy and fluid resuscitation as noted



FOLLOW-UP

DISPOSITION

Admission Criteria

- New diagnosis of polycythemia
- Hct > 60% without symptoms
- Symptoms of hyperviscosity
- Unstable vital signs/significant comorbidities

Discharge Criteria

- Previous diagnosis of polycythemia, Hct < 60, and asymptomatic
- Stable vital signs

Issues for Referral

All patients should be referred to a hematologist or primary care physician.

PEARLS AND PITFALLS

- Criteria for phlebotomy in polycythemia secondary to hypoxemia is not clear. While phlebotomy will decrease viscosity, it may decrease oxygen-carrying capacity.
- It is critical to distinguish PV from secondary causes of erythrocytosis since PV carries a high risk of thrombotic complications.
- Pruritus with water contact and erythromelalgia (pain, paresthesia and rubor in hands/feet) are unique features of PV.

ADDITIONAL READING

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- Tefferi A. Polycythemia vera and essential thrombocythemia: 2012 update on diagnosis, risk stratification and management. *Am J Hematol.* 2012;87(3):285–293.



CODES

ICD9

- 238.4 Polycythemia vera
- 289.0 Polycythemia, secondary

ICD10

- D45 Polycythemia vera
- D75.1 Secondary polycythemia

POLYNEUROPATHY

Sandra A. Deane

BASICS

DESCRIPTION

A peripheral nerve disorder in which many nerves throughout the body malfunction simultaneously:

- Acute polyneuropathy causes:
 - Infectious (toxin producing bacteria, viruses)
 - Autoimmune (Guillain–Barré)
 - Toxic (heavy metals):
 - Lead
 - Mercury
 - Drugs:
 - Anticonvulsants (phenytoin)
 - Antibiotics (chloramphenicol, nitrofurantoin, sulfonamides)
 - Chemotherapy (vinblastine, vincristine)
 - Sedatives (hexobarbital and barbital)
 - Cancer (multiple myeloma)
- Chronic polyneuropathy causes:
 - Diabetes (most common)
 - Alcohol abuse
 - Nutritional deficiencies (Thiamine, B₁₂)
 - Hypothyroidism
 - Liver failure
 - Kidney failure
 - Lung cancer
 - Chronic inflammatory demyelinating polyneuropathy (CIDM)

EPIDEMIOLOGY

Incidence and Prevalence Estimates

- In US, the prevalence of polyneuropathy is ~2% in the general population
- It is 8% in patients >55 yr of age
- The most common cause in US is diabetes and it occurs in ~50% of diabetics on insulin

ETIOLOGY

- Myelin dysfunction:
 - Parainfectious immune response triggered by antigens that cross-react with

- antigens in the peripheral nervous system:
 - Encapsulated bacteria (*Campylobacter* sp., diphtheria)
 - Viruses (enteric or influenza viruses, HIV)
 - Vaccines (influenza)
- Guillain–Barré syndrome:
 - Acute onset due to myelin dysfunction
 - Rapidly progressive weakness and may lead to respiratory failure
- CIDM:
 - Chronic illness of myelin dysfunction
 - Symptoms may recur or progress over months and years
- Vasa nervosum compromise:
 - Vascular supply to nerves compromised leading to nerve infarction
 - Causes:
 - Chronic atherosclerosis
 - Vasculitis
 - Infections
 - Hypercoagulable states
 - Axonopathy
 - Primary dysfunction of the axon
 - Most often the result of toxic–metabolic disorders:
 - Diabetes
 - Nutritional deficiencies
 - Drugs/chemicals

DIAGNOSIS

SIGNS AND SYMPTOMS

- May be acute or chronic
- May be predominately sensory, motor, combined sensory–motor, or autonomic dysfunction

History

- More commonly affects lower extremities than upper extremities and begins distally
- Typical complaints:
 - Dysaesthesias – numbness, burning, or tingling of the extremities
 - Weakness of extremities
 - Difficulty walking
 - Autonomic symptoms:
 - Constipation
 - Loss of bowel/bladder control
 - Sexual dysfunction

- Orthostatic dizziness
- Dry skin
- Decreased sweating

Physical-Exam

- Typically, findings are bilateral symmetrical and stocking glove distribution
- Typical findings:
 - Decreased sensation
 - Decreased vibratory and position sense
 - Decreased motor function
 - Decreased reflexes
 - Muscle atrophy
 - Fasciculations
 - Paralysis
- Findings in specific types of polyneuropathy:
 - Myelin dysfunction (Guillain–Barré – acute and CIDP – chronic):
 - Muscle weakness greater than expected for degree of atrophy
 - Paresthesias
 - Greatly diminished reflexes
 - Proximal and distal symptoms
 - Ischemia to nerve (atherosclerosis, vasculitis, infectious, hypercoagulable):
 - Painful, burning sensory disturbances
 - Decreased pain and temperature sensation
 - Muscle weakness proportional to atrophy
 - Reflexes spared
 - Usually spares proximal nerves
 - Cranial nerve involvement rare
 - Primary axon dysfunction (toxic-metabolic disorders):
 - Have symptoms of either myelin dysfunction, ischemia, or combined
 - Painful
 - Distally symmetrical
 - Stocking glove
 - Lower extremities before upper

ESSENTIAL WORKUP

- Thorough past medical history and physical exam should be obtained to guide testing
- Initial lab testing:
 - CBC
 - Electrolytes
 - Glucose
 - Renal and liver function
 - TSH

- ESR
- ANA
- Vitamin B₁₂
- Folate
- RPR
- HIV
- Hepatitis B and C
- Lyme
- CPK
- Serum protein electrophoresis
- Subsequent lab testing based on history:
 - Heavy metal levels (history of exposure)
 - Genetic testing for genetic neuropathies
 - Serum antibody testing for immune-mediated neuropathies

DIAGNOSIS TESTS & INTERPRETATION

Imaging

Should be guided by history and physical findings

Diagnostic Procedures/Surgery

- Electromyography (EMG)
- Nerve conduction studies
- Lumbar puncture:
 - Increased CSF protein level abnormal
 - Diagnostic of Guillain-Barré syndrome and CIDP
- Skin or nerve biopsy

DIFFERENTIAL DIAGNOSIS

- Primarily to differentiate between various causes of polyneuropathy:
 - Endocrine disease (diabetes)
 - Infections (Guillain-Barré, Lyme disease, HIV, syphilis)
 - Vitamin deficiency
 - Cancer/paraneoplastic
 - Toxins
 - Liver disease
 - Renal failure
 - Genetic disorders
 - Amyloidosis
- Other diseases with similar presentations:
 - Polio
 - Porphyria

- Spinal muscular atrophy
- Catecholamine disorders
- Psychological disorders



TREATMENT

PRE HOSPITAL

Primarily supportive care for ABCs

INITIAL STABILIZATION/THERAPY

- ABCs
- Respiratory support for respiratory failure

ED TREATMENT/PROCEDURES

- Pain control:
 - Parenteral or oral narcotics
 - Tricyclic antidepressants (amitriptyline)
 - Anticonvulsants (gabapentin)
- Plasma exchange or IV immune globulin for acute myelin dysfunction
- Corticosteroids or antimetabolite drugs for chronic myelin dysfunction
- Supportive care for autonomic dysfunction (IVF, pressors)
- Measure Negative Inspiratory Force (NIF) if concerned about respiratory compromise (Normal is \sim -60 cm H₂O)



FOLLOW-UP

DISPOSITION

Admission Criteria

Determined by medical condition and acuity of onset necessitating rapid diagnostic testing:

- Respiratory failure
- BP instability
- Inability to walk or care for self
- Inadequate pain control
- Poor control of underlying disease process
- Rapidly progressing symptoms

Discharge Criteria

- Underlying medical condition stabilized
- No evidence or low risk of respiratory failure or autonomic instability

- Able to care for self
- Adequate pain control
- Access to outpatient follow-up for further testing or management

Issues for Referral

All patients require referral to primary care physician or neurology for ongoing testing and/or management

FOLLOW-UP RECOMMENDATIONS

- Primary care physician
- Neurology
- Physical therapy

PEARLS AND PITFALLS

- Understanding that the potential causes of polyneuropathy are broad and a comprehensive search for the underlying cause will aid in management
- Recognizing those few causes that are at risk for respiratory failure or autonomic instability
- For most causes, treatment consists of controlling underlying disease process

ADDITIONAL READING

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CODES

ICD9

- 356.9 Unspecified hereditary and idiopathic peripheral neuropathy
- 357.4 Polyneuropathy in other diseases classified elsewhere
- 357.7 Polyneuropathy due to other toxic agents

ICD10

- G62.2 Polyneuropathy due to other toxic agents
- G62.9 Polyneuropathy, unspecified
- G63 Polyneuropathy in diseases classified elsewhere

POSTPARTUM HEMORRHAGE

AJ Kirk • Marco Coppola

BASICS

DESCRIPTION

- Postpartum hemorrhage (PPH) after 20 wk gestation
- Primary: Hemorrhage occurring ≤ 24 hr after delivery
 - Also known as early PPH
- Secondary: Hemorrhage occurring > 24 hr after delivery (but < 12 wk):
 - Also known as delayed PPH
- Definitions:
 - > 500 mL after vaginal delivery
 - $> 1,000$ mL after C-section
- Occurs in 4% of vaginal deliveries
- Occurs in 6% of C-sections
- Leading cause of death in pregnancy worldwide
 - Accounts for 25% of pregnancy-related deaths
 - $\sim 50\%$ of postpartum deaths are due to PPH
- 95% of PPH caused by:
 - Uterine atony (50–60%)
 - Retained placenta (20–30%)
 - Cervical/vaginal lacerations (10%)
- Complications:
 - Hypovolemic shock
 - Blood transfusion
 - Acute respiratory distress syndrome
 - Renal and/or hepatic failure
 - Sheehan syndrome
 - Loss of fertility
 - Disseminated intravascular coagulopathy (DIC)

ETIOLOGY

- 4 Ts:
 - Tone
 - Tissue
 - Trauma
 - Thrombin
- Immediate:
 - Uterine atony
 - Lower genital lacerations

- Retained placental tissue
- Placenta accreta
- Uterine rupture
- Uterine inversion
- Puerperal hematoma
- Coagulopathies
- Delayed:
 - Retained products of conception
 - Postpartum endometritis
 - Withdrawal of exogenous estrogen
 - Puerperal hematoma
- Coagulopathies:
 - Pre-existing idiopathic thrombocytopenic purpura
 - Thrombotic thrombocytopenic purpura
 - Von Willebrand disease
 - DIC
- Associated conditions:
 - If bleeding is present at other sites, consider coagulopathy
- Risk factors:
 - Prior PPH
 - Advanced maternal age
 - Multiple gestations
 - Prolonged labor
 - Polyhydramnios
 - Instrumental delivery
 - Fetal demise
 - Anticoagulation therapy
 - Placental abruption
 - Fibroids
 - Prolonged use of oxytocin
 - C-section
 - Placenta previa and accreta
 - Chorioamnionitis
 - General anesthesia

DIAGNOSIS

SIGNS AND SYMPTOMS

- Ongoing blood loss, usually painless
- Significant hypovolemia, resulting in:
 - Tachycardia
 - Tachypnea

- Narrow pulse pressure
- Decreased urine output
- Cool, clammy skin
- Poor capillary refill
- Altered mental status
- Maternal tachycardia and hypotension may not occur until blood loss > 1,500 mL

History

- Condition is typically recognized by obstetrician soon after delivery
- Delayed PPH presents as copious vaginal/perineal bleeding
- Key historical elements:
 - Complications of delivery
 - Episiotomy
 - Prior clotting disorders
- Symptoms of hypovolemia:
 - Decreased urine output
 - Lightheaded
 - Syncope
 - Pale skin

Physical-Exam

Thorough exam of perineum, cervix, vagina, and uterus:

- External inspection
- Speculum exam
- Bimanual exam

ESSENTIAL WORKUP

- Abdomen and pelvic exam to assess for uterine atony, retained products, or other anatomic abnormality
- Type and cross-match for packed red blood cells
- Rapid hemoglobin determination

DIAGNOSIS TESTS & INTERPRETATION

Diagnosis is chiefly based on clinical suspicion and exam

Lab

- CBC, platelets
- PT, PTT
- Fibrinogen level
- Type and cross-match

Imaging

US to evaluate for retained products in delayed PPH or for evaluation of fluid

concerning intrauterine or intra-abdominal hemorrhage

Diagnostic Procedures/Surgery

Manual exam preferred over ultrasonography:

- Greater sensitivity
- Both diagnostic and therapeutic

DIFFERENTIAL DIAGNOSIS

- Consider puerperal hematomas if perineal, rectal, or lower abdominal pain in conjunction with tachycardia and hypotension
- Retained products of conception



TREATMENT

ALERT

- Patients with PPH may be hemodynamically unstable
- IV access, and active resuscitation is important, considering both crystalloid and blood product resuscitation and closely following BP and mental status

PRE HOSPITAL

- Monitor hemodynamics
- Aggressive IV fluids to maintain BP

INITIAL STABILIZATION/THERAPY

- Attempt to simultaneously control bleeding and stabilize hemodynamic status
- Manage airway and resuscitate as indicated:
 - Supplemental oxygen
 - Cardiac monitor
- IV fluid resuscitation with normal saline or lactated Ringer solution
- Foley catheter

ED TREATMENT/PROCEDURES

- Management of uterine atony:
 - Bimanual massage
 - Oxytocin (Pitocin) administered IV/IM
 - Methylergonovine (Methergine) or ergonovine (Ergotrate) IM if oxytocin fails:
 - Avoid if known hypertensive
 - Onset in minutes
 - 15-methyl prostaglandin F^{2α} (PGF^{2α}; Hemabate) IM if above fails:
 - Relatively contraindicated in asthma
 - Surgery if medical intervention fails

- Inspect closely for genital tract laceration:
 - Repair required if ≥ 2 cm
 - Use 00 or 000 absorbable suture; continuous, locked recommended
- Management of uterine inversion (acute):
 - Reposition uterus using Johnson maneuver or Harris method:
 - Use left hand on abdominal wall to stabilize fundus of uterus
 - Place right hand with fingers spread into vagina and push steadily on inverted part to reduce
 - If unsuccessful, give terbutaline IV or magnesium sulfate to produce cervical relaxation, and reposition
 - Surgery if unsuccessful or if subacute or chronic inversion
- Management of coagulopathies in childbirth:
 - Fresh-frozen plasma, platelets, cryoprecipitate as indicated
 - Careful attention to volume status
 - Continuous reassessment
 - Active over expectant management
 - Immediate administration of uterotonics after delivery
 - Cord clamping and cutting without delay
 - Cord traction/uterine countertraction (Brandt–Andrews maneuver)
- Uterine tamponade
 - Can be used for atony or continued bleeding
 - Temporizing measures only
 - Balloon or packing can be used
 - May use a foley catheter, Rusch catheter, Sengstaken–Blakemore tube or
 - Surgical Obstetric Silicone (SOS) Bakri tamponade balloon
 - Specifically designed for control of PPH

MEDICATION

- Uterotonics—stimulate uterine contraction to control bleeding:
 - Ergonovine (Ergotrate): 0.2 mg IM; avoid if known hypertensive
 - Methylergonovine (Methergine): 0.2 mg IM; 0.2 mg PO q6h; avoid if known hypertensive
 - 15-methyl PGF^{2 α} (Hemabate): 0.25 mg IM; may repeat in 15–60 min
 - Oxytocin (Pitocin): 10 U IM or 20–40 U IV in 1 L normal saline; titrate to achieve uterine contractions
- Cervical relaxation agents facilitate uterine inversion reduction:
 - Magnesium sulfate 20%: 2 g IM bolus over 10 min
 - Terbutaline: 0.25 mg IV; avoid if hypotensive

First Line

- Uterotonics
- Oxytocin

- Methylergonovine

Second Line

- Surgical intervention:
 - Hysterectomy is required in management of PPH in 1/1,000 deliveries
- Radiologic embolization



FOLLOW-UP

DISPOSITION

Admission Criteria

- All patients with immediate PPH require admission to a closely monitored setting
- Early obstetrics consultation is recommended
- Early surgical intervention is dependent on cause
- ICU setting if DIC or evidence of hemodynamic compromise
- Patients with endometritis should be admitted for parenteral antibiotics

Discharge Criteria

- Delayed PPH that is easily controlled without excessive bleeding
- Outpatient management with methylergonovine 0.2 mg PO every 6 hr may be considered in consultation and close follow-up with obstetrician

FOLLOW-UP RECOMMENDATIONS

- Close follow-up with obstetrician
- Seek immediate care if bleeding recurs

PEARLS AND PITFALLS

- Active over expectant management
 - Most deaths are due to delayed diagnosis and/or inadequate resuscitation with blood products
- Uterotonics are the first line of treatment
- Aggressive use of fluid and blood products for resuscitation
- Manual exam is the preferred diagnostic approach
- Immediate obstetric consult

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Vaginal Bleeding
- Placenta Previa
- Placental Abruption
- Pregnancy, Trauma in
- Pregnancy, Uncomplicated
- Labor
- Delivery, Uncomplicated



CODES

ICD9

- 666.00 Third-stage postpartum hemorrhage, unspecified as to episode of care or not applicable
- 666.10 Other immediate postpartum hemorrhage, unspecified as to episode of care
- 666.20 Delayed and secondary postpartum hemorrhage, unspecified as to episode of care or not applicable

ICD10

- O72.0 Third-stage hemorrhage
- O72.1 Other immediate postpartum hemorrhage
- O72.2 Delayed and secondary postpartum hemorrhage

POSTPARTUM INFECTION

Noah White • Yvonne C. Chow • Marco Coppola

BASICS

DESCRIPTION

- Postpartum endometritis (PPE):
 - Early PPE
 - Develops within 48 hr
 - Most often complicating C-section
 - Occurs in 1–3% of uncomplicated vaginal deliveries
 - Classic triad: Fever, lower abdominal pain with uterine tenderness, foul-smelling lochia
 - Late PPE
 - Develops 3 days–6 wk after delivery
 - Usually follows vaginal delivery
 - Risk of PPE as high as 85–95% in high-risk nonelective C-section patient
- Complications of PPE: All are more common after C-section:
 - Pelvic thrombophlebitis
 - Pelvic abscess
 - Bacteremia
- Risk factors for PPE:
 - C-section
 - Prolonged labor
 - Prolonged rupture of membranes
 - Increased number of vaginal exams
 - Use of internal fetal monitoring
- Septic pelvic thrombophlebitis is a diagnosis of exclusion with 2 distinct clinical presentations, either of which may present with postpartum pulmonary embolus:
 - Acute thrombosis:
 - Most common in right ovarian vein
 - Usually occurs in 1st 48 hr as acute, progressive lower abdominal pain
 - Enigmatic fever: “Picket fence” spiking fevers and tachycardia
- Septic abortion:
 - Uncommon in developing countries
 - Usually an ascending infection through an open cervical os
 - Associated with:
 - Nonsterile techniques, instruments
 - Retained products of conception
- Mastitis:

- Ranges from mild breast redness to fever, systemic illness, and abscess
- Common (1–30% of postpartum patients)
- Occurs within the 1st 3 mo postpartum
- Peaks at 2–3 wk
- Recurs in 4–8%
- UTI/pyelonephritis:
 - Along with mastitis accounts for 80% of postpartum infections

ETIOLOGY

- PPE:
 - Polymicrobial infection result of ascending spread from lower genital tract
 - Anaerobic (up to 80%) and aerobic (~70%):
 - Gram-positive aerobes:
 - Group A, B streptococci
 - Enterococci
 - *Gardnerella vaginalis*
 - Gram-negative aerobes:
 - *Escherichia coli*
 - *Enterobacter*
 - Anaerobes:
 - *Bacteroides*
 - *Peptostreptococcus*
 - Other genital mycoplasmas common in late PPE:
 - *Ureaplasma urealyticum*
 - *Mycoplasma hominids*
 - *Chlamydia trachomatis*
- Septic abortion:
 - Usually polymicrobial
 - *E. coli*
 - *Bacteroides*
 - Anaerobic gram-negative rods
 - Group B streptococci
 - Staphylococcus
 - STD:
 - Gonorrhoea
 - *C. trachomatis*
 - *Trichomonas*
- Mastitis
 - *Staphylococcus aureus*
 - Group A and B hemolytic streptococci
 - *E. coli*
 - *Bacteroides*

SIGNS AND SYMPTOMS***History***

- Careful birth history:
 - C-section
 - Length of labor
 - Complications
 - Exposure to STDs
- Pre-existing immunocompromise or disease
- Endometritis:
 - Fever and chills
 - Abdominal pain
 - Foul-smelling lochia
- Septic abortion:
 - Similar to endometritis
 - Fever
 - Abdominal pain
 - May present with symptoms of shock including:
 - Dyspnea (acute respiratory distress syndrome [ARDS], pulmonary edema)
 - Bruising, bleeding (disseminated intravascular coagulation [DIC])
- Mastitis:
 - Fever
 - Breast pain, engorgement, redness
- Other sources of infection:
 - Wound infection:
 - Redness, pain, swelling
 - UTI/pyelonephritis:
 - Fever, dysuria, frequency, flank pain

Physical-Exam

- Abdominal and/or uterine tenderness
- Foul-smelling lochia
- Unilateral tender, engorged, erythematous breast in cases of mastitis
- Examine episiotomy infections
- Suprapubic or costovertebral angle tenderness in cases of UTI/pyelonephritis

ESSENTIAL WORKUP

- Abdominal and pelvic exam
- Cervical cultures for *Chlamydia*

- Transcervical endometrial cultures

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC
- Urinalysis and culture
- Blood cultures

Imaging

- CT or MRI for ovarian vein thrombosis
- US is sensitive for abscess or retained products of conception
- Plain x-rays may show retained foreign bodies or free air in septic abortion.

DIFFERENTIAL DIAGNOSIS

- Fever from other sources
- < 6 hr:
 - Early streptococcal infection
 - Transfusion reaction
 - Thyroid crisis
- < 48 hr:
 - Atelectasis
- < 72 hr:
 - UTI
 - Pneumonia
- 3–5 days:
 - Mastitis
 - Breast engorgement
 - Necrotizing fasciitis
- 3–7 days:
 - Mastitis
 - Septic thrombophlebitis
- 7–14 days:
 - Abscess
- > 2 wk:
 - Mastitis
 - Pulmonary embolism



TREATMENT

PRE HOSPITAL

- ABCs

- IV and IV fluids if signs of shock or impending shock

INITIAL STABILIZATION/THERAPY

Manage airway and resuscitate as indicated:

- Prompt evaluation of respiratory and hemodynamic status
- Supplemental oxygen, cardiac monitor, and pulse oximetry, as needed
- Venous access; support circulatory status with crystalloid and pressors, if needed

ED TREATMENT/PROCEDURES

- IV antibiotics and close observation
- Septic abortion is usually treated with dilatation and curettage and removal of any inciting agents
- Monitor for signs of impending shock, circulatory failure, ARDS, and/or sepsis.
- Heparin if suspicion or evidence of thrombophlebitis
- Infected wound or abscess should be opened to establish drainage
- Necrotizing fasciitis requires wide surgical débridement, parenteral antibiotics, and adjunctive hyperbaric oxygen therapy
- Peritonitis requires imaging to evaluate cause

MEDICATION

Per underlying infection. See corresponding chapters for complete list (consider safety in breast-feeding)

Endometritis

- Cefoxitin: 2 g IV q6h *or*
- Cefotetan: 2 g IV q12h *or*
- Piperacillin/tazobactam: 3.375 g IV q6–8h *or*
- Ampicillin/sulbactam: 1.5–3 g IV q6h *or*
- Clindamycin: 600–900 mg IV q8h +
- Gentamicin: 2 mg/kg load, then 1–1.5 mg/kg IV q8h

Septic Abortion

- Triple antibiotics
- Gram-positive coverage:
 - Ampicillin/sulbactam: 1.5–3 g IV q6h *or*
 - Cefoxitin: 2 g IV q6h *or*
 - Cefotetan: 2 g IV q12h
- Gram-negative coverage:
 - Gentamicin: 2 mg/kg load, then 1–1.5 mg/kg IV q8h
- Anaerobic coverage:
 - Clindamycin: 600–900 mg IV q8h *or*
 - Metronidazole: 500 mg IV q8h

Mastitis

- Dicloxacillin: 250 mg q6h PO for 10 days
- Mupirocin 2% ointment TID
- Cephalexin: 500 mg q6h PO for 10 days
- Clindamycin: 300 mg q6h PO for 10 days
- Erythromycin: 500 mg q6h PO for 10 days
- If MRSA positive: Vancomycin 1 g IV q12h

UTI/Pyelonephritis (Inpatient)

- Ciprofloxacin: 400 mg IV q12h *or*
- Ceftriaxone: 1–2 g IV q24h *or*
- Piperacillin/tazobactam: 3.375 g IV q6–8h



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients with endometritis or suspicion for septic pelvic thrombophlebitis should be admitted
- Septic abortion

Discharge Criteria

Nontoxic, mildly symptomatic patient may be considered for outpatient management in consultation and close follow-up with obstetrics

FOLLOW-UP RECOMMENDATIONS

Close follow-up with obstetrician and/or primary care physician to evaluate treatment

PEARLS AND PITFALLS

- Mastitis and UTI account for 80% of postpartum infections
- C-section increases risk for PPE
- Entertain broad differential with regard to source of infection
- Early broad-spectrum antibiotics are often indicated

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Mastitis
- Urinary Tract Infection
- Pyelonephritis



CODES

ICD9

- 670.00 Major puerperal infection, unspecified as to episode of care or not applicable
- 670.10 Puerperal endometritis, unspecified as to episode of care or not applicable
- 670.20 Puerperal sepsis, unspecified as to episode of care or not applicable

ICD10

- _ O85 Puerperal sepsis
- _ O86.4 Pyrexia of unknown origin following delivery
- _ O86.12 Endometritis following delivery

PREECLAMPSIA/ECLAMPSIA

Adam Z. Barkin

BASICS

DESCRIPTION

- Hypertension in pregnancy:
 - 1% of all pregnancies
 - 16% of maternal deaths
- Gestational hypertension (GH)
 - Hypertension associated with pregnancy
 - Resolves with delivery
 - 6–7% of all pregnancies
- Preeclampsia
 - GH PLUS proteinuria
 - 2.2–6.3% of all pregnancies
- Eclampsia
 - Preeclampsia with seizure
- Postpartum preeclampsia
 - Occurs within 6 wk of delivery
 - Usually no history of hypertension
 - Occurs in 5% patients
 - Most women are African American
- HELLP syndrome
 - May occur in women with preeclampsia or eclampsia
 - Hemolysis
 - Elevated liver function tests
 - Low platelets
- Superimposed preeclampsia
 - Preeclampsia in the setting of chronic hypertension
 - Complicates pregnancy in up to 25% of women with chronic hypertension
 - Risk factors:
 - African American
 - Antihypertensive medication use
- Chronic hypertension
 - Systolic BP (SBP) > 140 or diastolic BP (DBP) > 90
 - Measured twice prior to 20 wk gestation or lasting > 12 wk after delivery

ETIOLOGY

- Preeclampsia
 - Incomplete placental implantation and underperfusion

- Leads to decreased angiogenic growth factor and increased maternal placental debris in circulation
- Eclampsia
 - 1/3 of patients with eclampsia did not have hypertension prior to seizure
- Risk factors:
 - Extremes of reproductive age
 - Primigravida
 - Multiple gestations
 - Molar pregnancy, hydatidiform mole
 - Smoking
 - Increased body mass index
 - Diabetes, collagen vascular diseases
 - Pre-existing hypertension or renal disease
 - History of preeclampsia with prior pregnancies (7.5–10% increased risk)
 - Independent risk factors for eclampsia
 - Nulliparity
 - Maternal age
 - GH



DIAGNOSIS

- GH
 - Normotensive prior to 20 wk gestation
 - SBP > 140 or DBP > 90 on 2 separate measurements
 - Severe: SBP > 160 and DBP > 110
- Preeclampsia
 - GH and proteinuria
 - 300 mg protein on 24 hr urine
 - 1+ protein on urinalysis
 - Mild:
 - SBP < 160 mm Hg or
 - DBP < 110 mm Hg
 - Normal platelets
 - Normal liver function tests
 - No cerebral symptoms
 - Severe:
 - SBP > 160 or DBP > 110
 - 5 g protein on 24 hr urine
 - 3+ proteinuria on 2 occasions
 - Oliguria
 - Thrombocytopenia
 - Right upper quadrant pain
 - Impaired liver function

- Cerebral symptoms
- Intrauterine growth restriction
- Vision changes
- Pulmonary edema
- HELLP Syndrome
 - Hemolysis
 - Elevated liver enzyme
 - Low platelets
 - May present with:
 - Pulmonary edema
 - Renal failure
 - Liver failure
 - Sepsis
 - Pulmonary disease
 - Stroke

SIGNS AND SYMPTOMS

History

- History of preeclampsia
- Parity
- Weight gain
- Leg swelling
- Abdominal pain
- Nausea/vomiting
- Shortness of breath
- Headache
- Visual changes
- Jaundice
- Stroke symptoms

Physical-Exam

- Check serial BP
- Palpate abdomen carefully, especially RUQ
- Assess extremities for edema
- Perform neurologic exam:
 - Deep tendon reflexes
 - Mental status changes
 - Visual acuity

ESSENTIAL WORKUP

- Serial BP measurements
- Urinalysis

- CBC, LFTs, BUN/creatinine, uric acid
- US
- Fetal monitoring
- Head CT depending on severity of presentation

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Urinalysis:
 - Protein > 1+ correlates to 30 mg/dL
 - > 1+ requires 24 hr urine collection
 - Urine sediment for RBC, WBC, casts
- CBC
- LFTs
- BUN/creatinine
- Uric acid
- LDH
- d-dimer
- Fibrinogen levels
- Coagulation studies

Imaging

- US:
 - Gestational age
 - Fetal viability/distress
 - Oligohydramnios
- Fetal monitoring, nonstress test
- Head CT: Rule out mass or hemorrhage

Diagnostic Procedures/Surgery

- Lumbar puncture: Rule out infection or subarachnoid hemorrhage
- Urine toxicology: Rule out substance abuse:
 - Cocaine
 - Methamphetamine

DIFFERENTIAL DIAGNOSIS

- Essential hypertension
- Renal or collagen vascular disease
- Hydatidiform mole, hydrops fetalis
- Drug abuse
- Epilepsy
- Encephalitis
- Meningitis

- Encephalopathy
- Brain tumor
- Intracranial hemorrhage



TREATMENT

PRE HOSPITAL

- ABCs
- Oxygen
- Place patient in left lateral decubitus position

INITIAL STABILIZATION/THERAPY

- ABCs
- 100% oxygen
- Left lateral decubitus position (reduces pressure on inferior vena cava, enhancing cardiac return/output)
- Maternal cardiopulmonary monitoring
- Magnesium sulfate (MgSO_4) for seizures

ED TREATMENT/PROCEDURES

- Make arrangements for emergent C-section
- MgSO_4 for seizure treatment and prophylaxis
- Hydralazine or labetalol for BP control
 - Goal is to lower BP by 25% initially and then to $<160/100$ over subsequent hours
- Mg toxicity:
 - Hypotension
 - Loss of patellar reflex
 - Respiratory depression
 - Decreased urine output
 - Elevated creatinine
 - Calcium gluconate to reverse
- Intubate for airway protection/hypoxia or if seizures refractory to interventions
- Tocographic and fetal monitoring
- OB consult:
 - All cases along GH–preeclampsia–eclampsia spectrum
 - Expectant management if <30 wk gestation
 - Delivery >30 wk
 - Emergent delivery for severe symptoms: Induction vs. C-section

MEDICATION

First Line

- MgSO_4 : 10 g IM or 4 g IV; followed by 1–2 g/hr IV infusion:
 - MgSO_4 bolus should not exceed 1 g/min
 - Serum Mg goal: 4–7 mEq/L
- Hydralazine: 5–20 mg IV
- Labetalol: 10 mg IV initially, then 5–10 mg increments for desired effect

Second Line

- Valium: 5–10 mg IV if no response to MgSO_4
- Fosphenytoin: 15–20 mg phenytoin equivalents (PE) IV \times 1 (max. 150 mg PE/min IV)
- Phenytoin: 15–18 mg/kg IV, not to exceed 25–50 mg/min, for persistent seizure activity
- Calcium gluconate: 1 g IV



FOLLOW-UP

DISPOSITION

Admission Criteria

- Preeclampsia
- Eclampsia
- HELLP syndrome
- ICU, labor and delivery, OR

Discharge Criteria

- Isolated hypertension with workup negative for preeclampsia
- Asymptomatic
- Close obstetric follow-up assured

FOLLOW-UP RECOMMENDATIONS

- Follow-up with OB as above
- Return to ED:
 - Headache
 - Abdominal pain
 - Leg swelling
 - Decreased urination
 - Shortness of breath

PEARLS AND PITFALLS

- Delivery is the definitive treatment for preeclampsia and eclampsia
- BP of 130/80 mm Hg in a pregnant woman requires investigation
- Postpartum presentation: Consider preeclampsia/eclampsia in patient up to 30 days postpartum presenting with:
 - Edema
 - Shortness of breath
 - Headache
 - Seizure
- Airway considerations in preeclamptic or eclamptic patients:
 - Reduced internal diameter of airways due to engorgement
 - Airway edema may be present
 - Use smaller-diameter endotracheal tube
 - Use fiberoptic guidance if available
 - High risk for aspiration

ADDITIONAL READING

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- Leeman L, Fontaine P. Hypertensive disorders of pregnancy. *Am Fam Physician.* 2008;78:93–100.
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- Sibai BM. Etiology and management of postpartum hypertension-preeclampsia. *Am J Obstet Gynecol.* 2012;206:470–475.
- Yancey LM, Withers E, Bakes K, et al. Postpartum preeclampsia: Emergency department presentation and management. *J Emerg Med.* 2011;40:380–384.
- Yoder SR, Thornburg LL, Bisognano JD. Hypertension in pregnancy and women of childbearing age. *Am J Med.* 2009;122:890–895.



See Also (Topic, Algorithm, Electronic Media Element)

- HELLP Syndrome
- Hydatidiform Mole
- Seizure, Adult



CODES

ICD9

- 642.40 Mild or unspecified pre-eclampsia, unspecified as to episode of care
- 642.60 Eclampsia complicating pregnancy, childbirth or the puerperium, unspecified as to episode of care
- 642.64 Eclampsia, postpartum condition or complication

ICD10

- O14.90 Unspecified pre-eclampsia, unspecified trimester
- O15.2 Eclampsia in the puerperium
- O15.9 Eclampsia, unspecified as to time period

PREGNANCY, TRAUMA IN

Amin Antoine Kazzi • Ali F. Maatouk



BASICS

DESCRIPTION

- Fetal and maternal injury after the 1st trimester:
 - Increased rate of fetal loss, but not maternal mortality
- Likelihood of fetal injury increases with the severity of maternal insult
- Physiologic hypervolemia of pregnancy may lead to an underestimation of blood loss:
 - Clinical shock may be apparent only after a 30% maternal blood loss
- Abdominal findings are less evident in the gravid patient
- Minor trauma can also lead to fetal injuries (at least 50% of fetal losses)
- An Injury Severity Score >9 is associated with a worse outcome
- Less frequent bowel injury
- More frequent retroperitoneal hemorrhage due to the engorgement of pelvic organs and veins
- Increased morbidity and mortality with pelvic fractures due to pelvic and uterine engorgement
- Fetal or uterine trauma includes:
 - Placental abruption
 - Fetal–maternal hemorrhage (FMH)
 - Premature labor
 - Uterine contusion or rupture
 - Fetal demise
 - Premature membrane rupture
 - Hypoxemic or anatomic fetal injury (skull fracture)
- Abruption occurs in up to 60% of severe trauma and 1–5% of minor injuries:
 - Accounts for up to 50% of fetal loss
 - May occur with no external bleeding (20%)
 - Occurs after 16 wk of gestation
 - Can present with abdominal pain, cramping and/or vaginal bleeding
 - Hallmark is uterine contractions
- Uterine rupture:
 - Usually in patients with prior C-section
 - Nearly universal mortality
 - 10% maternal mortality
- Pelvic fracture:
 - May be an independent predictor of fetal death
 - Fatal insults to fetus can occur in all trimesters

- 10% fetal mortality in patients with minor injuries
- FMH occurs in > 30% of severe trauma:
 - Isoimmunization of Rh-negative mothers (with as little as 0.03 cc of FMH)
- Penetrating trauma results in direct injury to fetus, maternal shock, and premature delivery
- Falls and slips occur in 1 out of 4 pregnant women and may cause:
 - 4.4 fold increase in preterm birth (PTB)
 - 8 fold increase in placental abruption
 - 2.1 fold increase in fetal distress
 - 2.9 fold increase in fetal hypoxia
- Burns: If BSA involved is > 40% the maternal and fetal mortality approaches 100%
- Intentional trauma and domestic violence (DV) increases the risk for PTB 2.7 fold and low birth weight 5.3 fold
- Electrocution is a significant cause of fetal mortality

ETIOLOGY

- Trauma occurs in ~7% of all pregnancies
- Most common cause of nonobstetric morbidity and mortality in pregnancy
- Rate of fetal loss 3.4–38%
- Motor vehicle accidents (MVA; 48–84%)
- Domestic violence (DV)
- Falls
- Direct abdominal trauma
- Penetrating (stab or gunshot)
- Electrical or burn
- Higher rate in younger woman
- Substance abuse is a common accompaniment of MVA and DV
- Suicide and exposure to toxins



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Mechanism of injury
- Last menstrual period
- Abdominal pain
- Uterine contraction
- Vaginal bleeding or leakage of fluid
- Previous pregnancies, C-sections
- Substance use/abuse

Physical-Exam

- Perform with patient in left lateral recumbent position if possible
- Primary survey
- Secondary survey
- Tertiary survey
- Placental abruption:
 - Uterine tenderness
- Uterine rupture:
 - Uterine tenderness and variable shape
 - Palpation of fetal body parts
- Determine the gestational age (EGA) to assess viability:
 - Estimate last menstrual period
 - EGA = fundal height (FH; distance from pubic bone to top of uterus in cm after week 16)
- Vaginal exam to assess for:
 - Blood
 - Amniotic fluid
 - Cervical dilation and effacement

ESSENTIAL WORKUP

- Maintain spinal immobilization
- Identify maternal condition 1st:
 - Airway management and resuscitate as indicated
- Determine the EGA to assess viability:
 - EGA = FH after week 16
 - Doppler fetal heart tones
 - Sonography (may miss small abruptions)
- Fetal/maternal monitoring for > 4–6 hr:
 - Only monitor viable fetuses (typically with an EGA > 24 wk)
 - Abruption unlikely if no contractions during 1st 4 hr of monitoring
 - > 8 contractions/hr over 4 hr is associated with adverse outcome
 - If > 1 contraction every 10 min, there is a 20% incidence of abruption
 - The occurrence of bradycardia, poor beat-to-beat variability, or type II “late” deceleration indicates fetal distress
 - An abnormal tracing has a 62% sensitivity and 49% specificity for predicting adverse fetal outcomes
 - A normal tracing combined with a normal physical exam has a negative predictive value of nearly 100%

DIAGNOSIS TESTS & NTERPRETATION

Lab

- CBC, urinalysis

- Blood gas and electrolyte panel
- Type, Rh, and screening of blood
- The Kleihauer–Betke (KB) stain:
 - Identifies FMH in vaginal fluid or blood
 - Indicated when quantification of FMH is important

Imaging

- Shield the uterus if possible, but obtain necessary maternal radiographs
- *Inform the mother of the potential risks of radiation exposure*
- No definite evidence of increased risk for congenital malformation or intrauterine death
- Cancer risk is debated
- Radiation <1 rad (10 mGy) believed to carry little risk
- Increased risk of fetal malformation at 5–10 rad
- The radiation exposure is estimated at the following:
 - CXR (2 views): Minimal
 - Pelvis (anteroposterior): 1 rad
 - Cervical spine x-ray: Minimal
 - Thoracic spine x-ray: Minimal
 - Lumbar spine x-ray: 0.031–4.9 rads
 - CT head: <0.05 rads
 - CT thorax: 0.01–0.59 rads
 - CT abdomen: 2.8–4.6 rads
 - CT pelvis: 1.94–5 rads
- Ultrasonography:
 - Focused assessment with sonography for trauma (FAST) exam
 - Evaluate for solid-organ injury or hemoperitoneum
 - Fetal heart activity
 - Gestational age
 - Amount of amniotic fluid (amniotic fluid index)
 - Misses 50–80% of placental abruptions
- Test vaginal fluid with Nitrazine paper (turns blue) and for ferning
 - Likely rupture of membranes and presence of amniotic fluid
- With stable penetrating trauma, triple-contrast CT is advocated, particularly with stab wounds

Diagnostic Procedures/Surgery

As indicated by traumatic injury

DIFFERENTIAL DIAGNOSIS

Differential diagnosis is broad and should include careful exam for occult traumatic injuries



TREATMENT

PRE HOSPITAL

- Maintain spinal immobilization
- Patients in late 2nd and 3rd trimesters should be transported to a trauma center
- Advise trauma center early of pregnancy and EGA to facilitate mobilization of appropriate resources
- Place patient (while on backboard) in the left lateral recumbent position to avoid supine hypotension (after 20 wk EGA or earlier in multiple gestations)
- Mast suit inflation over the abdomen is contraindicated

INITIAL STABILIZATION/THERAPY

- Direct therapy to the mother with no delays due to pregnancy:
 - Manage airway and resuscitate as indicated
- Cardiac, pulse oximetry, and cardiotocographic monitoring
- Tilt patient or board 15–30° to the left (or manually displace uterus to the left)

ED TREATMENT/PROCEDURES

- Lactated Ringer preferred for IV fluids:
 - Large volumes of normal saline may induce hyperchloremic acidosis
- Replace estimated blood loss in a 3:1 ratio:
 - O-negative packed red blood cells if type-specific blood is not available
- In cases of severe hemorrhage transfusion of fresh frozen plasma, platelets and packed RBC at 1:1:1 ratio lowers the rate of coagulopathy and may improve survival
- Resort to transfusions after 1 L of estimated blood loss or if hypovolemia persists after 2 L of crystalloid
- Nasogastric tube decompression (higher risk of aspiration in pregnancy)
- Foley catheterization to assess urinary output
- Tube thoracostomy:
 - Use a higher intercostal space to avoid diaphragm
- Rapid sequence intubation:
 - Safe and preferred method
 - Avoid aspiration and deoxygenation
- If diagnostic peritoneal lavage is necessary, use supraumbilical open technique
- Use tocolytic therapy only for hemodynamically stable patients:
 - Contraindicated if cervix dilated > 4 cm or if FMH and abruption have not been reasonably ruled out
 - Use tocolytics only when > 8 contractions/hr have lasted > 4 hr
- A perimortem cesarean delivery may be attempted within 4–5 min of cardiopulmonary arrest. See Cesarean Section, Emergency.
- In minor trauma after week 20, fetal and maternal monitoring is best done in the

labor and delivery area

- If burns are $>50\%$ BSA + fetus in the 2nd or 3rd trimester consider delivery
- RhoGAM in all Rh-negative women (within 72 hr):
 - 50 μg IM in women <12 wk pregnant
 - 300 μg IM in women >12 wk pregnant
- 24 hr recheck for ongoing FMH:
 - Repeat Rh immune globulin if needed (if FMH >30 mL)
- Tocolytics: Magnesium sulfate 4 g IV
- Avoid aspirin, hypnotics, nonsteroidals, vasopressors when possible



FOLLOW-UP

DISPOSITION

Admission Criteria

- Vaginal bleeding or amniotic fluid leakage
- Fetomaternal hemorrhage
- Abdominal pain
- Uterine contractions
- Evidence of fetal distress
- Abruptio placenta
- Hemoperitoneum or visceral or solid-organ injury
- Fetal survival begins at week 24 (9.9%):
 - Survival becomes significant after week 26 (54.7%)

Discharge Criteria

- All the following criteria must be met:
 - No uterine contractions for >4 hr of tocodynamometry
 - No evidence of fetal distress
 - No vaginal bleeding or amniotic fluid leakage
 - No abdominal pain or tenderness
 - Timely obstetric follow-up
- Specific instructions to return if any of the above symptoms occur
- Discharge only in consultation with obstetrics.

FOLLOW-UP RECOMMENDATIONS

A pregnant trauma patient being discharged after appropriate evaluation and observation needs prompt follow-up with obstetrician.

PEARLS AND PITFALLS

- Minor trauma can lead to maternal and/or fetal death

- Stabilization of the mother is 1st priority
- Maternal stress may not occur until 1,500–2,000 mL of blood loss

ADDITIONAL READING

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- Wiencrot A, Nannini A, Manning SE, et al. Neonatal outcomes and mental illness, substance abuse, and intentional injury during pregnancy. *Matern Child Health J*. 2012;16:979–988.



See Also (Topic, Algorithm, Electronic Media Element)

- Cesarean Section, Emergency
- Placental Abruption



CODES

ICD9

- 641.20 Premature separation of placenta, unspecified as to episode of care or not applicable
- 656.00 Fetal-maternal hemorrhage, unspecified as to episode of care or not applicable
- 665.90 Unspecified obstetrical trauma, unspecified as to episode of care or not applicable

ICD10

- O43.019 Fetomaternal placental transfusion syndrome, unsp trimester
- O45.90 Premature separation of placenta, unsp, unsp trimester

- O71.9 Obstetric trauma, unspecified

PREGNANCY, UNCOMPLICATED

Jonathan B. Walker • James S. Walker



BASICS

DESCRIPTION

- Pregnancy is not a disease process but rather a physiologic state. It involves severe metabolic stresses on the mother to facilitate the growth and development of the fetus.
- All women of reproductive age with abdominal pain are considered pregnant until proven otherwise even with history of sterilization.
- The changes in pregnancy occur from the production of large amounts of placental hormones:
 - Placental progesterone and estrogen

Pediatric Considerations

- Range for menarche in US is 11–15 yr old
- Pregnant adolescents who present to the ED may be either unaware of the pregnancy or reluctant to admit it:
 - Assume pregnancy in adolescents, regardless of the chief complaint
 - Pediatric pregnancies have an increased risk of obstructive labor

ETIOLOGY

- Preceding signs and symptoms can be explained by elevations in various hormone levels or changes in anatomy that are a function of the progression of the pregnancy.
- Placental human chorionic gonadotropin (hCG):
 - Prevents the normal involution of the corpus luteum at the end of the menstrual cycle
 - Causes the corpus luteum to secrete even larger quantities of estrogen and progesterone
 - Elevated hCG levels are responsible for nausea and vomiting.
- Placental progesterone:
 - Causes decidual cells in the endometrium to develop and provide nutrition for the early embryo
 - Decreases contractility of the gravid uterus and risk of spontaneous abortion
 - Helps estrogen prepare the breasts for lactation
- Placental estrogen:
 - Responsible for enlargement of uterus, breasts, and mammary ducts
 - Enlargement of female external genitalia, relaxation of pelvic ligaments, symphysis pubis, and sacroiliac joints

DIAGNOSIS

The diagnosis of pregnancy and some of its potential complications focus on 3 diagnostic tools:

- History and physical exam
- Hormonal assays
- Ultrasonography

SIGNS AND SYMPTOMS

- Amenorrhea accompanied by nausea and vomiting in a sexually active woman
- Amenorrhea:
 - _ Most common cause of secondary amenorrhea in a woman of reproductive age is pregnancy
- Nausea and vomiting (morning sickness)
- Breast tenderness (mastodynia)
- Urinary frequency
- Headache
- Low back pain
- Pica
- Edema of feet and ankles
- Weight gain
- Easy fatigability, generalized malaise
- Increase in abdominal girth
- Constipation
- Heartburn
- Excessive eructation
- Skin darkening

History

- Determine 1st day of last menstrual period (FDLMP)
- 40% of women cannot accurately remember their FDLMP

Physical-Exam

Pelvic exam:

- Estimate expected date of delivery by determining uterine fundal height
- Centimeters from pubic bone to top of uterus approximates gestational age after 16 wk
- Detect abnormal pelvic pain or masses

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Pregnancy tests:

- β -subunit of hCG
- Quantitative hCG normally doubles every 2 days until 6–7 wk gestation
- Progesterone
- Measurement of β -hCG:
 - Most urine pregnancy tests have sensitivity at 25 mIU/mL:
 - False-negative tests with dilute urine and high vitamin C intake
 - Home pregnancy tests are not that accurate:
 - Detect pregnancy 9–12 days post conception
 - Positive home pregnancy tests should be confirmed by serum hCG levels.
 - Serum level of hCG:
 - Detectable 8–11 days post conception
 - hCG levels may remain detectable up to 60 days after an abortion.
- Serum progesterone level is an indicator of the viability of the pregnancy and may be used to predict the outcome of the pregnancy:
 - A serum progesterone level of < 5 ng/mL is indicative of a nonviable pregnancy (spontaneous abortion or ectopic pregnancy).
 - Progesterone level 25 ng/mL denotes a viable pregnancy.

Imaging

- Ultrasonography is used to confirm pregnancy in the setting of abdominal pain, vaginal bleeding, or some other potential obstetric complication:
 - Can estimate gestational age
 - Confirm intrauterine or ectopic pregnancy
 - Evaluate fetal viability
 - Identify fetal abnormalities
- Transabdominal US vs. transvaginal US:
 - Transvaginal US is more sensitive but more difficult to perform.
 - Intrauterine pregnancy seen at 4–5 wk in transvaginal US
 - Gestational sac seen at 5.5–6 wk in transabdominal US
 - Transvaginal US is contraindicated in the setting of premature rupture of membranes and 3rd-trimester bleeding.
- When used in combination with hCG levels, US is a very helpful tool in detecting abnormal/problem pregnancy.
- MRI: No significant side effects have been documented.
 - Often the study of choice to evaluate for appendicitis in pregnancy
- Plain radiography and CT:
 - Dose-dependent teratogen
 - Slight increase in risk of childhood cancer
 - Goal is to not exceed 5,000 mrad fetal dose of radiation:
 - CXR with abdominal shield: < 1 mrad
 - Abdominal plain film: 240 mrad
 - Chest CT: < 10 mrad
 - Head CT: < 10 mrad

- Abdominal CT with and without contrast: 2,000 and 1,000 mrad
- Cardiac catheterization: 1,300 mrad
- VQ scan: < 50 mrad

DIFFERENTIAL DIAGNOSIS

Any woman who is of the age to be sexually active who presents to the ED should be assumed to be pregnant until proven otherwise.



TREATMENT

PRE HOSPITAL

- Assume the patient is pregnant
- Administer medications only when necessary to avoid teratogenic side effects or placental–fetal compromise (e.g., epinephrine)
- If > 24 wk gestation, transport in left lateral recumbent position

INITIAL STABILIZATION/THERAPY

- Advanced cardiac life support, advanced trauma life support measures as needed: Oxygen, cardiac monitor, IV access, and fluids:
 - 1st objective is to resuscitate mother
- If > 24 wk gestation, place in the left lateral recumbent position

ED TREATMENT/PROCEDURES

The goal is to optimize maternal condition to improve fetal condition.

MEDICATION

- 1st trimester is when organogenesis is occurring.
- Fetal malformation continues beyond the 1st trimester.
- Before using any drug, refer to its Food and Drug Administration safety classification in pregnancy:
 - This classification system categorizes drugs as A, B, C, D, and X, with category A being the safest and category X being the most toxic.
- Analgesics: Acetaminophen is the preferred OTC analgesic
- Aspirin and NSAIDs are not teratogenic but are best used in consultation with an obstetrician
- Oxycodone, codeine, hydrocodone, meperidine, and morphine have no known teratogenic affect and can be used for the control of severe pain in pregnancy for short periods of time (3–4 days).
- Antibiotics: Selecting the right antibiotic in a gravid female depends on 3 factors:
 - Maternal drug allergies
 - Gestational age
 - Type of infections and associated pathogens
- Consider placing patient on prenatal vitamins

- Pain control:
 - Acetaminophen: 500 mg PO q6h; do not exceed 4g/d
- Antiemetic:
 - Ondansetron: 4 mg IM/IV q8h
 - Vitamin B₆ 25 mg TID or ginger can also help



FOLLOW-UP

DISPOSITION

Admission Criteria

- Pregnant women with the following obstetric complications should be admitted to the hospital:
 - Hyperemesis gravidarum with inability to tolerate oral fluids
 - Complicated UTI
 - Ectopic or molar pregnancy
 - Septic abortion
 - Preterm labor
 - Premature rupture of membranes
 - Preeclampsia/eclampsia
 - Severe pregnancy-induced HTN
- Pregnant women with medical conditions that would warrant admission in a nongravid female.

Discharge Criteria

Women without the above conditions may be discharged from the ED.

FOLLOW-UP RECOMMENDATIONS

Need OB follow up for prenatal care by 6–8 wk gestation

PEARLS AND PITFALLS

- All women are considered to be pregnant until proven they are not
- Review all medications' pregnancy safety classifications before administering or prescribing
- Minimize radiation exposure to fetus to <5,000 mrad

ADDITIONAL READING

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CODES

ICD9

- V22.0 Supervision of normal first pregnancy
- V22.1 Supervision of other normal pregnancy
- V22.2 Pregnant state, incidental

ICD10

- Z33.1 Pregnant state, incidental
- Z34.80 Encounter for suprvsn of normal pregnancy, unsp trimester
- Z34.90 Encntr for suprvsn of normal pregnancy, unsp, unsp trimester

PRIAPISM

David Barlas

BASICS

DESCRIPTION

- Penile erection (engorgement of corpora cavernosa) in the absence of sexual arousal that is prolonged and frequently painful
- Low-flow priapism:
 - _ Most common mechanism
 - _ Poor venous outflow
 - _ Usually painful
 - _ Ischemia and thrombosis from stagnant, hypoxic blood can occur after a few hours.
 - _ Fibrosis and erectile dysfunction are late sequelae.
- High-flow priapism:
 - _ Rare
 - _ Penile arterial laceration with uncontrolled inflow of arterial blood
 - _ Usually painless
 - _ Presentation may be later than in low-flow priapism.
 - _ Ischemia and erectile dysfunction are uncommon.

ETIOLOGY

- Idiopathic
- Pharmacologic agents:
 - _ Intracavernosal injectables for the treatment of erectile dysfunction:
 - Prostaglandin E1
 - Papaverine
 - Phentolamine
 - _ Psychotropics:
 - Phenothiazines
 - Butyrophenones
 - Trazodone
 - Sedative–hypnotics
 - Selective serotonin uptake inhibitors
 - _ Antihypertensives:
 - Prazosin
 - Hydralazine
 - Phenoxybenzamine
 - Guanethidine
 - _ Rarely implicated agents:

- Phosphodiesterase inhibitors: Sildenafil (Viagra), tadalafil (Cialis), vardenafil (Levitra)
- Anticoagulants
- Cocaine
- Marijuana
- Ethanol
- Androstenedione
- Toxins (Black Widow, Scorpion)
- Hematologic disorders predisposing to sludging of blood:
 - Sickle cell anemia (most common cause)
 - Leukemia
 - Multiple myeloma
 - Polycythemia
- Penile and perineal trauma (arterial laceration and high-flow priapism)
- Spinal trauma (loss of inhibitory adrenergic tone)
- Rare causes:
 - Pelvic neoplasms and infections
 - Infiltrative diseases (e.g., amyloidosis)
 - Dialysis
 - Parenteral nutrition solutions containing a fat emulsion

Pediatric Considerations

Sickle cell anemia is the cause of most priapism in children.

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Type of priapism may be determined by history:
- Low-flow priapism:
 - Painful
 - Predisposing condition or medication
- High-flow priapism:
 - Painless
 - Penile trauma

Physical-Exam

- Diagnosis is clinically apparent.
- Check for penile implants.
- Evaluate trauma (i.e., urethral, rectal injuries).
- Urinary retention

ESSENTIAL WORKUP

Lab tests and imaging should not delay urologic consultation and definitive management.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC
- Coagulation studies
- Sickle cell evaluation may be indicated.

Imaging

- Duplex Doppler US can verify and localize the arterial laceration in high-flow priapism.
- Angiography enables localization and embolization of the arterial laceration in high-flow priapism.

Diagnostic Procedures/Surgery

Intracavernosal blood gas analysis can help differentiate type of priapism if unsure:

- Because of the possibility of penile arterial injury, a urologist should perform this procedure.
- High-flow priapism: Near-normal values
- Low-flow priapism: Acidosis and hypoxia (pH <7.25; O₂ <30 Torr)

DIFFERENTIAL DIAGNOSIS

- Penile erection from sexual arousal is usually painless and transient.
- Penile implants are a benign cause of “priapism.”



TREATMENT

PRE HOSPITAL

- IV
- O₂
- Analgesia

INITIAL STABILIZATION/THERAPY

- O₂
- Analgesia and sedation
- IV hydration

ED TREATMENT/PROCEDURES

- Urgent urologic consultation

- Management of specific causes should begin concurrently with specific therapy outlined below:
 - Sickle cell anemia:
 - Packed RBC or exchange transfusion
 - Hyperbaric oxygen if other measures fail
 - Leukemia:
 - Chemotherapy
 - Arterial injury:
 - Expectant management is an option
 - Angiographic localization and embolization
- Terbutaline (β -agonist):
 - May be administered to initiate treatment of low-flow priapism, but may not be effective alone.
- Intracavernosal injection/aspiration is often required for low-flow priapism despite the above measures:
 - Ideally performed by urologist, but the ED physician may perform the procedure as follows if specialty care is not immediately available:
 - Sterile prep area
 - Consider IV sedation and analgesia
 - Administer local anesthesia, or perform a pudendal nerve block or penile nerve block (inject plain lidocaine around the base of the penis)
 - Position yourself to the right of the patient and grasp the penile shaft with your left hand
 - Enter the corpus cavernosum with a 19G butterfly needle and 10 mL syringe inserted laterally at 2- or 10-o'clock position and 45° angle to avoid the ventral urethra and the dorsal neurovascular bundle
 - Aspirate blood *slowly* while “milking” the penile shaft until arterial blood is obtained, often after 30–50 mL. Irrigation with saline may be necessary.
 - Aspirating both corpora cavernosa is unnecessary as they are connected by shunts.
 - Phenylephrine (preferred to limit systemic effects), or epinephrine may be injected through the butterfly needle if retumescence occurs. Monitor cardiac rhythm and BP if these agents are used and avoid in patients with cardiovascular or cerebrovascular disease, hypertension, or those taking monoamine oxidase inhibitors because of the risk of hypertensive crisis.
 - Repeated injections may be needed 5–15 min apart for 1 hr.
 - Surgical shunt (i.e., corpus cavernosum to spongiosum) may be necessary if the above measures fail.

First Line

- Terbutaline: 0.25–0.5 mg SC (may repeat in 15 min) or 5 mg PO
- Phenylephrine: Dilute 1 mg in 100 mL saline; inject 10 mL boluses in the corpus cavernosum.

Second Line

- Epinephrine: Dilute 1 mg in 100 mL saline; inject 1–3 mL boluses in the corpus cavernosum, up to 10 mL.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Persistent priapism despite noninvasive treatments
- Serious underlying disease (sickle cell anemia, leukemia)

Discharge Criteria

- Detumescence is complete and has not recurred after several hours of observation
- Urologic consultation has been obtained

Issues for Referral

Arrange short-term follow-up with a urologist for all patients.

FOLLOW-UP RECOMMENDATIONS

- Ensure underlying conditions are addressed.
- Discontinue offending medication(s).
- Advise patient to return to the ED if tumescence recurs.

PEARLS AND PITFALLS

- Intracavernosal injection of vasoactive medications during partial tumescence increases the risk of a systemic bolus and adverse effects.
- Management of underlying conditions should not delay timely direct therapy to reduce the risk of subsequent erectile dysfunction.
- Always document and warn of the possibility of subsequent complete erectile dysfunction even when timely and successful treatment has occurred.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Conscious Sedation
- Paraphimosis
- Penile Shaft Fracture
- Sickle Cell Disease



CODES

ICD9

607.3 Priapism

ICD10

- N48.30 Priapism, unspecified
- N48.33 Priapism, drug-induced
- N48.39 Other priapism

PROCEDURAL SEDATION

Christopher Ross • Theresa Schwab

BASICS

Administration of agents with or without analgesia to induce a state that allows diagnostic and therapeutic procedures to be performed successfully without significant pain and/or anxiety while maintaining cardiorespiratory function.

Preparation

- Obtain consent
- Equipment
 - Breathing masks, bag-valve ventilation device, oropharyngeal and nasal airways, laryngoscopes, endotracheal tubes, and stylets appropriate for size of patient
 - Defibrillator/automated external defibrillator
 - Suction
 - Emergency cart with all available medications if resuscitation needed
 - Flumazenil and naloxone
- Apply cardiorespiratory monitor, pulse oximeter, and BP monitor
- Gather medicines that will be used in procedure, label (preferably color-coded) syringes, and place at bedside.
- Apply oxygen delivery device (cannula or mask) and keep oxygen saturation > 95%.

Sedation Agents and Techniques

- Can be administered by various means
- Administer local or regional anesthesia if applicable.
- Perform the procedure.
- Closely observe and monitor patient during entire course of procedure as well as the recovery period afterward.
- Monitor patient until awake, alert, and back to baseline function.

DIAGNOSIS

SIGNS AND SYMPTOMS

History

Past medical history, anesthesia history, medications, allergies, review of systems and last meal.

Physical-Exam

- BP, heart rate, respiratory rate, pulse oximetry, cardiopulmonary and neurologic exams.

- Airway assessment:
 - _ Difficult bag-valve mask ventilation:
 - Beards
 - Abnormal facial contour
 - Morbid obesity
 - No teeth
 - Patients with COPD/asthma/congestive heart failure)
 - _ Difficult airway management:
 - Short neck
 - Large tongue
 - Small mandible
 - Mallampati scoring
 - Evidence of airway obstruction (stridor, drooling, and dysphagia).



TREATMENT

MEDICATION

ALERT

Many sedative and analgesic medications can cause dose-dependent respiratory depression. Combination of 2 or more of these agents may have a synergistic effect on respiratory depression that can lead to hypoxia and apnea.

- Painless procedures (single agents):
 - _ Methohexital
 - _ Choral hydrate (children)
 - _ Etomidate
 - _ Barbiturates (pentobarbital)
 - _ Benzodiazepines (midazolam)
 - _ Ketamine
- Painful procedures:
 - _ Ketamine (\pm barbiturates [midazolam] to reduce emergence reactions)
 - _ Ketamine and propofol (ketofol)
 - _ Opiates (fentanyl/remifentanyl) and midazolam
 - _ Nitrous oxide
 - _ Propofol and opiates (fentanyl)
 - _ Etomidate and opiates (fentanyl)
 - _ Dexmedetomidine
- Methohexital:
 - _ A short-acting barbiturate with rapid recovery that produces a state of unconsciousness and profound amnesia but has no analgesic properties
 - _ Dosage (IV): 0.75–1 mg/kg with subsequent titration at 0.5 mg/kg every 2

min to required effect

- Onset: < 1 min
- Duration of action: 10 min
- Side effects:
 - Respiratory depression with potential apnea
 - Hypotension due to myocardial depressant effect (caution with underlying myocardial disease)
- Pentobarbital:
 - A short-acting barbiturate used only for *painless* procedures as a sole agent for diagnostic modalities:
 - Dosage (IV): 2–5 mg/kg for children, and adults have a loading dose of 100 mg slow bolus repeated/titrated q3–5min to max. of 200–500 mg.
 - Dosage (IM): 4 mg/kg
 - Duration of action: 30–60 min
 - Onset: IV mode acts within 30 sec, and patient is appropriately sedated within 5 min.
 - Side effects: Central nervous system, respiratory depression, and bronchospasm (contraindicated in asthma/COPD)
- Benzodiazepines:
 - Commonly used for minimal sedation as it causes anxiolysis and amnesia
- Midazolam:
 - Short-acting agent that is lipophilic that allows penetration of the blood–brain barrier quickly
 - Provides anxiolysis and amnesia but not analgesia, so should not be the sole agent for painful procedures
 - Dosage (IV): 0.02–0.05 mg/kg (usual starting adult dose is 0.5–1 mg) with subsequent incremental doses at 3 min intervals to desired effect (single max. dose, 2.5 mg)
 - Dosage (IM): 0.05–0.2 mg/kg
 - Dosage (PO): 0.5–0.75 mg/kg (max. of 15 mg)
 - Dosage (nasal): 0.2–0.5 mg/kg (max. of 5 mg)
 - Onset/duration:
 - IV: Fairly rapid onset (1–2 min) and short duration of action (10–40 min)
 - IM, PO, and nasally: Slower onset and longer duration of action
 - Cautions for benzodiazepines:
 - Respiratory depression
 - Hypotension
 - Excessive sedation
 - Effects augmented by opioids, so reduce dose by 30–50% if opioid therapy utilized simultaneously
 - Effects may be reversed with flumazenil: 200 µg every 1–2 min, to effect
- Chloral hydrate:

- To be used in procedural sedation in children (< 2 yr old) undergoing painless diagnostic studies
- Dosage (PO): 50–100 mg/kg with usual dose of 50–75 mg/kg (max. 2 g)
- Dosage for rectal administration: Not recommended due to erratic absorption Max. dose 1 g in infants, 2 g noninfants
- Onset: 30–45 min
- Duration of action: 2–4 hr (effects can recur up to 24 hr)
- Side effects:
 - Nausea and vomiting
 - Respiratory depression
 - Prolonged sedation
 - Rarely paradoxical excitation
- **Dexmedetomidine:** A short-acting, rapidly cleared α_2 -adrenergic agonist with sedative, anxiolytic, and analgesic properties:
 - Dosage IV: 1 μ g/kg loading dose over 5–10 min followed by infusion at 0.2–1 μ g/kg/h (use half dose for elderly or less invasive procedures)
 - Onset: Progressive during loading dose cycle
 - Duration: 6 min after cessation of infusion
 - Side effects:
 - Moderate BP and heart rate reductions should be expected, but alternative agent should be used if bradycardia and/or severe heart block.
- **Etomidate:** Unique class of sedative–hypnotic:
 - Produces amnesia and sedation but *not* analgesia
 - Minimal cardiovascular and respiratory effects
 - Dosage IV: 0.1–0.15 mg/kg
 - Onset: < 1 min
 - Duration: 5 min
 - Side effects:
 - Myoclonus that seems to be related to dose and speed of administration
 - Nausea and vomiting
 - Hypotension and respiratory depression when combined with opioid or benzodiazepine
 - Adrenocortical suppression
- **Ketamine**
 - Produces analgesia, amnesia, and sedation due to its dissociative effect while maintaining spontaneous respirations and airway reflexes
 - Dosage (IV): 0.5–1 mg/kg (use midazolam 0.05 mg/kg and atropine 0.01 mg/kg concurrently) with onset of action 5–10 min
 - Dosage (IM): 2–4 mg/kg (combine atropine and midazolam in same syringe) with onset of action 15–25 min

- Dosage (PO): 5–10 mg/kg (use midazolam 0.5 mg/kg and atropine 0.02 mg/kg PO as well) with onset of 30–45 min
- Duration of action: IV 15–45 min; IM 30–90 min; PO 60–120 min
- Side effects:
 - Hypertension and tachycardia
 - Increases intracranial and intraocular pressure
 - Stimulates salivary and tracheobronchial secretions
 - Emergence reactions with hallucinations reported, but are less frequent in children <10 yr; the incidence can be reduced by premedication with midazolam in adults.
- Nitrous oxide
 - Inhalational agent administered in 50% nitrous oxide/oxygen concentration:
 - Excellent agent for quick procedures, as it provides analgesia, anxiolysis, and sedation without the need for IV placement
 - Onset of action: 30–60 sec
 - Duration of action: 3–5 min after ceasing inhalation
 - Side effects are rare but can cause deep sedation with respiratory depression (especially if concurrent narcotic) as well as nausea and vomiting.
 - Contraindications: Pregnancy, pneumothorax, and bowel obstruction
- Fentanyl
 - Ultra-short acting synthetic opioid
 - Analgesic properties but minimal sedative and no amnestic properties
 - Dosage (IV): 1–4 µg/kg (titrate)
 - Onset (IV): 30–60 sec with peak at 1–3 min
 - Transmucosal 10–15 µg/kg with onset in 15–20 min:
 - Oral lozenge (Oralet) allows patient to suck on drug, which then can be removed by physician or patient when adequate sedation achieved.
 - Duration of action: 30 min
- Remifentanyl
 - Potent ultra-short-acting synthetic with potent sedative and analgesic properties.
 - Dosage (IV): 0.5–1 µg/kg every 2 min titrated to effect
 - Onset (IV): <1 min
 - Duration of action: 5 min
 - Side effects of fentanyl and remifentanyl:
 - Respiratory depression with potential apnea
 - Hypotension
 - Chest wall rigidity is a rare complication when large doses given quickly.
 - Use <1/3 dose in children <6 mo.
 - Emesis with transmucosal preparation
- Propofol: Rapid onset and short duration make for excellent ED agent for

procedural sedation:

- Produces amnesia and sedation but *not* analgesia
- Onset of action: 15–45 sec
- Dosage (IV): 0.5–1 mg/kg bolus (usually given as 20 mg boluses every 10 sec in adults until desired effect is obtained) followed by infusion of 50–75 µg/kg/min; effective total dose for adults 20–150 mg
- Duration of action: < 2 min
- Side effects:
 - Dose-related respiratory depression with occasional apnea (care with COPD)
 - Hypotension (care with cardiomyopathy or hypovolemia)
 - Pain at injection site
- Care with patients in renal failure due to accumulation of active metabolite leading to prolonged sedation

Reversal agents

- **Naloxone:** Opioid antagonist:
 - For reversal of respiratory depression, apnea, and severe hypotension
 - Dosage: 0.1–0.2 mg/kg IV/IM in incremental doses (to total of 2 mg) q1–2min to the desired reversal effect; usual adult dose of 1–2 mg effective
 - Duration of action: 20–45 min

ALERT

Naloxone may induce severe opioid withdrawal (nausea and vomiting, agitation, abdominal pain with diarrhea) for those on chronic opioid therapy.

- **Flumazenil:** Benzodiazepine antagonist:
 - Reverses CNS depression and some degree of respiratory depression
 - Dosage (IV): 0.01 mg/kg per dose (max. initial dose 0.2 mg) repeated at 1-min intervals to desired effect or max. 0.05 mg/kg or 1 mg
 - Duration of action: 20–45 min

ALERT

Flumazenil may induce severe benzodiazepine withdrawal (seizures, agitation, psychosis, nausea and vomiting, and muscle spasm) for those on chronic benzodiazepine therapy.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Postprocedural sedation

- Inability to walk
- No responsible adult to accompany patient home
- Reason for undergoing conscious sedation still present
- Postprocedure complication

Discharge Criteria

- Patient is awake, alert, and at baseline
- Procedure was of sufficiently low risk that additional monitoring for complications is unnecessary
- Stable hemodynamically
- Ambulatory 30 min before discharge
- Able to urinate
- Able to retain oral fluids
- Pain controlled
- Under observation of a responsible person and have transportation from the hospital

PEARLS AND PITFALLS

- All airway adjuncts should be readily available in case of respiratory compromise.
- All reversal agents should be readily available in case of inadvertent overdose of medications.
- Patients must have continuous cardiorespiratory monitoring during and after procedural sedation.

ADDITIONAL READING

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PROSTATITIS

Nicole M. Franks

BASICS

DESCRIPTION

- Acute (bacterial) prostatitis:
 - Acute febrile illness
 - Systemic symptoms may appear days before localizing urinary symptoms appear.
 - Patients may appear toxic and usually have a concurrent cystitis.
- Prostatic abscess:
 - Once common after acute prostatitis, now rare except in immunocompromised patients
 - Fever, rectal pain, and leukocytosis despite treatment
 - Fluctuant mass on rectal exam
- Chronic bacterial prostatitis:
 - ~10% of cases of prostatitis
 - Most common cause of recurrent UTI in men
 - WBC and bacteria may be present in expressed prostatic secretions (EPS).
- Chronic nonbacterial prostatitis (also called prostaticitis):
 - Same symptoms as chronic bacterial prostatitis but unable to culture organisms from urine or EPS
- Chronic pelvic pain syndrome (CPPS):
 - Symptoms referable to the prostate
 - No inflammatory cells are found
 - No bacteria cultured from the urine or EPS

ETIOLOGY

- Usually a single-organism bacterial infection of the prostate
- Acute prostatitis:
 - Age < 35 yr:
 - *Neisseria gonorrhoeae* and *Chlamydia trachomatis* are usual etiologies.
 - Age ≥ 35 yr:
 - Enterobacteriaceae or *Escherichia coli* (usual), *Klebsiella*, *Pseudomonas*, *Enterococcus*, and *Proteus* also seen
 - Rarely may be caused by *Salmonella*, *Clostridia*, tuberculosis, or fungi.
 - *Cryptococcus neoformans* in AIDS patients
- Chronic bacterial prostatitis:
 - Enterobacteriaceae (80%), *Enterococcus* (15%), and *Pseudomonas aeruginosa*
- Chronic nonbacterial prostatitis:

- Possible role for *Chlamydia*, *Ureaplasma urealyticum*, *Trichomonas vaginalis*, and *Mycoplasma hominis*

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Irritative voiding symptoms:
 - Frequency, urgency, dysuria
- Low back pain
- Perineal, suprapubic, or testicular pain
- Bladder outlet obstruction and urinary retention
- Ejaculatory symptoms such as hematospermia
- Acute prostatitis:
 - Fever, chills
 - Malaise
 - Arthralgias or myalgias
- Primary symptom in chronic prostatitis is relapsing dysuria.

Physical-Exam

- Acute prostatitis:
 - Exquisitely prostate tenderness
 - Warm, swollen
 - Firm or boggy prostate
 - Acutely inflamed prostate should not be massaged because that may precipitate hematogenous spread of organisms.
- In chronic prostatitis, the exam is usually normal.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Urinalysis (with microscopy) and culture
- Acute prostatitis:
 - CBC, electrolytes, and blood cultures may be helpful in the acutely ill patient.
 - If < 35 yr old or suspected sexual transmission, test for syphilis:
 - Venereal Disease Research Lab or rapid plasma reagin
- Chronic prostatitis/CPPS:
 - Prostatic massage between voiding may be used to capture EPS for Gram stain and culture if organism or white cells not present in the urine.

Imaging

- Not indicated in acute prostatitis
- If prostatic abscess suspected, transrectal US or pelvic CT with IV and rectal contrast will confirm diagnosis.

Diagnostic Procedures/Surgery

Not applicable in ED

DIFFERENTIAL DIAGNOSIS

- Benign prostatic hyperplasia
- Cystitis
- Epididymitis
- Orchitis
- Perirectal/perianal abscess
- Proctitis
- Prostatic carcinoma
- Prostatic infarction
- Pyelonephritis
- Seminal vesiculitis
- Urethritis
- Urolithiasis
- Vesicular calculi
- Other causes of lower back pain (strain, disc disease, sacroiliac joint disease, etc.)



TREATMENT

INITIAL STABILIZATION/THERAPY

Initial resuscitative measures as indicated

ED TREATMENT/PROCEDURES

- Prostatic abscess requires urgent urologic consultation and transrectal US-guided aspiration.
- Antibiotic therapy should be initiated in ED (see Medications).
- Urinary tract instrumentation should be avoided:
 - If patient has painful urinary retention in acute prostatitis, suprapubic needle aspiration or suprapubic catheter placement should be performed.
- Many patients will benefit from IV fluid.
- Pain control with NSAIDs and narcotic analgesics as needed
- Stool softeners
- Bed rest
- Irritative voiding symptoms may persist for months after antibiotic therapy and may be treated with NSAIDs.

MEDICATION

- Analgesia:
 - Narcotic, analgesic combinations such as hydrocodone/acetaminophen: 1–2 tabs PO q4h
 - NSAIDs such as ibuprofen: 800 mg PO TID
- Parenteral antibiotic therapy for acute prostatitis:
 - Levofloxacin: 750 mg IV daily
 - Ampicillin/sulbactam: 3 g IV q6h
 - Cefotaxime: 2 g IV q8h
 - Ceftriaxone: 2 g IV daily
 - Ciprofloxacin: 400 mg IV BID
 - Ofloxacin: 200 mg IV BID
 - Piperacillin/tazobactam: 3.375 g IV q6h or 4.5 g IV q8h
 - Ticarcillin/clavulanate: 3.1 g IV q6h
- Antibiotics for outpatient treatment of acute (≤ 35 yr old) prostatitis, suspected etiology *N. gonorrhoeae* or *C. trachomatis*:
 - Ceftriaxone: 250 mg IM, then doxycycline: 100 mg PO BID \times 10–14 days
 - Levofloxacin: 500 mg PO every day for 10–14 days
 - Ofloxacin: 400 mg PO \times 1, then 300 mg PO BID \times 10–14 days
- Antibiotics for outpatient treatment of acute (> 35 yr old) prostatitis, suspected etiology Enterobacteriaceae (coliforms); some authorities recommend 3–4 wk of therapy:
 - Ciprofloxacin: 500 mg PO BID \times 14 days
 - Levofloxacin: 500 mg PO every day for 14 days
 - Ofloxacin: 200 mg PO BID \times 14 days
 - Trimethoprim/sulfamethoxazole: 1 double-strength (DS) tab or 2 regular-strength tabs PO BID \times 28 days
- Outpatient therapy for chronic bacterial prostatitis (Enterobacteriaceae, *Enterococcus*, or *P. aeruginosa*):
 - Ciprofloxacin: 500 mg PO BID for 4 wk
 - Levofloxacin: 500 mg PO every day for 4 wk
 - Ofloxacin: 300 mg PO BID for 6 wk
 - Trimethoprim/sulfamethoxazole DS: 1 tab PO BID for 1–3 mo
- CPPS:
 - Tamsulosin: 0.4 mg PO every day
 - Doxazosin: 1 mg PO (immediate release) every day
 - Peripheral β -adrenergic blocking agents have been used with some success; consult a urologist.
 - Prazosin: 1 mg PO BID/TID
 - Terazosin: 1 mg PO qhs



FOLLOW-UP

DISPOSITION

Admission Criteria

- Acute prostatitis:
 - Patients who appear ill or toxic
 - Hypotension
 - Urinary retention
- Chronic prostatitis:
 - Admission generally not warranted unless patient has signs or symptoms of acute prostatitis.

Discharge Criteria

- Acute prostatitis:
 - Patient must be nontoxic.
 - Able to take fluids and oral medications (analgesia and antibiotics)
 - Urinate without difficulty
 - Immunocompetent
 - Relatively free of concurrent underlying disease
 - Have appropriate follow-up care
- Chronic prostatitis: Appropriate follow-up care should be available.

Issues for Referral

Patient with either acute or chronic prostatitis should be referred to an urologist.

PEARLS AND PITFALLS

- Obtain a good history to distinguish acute from chronic prostatitis, as longer antibiotic therapy may be warranted.
- Consider this diagnosis even in sexually active adolescent males.
- Acutely ill males with antibiotic treatment failure for prostatitis should be evaluated for abscess regardless of immunocompetence.

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CODES

ICD9

- 601.0 Acute prostatitis
- 601.1 Chronic prostatitis
- 601.9 Prostatitis, unspecified

ICD10

- N41.0 Acute prostatitis
- N41.1 Chronic prostatitis
- N41.9 Inflammatory disease of prostate, unspecified

PRURITUS

Christine Tsien Silvers

BASICS

DESCRIPTION

- Unpleasant sensation that provokes a desire to scratch
- Mediated by unmyelinated C fibers in upper portion of dermis:
 - Transmitted to dorsal horn of spinal cord
 - Via spinothalamic tract to cerebral cortex
- Peripheral mediators (e.g., histamine and peptides such as substance P that release histamine) stimulate C fibers and induce itching
- Prostaglandins (PGE₂, PGH₂) lower threshold to pruritus
- Opiates cause pruritus by acting on central receptors
- No single pharmacologic agent effectively treats all causes of pruritus
- “Itch–scratch–itch” cycle:
 - Itching triggers scratching
 - Scratching damages skin and stimulates nerve endings, thereby producing even greater itching

ETIOLOGY

4 categories in proposed itch classification:

- Pruritoceptive: Generated in the skin from localized irritation or inflammation
- Neurogenic: Generated in the CNS due to circulating pruritogens
- Neuropathic: Due to CNS or PNS lesions
- Psychogenic

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Onset:
 - Shortly after freshwater bathing in swimmer’s itch
 - More intense at night with scabies
 - Paroxysmal with multiple sclerosis
 - With sudden changes in temperature in polycythemia vera
- Character: Paroxysmal, burning, pricking
- Time of occurrence, duration
- Severity; impact on quality of life
- With or without skin lesions

- Anatomic area (e.g., exposed skin only)
- Exacerbating or alleviating factors (e.g., water, heat, dryness, dampness, coolness)
- Medications
- New products (e.g., soap, cosmetics, laundry detergents, fabric softeners)
- Age
- Family history of atopic dermatitis or skin disease
- Personal history of allergies or asthma
- Pruritus in other family members
- Systemic or associated symptoms (e.g., night sweats, fever, tremors, weight loss, fatigue, jaundice, anemia, neurologic symptoms)
- Sexual history, history of HIV or AIDS
- Social: Occupation, hobbies, pets, travel

Physical-Exam

- Dermatologic:
 - _ Absence of rash
 - _ Diffuse or localized rash
 - _ Location: Genitals, interdigital webs, axilla, wrists, etc.
 - _ Generalized morbilliform eruptions
 - _ Discrete weeping patches with vesicles
 - _ Dry skin
 - _ Jaundice
 - _ Follicular (around the hair)
 - _ Nonfollicular (e.g., insect bites, scabies)
 - _ Primary lesions:
 - Papular, pustular, urticarial, or polymorphic
 - _ Secondary lesions:
 - Excoriations
 - Lichenification
 - Hyperpigmentation
 - Prurigo papules: Thickened papular areas of skin from constant rubbing
- Psychogenic: Constant rubbing in areas patient can readily reach

ESSENTIAL WORKUP

- Detailed history is key in the ED workup
- Physical exam to characterize skin lesions
- Look for evidence of systemic disease

DIAGNOSIS TESTS & INTERPRETATION

Lab

Indications for specific studies (e.g., CBC and differential, ESR, CRP, BUN/creatinine,

glucose, LFTs, TSH, free T4, HIV, RPR, cancer screening, CXR, abdominal ultrasound, CT/MRI) vary based on the clinical presentation and should be guided by clinical judgment.

Diagnostic Procedures/Surgery

- Skin scrapings for scabies and dermatophytoses
- Skin biopsy performed by dermatologist at follow-up visit
- Skin culture for bacterial, viral, or fungal infection

DIFFERENTIAL DIAGNOSIS

Dermatologic

- Xerosis (dry skin)
- Insect infestations:
 - Scabies: Vesicles and burrows on intertriginous areas
 - Pediculosis (lice)
- Insect bites: Localized clusters of papules
- Dermatitis:
 - Atopic dermatitis
 - Contact dermatitis (e.g., poison ivy contact)
 - Nummular dermatitis: Round eczematous or vesicular eruption
- Drug induced (suspect when no rash):
 - Opiates and derivatives
 - Aspirin/NSAIDs
 - Quinidine; amiodarone
 - Certain antibiotics, antifungals, antimalarials
 - Phenothiazines
 - Estrogens, progestins, testosterone
 - Statins
 - Others
- Lichen planus: Lichenification, hyperpigmentation, skin thickening
- Urticaria
- Bullous pemphigoid
- Eosinophilic folliculitis
- Psoriasis
- Dermatitis herpetiformis: Burning itch
- Sunburn
- Aquagenic pruritus
- Fiberglass dermatitis
- Seborrheic dermatitis: Scaly plaques on sebaceous gland-bearing areas
- Swimmer's itch, schistosome cercarial dermatitis, or schistosomiasis:
 - Repeated freshwater exposure
 - Itching starts as water evaporates

- Highly pruritic papules develop hours later
- Miliaria rubra (prickly heat)

Pregnancy Considerations

- Polymorphic eruption of pregnancy
- Pemphigoid gestationis
- Intrahepatic cholestasis of pregnancy
- Atopic eruption of pregnancy

Infectious

- HIV
- Parasites:
 - Ankylostomiasis/helminthiasis (hookworm)
 - Onchocerciasis/river blindness (nematode)
 - Ascariasis (roundworm)
 - Trichinosis (roundworm)

Cholestatic

- Obstructive biliary disease
- Primary biliary cirrhosis
- Hepatic cholestasis secondary to drugs
- Intrahepatic cholestasis of pregnancy
- Extrahepatic biliary obstruction
- Chronic hepatitis, especially hepatitis C

Hematologic

- Polycythemia vera
- Iron-deficiency anemia
- Paraproteinemia
- Waldenström macroglobulinemia
- Mastocytosis

Neoplastic

- Lymphoma, including Hodgkin lymphoma
- Mycosis fungoides
- Leukemia
- CNS tumors
- Multiple myeloma
- Carcinoid
- Visceral malignancies (breast, stomach, lung)

Metabolic-Endocrine

- Uremia
- Hyperthyroidism
- Hypothyroidism
- Hyperparathyroidism
- Diabetes mellitus
- Carcinoid

Neurologic

- Multiple sclerosis: Paroxysmal itching
- Notalgia paraesthetica: Local itch of back, medial shaft scapula
- Brain abscess
- CNS infarct
- Cerebral tumor
- Creutzfeldt-Jakob disease

Renal

- Chronic renal failure
- Chronic hemodialysis

Rheumatologic

- Sjögren syndrome
- Dermatomyositis

Psychiatric

- Stress, anxiety, neurotic excoriation
- Delusions of parasitosis
- Psychogenic pruritus



TREATMENT

ED TREATMENT/PROCEDURES

- Start with antihistamines for pruritus of undetermined etiology
- Emollients indicated for dry skin
- Coolants to alleviate itching, for neural modulation: Menthol, camphor, eucalyptus oil, calamine lotion, ice, topical anesthetics
- Substance P evacuators (capsaicin) block C fibers:
 - Burning sensation during 1st weeks of use
 - Anesthetic can be applied prior
- Topical glucocorticoids for contact dermatitis
- Permethrin cream for scabies and lice when rash is suggestive
- Topical antihistamines (e.g., doxepin) for eczema, urticaria, bites
- Swimmer's itch:

- Control with antihistamines, cool compresses, calamine lotion
- Topical steroids to suppress intense inflammation
- Towel dry immediately after leaving water as preventive measure
- Discontinue medications that may cause allergic reaction
- UV light for uremic pruritus
- Treat the underlying cause for pruritus associated with a systemic disease

MEDICATION

- Oral antihistamines:
 - Chlorpheniramine 4 mg (peds: 0.35 mg/kg/24h div. q 4–6h PRN; 2–6 yr max. 4 mg/24h; 6–12 yr max. 12 mg/24h) PO q4–6h PRN; max. 24 mg/24h
 - Diphenhydramine 25–50 mg (peds: 5 mg/kg/24h div. q6h PRN; 2–5 yr max. 37.5 mg/24h; 6–11 yr max. 150 mg/24h; > 12 yr max. 400 mg/24h) PO q4–6h PRN; max. 400 mg/24h
 - Hydroxyzine 25–100 mg (peds: 2 mg/kg/24h div. q6h PRN) PO q6–8h PRN; max. 600 mg/24h
- Topical treatments:
 - Capsaicin 0.025%, 0.075% cream: Apply TID–QID
 - Doxepin 5% cream: Apply QID for up to 8 days (to max. of 10% of the body)
 - EMLA (2.5% lidocaine + 2.5% prilocaine): Apply prior to capsaicin
 - Hydrocortisone 0.5%, 1%, 2.5%: Up to QID
 - Permethrin 5% cream (for scabies):
 - Apply from neck down after bath
 - Wash off thoroughly with water in 8–12 hr
 - May repeat in 7 days
 - Permethrin 1% cream rinse (for lice):
 - Shampoo, rinse, towel dry, saturate hair and scalp (or other affected area), leave on 10 min, then rinse
 - May repeat in 7 days
 - White petroleum emollients: Apply after short bath/shower in warm (not hot) water
- Other treatments for specific diseases



FOLLOW-UP

DISPOSITION

Admission Criteria

- Anaphylaxis
- Generalized exfoliating lesions
- Manifestations of systemic diseases requiring admission

Discharge Criteria

Vary by etiology

Issues for Referral

- Refer patients with skin lesions to primary care physician or dermatologist
- Stable patients with pruritus without skin lesions should be discharged on antipruritic medication and referred to a physician for an underlying systemic illness

FOLLOW-UP RECOMMENDATIONS

- Practical recommendations for dry skin:
 - Take baths with baking soda, bath oils, or colloidal oatmeal
 - Use moisturizers frequently during day and immediately after bathing
- Avoid:
 - Dry air (humidity < 40%)
 - Contact irritants (e.g., wool, cleansers)
 - Alkaline soaps and overwashing
 - Alcohol, caffeine, peppery foods
 - Overexposure to heat, hot water

PEARLS AND PITFALLS

- Detailed history is key in the ED workup
- Pruritus can be indicative of systemic illness
- No single treatment for all causes of pruritus

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Anaphylaxis
- Anemia

- Contact Dermatitis
- Eczema/Atopic Dermatitis
- Hepatitis
- HIV/AIDS
- Hyperparathyroidism
- Hyperthyroidism
- Hypothyroidism
- Leukemia
- Multiple Myeloma
- Multiple Sclerosis
- Pediculosis
- Polycythemia
- Psoriasis
- Rash
- Renal Failure
- Scabies
- Seborrheic Dermatitis
- Tinea Infections, Cutaneous
- Urticaria



CODES

ICD9

- 120.3 Cutaneous schistosomiasis
- 693.0 Dermatitis due to drugs and medicines taken internally
- 698.8 Other specified pruritic conditions

ICD10

- B65.3 Cercarial dermatitis
- L27.1 Loc skin eruption due to drugs and meds taken internally
- L29.8 Other pruritus

PSEUDOTUMOR CEREBRI

Ian Reilly



BASICS

DESCRIPTION

- Buildup of CSF pressure without mass lesion or clear etiology
- Also known as idiopathic intracranial hypertension
- 2 proposed mechanisms:
 - Increased abdominal pressure or intracranial venous stenosis may decrease venous drainage from the head
 - Vitamin A levels above the saturation of the liver can damage cell membranes in the arachnoid granulations
- Associated with obesity
- Average age of onset 30 yr
- Female predominance (7:1)
- Uncommon, ~1–5 cases per 100,000

ETIOLOGY

Proposed causative agents:

- Obesity
- Obstruction of intracranial venous drainage
- Hypervitaminosis A
- Steroids/steroid withdrawal
- Tetracycline antibiotics
- Oral contraceptive pills
- Hypertension
- Recent weight gain
- Chronic carbon dioxide retention with elevated intracranial pressure



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Headache:
 - Typically described as constant, bilateral
 - Pressure like
 - Worse in the morning
 - Worse with Valsalva maneuver
- Nausea and vomiting

- Tinnitus or pulsatile intracranial noise
- Diplopia
- Dizziness
- Scotoma
- Transient visual obscurations lasting seconds
- Blind spots
- Constriction of vision

Physical-Exam

- Visual field defects (in up to 90%):
 - Typically inferior nasal visual field loss
- Papilledema
- Lumbar puncture improves symptoms
- 6th cranial nerve palsy
- Loss of visual acuity
- Otherwise normal neurologic exam except:
 - Visual changes
 - Abducens palsy
 - Rarely 7th cranial nerve palsy

Pediatric Considerations

- Usually presents with strabismus as opposed to headache and visual field loss
- Also associated with obesity and medications (tetracycline antibiotics, steroids)

ESSENTIAL WORKUP

- Thorough history and physical exam
- Detailed neurologic assessment and fundoscopic exam

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Lumbar puncture: CSF normal or low protein with a normal cell count
- Opening pressure > 25 cm H₂O or > 20 cm H₂O in nonobese, relaxed patient
- Consider CBC, coagulation studies prior to lumbar puncture
- Improvement of symptoms with lumbar puncture

Imaging

- Head CT/MRI to rule out mass lesions (prior to lumbar puncture)
- Classically, the head CT will demonstrate slitlike frontal horns of the lateral ventricles
- MRI recommended in the full workup:
 - Can be done as an outpatient

- Cerebral venous thrombosis can mimic pseudotumor cerebri in all regards including normal head CT

Diagnostic Procedures/Surgery

- Modified Dandy criteria for diagnosis:
 - Symptoms of raised intracranial pressure
 - No localizing symptoms with exception of 6th nerve palsy
 - Patient is awake and alert
 - Normal CT/MRI findings without evidence of thrombosis
 - Lumbar puncture opening pressure > 25 cm H₂O (some suggest > 20 cm H₂O in nonobese, relaxed patients)
- Lumbar puncture
 - Opening pressure should be performed in lateral decubitus position with neck and legs straight
 - Observing respiratory variation ensures good transmission of pressure
 - Improvement of symptoms may occur with lumbar puncture

DIFFERENTIAL DIAGNOSIS

- Migraine headache
- Hypertensive headache
- Anoxic headache
- Tension headache
- Cluster headache
- Subarachnoid hemorrhage
- Aneurysm/arteriovenous malformation
- Meningitis/encephalitis
- Subdural hematoma
- Epidural hematoma
- Tumor
- Abscess
- Trigeminal neuralgia
- Giant cell/temporal arteritis
- Sinusitis
- Glaucoma
- Central retinal vein/artery occlusion
- Congenital optic nerve head elevation
- Optic nerve drusen
- Labyrinthitis
- Optic neuritis
- Cerebral venous thrombosis
- Chronic carbon dioxide retention



TREATMENT

PRE HOSPITAL

Pain control as appropriate

INITIAL STABILIZATION/THERAPY

- Airway and circulation management as indicated
- IV fluid hydration

ED TREATMENT/PROCEDURES

- Large-volume lumbar puncture of 20–30 mL of CSF:
 - Only if confident of correct diagnosis and head CT demonstrates open basilar cisterns and 4th ventricle
- Acetazolamide
- Pain control
- Neurology consult
- Ophthalmology consult
- Neurosurgery consult for acute or impending visual loss unresponsive to diuretics (for lumboperitoneal shunt)
- Optic nerve fenestration is another surgical option
- Venous sinus stenting if stenosis is present
- Weight loss
- Discontinue any drugs that could be causative
- Typically resolves spontaneously

MEDICATION

- Acetaminophen: 500 mg PO (peds: 10–15 mg/kg; do not exceed 5 doses/24 h) PO q6h; do not exceed 4 g/24 h
- Acetazolamide: 500 mg slow-release PO BID (peds: 25 mg/kg/d div. TID/QID) PO/IV
- Ibuprofen: 600–800 mg (peds: 10 mg/kg) PO q8h
- Lasix: 0.5–1 mg/kg IV/PO
- Morphine: 0.1 mg/kg IV/IM
- Prednisone: Helpful when severe visual symptoms present, 5-day course recommended (longer treatment not recommended)

First Line

- Acetazolamide
- NSAIDs

Second Line

Topiramate has been suggested as a 2nd-line agent but is not FDA approved for this use



FOLLOW-UP

DISPOSITION

Admission Criteria

Acute or impending visual loss

Discharge Criteria

- Consultation obtained from neurology and ophthalmology
- Appropriate follow-up arranged
- Tolerating oral diuretics
- Pain under control

Issues for Referral

Timely referral and return precautions:

- Visual loss
- Focal neurologic deficit
- Worsening headache

FOLLOW-UP RECOMMENDATIONS

Follow-up is recommended with neurology and ophthalmology

PEARLS AND PITFALLS

- Consider this diagnosis in younger patients with chronic headache
- Consider measuring opening pressure when performing lumbar puncture for headache
- Measure opening pressure with neck and legs straight in lateral decubitus position
- Visual changes can portend visual loss

ADDITIONAL READING

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- Galgano MA, Deshaies EM. An update on the management of pseudotumor cerebri. *Clin Neurol Neurosurg*. 2013;115:252--259.
- Lee AG, Wall M. Idiopathic Intracranial hypertension. In: UpToDate, Basow DS, eds. *UpToDate*: Waltham, MA, 2013.
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See Also (Topic, Algorithm, Electronic Media Element)

- Giant Cell Arteritis
- Headache
- Headache, Migraine
- Labyrinthitis
- Trigeminal Neuralgia



CODES

ICD9

348.2 Benign intracranial hypertension

ICD10

G93.2 Benign intracranial hypertension

PSORIASIS

Allison Tadros • Erica B. Shaver

BASICS

DESCRIPTION

- Chronic, noncontagious, inflammatory skin condition
- Disease of hyperproliferation
- Recently classified as an autoimmune disease
- Presents with erythematous plaques with silver scaling
- Most commonly affects the elbows, knees, lumbar area, gluteal cleft, and glans penis
- Up to 1/3 of patients develop associated arthritis and 10% have ocular manifestations
- Course is unpredictable; marked variability in severity over time and remissions may be seen
- Exact cause unclear: triggers may be infectious, stressors, medications, or trauma
- Tends to show improvement in summer months, possibly related to UV exposure
- Associated with metabolic syndrome
- Caucasians and atopics most affected
- 2 peaks of onset: between ages 20–30 and 50–60
- Affects 2.2% of US population, slightly higher incidence in females
- Rare cause of mortality, but at least 100 deaths annually in US related to severe disease and/or treatment adverse effects
- Several clinical presentations:
 - Plaque-type psoriasis (psoriasis vulgaris):
 - Most common form (75–80%) with erythematous, raised plaques with well-demarcated borders distributed over the scalp, back, and extensor side of the knees and elbows
 - Guttate psoriasis:
 - Abrupt appearance of multiple, discrete, salmon-colored, “drop-like” papules with a fine scale in a patient with no prior history of psoriasis
 - Most commonly seen on the trunk and proximal extremities
 - Often preceded by a streptococcal infection and resolves spontaneously
 - Pustular psoriasis:
 - Occasionally isolated to the palms and soles, but can present as widespread erythema, scaling, and sheets of superficial pustules with erosions
 - Patient may appear toxic and have other systemic symptoms, like

- malaise, fever, and diarrhea
 - Potentially severe and life threatening and treated as an inpatient if generalized
- Erythrodermic psoriasis:
 - Generalized erythema and pruritis with a fine scale
 - Increased risk for infection, dehydration
 - Often treated as inpatients
- Nail psoriasis
 - Pitting over the nail plate or change in nail bed
 - Nail changes in up to 50% of patients
- Inverse flexural psoriasis:
 - A variant that causes lesions in flexural areas and in skin folds that do not exhibit scaling due to moisture in these areas
- HIV-induced psoriasis:
 - May be the first manifestation of AIDS, more frequent and severe in HIV population
- Genetics:
 - There is a genetic predisposition and gene loci have been identified
 - 40% of patients with psoriasis have a family history in a 1st degree relative

ETIOLOGY

- Typical findings of erythema and scaling are the result of increased number of epidermal stem cells and keratinocyte hyperproliferation, shortened cell cycles, inflammatory infiltrates, and vascular changes
- Triggers include:
 - Drugs:
 - Lithium
 - β -blockers
 - Antimalarials
 - Steroid withdrawal
 - NSAIDs
 - Alcohol
 - Potassium iodide
 - Infections:
 - Streptococcal pharyngitis
 - HIV
 - Staph
 - Local trauma:
 - Frostbite
 - Sunburn
 - Recent skin trauma (Koebner phenomenon)
 - Stress: Emotional and physical
 - Winter:

- Low light exposure
- Dry weather
- Cigarette smoking
- Elevated BMI

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Long standing area of scaling erythema
- May give a history of previous diagnosis of psoriasis
- May be mildly pruritic
- May relate to one of the above triggers
- Specific location of lesions
- Family history of the disease
- History of improvement with sun exposure, or if recurrent, success of prior regimens
- Systemic symptoms like fevers or joint pains

Physical-Exam

- The classic skin lesion is a round, red patch with a central plaque of silvery, white scale that appears on extensor surfaces
- Redness and scaling around the umbilicus is highly suggestive of psoriasis
- Positive Auspitz sign:
 - Erythema and punctate bleeding when scales are removed
- In dark-skinned patients lesions may be grey
- Scalp lesions may be confused with seborrhea:
 - Lesions that extend beyond the hair borders indicate psoriasis
- Stippling and pitting of nail and onycholysis:
 - Yellow or brown band across the nail may help differentiate psoriasis (+ band) from onychomycosis (– band)
- Patients with plaque psoriasis may have concomitant psoriatic arthritis:
 - Often affects the DIP joints of the hands and feet
- Asymmetric oligoarticular arthritis:
 - Present in 70% of these patients
 - Swelling of the juxta-articular tissue
 - “Sausage-shape” to the affected digits

ESSENTIAL WORKUP

- The diagnosis is clinical
- Rarely, a biopsy is necessary to confirm the diagnosis or rule out other conditions

DIAGNOSIS TESTS & INTERPRETATION

Lab

- No lab test confirms the diagnosis
- Elevated sedimentation rate in erythrodermic and pustular forms
- Positive streptococcal cultures and titers with guttate psoriasis
- Hypocalcemia and leukocytosis in pustular disease
- Negative rheumatoid factor
- Uric acid may be elevated
- If starting medications, consider checking baseline CBC, LFTs, and renal function, as well as TB screening

Imaging

- Plain radiographs of the hands or feet may be abnormal with psoriatic arthritis
- Sacroiliitis and ankylosing spondylitis may also be seen on radiographs

DIFFERENTIAL DIAGNOSIS

Best thought of by region

- Scalp: Seborrhea
- Flexure creases:
 - Candidiasis
 - Intertrigo
 - Eczema
- Nails: Onychomycosis
- Trunk and extremities:
 - Nummular eczema
 - Pityriasis rosea or rubra pilaris
 - Tinea
 - Systemic lupus erythematosus
 - Syphilis
 - Lichen simplex chronicus
 - Atopy, drug eruption
 - Mycosis fungoides
 - Squamous cell carcinoma



TREATMENT

PRE HOSPITAL

- Maintain universal precautions
- IV access and pain control as necessary

INITIAL STABILIZATION/THERAPY

- General resuscitation efforts aimed at correcting fluid and electrolyte abnormalities
- Treating sepsis if present:
 - Cultures of lesions, blood, and urine
- Systemic steroids should not be used as they may predispose to severe complications

ED TREATMENT/PROCEDURES

- Patients should be educated on the chronic nature of psoriasis and that there is no cure even with treatment
- Treatments can be expensive and compliance is often poor
- Some patients may decline treatment in milder cases
- 3 basic types of treatment for psoriasis:
 - Topical therapy
 - Systemic therapy
 - Phototherapy
- Topical therapy is the most commonly prescribed treatment modality from the ED
- Systemic therapy is usually employed only after failure of topical and phototherapy and in conjunction with a dermatologist
- Exceptions where systemic therapy may be used:
 - Generalized pustular psoriasis
 - Very active psoriatic arthritis
 - Psoriasis that is considered severely disabling
- Phototherapy is not an ED treatment modality
- Dermatology consult should be obtained in severe cases

MEDICATION

- Mild to moderate disease:
 - Usually topical treatment only
 - No single topical agent works best for all patients.
 - Emollients:
 - Works well for limited plaque psoriasis
 - Greasier choices work best, but may be poorly tolerated by patients.
 - Topical steroids
 - Major form of therapy for those with limited disease
 - Can be used as monotherapy, 1–2 times a day, or in combination with emollients
 - Once improvement is achieved, consider tapering use
 - May need to rotate drugs
 - Occlusive dressing improves efficacy
 - Salicylic acid
 - Topical keratolytic agent
 - Precaution if already on systemic aspirin

- Coal-tar preparations:
 - Usually used with topical steroids
 - Newer forms are less messy
- Vitamin D analogs:
 - Calcipotriene and calcitriol
- Tazarotene
 - Topical retinoid, 0.1% cream
 - Pregnancy class X
- Tacrolimus
 - Topical treatment for inverse psoriasis or facial lesions
- Is steroid sparing and reduces risk of atrophy from steroids
- Moderate to severe disease:
 - The above-named agents may be employed along with phototherapy and systemic medications
 - Phototherapy:
 - UV radiation is thought to have antiproliferative and anti-inflammatory effects
 - Ultraviolet B light is usually combined with ≥ 1 topical agents and has reports of 80% remission
 - Ultraviolet B may be used alone in guttate psoriasis
 - Psoralen ultraviolet A (PUVA) light therapy combines a systemic agent (psoralen) that sensitizes the skin to UVA light
 - Therapy is usually given 2–3 times per week
 - Systemic agents: May be used in various combinations with the above modalities:
 - Should not be initiated without dermatology consultation
 - Methotrexate (immunosuppressant): Assess renal, liver, and hematologic function prior to therapy; not to be used during pregnancy
 - Retinoids: May cause dryness, scaling, redness, and tenderness of the skin
 - Systemic corticosteroids: Not favorable due to iatrogenic Cushing syndrome; it may have a role in acute erythrodermic psoriasis if patient is extremely ill
 - Cyclosporine: Use in conjunction with dermatology consult
 - Injectable immunosuppressants: Etanercept and Alefacept



FOLLOW-UP

DISPOSITION

Admission Criteria

ALERT

Acute erythroderma and acute pustular psoriasis warrant admission for supportive therapy and systemic treatment, as noted above.

Discharge Criteria

- Advise patients that the disease is not contagious
- Warn patients to avoid skin trauma and sunburns
- Educate the patient on avoiding medications that trigger relapses
- Refer patients to the National Psoriasis Foundation, www.psoriasis.org

Pediatric Considerations

- About 10–15% of cases occur \leq age 10
- Involvement of face and flexural areas more common; pustular and erythrodermic less common
- May have significant psychosocial impact on this population

Pregnancy Considerations

Many of the drugs used to treat psoriasis are contraindicated in pregnancy

Issues for Referral

- Referral to dermatology is indicated for most patients with psoriasis
- Patients with psoriasis may also need referral to primary care doctor and/or psychiatry to cope with impaired quality of life

FOLLOW-UP RECOMMENDATIONS

Follow-up with dermatology and/or primary care doctor to evaluate efficacy of treatment

PEARLS AND PITFALLS

- Patients with pustular psoriasis are at risk for severe systemic infections
- Patients with erythrodermic psoriasis are at risk for dehydration and may need to be treated similarly to a major burn patient
- Improvement occurs in weeks, not days

ADDITIONAL READING

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CODES

ICD9

- 696.0 Psoriatic arthropathy
- 696.1 Other psoriasis

ICD10

- L40.0 Psoriasis vulgaris
- L40.4 Guttate psoriasis
- L40.9 Psoriasis, unspecified

PSYCHIATRIC COMMITMENT

Danielle B. Kushner • Rohn S. Friedman

BASICS

DESCRIPTION

- Psychiatric “civil commitment”: The state-sanctioned *involuntary* hospitalization of mentally disordered individual
- Voluntary psychiatry hospitalization: Voluntary psychiatric admission of competent adult who agrees to hospitalization
- Commitment criteria (review specific laws in your state):
 - Individual is mentally ill (many states exclude mental retardation, antisocial behavior, medical illness, and substance abuse)
 - Likelihood of serious harm defined as:
 - Substantial risk of physical harm to self
 - Substantial risk of physical harm to other persons
 - “Gravely disabled”: Inability to care for basic needs, including food, clothing, shelter, medical care, and safety
 - No less-restrictive alternative to hospitalization would attenuate risk
- 2 stages of commitment:
 - Emergency detention and admission (sometimes called emergency hold)—an emergency admission with minimum of legal process. Usually 72 hr
 - Longer-term commitment—requires judicial approval of continued confinement in an adversarial proceeding where patient may have legal representation

ETIOLOGY

- Underlying genetic/biologic predisposition to psychiatric illness
- Precipitating psychosocial events can trigger onset or worsening of symptoms
- Substance abuse can worsen symptoms or lead to disinhibition causing worsening safety concerns

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Recent change in behavior or thinking
- Psychotic symptoms
 - Hallucinations
 - Delusions

- Disorganized thought
- Panic, anxiety, or agitation
- Depressed mood
- Neurovegetative symptoms (disturbance of sleep, appetite, libido, energy, concentration, energy, psychomotor state)
- Manic symptoms (elevated or irritable mood, grandiosity, racing thoughts, excessive involvement in pleasurable activities at high potential risk)
- Specific evidence of commitment criteria:
 - Actual or threatened violence toward self
 - Actual or threatened violence toward others
 - Inability to care for self or protect self in the community
- Past psychiatric diagnoses, treatment, hospitalizations, history of harm to self or others
- Medication compliance
- Drugs of abuse: Amount and time of last use
- Access to weapons

Physical-Exam

- Signs of toxidromes/overdose
 - Slurred speech, ataxia (alcohol intoxication)
 - Tremor, nystagmus, diaphoresis, tachycardia (alcohol withdrawal)
 - Dilated pupils, piloerection, rhinorrhea (opiate withdrawal)
 - Hyperreflexia, myoclonus, diaphoresis, dilated pupils (serotonin syndrome)
- Signs of self-injury (scars, fresh wounds)
- Hypoactivity, rigidity, catalepsy, posturing (catatonia, NMS)
- Mental status exam
 - Expansive, irritable, or depressed mood
 - Disordered thought process, loose associations
 - Hallucinations
 - Delusions
 - Suicidal ideation (active vs. passive), intent, or plan
 - Homicidal ideation (identified target, intent, plan)
 - Impaired cognition (delirium, dementia)

ESSENTIAL WORKUP

- Thorough medical evaluation to rule out medical causes of change in thinking or behavior
- Psychiatric evaluation to clarify diagnosis and need for hospital level of care

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Basic screening labs for medical clearance as indicated by history and physical:

- Electrolytes, BUN, creatinine, glucose
- Liver function tests
- CBC and differential
- Toxicology screens and medication levels
- Urinalysis if infection suspected
- TFTs

Imaging

CT or MRI of head if injury or structural CNS pathology suspected

Diagnostic Procedures/Surgery

- EEG if seizure/postictal state suspected
 - Repetitive pattern
 - Aura usually somatosensory psychiatric autonomic symptoms
 - Postictal period of either confusion or dysphasia
- LP
 - Fever or nuchal rigidity
 - Seizures

DIFFERENTIAL DIAGNOSIS

- Substance intoxication or withdrawal
- Delirium
- Dementia
- Traumatic brain injury/subdural
- Temporal lobe seizures
- Encephalitis
- Meningitis
- Antisocial behavior (patient feigning psychiatric symptoms, homicidal or suicidal ideation to obtain medications or shelter or avoid arrest)



TREATMENT

PRE HOSPITAL

- Patient or concerned family may call the ED and describe an emergency circumstance
- Depending on state, police, social worker, psychologist, or psychiatrist can send patient to ED for emergent assessment and possible involuntary hospitalization
- EMTs can restrain and bring patient to hospital involuntarily with appropriate legal paperwork

INITIAL STABILIZATION/THERAPY

- Ensure patient and staff safety

- Make sure patient does not have weapon or medications
- Room without hazards (sharps, medications, etc)
- Constant observation to prevent elopement
- Assess for overdose or self-injury requiring immediate treatment
- Ensure that proper legal paperwork is in place to hold patient in the ED

ED TREATMENT/PROCEDURES

- Medical workup and clearance
- Psychiatric consultation, if available
- Determine if patient requires psychiatric hospitalization.
- Restrain patients at risk of harming themselves or others, using least restrictive means required to maintain safety:
 - Nurse or security guard standing outside room
 - Pharmacologic restraint given PO, IM, or IV
 - Physical restraints
- Continue patient's confirmed home medications if appropriate
- Offer symptomatic medications such as antianxiety, antipsychotic, or sleep aid as needed
- Treat alcohol or drug withdrawal as needed

MEDICATION

- First line is Olanzapine 5--10 mg PO/IM OR haloperidol 5 mg IM with lorazepam 2 mg IM and benztropine 1 mg IM.
- First Line for EtOH/BZP withdrawal:
 - Diazepam 5–10 mg PO q1h prn (monitored with standardized symptoms assessment such as CIWA scale)
- 1st line for delirium not associated with alcohol withdrawal or anticholinergic excess:
 - Haloperidol 1–2 mg PO/IM/IV
- 1st line for agitation not associated with psychosis, delirium, or alcohol withdrawal:
 - Lorazepam 1 mg PO/IM/IV



FOLLOW-UP

DISPOSITION

Admission Criteria

- Danger to self
- Danger to others
- Severely disabled and unable to care for self/protect self in community
- Follow commitment process for your state

Discharge Criteria

- Patients may be discharged after medical and psychiatric evaluation if they:
 - _ Can care for themselves adequately *and*
 - _ The risk of harm to self or others is assessed to be safely manageable in a less-restrictive alternative, such as a partial hospital, a crisis stabilization/observation unit, or outpatient treatment

Issues for Referral

- Patient with acute psychiatric illness who does not meet the criteria for hospitalization usually requires 1 or more of the following:
 - _ Crisis stabilization or observation unit
 - _ Partial hospitalization, day program, or intensive outpatient program referral
 - _ Psychiatrist and/or therapist follow-up appointment within 3–5 days
 - _ Crisis Line phone number
- Patient may need to call insurer for list of or referral to outpatient providers covered by his or her insurance and may need a prior authorization



FOLLOW-UP

FOLLOW-UP RECOMMENDATIONS

Patient instructed to return to ED if feels unsafe, has increasing suicidal/homicidal thoughts, or other symptoms worsen

PEARLS AND PITFALLS

- Psychiatric civil commitment involves involuntary hospitalization due to mental illness and 1 of the following:
 - _ Substantial risk of harm to self
 - _ Substantial risk of harm to others
 - _ Inability to care for/protect self
- The details vary by state, so you need to know the specifics of your jurisdiction:
 - _ Mental retardation, antisocial behavior, organic causes such as dementia or delirium, and substance abuse may not qualify as a mental illness for which a person can be committed
 - _ The definition of the 3rd criterion for commitment (“gravely disabled,” unable to care for or protect self, or in need of treatment) varies
 - _ Time frames and procedures differ
- Need to complete thorough psychiatric and medical evaluation to evaluate causes of change in patient’s behavior
- Physician must weigh the ethical considerations inherent in involuntary hospitalization, balancing patient rights against the safety of patient or others.

ADDITIONAL READING

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- Wilson MP, Pepper D, Currier GW, et al. The psychopharmacology of agitation: Consensus statement of the American Association for Emergency Psychiatry Project Beta psychopharmacology workgroup. *West J Emerg Med*. 2012;13(1):26–34.



See Also (Topic, Algorithm, Electronic Media Element)

- Agitation
- Altered Mental Status
- Depression
- Psychosis, Acute
- Psychosis, Medical vs. Psychiatric
- Violence, Management of



CODES

ICD9

- 298.9 Unspecified psychosis
- 300.9 Unspecified nonpsychotic mental disorder
- V62.84 Suicidal ideation

ICD10

- F29 Unsp psychosis not due to a substance or known physiol cond
- F99 Mental disorder, not otherwise specified
- R45.851 Suicidal ideations

PSYCHOSIS, ACUTE

Celeste N. Nadal • Serena A. Fernandes



BASICS

DESCRIPTION

- Disorder of brain function characterized by loss of contact with reality, abnormal perceptions, disorganization of emotions, thought, and behavior
- Dopamine pathways are strongly implicated
- May be secondary to psychiatric or medical, nonpsychiatric causes
- Medical causes of psychosis can be secondary to focal or systemic medical insults, neurologic impairment, or pharmacologic agents

ETIOLOGY

Medical, Nonpsychiatric

- Neurologic disease:
 - Head injury (history of)
 - Dementias (Alzheimer, Lewy body)
 - Cerebrovascular accident
 - Seizures
 - Space occupying lesions (neoplasm, malignancy, abscesses, cysts)
 - Hydrocephalus
 - Migraines
 - Demyelinating diseases (multiple sclerosis)
 - Neuropsychiatric disorders (Parkinson, Huntington, Wilson disease)
- Infectious disease:
 - Any focal infection (UTI, PNA, cellulitis)
 - HIV infection
 - Neurosyphilis
 - Lyme disease
 - Encephalitis, meningitis or cerebritis:
 - Bacterial (TB, Lyme)
 - Viral (HSV, CMV, EBV)
 - Fungal (Cryptococcus)
 - Prion diseases
- Metabolic:
 - Electrolyte imbalance
 - Hypoxia
 - Hypoglycemia
 - Hypercarbia

- Porphyria
- Intoxication or withdrawal syndrome
- Organ failure:
 - Liver (hepatic encephalopathy)
 - Renal
 - Cardiac (CHF, arrhythmias)
- Endocrine:
 - Thyroid disease
 - Parathyroid disease
 - Cushing syndrome
 - Addison disease
- Nutritional deficiencies:
 - Niacin
 - Thiamine
 - Vitamin B₁₂ and folate
- Autoimmune disease:
 - SLE
 - Paraneoplastic syndrome
 - Myasthenia gravis

Pharmacologic

- Medications:
 - All medications can cause psychosis
 - Sedative–hypnotics: Benzodiazepines (lorazepam, diazepam, alprazolam), barbiturates (butalbital), other (zolpidem)
 - Anticholinergic and antihistaminergic agents (diphenhydramine, cimetidine)
 - Steroids (prednisone)
 - Antiepileptic agents
 - Antiparkinsonian agents (amantadine, levodopa)
 - Cardiovascular agents (digoxin, reserpine)
 - Anti-infectious medications: Antibiotics (isoniazid, rifampin, fluoroquinolones, TMP/SMX), antivirals (oseltamivir, interferon), antiparasitics (metronidazole)
 - Chemotherapeutic agents (vincristine)
 - Muscle relaxants (dicyclomine, carisoprodol)
- Substances associated with intoxication:
 - Alcohol
 - Amphetamines
 - Cocaine
 - Opioids
 - Hallucinogens
 - Cannabis

- Sedative–hypnotics
- Other: LSD, MDMA, PCP, ketamine
- Substances associated with withdrawal:
 - Alcohol and sedative–hypnotics
- Toxins (heavy metals, organophosphates, carbon monoxide)

Psychiatric

- Brief psychotic disorder:
 - Abrupt onset, usually due to psychosocial stressors, lasting < 1 mo
- Delusional disorder:
 - Circumscribed delusions
- Schizophreniform disorder:
 - Symptoms present 1–6 mo
- Schizophrenia
- Schizoaffective disorder
- Mood disorder with psychotic features
- Postpartum psychosis



DIAGNOSIS

SIGNS AND SYMPTOMS

- Delusions are fixed, false beliefs that are:
 - Impervious to outside logic
 - Often persecutory, religious, or somatic content
- Hallucinations:
 - Sensory experiences in the absence of external stimuli
 - Can involve any sensory modality; auditory and visual are most common.
- Thought disorder:
 - Disorganized speech ranging from odd, idiosyncratic logic (loose associations) to incoherence (neologisms, word salad) or poverty of content
- Disorganized or catatonic behavior:
 - Odd, stereotyped behavior (waxy flexibility, echopraxia)
- Negative symptoms:
 - Flattened affect
 - Apathy
 - Anhedonia
 - Social isolation
- Features suggesting a nonpsychiatric etiology:
 - Sudden onset
 - > 30 yr old
 - Fluctuating course
 - Focal neurologic symptoms

- Abnormal vital signs
- Visual, olfactory, gustatory or tactile hallucinations
- Impairment of orientation, attention, or cognitive function

History

- Screen for psychosis, including onset, duration, triggers, and content:
 - Delusions:
 - “Do you feel anyone is trying to harm you or that you are being followed?”
 - “Is anyone trying to send you messages, steal, control, or block your thinking?”
 - Hallucinations:
 - “Do you ever see or hear things that other people cannot see or hear?”
 - “Do you ever hear voices telling you to do things such as to harm yourself or to harm others?”
- Suicidal or homicidal behavior or threats
- Past medical and psychiatric history
- Social situation and ability to care for self
- Recent use, increase or cessation of medications, drugs, or alcohol
- Obtain history from friends, family, and treaters

Physical-Exam

Look for signs of a medical etiology:

- Vital signs
- Eye exam (pupils, EOM, fundi)
- General exam with particular attention to the signs and symptoms of endocrine, liver, and renal disease
- Neurologic exam, including cognitive exam
- Careful assessment for signs of delirium

ESSENTIAL WORKUP

The workup is case specific and primarily based on the suspected etiology

DIAGNOSIS TESTS & NTERPRETATION

Lab

- Electrolytes, BUN, creatinine, glucose, calcium
- Toxicology screen
- CBC with differential
- TSH
- Urinalysis
- Further specific studies should be guided by the suspected underlying etiologies

Imaging

Consider head imaging for new-onset psychotic symptoms of unclear etiology, especially in setting of focal neurologic symptoms.

Diagnostic Procedures/Surgery

When clinically warranted consider:

- Lumbar puncture
- EEG
- EKG (monitor QT)

DIFFERENTIAL DIAGNOSIS

See Etiology.



TREATMENT

PRE HOSPITAL

- Patients can display unpredictable and violent behavior toward themselves and others
- Patients may require police presence or restraints to maintain safety
- Local laws vary regarding involuntary restraint

INITIAL STABILIZATION/THERAPY

- Safety of patient and staff is paramount and may require presence of security
- Behavioral interventions should be used first
 - Provide a calm, containing environment
 - Remove all potentially dangerous items
 - Use a reassuring voice and calm demeanor to set boundaries and verbally redirect
- If safety is a concern, patient needs to be under constant observation
- Physical or chemical restraints as necessary

ED TREATMENT/PROCEDURES

- If a nonpsychiatric etiology is suspected, identify and treat underlying medical condition
- If a psychiatric etiology is suspected, consider psychiatric consultation or referral
- Acute agitation is reduced with antipsychotics:
 - Encourage voluntary PO medications prior to IM administration
 - Avoid polypharmacy
- Rapid tranquilization may be achieved with the addition of a benzodiazepine
- Monitor for and treat adverse effects from antipsychotic medications:
 - Extrapyramidal symptoms (dystonia, akathisia, pseudoparkinsonism, and tardive dyskinesia)

- Neuroleptic malignant syndrome is a life-threatening complication:
 - o Characterized by hyperthermia, muscle rigidity, autonomic instability, and altered consciousness

MEDICATION

- 1st line antipsychotics:
 - Haloperidol: 2–10 mg PO/IV/IM, repeat q20–60min prn to max. 100 mg/d; elderly 0.5–2 mg/dose
 - o Commonly augmented with lorazepam
- 2nd line antipsychotics:
 - Aripiprazole: 2–15 mg PO/IM, may repeat q2h prn to max. 30 mg/d
 - Chlorpromazine: 25 mg PO/IM, repeat 25–50 mg q60min prn to max. 1,000 mg/d. Caution: Sedating, postural hypotension, do not use in elderly
 - Olanzapine: 2.5–20 mg PO/IM, may repeat dose q2–4h prn to max. 20 mg/d; elderly 2.5–5 mg/dose. Caution: Concurrent use of IM olanzapine and IV benzodiazepines may increase risk of cardiopulmonary collapse
 - Risperidone: 1–2 mg PO, may repeat 2 times; elderly 0.25–0.5 mg/dose. Caution: Orthostatic hypotension
 - Quetiapine: 25–50 mg PO BID, increase by 100 mg/d to max. 800 mg/d; elderly 12.5–25 mg/dose, increase by 25–50 mg/d
 - Ziprasidone: 20–40 mg PO BID, max. 80 mg PO BID; 10 mg IM q2h or 20 mg IM q4h prn to max. 40 mg/d IM, no more than 3 days. Caution: Monitor QT
- Benzodiazepines:
 - Lorazepam to augment tranquilization: 1–2 mg PO/IM/IV; elderly 0.25–0.5 mg PO/IM/IV

Geriatric Considerations

Black box warning: Elderly patients with dementia-related psychoses treated with antipsychotic drugs are at increased risk of death.



FOLLOW-UP

DISPOSITION

Admission Criteria

- If nonpsychiatric etiology, admit to appropriate medical service
- If psychiatric etiology and patient is medically stable, patient may require admission to a psychiatric hospital if patient:
 - Is a danger to self or others
 - Is gravely disabled and unable to care for self due to psychosis
 - Has new-onset psychosis and medical etiology has been ruled out

- Criteria for involuntary hospitalization vary

Discharge Criteria

- Patient is not a danger to self or others and is able to perform activities of daily living
- Psychotic symptoms resolved after causative medical issue addressed and patient is medically stable for discharge

Issues for Referral

Consider psychiatric consultation for complicated cases or for psychiatric admission.

FOLLOW-UP RECOMMENDATIONS

- Plan appropriate outpatient medical follow-up
- In patients with psychiatric disorders not requiring admission, plan outpatient psychiatric follow-up within 1 wk
- Consider referral for detoxification in patients with problems related to substance use

PEARLS AND PITFALLS

- Psychotic symptoms should be evaluated for treatable medical causes and not assumed to be solely psychiatric in nature even in patients with known mental illness
- Visual, olfactory, gustatory, or tactile hallucinations should prompt medical workup
- Avoid using IM olanzapine with IV benzodiazepines as this increases risk for cardiopulmonary collapse
- Patients who have recently started or increased their antipsychotics who present with fever, rigidity, autonomic instability, and mental status changes should be assessed for neuroleptic malignant syndrome

ADDITIONAL READING

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- Wilson MP, Pepper D, Currier GW, et al. The psychopharmacology of agitation: Consensus statement of the American Association for Emergency Psychiatry Project Beta medical evaluation workgroup. *West J Emerg Med*. 2012;13(1):26–34.



See Also (Topic, Algorithm, Electronic Media Element)

- Delirium
- Dystonic Reaction
- Neuroleptic Malignant Syndrome
- Psychosis, Medical vs. Psychiatric
- Schizophrenia
- Violence, Management of



CODES

ICD9

- 292.9 Unspecified drug-induced mental disorder
- 298.8 Other and unspecified reactive psychosis
- 298.9 Unspecified psychosis

ICD10

- F19.959 Oth psychoactv substance use, unsp w psych disorder, unsp
- F23 Brief psychotic disorder
- F29 Unsp psychosis not due to a substance or known physiol cond

PSYCHOSIS, MEDICAL VS. PSYCHIATRIC

Richard E. Wolfe



BASICS

DESCRIPTION

Mental derangement involving hallucinations, delusions, or grossly disorganized behavior resulting in loss of contact with reality

- Complex and poorly understood pathophysiology
- An excess in dopaminergic signaling may be a contributing factor
- Psychosis ranges from a relatively mild derangement to catatonia
- CNS impairment leading to a psychotic presentation may be due to:
 - _ Neurologic disorders
 - _ Metabolic conditions
 - _ Toxins or drug effects
 - _ Infections
- Higher risk for underlying psychiatric disorder:
 - _ Hallucinations and illusions incorporated into delusional system
 - _ Late adolescence/early adulthood
 - _ Normal orientation
- Higher risk for underlying medical disorder:
 - _ Middle- to late-life presentation
 - _ Acute onset
 - _ History of substance abuse
 - _ No pre-existing psychiatric history
 - _ Absence of a family history of major mental illness
 - _ Presence of pre-existing medical disorders
 - _ Lower socioeconomic level
 - _ Recent memory loss
 - _ Disorientation or distractibility
 - _ Abnormal vital signs
 - _ Visual hallucinations:
 - Delirium
 - Dementia
 - Migraines
 - Dopamine agonist therapy (i.e., carbidopa)
 - Posterior cerebral infarcts
 - Narcolepsy

ETIOLOGY

- Neurologic:

- Head trauma
 - Space-occupying lesions
 - Cerebrovascular accident
 - Seizure disorders
 - Hydrocephalus
- Neuropsychiatric disorders: (Parkinson, Huntington, Alzheimer, Pick, Wilson disease)
- Infectious:
 - Focal infections in the elderly (UTI, pneumonia)
 - HIV
 - Neurosyphilis
 - Encephalitis
 - Lyme disease: Neuroborreliosis
 - Parasites:
 - Cerebral malaria
 - Neurocysticercosis
 - Schistosomiasis
 - Toxoplasmosis
 - Trypanosomiasis
- Metabolic:
 - Electrolyte imbalance
 - Hypoglycemia
 - Hypoxia
 - Porphyria
 - Withdrawal syndromes
- Endocrine:
 - Thyroid disorders
 - Parathyroid disorders
 - Diabetes mellitus
 - Pituitary abnormalities
 - Adrenal abnormalities
- End-organ failure:
 - Cardiac/respiratory
 - Renal
 - Hepatic
- Nutritional deficiencies:
 - Pernicious anemia
 - Wernicke–Korsakoff syndrome
 - Pellagra
 - Pyridoxine deficiency
- Autoimmune disorders:
 - Systemic lupus erythematosus
 - Sarcoidosis

- Myasthenia gravis
 - Paraneoplastic syndromes
- Demyelinating disease:
 - Multiple sclerosis
 - Leukodystrophies
- Postoperative states:
 - Delirium
- Intoxicants:
 - Alcohol
 - Benzodiazepines
 - Barbiturates
 - Stimulants (cocaine, amphetamines)
 - Hallucinogens
 - Opiates
 - Anticholinergic compounds
 - Inhalants
 - Cannabis
- Toxins:
 - Bromide
 - Carbon monoxide
 - Heavy metals
 - Organic phosphates
- Medication side effects:
 - Corticosteroids
 - Anticholinergics
 - Sedative–hypnotics
- Psychiatric:
 - Antidepressants
 - Antipsychotics
 - Lithium carbonate
- Antiparkinsonian drugs
- Anticonvulsants
- Antibiotics (quinolones, isoniazid)
- Antihypertensive agents
- Cardiac (digitalis, lidocaine, propranolol, procainamide)
- Interferon
- Muscle relaxants
- Over-the-counter medications:
 - Pseudoephedrine
 - Antihistamines
- Psychiatric:
 - Schizophrenia
 - Schizoaffective disorder

- Delusional disorder
- Bipolar disorder with psychotic features
- Major depression with psychotic features
- Stress reactions including post-traumatic stress disorder
- Narcolepsy (hallucinations at edge of sleep/wake cycle)
- Postpartum psychosis

DIAGNOSIS

SIGNS AND SYMPTOMS

- Psychosis characterized by:
 - Impaired reality testing
 - Inappropriate affect
 - Poor impulse control
- Focal and diffuse CNS impairment may result in derangements of:
 - Perception
 - Thought content
 - Thought process
- Hallucinations:
 - Sensory perception that has the compelling sense of reality of a true perception without external stimulation of the relevant sensory organ
- Delusions
 - Beliefs held with certainty, incorrigibility, and impossibility
 - Categorized by type and theme:
 - Bizarre or nonbizarre
 - Mood congruent or neutral
 - Persecutory or grandiose
 - Primary or secondary
- Thought disorder
- Affective symptoms may include mania, depression, or catatonia.

History

- Time course: Acute, episodic, chronic
- Collateral from family or outpatient providers
- Substance use
- Medications and medication adherence
- Family history
- Associated symptoms: Fever, weight loss, appetite, recent surgery and trauma

Physical-Exam

- Vital signs
- Neurologic exam:

- Cognitive exam: Attention and orientation
- Motor exam: Tone, abnormal movements

ESSENTIAL WORKUP

Detailed history and physical exam, including neurologic exam

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Low likelihood of clinically significant findings if there is a past history of psychosis, a benign history, and normal physical exam
- 1st line:
 - CBC
 - Electrolytes including calcium, BUN/creatinine, glucose
 - Urine and serum toxicology screen
 - Urinalysis
 - Liver function tests
 - Thyroid function tests
 - Vitamin B₁₂ and folate
- 2nd line guided by history and physical findings:
 - Ammonia level
 - HIV testing
 - Fluorescent treponemal antibody absorption (to rule out neurosyphilis; rapid plasmin reagin not sufficient as screen)
 - Ceruloplasmin
 - Urine heavy metals
 - ESR, C-reactive protein, antinuclear antibody

Imaging

- Routine CT or MRI scans are of little benefit
- Indications:
 - History or exam suggests a neurologic disorder
 - 1st-episode psychosis, 50 yr and older
- No clear clinically relevant benefit for MRI over CT

Diagnostic Procedures/Surgery

- EKG with attention to corrected QT interval
- Not recommended for routine screening:
 - Lumbar puncture
 - EEG

DIFFERENTIAL DIAGNOSIS

- Martha Mitchell effect:

- Process by which a clinician mistakes the patient's perception of real events as delusional
- Locked-in syndrome
- Periodic paralysis
- Conversion disorder



TREATMENT

PRE HOSPITAL

- Ensure safety of patient, bystanders, and medical personnel.
- Monitor vital signs, check finger stick.

INITIAL STABILIZATION/THERAPY

- Safety
- Evaluation
- Check O₂ saturation and serum glucose
- If uncooperative and dangerous, control behavior

ED TREATMENT/PROCEDURES

- Treat underlying medical illness or substance abuse disorder.
- Control psychotic behavior with psychotropic medications
- Check for prolonged QT before administering neuroleptic agents
- Haloperidol in combination with lorazepam:
 - Safe, fast; least disruptive of ongoing medical exam of patient
- Atypical neuroleptics:
 - Few extrapyramidal side effects
 - Olanzapine and ziprasidone can be given IM
 - Olanzapine (Zydis) and Risperdal M-tab are available in dissolving wafer preparations.
 - Avoid IM lorazepam with IM olanzapine due to risk of respiratory depression.

MEDICATION

First Line

- Haloperidol 2–10 mg IM or IV with lorazepam 0.5–2 mg IM or IV

Second Line

- Neuroleptics:
 - Olanzapine: 5–10 mg PO, SL, or IM
 - Risperidone: 1–2 mg PO or SL
 - Quetiapine: 25–100 mg PO

- Benzodiazepines:
 - Diazepam: 5–10 mg IV

Geriatric Considerations

- Increased mortality risk in patients > 65 yr on typical and atypical antipsychotics
- Start with lower doses (Haloperidol 2 mg IV), Olanzapine 2.5–5 mg PO, SL, or IM).
- Use benzodiazepines cautiously, given risk of disinhibition; avoid in delirious patients.

Pregnancy Considerations

Best evidence of safety of antipsychotic use in pregnancy is for 1st-generation (typical) antipsychotics such as haloperidol.



FOLLOW-UP

DISPOSITION

Admission Criteria

- If primarily medical etiology, admission to medical service, criteria dictated by specific medical condition
- If primarily psychiatric etiology (e.g., schizophrenia), admit to psychiatric service if:
 - Danger to self or others
 - Inability to care for self
 - Deranged thought pattern that can be threat to self or others
 - 1st episode: Evaluation and stabilization
 - Laws for involuntary hospitalization vary by state.

Discharge Criteria

- Stable medical condition
- Not suicidal/homicidal
- Able to care for self
- Capable of making medical decisions

Issues for Referral

- Insurance coverage determines inpatient and outpatient psychiatric disposition options.
- Case management or social services necessary for psychiatric disposition.

FOLLOW-UP RECOMMENDATIONS

- If psychosis is primarily psychiatric, confirm follow-up appointment with mental health provider within 1–2 wk.

- Reassess risk/benefit of continuing on antipsychotic medication at follow-up.

PEARLS AND PITFALLS

- Patients with psychosis may not be able to explain their symptoms in a typical way. Get collateral and maintain a high degree of suspicion.
- Important to rule out organic causes prior to ascribing psychosis to a psychiatric disorder.

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See Also (Topic, Algorithm, Electronic Media Element)

- Agitation, Management of
- Psychosis, Acute
- Schizophrenia



CODES

ICD9

- 292.9 Unspecified drug-induced mental disorder
- 298.9 Unspecified psychosis
- 780.1 Hallucinations

ICD10

- F19.959 Oth psychoactv substance use, unsp w psych disorder, unsp
- F23 Brief psychotic disorder
- R44.3 Hallucinations, unspecified

PULMONARY CONTUSION

Douglas W. Lowery-North

BASICS

DESCRIPTION

- Transfer of kinetic energy to the lung, causing direct damage to the lung parenchyma, resulting in both hemorrhage and edema in the absence of a pulmonary laceration
- Mortality rate is 10–25%.
- Independent risk factor for:
 - Acute respiratory distress syndrome
 - Pneumonia
 - Long-term respiratory dysfunction

PATHOPHYSIOLOGY

- Development of pulmonary contusion:
 - Takes place in 2 stages:
 - 1st stage, which is related to the direct injury, results in disruption of the alveolocapillary membrane, which leads to extravasation of blood into the interstitial and alveolar space.
 - 2nd stage is related to the indirect worsening of the injury as a result of measures that occur during the resuscitation of the patient, in particular, administration of IV fluids.
- Leads to:
 - Increased intrapulmonary shunting
 - Increased resistance to airflow
 - Decreased lung compliance
 - Increased respiratory work
 - Hypoxemia and acidosis
 - Respiratory failure

ETIOLOGY

- Blunt or penetrating thoracic trauma
- Sudden deceleration–compression
- Fall from height
- Motor vehicle accident
- Assault
- Missile

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Blunt or penetrating thoracic trauma by any mechanism
- Mechanism as described by patient, family or emergency medical services personnel:
 - Seat belt use
 - Steering wheel damage
 - Air bag deployment
- Chest pain
- Dyspnea
- Hemoptysis

Physical-Exam

- Auscultation:
 - Initially normal or diminished breath sounds
 - Progresses to crackles, rales, absent breath sounds
- Localized ecchymosis, edema, erythema, and tenderness of chest wall
- Bony deformities, crepitus, point tenderness, and paradoxical movements associated with rib fractures
- Ecchymosis from seat belt, aka “seat belt sign”
- Ecchymosis from steering wheel impact
- Splinting respirations
- Cyanosis, tachycardia, hypotension
- Dyspnea, tachypnea

ALERT

Insidious onset increasing 6–12 hr post injury

ESSENTIAL WORKUP

CXR:

- Radiographic findings may not appear until 6–12 hr post injury.
- Patchy alveolar infiltrates to frank consolidation.
- Associated intrathoracic injury:
 - Rib fractures
 - Pneumothorax, hemothorax
 - Widened mediastinal silhouette

DIAGNOSIS TESTS & NTERPRETATION

Lab

Arterial blood gas may reveal hypoxemia and elevated alveolar–arterial gradient.

Imaging

- Chest radiograph:
 - Percentage of contusion can help predict the need for intubation:
 - <18%: Usually will not need intubation
 - >28%: Usually leads to intubation
- Thoracic CT is useful in detecting pulmonary injury and associated intrathoracic injuries not identified on CXR:
 - Studies that have shown injury size on CT can also assist with prognosis.
 - >20% of the total lung volume is predictive of the need of assisted ventilation.
- US has been studied and could prove to be a fast, sensitive method for diagnosing pulmonary contusion.

DIFFERENTIAL DIAGNOSIS

- Adult respiratory distress syndrome
- CHF
- Hemothorax
- Noncardiogenic causes of pulmonary edema
- Pneumonia, abscess, or other infectious process
- Pneumothorax
- Pulmonary laceration, infarction, or embolism



TREATMENT

PRE HOSPITAL

Thoracic trauma with significant mechanism or pre-existing pulmonary disease should be routed to the nearest available trauma center.

INITIAL STABILIZATION/THERAPY

- Manage airway and resuscitate as indicated.
 - Stabilize associated chest wall injuries (open chest, flail chest)
- IV line, O₂, continuous cardiac monitoring, and pulse oximetry
- Control airway:
 - Endotracheal intubation indications:
 - Severe hypoxemia (PaO₂ <60 mm Hg on room air, <80 mm Hg on O₂)
 - Significant underlying lung disease
 - Impending respiratory failure
 - Early intubation and institution of positive end expiratory pressure:
 - Correct hypoxemia and acidosis.
 - Decrease the work of breathing.

ED TREATMENT/PROCEDURES

- Maintain adequate oxygenation and ventilation.
- Monitor O₂ saturation and respiratory rate.
- In conscious and alert patients, O₂ administration via face mask is 1st-line therapy.
- If patient cannot maintain a PaO₂ > 80 mm Hg on high-flow oxygen:
 - _ Continuous positive airway pressure via mask
 - _ Nasal bilevel positive airway pressure (BiPAP)
 - _ Early endotracheal intubation and mechanical ventilation
- In patients with severe unilateral injuries with significant hemoptysis or air leaks, consider selective bronchial intubation.

ALERT

- Avoid excessive fluid administration:
 - _ IV crystalloid administration needed for resuscitation must be balanced with the risk of increasing interstitial pulmonary edema.
- Frequent re-exam and serial chest radiographs are required to monitor alveolar fluid accumulation.
- Mental status must be appropriate and patient must be alert and cooperative for BiPAP/CPAP:
 - _ Often this is only a temporizing intervention and should not delay intubation in worsening patients.

MEDICATION

- Adequate pain control is key for optimal outcome.
- Steroids have no proven benefit.
- Prophylactic antibiotics are not indicated.

Pediatric Considerations

Increased pliability of the chest wall increases the frequency of pulmonary contusions.

Geriatric Considerations

- Suboptimal cardiopulmonary reserve in combination with large-volume fluid resuscitation increases the likelihood of worsening of pulmonary contusions in the elderly.
- Pulmonary contusion has been identified as a marker for bad outcomes in elderly patients with isolated blunt chest trauma.



FOLLOW-UP

DISPOSITION

Admission Criteria

Patients with pulmonary contusion must be admitted to the hospital for observation in anticipation of delayed-onset respiratory compromise.

Discharge Criteria

- Patients with minimal chest trauma
- No evidence of respiratory distress or hypoxemia:
 - Normal respiratory rate
 - Reassuring pulse oximetry
 - Negative chest radiograph
- Strict return criteria should be discussed with the patient prior to discharge:
 - Shortness of breath
 - Hemoptysis
 - Inadequate pain control or increased pain
 - Cough

PEARLS AND PITFALLS

- Avoid underestimating the severity of pulmonary injury based on initial chest x-ray.
- Failure to recognize this injury in the ED can lead to unexpected deterioration.
- Comorbid conditions such as chronic lung disease and renal failure increase the likelihood of requiring mechanical ventilation.
- Careful monitoring and reassessment is key.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Dyspnea
- Chest Trauma, Blunt

- Flail Chest
- Trauma, Multiple

Acknowledgment

The author gratefully acknowledges Nicholas C. Mosely for his work on the previous edition of this chapter.



CODES

ICD9

- 861.21 Contusion of lung without mention of open wound into thorax
- 861.31 Contusion of lung with open wound into thorax

ICD10

- S27.321A Contusion of lung, unilateral, initial encounter
- S27.322A Contusion of lung, bilateral, initial encounter
- S27.329A Contusion of lung, unspecified, initial encounter

PULMONARY EDEMA

Timothy C. Peck • Shamai A. Grossman

BASICS

DESCRIPTION

Imbalance in Starling forces causes an accumulation of alveolar fluid secondary to leakage from pulmonary capillaries into the interstitium and alveoli of the lungs.

- **Cardiogenic:**
 - Abnormality in cardiac function leading to inadequate tissue perfusion
 - Acute decompensated cardiac failure: Acute fluid overload in the setting of chronic HF
 - Acute vascular failure: Decreased contractility and increased vascular resistance
- **Noncardiogenic:**
 - Increased alveolar–capillary membrane permeability, and accumulation of fluid in the alveoli without a cardiac etiology
 - Acute lung injury: Lower severity
 - Acute respiratory distress syndrome (ARDS): $\text{PaO}_2/\text{FiO}_2$ ratio of ≤ 200 mm Hg
- **New York Heart Association classification:**
 - Class I: Not limited in normal physical activity by symptoms
 - Class II: Ordinary physical activity results in fatigue, dyspnea, or other symptoms
 - Class III: Marked limitation in normal activity
 - Class IV: Symptoms at rest or with any activity
- **Epidemiology:**
 - 5.8 million patients in US
 - Increases with increasing age and affects 10% of population >75 yr.
 - 30–40% of patients with HF are hospitalized every year.
 - 11% 1 mo mortality after AHF admission

ETIOLOGY

- **Cardiogenic etiologies:**
 - Contractile dysfunction:
 - Ischemic heart disease
 - Idiopathic cardiomyopathy
 - Myocarditis
 - Systolic pressure overload:
 - Aortic stenosis
 - Systemic hypertension

- Systolic volume overload:
 - Aortic regurgitation
 - Mitral regurgitation
- Restricted diastolic filling:
 - Mitral stenosis
 - Left atrial myxoma
 - Hypertrophic cardiomyopathy
- High-output states:
 - Hyperthyroidism
 - Anemia
 - Arteriovenous fistula
 - Wet beriberi
- Congenital heart disease
- Endocarditis
- Rheumatic heart disease
- Noncardiogenic etiologies:
 - Sepsis
 - Acute pulmonary infection, aspiration
 - Inhalation injuries
 - Aspiration
 - Near drowning
 - Disseminated intravascular coagulation
 - Pancreatitis
 - Pulmonary contusion
 - Severe (nonthoracic) trauma
 - Cardiopulmonary bypass
 - Uremia
 - High-altitude pulmonary edema
 - Neurogenic pulmonary edema
 - Narcotic overdose
 - Salicylate overdose
 - Pulmonary embolism
 - Fat embolism
 - Transfusion-related acute lung injury

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Risk factors:
 - Prior CHF diagnosis

- History of coronary artery disease or myocardial infarction
- Diabetes
- Severe systemic illness
- Symptoms:
 - Dyspnea on exertion progressing to dyspnea at rest
 - Orthopnea
 - Peripheral edema
 - Paroxysmal nocturnal dyspnea
 - Acute weight gain
 - Weakness/fatigue
 - Cough

Physical-Exam

- Vital signs
 - May be hypertensive or hypotensive
 - Tachypnea
 - Low oxygen saturation
- General
 - Diaphoresis
 - Cold, ashen, or cyanotic skin
- Respiratory:
 - Rales
 - Wheezes
 - Accessory muscle use
- Cardiovascular
 - Tachycardia
 - Jugular venous distention
 - Increased P2, S3, S4
 - Hepatojugular reflex
- Extremities
 - Peripheral edema
- Noncardiogenic
 - Similar pulmonary but rarely peripheral signs

ESSENTIAL WORKUP

- ECG to evaluate for cardiac ischemia and dysrhythmias.
- Chest x-ray to confirm the diagnosis and assessing illness severity.
- Labs: B-type natriuretic peptide (BNP), cardiac enzymes, and creatinine

DIAGNOSIS TESTS & NTERPRETATION

Lab

- BNP:

- Lab parameter for the detection and follow-up of heart failure:
 - <100 pg/mL: CHF unlikely
 - 100–500 pg/mL: Indeterminate
 - >500 pg/mL: Most consistent with CHF
- May not be elevated in very acute CHF or ventricular inflow obstruction
- May be falsely elevated in patients with renal failure undergoing dialysis due to LVH
- N-terminal pro-BNP:
 - Similar test characteristics to BNP
- Cardiac troponins:
 - May be elevated due to myocardial ischemia causing AHF or as result of AHF's effects on cardiac myocytes
 - Elevated in 20% of AHF episodes
 - Strong negative prognostic factor
- Serum chemistry panel:
 - Creatinine elevation:
 - Predicts all-cause mortality in chronic heart failure
 - Indication of acute end-organ hypoperfusion
 - Indication for admission or observation
 - Hyponatremia: Marker of severe HF
 - Electrolyte abnormalities are common due to various HF treatments.
- Elevated alanine aminotransferase, aspartate aminotransferase, or bilirubin suggests congestive hepatopathy.
- Serum lipase if pancreatitis is suspected as the underlying cause
- Arterial blood gas: Evaluates hypoxemia, ventilation/perfusion mismatch, hypercapnia, and acidosis.

Imaging

- CXR:
 - Pulmonary redistribution: Cephalization of vessels
 - Cardiomegaly: Cardiac silhouette >50% of thoracic width on PA exam only
 - Interstitial edema:
 - Pleural effusions
 - Kerley B lines
 - Bilateral perihilar alveolar edema producing a characteristic butterfly pattern
 - Noncardiogenic: Bilateral interstitial or alveolar infiltrates in a homogeneous pattern, typically without enlarged heart shadow
 - Radiographs are often normal in the 1st 12 hr of the disease process.
- ECG:
 - Assess for underlying cardiac disorders:
 - Acute dysrhythmias
 - Signs of acute coronary syndromes

- Signs of electrolyte abnormalities
- Atrial fibrillation occurs in 30–42% of patients admitted for acute heart failure.
- Both tachy- and bradydysrhythmias can lead to decreased cardiac output.
- Echocardiography:
 - Evaluates left ventricle function
 - Assesses acute valvular or pericardial pathology
 - Measures cardiac output
- Bedside ultrasonography:
 - Bilateral B-lines: Comet-tail artifacts arising from pleural line extending to the far field without a decrease in intensity on both the left and the right thorax

DIFFERENTIAL DIAGNOSIS

- COPD exacerbation
- Pneumonia
- Asthma
- Pulmonary embolism
- Pericardial tamponade
- Pneumothorax
- Pleural effusion
- Anaphylaxis
- Acidosis
- Hyperventilation syndrome



TREATMENT

PRE HOSPITAL

- IV access
- Supplemental oxygen
- 100% nonrebreather mask
- Cardiac monitor
- Pulse oximetry
- Sublingual nitrates
- If bag valve mask is needed, should use PEEP valve if available
- Endotracheal intubation in severe cases.

INITIAL STABILIZATION/THERAPY

- Assess and gain control of airway, breathing, and circulation.
- Noninvasive ventilation or endotracheal intubation for impending respiratory failure.

- IV access
- Supplemental oxygen
- Cardiac monitor
- Pulse oximetry
- Place patient in an upright position.
- Inotropic therapy for hypotensive patient with signs of end-organ dysfunction

ED TREATMENT/PROCEDURES

- Treatment decisions should be based on the underlying cause of pulmonary edema.
- Supplemental O₂
- Volume restriction
- Urine output monitoring with or without urinary catheter
- BiPAP/CPAP:
 - _ Improves oxygenation, reduces respiratory work, decreases left ventricular afterload
 - _ Reduces need for intubation, length of stay, and mortality
 - _ Efficacy of BiPAP = CPAP
- Noncardiogenic causes: Frequently require positive-pressure ventilation:
 - _ Low-volume ventilation recommended (6 mL/kg)
- Positive-end expiratory pressure: Most useful strategy for oxygenation
- Hypotensive patients:
 - _ Avoid nitrates, angiotensin-converting enzyme inhibitors (ACEIs), and morphine.
 - _ Initiate inotropes:
 - Dobutamine, Dopamine, Norepinephrine, or Milrinone
 - _ Direct cardioversion for new onset unstable atrial fibrillation
- Normotensive or hypertensive patients:
 - _ Nitrates (nitroglycerin vs. nitroprusside)
 - _ Diuretics (furosemide vs. bumetanide) may be most effective after initial stabilization
- Noncardiogenic: Treat underlying cause.

MEDICATION

- Aspirin: 325 mg PO/PR if myocardial infarction suspected
- Bumetanide: 1–3 mg IV
- Captopril: 6.25 mg SL
- Dobutamine: 2–10 µg/kg/min IV, titrate. May lower BP due to vasodilatory effects.
- Dopamine: 2 × 20 µg/kg/min IV; titrate
- Enalapril: 0.625–1.25 mg IV
- Furosemide: 20–80 mg IV
- Torsemide: 10–20 mg IV
- Milrinone: 50 µg/kg IV; titrate; inotropic effects comparable to dobutamine
- Nitroglycerin: 0.4 mg SL; 1–2 in; 5–20 µg/min IV and titrate; Nitropaste is not

- preferred as it is more difficult to titrate and to use in diaphoretic patients.
- Nitroprusside: 0.25–0.3 µg/kg/min, titrate up by 0.5 µg/kg/min q2–3 min until desired effect
- Norepinephrine: 2–12 µg/min IV; titrate



FOLLOW-UP

DISPOSITION

Admission Criteria

- ICU:
 - Positive-pressure ventilation
 - Inotropic support
 - Acute cardiac ischemia or infarction
 - ARDS
- Monitored unit:
 - New-onset pulmonary edema
 - Electrocardiographic changes
 - Patients presenting with risk factors for mortality, including advanced age, renal dysfunction, hypotension, digoxin use, and anemia

Discharge Criteria

- Most patients with pulmonary edema should be admitted or observed for 24 hr.
- Patients with mild underlying disease and a mild exacerbation that responds fully to ED management and have no risk factors for in-house mortality (see above) may be discharged.
- Ensure close outpatient follow-up.

FOLLOW-UP RECOMMENDATIONS

- Contact patient's primary physician and/or cardiologist to establish close follow-up.
- Continue diuresis.
- Low-salt diet
- Daily weights

PEARLS AND PITFALLS

- Nitrates, SL and IV, are 1st-line therapy to reduce preload.
- BNP can reliably differentiate between AHF syndromes and other causes of dyspnea.
- AHF chest radiography findings can be absent early in disease course.
- Aggressive, early treatment of normotensive and hypertensive patients with

nitrates, diuretics, and ACEIs can rapidly reverse the clinical course.

- Positive-pressure ventilation is an essential intervention in noncardiogenic pulmonary edema and can reduce rates of intubation and mortality in AHF.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Congestive Heart Failure



CODES

ICD9

- 428.1 Left heart failure
- 514 Pulmonary congestion and hypostasis
- 518.82 Other pulmonary insufficiency, not elsewhere classified

ICD10

- I50.1 Left ventricular failure
- J80 Acute respiratory distress syndrome
- J81.0 Acute pulmonary edema

PULMONARY EMBOLISM

Alan M. Kumar



BASICS

DESCRIPTION

- The majority of pulmonary embolisms (PEs) arise from thrombi in the deep veins of the lower extremities and pelvis.
- Thrombi also originate in renal and upper extremity veins.
- After traveling to lungs, the size of the thrombus determines signs and symptoms.

ETIOLOGY

- Most patients with PE have identifiable risk factor:
 - Recent surgery
 - Pregnancy
 - Previous deep vein thrombosis (DVT)/PE
 - Stroke or recent paraplegia
 - Malignancy
 - Age > 50 yr
 - Obesity
 - Smoking
 - Oral contraceptives
 - Major trauma
- Hematologic risk factors:
 - Factor 5 Leiden
 - Protein C or S deficiency
 - Antithrombin III deficiency
 - Antiphospholipid antibody syndrome
 - Lupus anticoagulant

Pediatric Considerations

- Thromboembolic disease is quite rare.
- Risk factors in children:
 - Presence of central venous catheter
 - Immobility
 - Heart disease
 - Trauma
 - Malignancy
 - Surgery
 - Infection

DIAGNOSIS

SIGNS AND SYMPTOMS

- Variability in signs and symptoms make diagnosis difficult
- Most common:
 - Dyspnea
 - Pleuritic chest pain
 - Tachypnea
- General:
 - Fevers (rarely $> 102^{\circ}\text{F}$)
 - Diaphoresis
- Pulmonary:
 - Cough
 - Hemoptysis (rarely massive)
 - Rales
- Cardiovascular:
 - Tachycardia
 - Syncope
 - Murmur
- Extremities:
 - Cyanosis
 - Evidence of thrombophlebitis
 - Lower-extremity edema
- Abdominal pain
- Symptoms similar in elderly but typically more subtle if age < 40 yr

ESSENTIAL WORKUP

- Routine labs are nonspecific.
- CXR:
 - Used to rule out other causes
 - Most common findings with PE:
 - Normal
 - Nonspecific parenchymal abnormality
 - Atelectasis
 - Other findings with PE:
 - Pleural effusions
 - Pleural-based opacities (Hampton hump)
 - Elevated hemidiaphragm
 - Local oligemia (Westermark sign)
- ECG:
 - To rule out cardiac etiology
 - Usually normal in PE
 - Other findings include:

- Nonspecific ST–T-wave changes (most common abnormality)
- Sinus tachycardia
- Left axis deviation
- Right bundle branch block pattern
- S1Q3T3 pattern is uncommon and not specific enough to rule in/out diagnosis.
- Modified Wells criteria:
 - Popular decision rule that can assist with risk stratification in combination with d-dimer
 - Each criterion is given numeric value and if total value <4, along with negative d-dimer, risk of PE is <2%:
 - Clinical signs/symptoms of DVT: 3 pts
 - PE is no. 1 diagnosis: 3 pts
 - Heart rate >100 bpm: 1.5 pts
 - Surgery or immobilization for 3 days within last 4 wk: 1.5 pts
 - Previous PE or DVT: 1.5 pts
 - Hemoptysis: 1 pt
 - Malignancy with treatment within last 6 mo: 1 pt
- Pulmonary Embolism Rule-out Criteria (PERC)
 - Useful in low prevalence setting (ED) in combination with low clinical suspicion.
 - Age <50 yr
 - Heart rate <100 bpm
 - O₂ saturation ≥95%
 - No hemoptysis
 - No estrogen use
 - No prior DVT or PE
 - No unilateral leg swelling
 - No surgery or trauma requiring hospitalization within the past 4 wk
 - <1% risk for PE/DVT in 45 days if PERC score 0

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Arterial blood gas:
 - Can show hypoxemia, hypocapnia, respiratory alkalosis, or increased alveolar–arterial (A–a) gradient
 - PE still possible with normal A–a gradient
 - Does not aid in diagnosis of PE
- CBC:
 - Anemia may be contributing factor to dyspnea.
- d-dimer enzyme-linked immunosorbent assay:

- d-dimers are detectable at levels > 500 ng/mL in nearly all patients with PE.
- High sensitivity (close to 100%) with low specificity for PE
- Almost always elevated in patients with malignancy or surgery within the last 3 mo
- Multiple studies confirm that negative enzyme-linked immunosorbent assay d-dimer in combination with low clinical suspicion effectively rules out PE.

Imaging

- **Spiral chest CT with IV contrast:**
 - Has ability to also detect alternative pulmonary abnormalities
 - Accurate for identifying PE in proximal pulmonary tree:
 - In patients with high pretest probability, positive predictive value of 96%
 - In patients with low pretest probability, negative predictive value of 96%
- **Ventilation–perfusion scan (V/Q):**
 - Results reported in probabilities and correlated to clinical suspicion
 - Probability of PE with V/Q results:
 - Normal or near normal V/Q scan: 4% probability for PE
 - Low-probability V/Q scan with low clinical suspicion: 4% probability for PE
 - Low-probability V/Q scan with high clinical suspicion: 16–40% probability for PE
 - Intermediate V/Q scan: 16–66% probability for PE
 - High-probability V/Q scan with low clinical suspicion: 56% probability for PE
 - High-probability V/Q scan with high clinical suspicion: 96% probability for PE
- **Lower-extremity duplex US:**
 - Used in patients who would otherwise require pulmonary angiogram
 - Presence of DVT requires same anticoagulation as PE.
 - Negative lower-extremity duplex does not rule out PE.
- **Echocardiogram:**
 - Used to assess for right heart strain or patent foramen ovale when thrombolysis is a possibility

Diagnostic Procedures/Surgery

Pulmonary angiogram:

- Gold standard for diagnosis
- Used when diagnosis not confirmed or excluded
- Higher complication rate than other modalities

DIFFERENTIAL DIAGNOSIS

- Anxiety disorder
- Aortic dissection
- Asthma
- Cardiac dysrhythmias
- Costochondritis
- Myocardial infarction
- Pericarditis
- Pneumonia
- Pneumothorax
- Rib fracture



TREATMENT

PRE HOSPITAL

- Initial supplemental oxygen
- Establish IV access
- Cardiac monitor

INITIAL STABILIZATION/THERAPY

- Airway, breathing, and circulation
- Provide supplemental oxygen to maintain adequate oxygen saturation.
- Intubate if unable to provide adequate oxygenation.
- Administer IV fluids carefully for hypotensive patients:
 - _ Excessive fluid expansion may worsen right heart failure.
- IV vasopressor therapy is indicated if hypotension does not resolve with IV fluids.

ED TREATMENT/PROCEDURES

- Anticoagulation:
 - _ Prevents additional thrombus from forming
 - _ Stabilizes existent clot to prevent migration
 - _ Risk of minor/major bleeding with therapy
- Unfractionated heparin:
 - _ Dose titration fraught with difficulty leading to inadequate therapy
 - _ Goal to maintain partial thromboplastin time test between 1.5 and 2.5 times the control value (60–80 sec)
- Low-molecular-weight heparin:
 - _ At least as effective as unfractionated heparin in multiple prospective randomized trials
 - _ Therapeutic goal automatic with weight-based dosing
 - _ Easier administration and monitoring than heparin with some cost benefit
- Warfarin:

- Oral therapy for long-term anticoagulation
- Goal is international normalized ratio (INR) of 2–3
- Rivaroxaban:
 - Oral factor 10a inhibitor
 - Recently approved for treatment of PE
 - Does not require lab monitoring
 - Not recommended in renal/hepatic insufficiency or pregnancy
 - No specific antidote but has short half-life in case of bleeding
- Thrombolysis:
 - Initiate in hemodynamically unstable patients with confirmed PE.
 - Consider in stable patients with PE and severe hypoxemia, massive PE, or right ventricular dysfunction.
- Inferior vena cava filter:
 - Indicated in patients who have contraindications to anticoagulation or have been therapeutic on anticoagulation but failed prevention of PE
- Surgical or catheter embolectomy:
 - Consider in those with thrombolysis contraindications or failure, or deemed unstable for medical management.
 - Case-by-case basis

MEDICATION

- Alteplase: 100 mg (peds: N/A) IV over 2 hr
- Enoxaparin: 1 mg/kg (peds: 0.75 mg/kg) SC q12h
- Reteplase: 10 U (peds: N/A) IV bolus q30min × 2
- Streptokinase: 250,000 U (peds: 3,500–4,000 U/kg) IV bolus over 30 min, then 100,000 U (peds: 1,000–1,500 U/kg) IV maintenance over 24 hr
- Unfractionated heparin:
 - Bolus: 80 U/kg (peds: 75 U/kg) IV over 10 min
 - Maintenance: 18 U/kg (peds: 20 U/kg) IV drip
 - Do not use TBW to calculate dose in obese patients.
- Warfarin: 5 mg (peds: 0.05–0.34 mg/kg/d) PO per day, adjust for INR goal 2–3
- Rivaroxaban: 15 mg BID × 3wks then 20mg QD



FOLLOW-UP

DISPOSITION

Admission Criteria

- Admit all patients with PE for continued anticoagulation and observation.
- Clinically stable patients with a high suspicion for PE, no contraindication to anticoagulation, and a lack of V/Q scanning or angiographic availability may be anticoagulated and studied when resources are available in the morning.

PEARLS AND PITFALLS

- Clinical presentation is variable and nonspecific, making diagnosis difficult in many cases.
- Patients with malignancy are at higher risk for Coumadin failure and recurrent PE even with therapeutic INR.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Chest Pain
- Dyspnea



CODES

ICD9

- 415.11 Iatrogenic pulmonary embolism and infarction
- 415.19 Other pulmonary embolism and infarction
- 673.20 Obstetrical blood-clot embolism, unspecified as to episode of care or not applicable

ICD10

- I26.99 Other pulmonary embolism without acute cor pulmonale
- O88.219 Thromboembolism in pregnancy, unspecified trimester

PURPURA

Richard E. Wolfe • Ashley L. Greiner

BASICS

DESCRIPTION

- Skin lesions caused by extravasation of blood into the skin or subcutaneous tissue
- Can be caused by fragile capillaries, poor dermal support, and/or platelet dysfunction
- The resultant lesions do not blanch completely with pressure (as seen when pressing down through a glass slide)
- Nomenclature varies by the size of the lesions:
 - Petechiae (≤ 4 mm)
 - Purpuric lesions (5–10 mm)
 - Ecchymoses (> 10 mm)
- Color determined by depth and time of onset:
 - Red if superficial and recent onset
 - Purple if deep
 - Deep purple, brown, orange, or blue-green with later presentations
- Nonpalpable purpura:
 - Simple hemorrhage or microvascular occlusion with ischemic hemorrhage
 - Generally due to a platelet disorder:
 - Diminished production
 - Altered distribution
 - Increased destruction
 - Abnormal function
- Palpable purpura:
 - Generally due to vasculitis:
 - Autoimmune, small-vessel leukocytoclastic vasculitis
 - Hypersensitivity to various antigens
 - Formation of circulating immune complexes deposited in walls of postcapillary venules; activate complement that is chemotactic for polymorphonuclear leukocytes
 - Released enzymes damage vessel walls and cause leakage of blood
 - Vasculitic lesions may not be palpable in immunocompromised patients

ETIOLOGY

- Nonpalpable purpura:
 - Viral:
 - Echovirus

- Coxsackie
- Measles
- Parvovirus B19

– Drugs:

- Acetaminophen
- Allopurinol
- Anticoagulants
- Aspirin
- Digoxin
- Furosemide
- Gold salts
- Lidocaine
- Methyldopa
- Nonsteroidal anti-inflammatory drugs
- Penicillin G
- Phenylbutazone
- Quinidine
- Quinine
- Rifampin
- Steroids
- Sulfonamides
- Thiazides

– Nutritional deficiencies:

- Vitamin K deficiency
- Vitamin C deficiency (Scurvy)

– Bone marrow disease

– Hypersplenism

– Idiopathic thrombocytopenic purpura (ITP)

– Disseminated intravascular coagulation (DIC)

– Thrombotic thrombocytopenic purpura

– Liver or renal insufficiency

– Thrombocytopenia ($< 50,000$ plt/cc)

– Thrombocytosis ($> 1,000,000$ plt/cc)

– Spiking elevations of intravascular pressure (childbirth, vomiting, paroxysmal coughing)

– Hemophilia

– Solar purpura (only on sun-exposed areas)

– Post-transfusion

● Palpable purpura:

– Viral:

- Echovirus type 9
- Coxsackie
- Hepatitis B

- *Streptococcal pharyngitis*
- Drugs:
 - Allopurinol
 - Anti-influenza vaccines
 - Cephalosporins
 - Gold
 - Heparin
 - Hydralazine
 - Iodides
 - Levamisole
 - Metoclopramide
 - Penicillin G
 - Phenylbutazone
 - Phenytoin
 - Quinidine
 - Quinine
 - Streptomycin
 - Sulfonamides
 - Thiazides
 - Ticlopidine
- Malignancies
- Autoimmune and connective tissue diseases
- *Gonococcus*
- *Meningococcus*
- *Pseudomonas* (ecthyma gangrenosum)
- Rocky Mountain spotted fever
- In immunocompromised hosts: *Candida*, *Aspergillus*
- Occlusion due to organisms living in vessels (generally immunocompromised hosts): Mucormycosis, aspergillosis, and disseminated strongyloidiasis
- Occlusion due to microvascular platelet plugs (heparin necrosis)
- Cold-related gelling or agglutination (cryoglobulinemia)
- Local or systemic coagulation abnormalities: Scarlet fever, *Vibrio vulnificus* bacteremia; “malignant chickenpox” and “black measles” (both rare in US); Coumadin necrosis
- Embolization: Cholesterol, crystal, thrombus (atrial myxoma, septic endocarditis, multiple myeloma)

Pediatric Considerations

- Henoch–Schönlein purpura
- Hemolytic uremic syndrome
- Kawasaki disease
- Neonatal:

- Extramedullary erythropoiesis in rubella and cytomegalovirus (blueberry muffin baby)
- Purpura fulminans (protein C and S deficiency)
- Maternal ITP
- Wiskott–Aldrich syndrome

DIAGNOSIS

SIGNS AND SYMPTOMS

- Palpable or nonpalpable, nonblanching lesions
- Size:
 - Petechiae (≤ 4 mm)
 - Macular (5–10 mm)
 - Ecchymoses (> 1 cm)
- Shape:
 - Round lesions: Caused by leukocytoclastic emboli
 - Irregular (retiform) lesions: Caused by infectious emboli
 - Annular or erythema multiforme (target lesions)
- Distribution:
 - Generalized: Consider DIC and meningococemia
 - Dependent: Most common. Seen in the lower extremities (increased hydrostatic force)
 - Acral: Found in the extremities only
 - Oral/mucous membranes: Consider ITP
- Hypotension
- Altered mental status
- Gingival hemorrhage
- Epistaxis
- Hematuria
- Fever
- Malaise
- Arthralgias/hemarthroses
- Myalgias
- Purpura fulminans:
 - Large, irregular ecchymoses
 - Fever
 - Shock
 - DIC
- *Pseudomonas* (ecthyma gangrenosum):
 - Begins as edematous, erythematous papules
 - Bullae formation in girdle region
- Disseminated gonococcal infection:

- Usually < 10 lesions, purpuric papules, or vesicopustules on the extensor surface of hands, dorsal aspect of ankles and toes
- Fever
- Arthralgias
- Meningococemia:
 - Small areas of skin infarction cause retiform purpura
 - May involve head, palms, soles, mucous membranes, including conjunctivae
 - Fever
 - Headache
- Rocky Mountain spotted fever:
 - After 4–7 days of generalized symptoms, erythematous macules on distal extremities including palms and soles, then petechial
 - Fever
 - Headache
- Henoch–Schönlein purpura:
 - Appears on extensor aspects of lower extremities and buttocks
 - Fades in about 5 days
 - Fever
 - Arthralgias
 - Abdominal pain
 - Hematuria
- Kawasaki disease:
 - Purpura is rare
 - Fever, plus 4 of the following: Polymorphous exanthem, peripheral extremity changes, bilateral conjunctivitis, changes of lips and mouth, unilateral cervical lymphadenopathy
- Levamisole adulterated cocaine:
 - Retiform, necrotic purpura involving the ears/face but can occur anywhere
 - Neutropenia
 - Recurrent symptoms with continued abuse

ESSENTIAL WORKUP

- Obtain a complete medical history
- Previous bleeding problems
- Deep venous thrombosis/pulmonary embolism suggesting factor V Leiden mutation
- Splenectomy
- Alcohol and drug abuse
- Family history of bleeding disorders
- High-risk medications

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Platelet count: Abnormal counts must be verified by manual exam of a peripheral smear
- DIC screen: Indicated when patient appears toxic
- PT/PTT
- Chemistry panel including liver function tests
- Rapid strep test
- Urinalysis
- Studies for outpatient management:
 - Bleeding time
 - Hepatitis B and C serologies
 - Strep throat culture or anti-streptolysin O titer
 - Antinuclear antibodies
 - Cryoglobulins
 - Platelet function studies
 - Serum complements
 - Serum protein electrophoresis
 - von Willebrand disease screen

DIFFERENTIAL DIAGNOSIS

- Disorders with telangiectasias:
 - Cherry angiomas
 - Hereditary hemorrhagic telangiectasia
 - Chronic actinic telangiectasia
 - Scleroderma
 - CREST syndrome
 - Ataxia-telangiectasia
 - Chronic liver disease
 - Pregnancy-related telangiectasia
- Kaposi sarcoma and other vascular sarcomas
- Fabry disease
- Neonatal extramedullary hematopoiesis
- Angioma serpiginosum



TREATMENT

PRE HOSPITAL

- IV access
- Monitor for:
 - Fever
 - Hypotension
 - Altered mental status

INITIAL STABILIZATION/THERAPY

- For fever, hypotension, altered mental status, or generalized ecchymoses:
 - Airway support
 - IV access
 - Fluid resuscitation
 - IV antibiotics as soon as possible

ED TREATMENT/PROCEDURES

- Presumptive treatment of infectious etiology:
 - *Meningococcus*: Ceftriaxone (Prophylaxis: Rifampin or Ciprofloxacin)
 - *Pneumococcus*: Ceftriaxone, consider penicillin
 - *Rickettsia rickettsii*: Doxycycline, Chloramphenicol in pregnancy

MEDICATION

- Ceftriaxone: 2 g (peds: 100 mg/kg/24h) IV BID
- Ciprofloxacin (prophylaxis): 500 mg PO once
- Chloramphenicol: 75 mg/kg/24h PO or IV QID
- Doxycycline 100 mg (peds: 4 mg/kg/24h) PO or IV BID
- Penicillin G: 4 million U (peds: 240,000 U/kg/24h) IV q4h
- Rifampin (prophylaxis): 600 mg PO BID for 2 days
- Neonatal sepsis: Ampicillin 100 mg/kg/24h IV q6h *and* gentamicin 7.5 mg/kg/24h IV q8h (or cefotaxime 200 mg/kg/24h IV q6h)



FOLLOW-UP

DISPOSITION

Admission Criteria

- Unstable vital signs
- Altered mental status
- Fever

Discharge Criteria

Exclusion of life-threatening etiologies:

- Serious bacterial infections
- Critical thrombocytopenia

Issues for Referral

- Serious hematologic, rheumatologic features and malignancies require an in-depth outpatient assessment if the patient is not admitted
- No contact sports or antiplatelet agents until cleared by a physician

FOLLOW-UP RECOMMENDATIONS

- Appropriate close follow-up scheduled
- Consider follow-up with dermatology (skin biopsy) and hematology

PEARLS AND PITFALLS

Consider empiric antibiotics to cover for meningococemia, Rocky Mountain Spotted fever, and/or sepsis if any doubt of underlying infection

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Rash, Pediatric



CODES

ICD9

- 287.0 Allergic purpura
- 287.2 Other nonthrombocytopenic purpuras
- 287.31 Immune thrombocytopenic purpura

ICD10

- D69.0 Allergic purpura
- D69.2 Other nonthrombocytopenic purpura
- D69.3 Immune thrombocytopenic purpura

PYELONEPHRITIS

Matthew D. Bitner



BASICS

DESCRIPTION

- Complication of a lower UTI by bacterial ascension into the upper urinary tract
- Primarily a clinical diagnosis
- Incidence lower in males in every age group
- Male/female ratio:
 - _ 1:10 in 1st years of life
 - _ 1:5 in children
 - _ 1:50 in reproductive years
 - _ 1:1 in 5th decade and later
- Bilateral infection in up to 25% of cases, hence no lateralizing signs (in some studies)

ETIOLOGY

- Bacteriology:
 - _ *Escherichia coli* 80–95% predominates
 - _ Uropathogens:
 - *Klebsiella* species
 - *Citrobacter* species
 - *Enterobacter* species
 - _ Others:
 - *Staphylococcus saprophyticus* 5–15%
 - *Proteus mirabilis*
 - *Serratia* species
 - *Pseudomonas* species
 - *Staphylococcus aureus* (increasing)
- Predisposing factors (consider complicated infections):
 - _ Recent instrumentation:
 - Catheterization
 - Cystoscopy
 - _ Urinary retention:
 - Mechanical (see Obstruction below)
 - Medications (e.g., anticholinergics)
 - Other infections (e.g., herpes simplex)
 - _ Urinary obstruction:
 - Stricture
 - Renal calculi

- Prostatic hypertrophy
- Anatomic abnormalities:
 - Hypospadias
 - Ureteral ectopia
 - Bifid ureter
 - Renal scarring
 - Ureterovesicular reflux (UVR)
 - Posterior urethral valves
- Neurologic conditions:
 - Neurogenic bladder
 - Spinal cord injury
- Abnormal urodynamics
- Previous UTIs (in childhood, > 3 in last year)
- Recent pyelonephritis (within 1 yr)
- Diabetes mellitus
- Immunosuppression
- Pregnancy



DIAGNOSIS

SIGNS AND SYMPTOMS

- Dysuria, urgency, frequency
- Back, flank, or abdominal pain
- Fever, chills
- Arthralgias, myalgias, malaise
- Nausea and/or vomiting
- Costovertebral angle/suprapubic tenderness
- Ill/toxic appearing
- Dehydration
- Occult pyelonephritis:
 - Invasion of upper urinary tract without clinical symptoms:
 - Suspect in lower UTI that does not resolve with standard treatment.

Pediatric Considerations

- Fever, irritability, lethargy, poor feeding, or jaundice may be only symptom in infants.
- Enuresis in previously toilet-trained child
- Common cause of a serious bacterial infection (SBI) in neonates, young children, and the immunocompromised (hematogenous spread)
- Renal scarring:
 - More common sequelae in young children than in adults
- Group B streptococci

- Etiologic agents in neonates

Geriatric Considerations

Commonly present atypically:

- Absence of classic dysuria/frequency
- Instead nausea/vomiting, diarrhea, fever, or altered mental status may predominate.

ESSENTIAL WORKUP

- Urinalysis (UA):
 - Clean-catch or catheterized urine specimen; catheterized specimen if:
 - Vaginal discharge or bleeding
 - Contaminated specimen
 - Pyuria: 5–10 WBCs, plus leukocyte esterase, plus nitrites:
 - If not present, consider alternate diagnosis.
 - Nitrite represents a gram-negative pathogens are present that is converting dietary nitrates to nitrites.
 - Note that some uropathogens such as *Pseudomonas*, *Enterococcus*, and *S. Saprophyticus* are not nitrate reducers
 - Hematuria:
 - White cell cast: Renal origin of pyuria
- Urine culture and sensitivity:
 - Obtain in:
 - Suspected pyelonephritis
 - Unclear diagnosis
 - Treatment failures, recurrent infections
 - High clinical suspicion, with negative UA
 - >100,000 colony-forming units (CFU)/mL is positive.
 - 10^2 – 10^4 CFU considered positive in:
 - Early infection
 - Clinical scenario consistent with UTI
 - Catheter or suprapubic specimen
 - Males

DIAGNOSIS TESTS & NTERPRETATION

Lab

- CBC:
 - Leukocytosis
 - Does not rule in or out upper tract infection
- Blood cultures:
 - Not needed unless patient is septic; positive cultures do not correlate with

more severe disease.

– Bacteria identified more readily on urine culture

- **Chemistries:**

– For patients with significant risk for electrolytes abnormalities (severe nausea/vomiting, or medication use)

Imaging

- Imaging is required to differentiate pyelitis (no parenchymal involvement) and pyelonephritis (parenchymal involvement); however, this typically does not alter ED treatment.

- **Bedside renal US:**

– Limited value for characterization except for detecting hydro/pyonephrosis/obstruction

- **Helical CT:**

– Superior to renal US in detecting abnormalities/characterizing extent of disease

– Consistent or concerning findings:

- Stranding or inflammation and edema of parenchyma

- Perinephric fluid

- Calculi, obstruction

- Renal/perinephric abscess

- Intraparenchymal gas formation (emphysematous pyelonephritis)

- **MRI:**

– Useful in:

- Pregnant patients (lack of radiation)

- Renal failure (lack of iodinated contrast)

– Cost/availability limit usefulness in the ED

– Obtain imaging if:

- Concomitant stone/obstruction

- At risk for emphysematous pyelonephritis/abscess (diabetes mellitus, immunocompromised, elderly)

- Elective evaluation of genitourinary tract in males with pyelonephritis

Pediatric Considerations

- **Obtain catheter urine specimen:**

– Vast majority of bag urine specimens will result in positive cultures (contaminants).

– Helpful only for excluding disease if culture is negative

– Catheterized or suprapubic specimen with > 1,000 CFU is positive.

- Blood cultures usually performed for children < 1 yr of age (due to risk for SBI)

- All children with 1st episode of pyelonephritis should have urinary tract imaging

performed later to evaluate for UVR.

- Renal US:
 - Within 48 hr if no clinical improvement
 - Within 3–6 wk if clinical improvement

Diagnostic Procedures/Surgery

Suprapubic bladder aspiration:

- When urethral catheterization is not successful, or not possible (phimosis, urethral stricture, etc.)
- Contraindicated when there is an overlying infection, a known anatomic abnormality (tumor), recent complete voiding/micturition

DIFFERENTIAL DIAGNOSIS

- Abdominal aortic aneurysm or dissection
- Appendicitis
- Cholecystitis
- Cystitis
- Diverticulitis
- Cervicitis/pelvic inflammatory disease
- Endometritis/salpingitis
- Inferior pneumonia
- Prostatitis/epididymitis
- Nephrolithiasis
- Renal/perinephric abscess
- Urethritis



TREATMENT

PRE HOSPITAL

IV access for the ill/toxic-appearing patient with appropriate fluid resuscitation

INITIAL STABILIZATION/THERAPY

Treat shock with 0.9% normal saline 500 mL–1 L (peds: 20 mL/kg) IV fluid bolus

- While shock needs to be treated aggressively, be cognizant of fluid overload in patients with comorbidities (renal failure, congestive heart failure).

ED TREATMENT/PROCEDURES

- Parental antibiotics for:
 - Inability to tolerate oral therapy
 - Extremes of age, immunosuppression, and pregnancy
 - Failure of oral/outpatient therapy
 - Urinary obstruction

- Suspected antibiotic-resistant organisms
- Empiric IV antibiotics:
 - Fluoroquinolones (not approved in children)
 - Aminoglycoside (gentamicin) plus ampicillin
 - 3rd-generation cephalosporin (ceftriaxone)
 - In pregnancy:
 - 3rd-generation cephalosporin
 - Gentamicin/ampicillin
 - Cefazolin
 - Aztreonam
- Outpatient oral antibiotics:
 - For nontoxic and otherwise healthy patient:
 - Fluoroquinolone: 7–14 day course
 - May administer 1 dose of parenteral antibiotics prior to oral antibiotics:
 - Ensures prompt cessation of bacterial proliferation
 - No literature addressing efficacy
- Antiemetics and analgesics

MEDICATION

- Oral antibiotics:
 - Ciprofloxacin: 500 mg PO BID
 - Ciprofloxacin ER: 1,000 mg PO daily.
 - Levofloxacin: 750 mg PO daily (5 days)
 - Ofloxacin: 200 mg PO BID
 - Amoxicillin/clavulanic acid: 875 mg/125 mg PO BID
- IV antibiotics:
 - Ceftriaxone: 1 g IV q24h
 - Ciprofloxacin: 400 mg IV q12h
 - Ampicillin/sulbactam: 3 g IV q6h
 - Cefazolin: 1–1.5 g IV q8h
 - Gentamicin: 3–5 mg/kg IV load
 - Levofloxacin: 500 mg IV daily
 - Piperacillin–tazobactam: 3.375 g IV q8h

Pediatric Considerations

- Oral antibiotic liquid preparations for children:
 - Amoxicillin: 30–50 mg/kg/24h PO TID
 - Amoxicillin/clavulanic acid: 45 mg/kg/24h PO TID
 - Cefixime: 8 mg/kg PO daily
 - Cefpodoxime: 10 mg/kg/24h PO BID
 - Cephalexin: 50–75 mg/kg/24h PO QID
 - Erythromycin/sulfisoxazole: 50 mg erythromycin/kg/24h PO QID
- Parenteral antibiotics for admitted children:

- Age 0–3 mo:
 - Cefotaxime (50–180 mg/kg/d TID) + ampicillin (50–100 mg/kg/d QID)
 - Gentamicin (1–2.5 mg/kg/d TID) + ampicillin
- Age > 3 mo:
 - May substitute ceftriaxone (50–100 mg/kg/d BID to daily) for cefotaxime



FOLLOW-UP

DISPOSITION

Admission Criteria

- Sepsis, ill/toxic appearance
- Inability to tolerate oral therapy
- Intractable nausea/vomiting
- Social situation prevents compliance.
- Pregnancy
- Indwelling urinary catheter
- Urinary obstruction/anatomic abnormalities
- Proximal obstruction,
- Immunosuppression/diabetes mellitus
- Extremes of age (children < 2–6 mo)
- Failure of outpatient therapy/recent antibiotics

Discharge Criteria

- Clinical course improving in ED
- Ability to maintain oral hydration
- Pain controlled with oral analgesic
- Normal renal function
- Follow-up in 48–72 hr

FOLLOW-UP RECOMMENDATIONS

- Uncomplicated cases in patients without comorbidities can safely follow up with their primary care physicians.
- If cultures were obtained, patient will need to follow up on results for possible therapy change once antibiotic sensitivities are known.
- Pediatric patients all need to follow up with their pediatrician for required imaging for anatomic abnormalities
- Pregnant patients need repeat UA to assess for resolution/recurrence and possible suppressive therapy.
- Patients with recurrent infections and those with identified unusual or resistant

organisms require close follow-up with urologic and/or infectious disease consultation.

PEARLS AND PITFALLS

- Primarily a clinical diagnosis with minimal lab work required
- Treat young, old, immunosuppressed, and pregnant patients aggressively.
- Consider other diagnoses (e.g., gynecologic etiologies, abdominal aortic aneurysm)

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Pelvic Inflammatory Disease
- Urinary Tract Infection, Adult
- Urinary Tract Infection, Pediatric

ACKNOWLEDGMENT

The author gratefully acknowledges the contribution of Ingrid Carter for previous editions of this chapter.



CODES

ICD9

- 590.10 Acute pyelonephritis without lesion of renal medullary necrosis
- 590.80 Pyelonephritis, unspecified
- 592.0 Calculus of kidney

ICD10

- N10 Acute tubulo-interstitial nephritis
- N12 Tubulo-interstitial nephritis, not spcf as acute or chronic

- N20.0 Calculus of kidney

PYLORIC STENOSIS

Roger M. Barkin



BASICS

DESCRIPTION

- Postnatal hypertrophy and hyperplasia of the circular smooth muscle cell layer causing a thickened pylorus and antrum leading to progressive gastric outlet obstruction
- Neuronal nitric oxide synthase (NOS-1) may be a genetic susceptibility locus.
- Administration of erythromycin in infants during 1st 2 wk of life may increase risk of hypertrophic pyloric stenosis.
- Jaundice due to transient glucuronyl transferase deficiency
- Adult: Caused by peptic ulcer disease

ETIOLOGY

- Most common cause of GI obstruction in infants; incidence 1/150 males, 1/750 females (average: 3/1,000 live births)
- Males affected 5× more commonly than females; firstborn most common
- Familial, 15%:
 - Child of affected parent has 7% incidence.
 - Recurrence risk in subsequent male children is 10%; 2% in females.



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Vomiting:
 - Gradual onset, usually beginning at around 3–6 wk of age; rarely after 12 wk of age
 - Progressive, usually becoming projectile
 - Nonbilious
 - May be blood tinged (secondary to esophagitis, gastritis, gastric ulceration)
 - Progressively worsening
 - Postprandial
- Represents the hypertrophied pylorus:
 - Confirms diagnosis
- Constipation or small amount of stools
- “Lean and hungry” infant early in course; dehydrated and uninterested in feeding late in course; failure to thrive

- Variable dehydration and wasting depending on duration of symptoms
- Jaundice in 8% of children
- Adult presents with vomiting, anorexia, early satiety, and epigastric pain.

Physical-Exam

- Often normal unless a relaxed abdomen
- May feel olive-shaped mass at lateral margin of the right rectus abdominis muscle in the right upper quadrant (80% of patients), often after vomiting:
 - Best felt immediately after vomiting or after the stomach is emptied via gastric suction as the dilated body of the stomach overlies the pylorus
 - Represents the hypertrophied pylorus:
 - Helps confirm diagnosis
 - Peristaltic waves moving from the left to right in the left upper quadrant, seen best after feeding or just prior to vomiting

ESSENTIAL WORKUP

If “olive” palpable, further diagnostic evaluation may be unnecessary and surgical consultation should be sought; otherwise, imaging studies are indicated.

DIAGNOSIS TESTS & NTERPRETATION

Lab

- Electrolytes, BUN/creatinine, glucose:
 - Hypokalemic, hypochloremic metabolic alkalosis
 - Normal electrolytes do not exclude the diagnosis.
- Bilirubin elevated, usually unconjugated
- CBC if blood in emesis
- Urinalysis for hydration

Imaging

- Abdominal US:
 - Study of choice
 - US diagnosis hinges on identification and measurement of pyloric muscle mass (3 mm ring thickness, 1.5 cm pylorus channel or muscle length, and 10–14 mm pylorus diameter) and observation of fluid movement through the pylorus.
 - Positive predictive value approaches 100%; 19% false negatives.
 - Serial US for equivocal or negative study
- Upper GI series:
 - String sign representing contrast passing through a narrowed gastric outlet
 - 95% accurate
 - Remove contrast from the stomach after the study to prevent aspiration.
- Supine abdominal film:

- Not diagnostic; rarely helpful
- Dilated stomach and no air distal to the pylorus
- Most useful with other views to begin evaluation for other abdominal pathology

DIFFERENTIAL DIAGNOSIS

- GI anatomic/functional disorder:
 - Gastroesophageal reflux
 - Hiatal hernia
 - Obstruction/atresia
 - Gastric or duodenal web
- Infection:
 - Gastroenteritis
 - UTI
 - Sepsis
- Metabolic:
 - Adrenal insufficiency
 - Inborn error of metabolism
- Feeding problems:
 - Psychosocial: Poor maternal interaction or stress
 - Chhalasia
 - Formula intolerance
 - Overfeeding
- Drug withdrawal
- Increased intracranial pressure



TREATMENT

PRE HOSPITAL

Fluid resuscitation if significant volume deficit

INITIAL STABILIZATION/THERAPY

- IV access
- Rapid bedside glucose test to exclude hypoglycemia
- Correct volume deficit with 20 mL/kg bolus of 0.9% normal saline IV; may repeat.

ED TREATMENT/PROCEDURES

- Correct electrolyte abnormalities.
- Hydrate with dextrose-containing solution after fluid resuscitation at 1–1.5 × maintenance rate:
 - Add potassium after ensuring adequate urine output.
- Insert nasogastric tube to decompress the stomach.
- Restrict oral intake.

- Consult pediatric surgeon for pyloromyotomy.
- Adult: Proton pump antagonist (lansoprazole or omeprazole)

MEDICATION

Adults

- Lansoprazole: 30 mg daily PO
- Omeprazole: 20 mg daily PO



FOLLOW-UP

DISPOSITION

Admission Criteria

- All pediatric patients should be admitted to the hospital for rehydration and surgical correction with either an umbilical pyloromyotomy or laparoscopic pyloromyotomy.
- Adult patients: Admit as necessary for rehydration; may be scheduled for elective pyloromyotomy if proton pump inhibitors fail to improve this condition.

Discharge Criteria

None

Issues for Referral

Surgical consultation concurrent with correction of electrolytes and fluid deficits

FOLLOW-UP RECOMMENDATIONS

Follow growth pattern after surgery.

PEARLS AND PITFALLS

Suggestive clinical presentation combined with lab evaluation should lead to imaging and correction of electrolyte abnormalities.

ADDITIONAL READING

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CODES

ICD9

537.0 Acquired hypertrophic pyloric stenosis

ICD10

K31.1 Adult hypertrophic pyloric stenosis

QT SYNDROME, PROLONGED

Jason A. Tracy

BASICS

DESCRIPTION

A disorder of myocardial repolarization characterized by a prolonged QT interval on the electrocardiogram

- The pathophysiology is complex and incompletely understood:
 - Alteration in cardiac sodium, potassium, or calcium ion flow
 - Imbalance in the sympathetic innervation of the heart
- Prolonged ventricular repolarization results in lengthening of QT interval on surface ECG:
 - “Pause-dependent” lengthening due to short–long–short sequence in which a sinus beat is followed by an extrasystole (short), then a postextrasystolic pause (long), concluding with a ventricular extrasystole (short)
 - “Adrenergic-dependent” pauses found in congenital cases
- Symptoms often preceded by vigorous exercise, emotional stress, or loud noise.
- Nocturnal bradycardia can lengthen QT interval, causing sleep-related symptoms.
- Re-entrant rhythm can lead to torsades de pointes, ventricular tachycardia, and ventricular fibrillation.
- Hemodynamic compromise following dysrhythmia leads to syncope or death.
- Independent risk factor for sudden cardiac death.

RISK FACTORS

Genetics

- 10 genes linked to long QT syndrome:
 - Autosomal recessive form associated with deafness (Jervell and Lange–Nielsen syndromes)
 - Autosomal dominant form not associated with deafness (Romano–Ward syndrome)
 - Adrenergic stimulation (fright, exertion, delirium tremens, and loud auditory stimulus) becomes prodysrhythmic in certain genotypes, while sleep-related symptoms are found in others.
- 10–15% of carriers have baseline normal QTc.
- Death occurs in 1–2% of untreated patients per year.
 - Drug-induced QT prolongation may also have a genetic background.
 - Congenital form occurs in 1 in 3,000–5,000, with mortality of 6% by age 40 yr.

Pediatric Considerations

- Diagnosis suspected in the young with syncope, cardiac arrest, or sudden death
- Syncope following emotional stress or exercise suggestive
- Death occurs without preceding symptoms in 10% of pediatric patients.

ETIOLOGY

- Drugs:
 - Complete list at www.QTDrugs.org
 - Class Ia antidysrhythmics—quinidine, procainamide, disopyramide
 - Class III antidysrhythmics—sotalol, ibutilide, amiodarone
 - Antibiotics—erythromycin, pentamidine, chloroquine, trimethoprim-sulfamethoxazole
 - Antifungal agents—ketoconazole, itraconazole
 - Psychotropic drugs—phenothiazines, haloperidol, risperidone, STCAs
 - Cisapride
 - Antihistamines
 - Organophosphates
 - Narcotics—methadone
- Electrolyte abnormalities
 - Hypokalemia
 - Hypomagnesemia
 - Hypocalcemia
- Cardiac
 - Bradyarrhythmias
 - Arteriovenous block
 - Mitral valve prolapse
 - Myocarditis
 - Myocardial ischemia
- CNS
 - Subarachnoid hemorrhage
 - Stroke
- Congenital (idiopathic)
- Other
 - Protein-sparing fasting
 - Anorexia nervosa
 - Hypothyroidism
 - Hypothermia

DIAGNOSIS

SIGNS AND SYMPTOMS

- Palpitations
- Light-headedness

- Dizziness

History

- Syncope
- Near syncope
- Seizure
- Family history of syncope or sudden death
- Congenital deafness
- Medication use

ESSENTIAL WORKUP

Cardiac monitor:

- ECG
- QTc (QT corrected for heart rate) > 0.44 sec in men and > 0.46 sec in women
- QT measured from beginning of quasi-random signal to end of T wave:
 - Measured best in the limb leads and should be averaged over 3–5 beats
 - There is no expert consensus on best heart rate correction (QTc) formula.
 - Bazett formula (QT divided by square root of RR interval) is most commonly used
 - Increase in QT variability
- T-wave abnormalities (T-wave alternans, biphasic)
- Appearance of U waves
- Ventricular tachycardia
- Ventricular fibrillation
- Torsades de pointes

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Full electrolytes including calcium and magnesium
- Toxicology screen

Imaging

Echocardiography to exclude other cardiac causes

Diagnostic Procedures/Surgery

- ECG stress testing to induce a prolonged QT interval in suspected cases
- Holter monitoring of QTc
- Genetic counseling/testing in suspected congenital forms
- Familial ECG testing

DIFFERENTIAL DIAGNOSIS

- Myocardial infarction

- Hypertrophic cardiomyopathy
- Valvular defect



TREATMENT

PRE HOSPITAL

- Supplemental oxygen
- IV access
- Monitor

ALERT

- Stable patients with prolonged QT transported without intervention
- Cardioversion for unstable patients with confirmed torsades de pointes
- Magnesium sulfate for stable patients with evidence of torsades de pointes

INITIAL STABILIZATION/THERAPY

- IV access
- Monitor
- Determine hemodynamic stability
- Unstable patients require immediate cardioversion

ED TREATMENT/PROCEDURES

- IV magnesium sulfate for torsades de pointes
- IV potassium to serum levels of 4.5–5 mEq/L
- Temporary transvenous cardiac pacing (rates from 100–120 beats/min) for recurrences of torsades de pointes refractory to magnesium sulfate therapy (shortens QTc)
- IV isoproterenol for refractory cases or hemodynamically unstable patients with acquired long QT (ineffective in congenital cases) who do not respond to transvenous pacing
- Remove any offending medications and correct metabolic derangements.
- Consult with cardiology in those with symptomatic long QT regarding use of β -blockers at maximum doses.
- No ED treatment needed (in consultation with cardiology) for those with suspected idiopathic long QT and no history of syncope, family history of sudden cardiac death, or ventricular arrhythmias.
- Pacemaker or defibrillator placement with or without cervicothoracic stlectomy (to reduce adrenergic stimulation) may be required in high-risk patients.
- β -Blockers prevent 70% of cardiac events in congenital cases.

MEDICATION

First Line

- Magnesium sulfate: 2 g (peds: 25–50 mg/kg) IV bolus over 2–3 min followed by IV infusion at 2–4 mg/min
- Isoproterenol: 1 µg/min (peds: 0.05–0.1 µg/kg/min) IV continuous infusion, titrate for effect, up to 10 µg/min

Second Line

Propranolol: 2–3 mg/kg/d (peds: 2–3 mg/kg/d) PO (in consultation with cardiology)



FOLLOW-UP

DISPOSITION

Admission Criteria

- Symptomatic prolonged QT
- Syncope
- Cardiac dysrhythmia
- Possible cardiac or ischemic event
- Metabolic abnormality

Discharge Criteria

Asymptomatic prolonged QT in consultation with cardiology

FOLLOW-UP RECOMMENDATIONS

Follow-up recommended in all patients with a new diagnosis of prolonged QT

PEARLS AND PITFALLS

- Suspect prolonged QT in patients with syncope
- Prolonged QT is an independent risk factor for sudden cardiac death.
- Correct electrolyte abnormalities and discontinue offending drugs in those with prolonged QT.
- Magnesium sulfate followed by pacing for torsades de pointes.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

www.QTDrugs.org



CODES

ICD9

426.82 Long QT syndrome

ICD10

I45.81 Long QT syndrome

RABIES

Herbert Neil Wigder • Ashley Alwood • Matthew A. Kippenhand

BASICS

DESCRIPTION

CNS infectious disease of mammals caused by the rabies virus:

- Highest case fatality rate of any known infectious disease

ETIOLOGY

- Epidemiology:
 - _ 30,000–70,000 people die/yr worldwide
 - _ Especially common in Southeast Asia, Philippines, Africa, South America, and Indian subcontinent
 - _ US has 2–3 human cases per year.
 - _ Most clinical cases in US from foreign travel and bat exposures
 - _ Raccoons, skunks, foxes, bats, dogs, woodchucks, groundhogs are reservoirs.
 - _ In US bats are the most common reservoir while abroad dogs are more common.
 - _ Squirrels, rats, mice, hamsters, guinea pigs, gerbils, chipmunks, and rabbits can also be infected but there has never been a reported case of human transmission.
- Pathophysiology:
 - _ Negative-stranded RNA genome, family Rhabdoviridae, genus Lyssavirus
 - _ Mode of transmission:
 - Contact with infected saliva of host
 - Bite: Most common
 - Nonbite: Saliva or bat aerosol exposure to an open wound or mucous membrane
 - Transplant procedures are the only well-documented person-to-person transmission
 - Not considered a transmission risk: Petting rabid animal or contact with the blood, urine, or feces of a rabid animal
 - _ Progression after infection:
 - Virus multiplies in local tissue and is taken up into muscle through n-acetylcholine receptors.
 - Virus enters peripheral nerves and is transported to CNS via retrograde axoplasmic flow at ~1--4 inches per day
 - Once in CNS, rapid replication and dissemination cause encephalitis.
 - Centrifugal spread of virus to peripheral nerves, including salivary glands

SIGNS AND SYMPTOMS

- Once a patient exhibits clinical signs course is almost universally fatal.
- 5 stages: Incubation, prodrome, encephalitis, coma, death (or recovery):
 - _ Incubation: 1–3 mo (range 10 days to 1 yr):
 - Virus amplifies in peripheral tissues
 - Time depends on amount inoculated and proximity to CNS, thus shorter incubation for head or neck bites
 - _ Prodrome: 1–7 days:
 - Nonspecific symptoms: Fever, headache, malaise, myalgias, anorexia, sore throat, nausea, and vomiting
 - Paresthesias or fasciculations around bite site give clue to diagnosis.
 - _ Encephalitis (classic form): 2–7 days:
 - Anxiety, agitation, hallucinations, confusion or delirium, muscle spasms, opisthotonos, and seizure
 - Aerophobia (pathognomonic): Pharyngeal spasm from draft of air
 - Hydrophobia (pathognomonic): Violent involuntary muscle contraction of diaphragm, pharyngeal, laryngeal, and accessory respiratory muscles when attempting to swallow (seen in up to half of cases)
 - Dysrhythmias, myocarditis, autonomic instability, and fevers
 - Brainstem involvement: Diplopia, facial paralysis
 - _ Coma:
 - Apnea from respiratory center involvement, vascular collapse, flaccid paralysis, adult respiratory distress syndrome, syndrome of inappropriate diuretic hormone
 - Most die within 2 wk
 - _ Death (or recovery):
 - Almost universally fatal if no pre- or postexposure prophylaxis (PEP) given
 - Rare case reports of survival without prophylaxis
 - Known survivors have some residual neurologic deficits
- 3 manifestations of disease:
 - _ Classic or encephalitic rabies accounts for ~80% of cases. See above.
 - _ Paralytic rabies (~20%): Ascending paralysis mimicking Guillain–Barré syndrome
 - _ Atypical rabies (<1%): Seen with bat-associated rabies. Characterized by neuropathic pain, sensory or motor deficits, choreiform movements, myoclonus, and seizures

- Bite wound or other known exposure
- Bat found in room with person unable to give history (e.g., child or intoxicated): Assume exposure
- Travel to endemic areas with associated dog exposure
- Rabid animals more likely to attack unprovoked. Any handling of the animal prior to bite is considered a provoked attack.

Physical-Exam

- Fever
- A bat bite wound often not visible on exam
- Altered mental status, seizures, encephalopathy
- Percussion myoedema: Muscle mounds at percussion site
- Autonomic manifestations: Dilated pupils, perspiration, hypersalivation, orthostatic hypotension

ESSENTIAL WORKUP

- Saliva:
 - Rabies RNA by reverse transcription polymerase chain reaction (RT-PCR)
 - Virus isolation in cell culture
- Serum:
 - Rabies antibodies are diagnostic only if not vaccinated.
 - Earliest positive, day 6
- CSF:
 - Mildly elevated WBC and protein, normal glucose
 - Virus isolation
 - Rabies antibodies in CSF are diagnostic, even if immunized.

DIAGNOSIS TESTS & NTERPRETATION

Lab

- CBC: May have leukocytosis
- Electrolytes, BUN, creatinine, glucose
- Blood cultures/urinalysis:
 - Search for other infection/illness
- Neck biopsy: RT-PCR, immunofluorescent staining for viral antigen

Imaging

- CT head: Usually normal but may show cerebral edema, evaluate for other causes of symptoms
- Chest radiograph: Other infectious etiologies

Diagnostic Procedures/Surgery

Lumbar puncture

DIFFERENTIAL DIAGNOSIS

- Other causes of encephalitis:
 - Herpesviruses: HSV1, VZV
 - Enterovirus (Coxsackie, echovirus, poliovirus)
 - Arboviruses (West Nile, eastern/western equine encephalitis, St. Louis)
- Tetanus
- Delirium tremens
- Psychosis
- Paralytic form:
 - GBS
 - Polio
 - Tick-bite paralysis
 - Immune-mediated polyneuritis
 - Botulism



TREATMENT

PRE HOSPITAL

- Thoroughly wash wound with soap and water.
- If safely able, capture wild animal for sacrifice and testing.

INITIAL STABILIZATION/THERAPY

- Airway, breathing, and circulation
- Intubation as needed
- Treatment of seizures

ED TREATMENT/PROCEDURES

- Wound cleansing and irrigation
- Tetanus immunization
- Determine if exposure requires prophylaxis:
 - Consult local health department
 - Domestic animal bite:
 - Home monitoring of animal for 10 days
 - If animal displays no signs of illness, patient does not need PEP.
 - Wild animal bite:
 - Rabies testing of sacrificed animal head-Negri bodies are diagnostic
 - Start PEP and stop if test is negative
 - Treat if animal not captured.
 - Unprovoked attacks should be assumed high risk for exposure.
- PEP:
 - Passive immunization with human rabies immune globulin (HRIG)
 - HRIG: 20 IU/kg:

- Majority infiltrated in and around wound
- Remainder given IM (gluteus)
- Active immunization with rabies vaccine
- Rabies vaccine: 1 mL (2.5 IU) IM days 0, 3, 7, 14, add day 28 if immunocompromised
- 3 vaccines approved in US:
 - Imovax–human diploid cell culture
 - RabAvert–chick embryo cell culture
 - Rabies vaccine adsorbed–inactivated virus, for US military
- Administration location:
 - Deltoid in adults or anterior thigh in small children or infants
- For those with pre-exposure prophylaxis and rabies exposure:
 - Do not require HRIG
 - Need vaccine booster on days 0 and 3
- If care delayed after rabies exposure:
 - HRIG not indicated > 7 days after exposure
 - Vaccine should be administered as usual
- Pre-exposure prophylaxis:
 - Rabies vaccine on days 0, 7, 21, 28
 - Target groups: Veterinarians, animal handlers, virus lab workers, foreign travelers in endemic regions

Pediatric Considerations

Treat as in adults.

Pregnancy Considerations

Treatment considered safe during pregnancy.



FOLLOW-UP

DISPOSITION

Ensure adequate access for subsequent vaccine administration post rabies exposure.

Admission Criteria

Patient with clinical signs of rabies

Discharge Criteria

- Stable patient
- No evidence of reaction to vaccine

Issues for Referral

Public health and CDC for suspicious cases

FOLLOW-UP RECOMMENDATIONS

- Ensure access to subsequent vaccine doses
- Patient should follow up with animal control if source animal has been sacrificed or is being observed.

PEARLS AND PITFALLS

- PEP is only proven treatment after exposure.
- PEP should be given in all high-risk exposures regardless of timing
- Vaccine should only be given in deltoid in adults: Treatment failures reported with inadvertent SC administration in gluteus injections.

ADDITIONAL READING

- American Academy of Pediatrics. *Report of the Committee on Infectious Report*. 29th ed. Elk Grove Village, IL: American Academy of Pediatrics; 2012.
- Centers for Disease Control and Prevention. Rabies. Available at <http://www.cdc.gov/rabies/> Updated December 13, 2012.
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- Franka R, Rupprecht CE. Treatment of rabies in the 21st century: Curing the incurable. *Future Microbiol*. 2011;6:1135–1140.
- Manning SE, Rupprecht CE, Fishbein D, et al. Human rabies prevention—United States, 2008: Recommendations of the Advisory Committee on Immunization Practices. *MMWR Recomm Rep*. 2008;57:1–28.
- Willoughby RE Jr, Tieves KS, Hoffman GM, et al. Survival after treatment of rabies with induction of coma. *N Engl J Med*. 2005;352:2508–2514.



See Also (Topic, Algorithm, Electronic Media Element)

- Encephalitis
- Meningitis



CODES

ICD9

- 071 Rabies
- V01.5 Contact with or exposure to rabies

ICD10

- A82.9 Rabies, unspecified
- Z20.3 Contact with and (suspected) exposure to rabies

RADIATION INJURY

Robert J. Feldman



BASICS

DESCRIPTION

- *Radiation* in this chapter refers to ionizing radiation.
- *Alpha* (α)—*helium nucleus; does not penetrate skin*
- *Beta* (β)—*electron; penetrates tissue a few cm*
- *Gamma* (γ)—*photon; penetrates body*
- *Neutron*—*very penetrating; not detected by Geiger counter, but neutron emitters also emit γ radiation*
- *Radioisotope/radionuclide*—*chemical element that emits radiation from its nucleus:*
 - Radioactivity cannot be destroyed, only relocated or shielded.
 - Being radioactive does not change element's other chemical and physical properties, such as heavy metal toxicity.
- *Exposure/irradiation*—*patient has been in presence of ionizing radiation:*
 - Whole body or only certain areas may be exposed.
- *Contamination*—*radioactive material where it is not desired:*
 - Internal—within body (e.g., lung)
 - External—outside body (skin, hair, clothing)
- *Dose*—*amount of radiation energy absorbed by tissue:*
 - Units and conversions:
 - 1 gray (Gy) = 100 rad
 - 1 sievert (Sv) = 100 rem
 - For β and γ radiation:
 - 1 Gy = 1 Sv = 100 rad = 100 rem

ALERT

Contact regional or federal authorities for guidance if radiation incident is suspected.

Pediatric Considerations

- Children are more sensitive to radiation injury.
- Potassium iodide is most protective for children and should be given promptly if contamination with radioactive iodine (I-131) is suspected.

Pregnancy Considerations

- Developing fetus is very sensitive to radiation.
- Pregnant staff should not care for radioactively contaminated patients.

ETIOLOGY

- Ionizing radiation leads to cellular injury.
- Damage to blood vessels leads to endarteritis and loss of tissue blood supply.
- Higher rates of cell division within an organ make it more sensitive to radiation:
 - Bone marrow and GI tract are very sensitive.
 - Skin and nerve are less sensitive.
- *Acute radiation syndrome* (ARS) occurs in stages following whole-body exposure:
 - Prodromal: Acute radiation injury leads to acute inflammation (0–48 hr).
 - Latent: If the acute phase of injury is survived, inflammation and symptoms subside (0–2 wk).
 - Manifest illness: At higher radiation doses, organ failure then develops.
 - Recovery or death (usually from infection) follows.
- Sources of radiation include medical devices, therapeutics, nuclear weapons, and industry.



DIAGNOSIS

- Diagnosing contamination is fairly easy.
- Diagnosing and quantifying exposure is more difficult and probably require expert consultation.

SIGNS AND SYMPTOMS

- Vary based on dose; see: <http://www.afrrri.usuhs.mil/outreach/pdf/AFRRI-Pocket-Guide.pdf> for quick reference.
- Overall:
 - Whole-body exposure: Syndrome similar to high-dose chemotherapy toxicity
 - ARS progresses more rapidly the higher the absorbed dose.
- Local exposure:
 - Early resembles thermal or UV burn
 - Later resembles ischemic ulcer

History

- Recognized exposure:
 - Occupational, medical, transportation accident
- Unrecognized or clandestine exposure:
 - Radiologic dispersal device (RDD), concealed or unrecognized source
 - Industrial and medical radiography sources may be pellets, only a few millimeters in diameter and are highly radioactive.
 - Suspect if multiple patients present with symptoms of ARS at any stage, burns without history of thermal exposure, or ischemic ulcers in unusual locations (e.g., hand from handling unrecognized source, hip from placing source in pocket).

Physical-Exam

- Whole-body exposure:
 - Nausea, vomiting:
 - Within 3–6 hr for >100 rad exposure; sooner with higher exposures
 - Vomiting within 1 hr of exposure indicates potentially lethal injury (>600 rad).
 - Confusion and weakness (>200 rad)
 - Fever:
 - Acutely, from inflammation
 - During manifest illness, from infection
 - Hair loss, hemorrhage, diarrhea may develop with doses >300 rad.
- Dermal exposure:
 - Initial erythema
 - Blistering and ischemic necrosis may follow.

ESSENTIAL WORKUP

- *Survey for radiation* using a *Geiger counter*, which can be found in any nuclear medicine or radiation therapy department:
 - Any probe style is acceptable for survey.
 - Cover probe with exam glove:
 - Prevents contamination of probe
 - Blocks α radiation but detects β/γ
 - Measure background radiation away from patient.
 - Move probe slowly over patient's skin:
 - 1–2 cm from skin
 - Move probe only 2–3 cm/sec.
 - Contamination is $>2 \times$ background radiation level.
 - Note any contaminated areas.
 - Follow systematic pattern to avoid missing areas.
 - Remember to survey palms, soles, hair.
- *Absolute lymphocyte count (ALC)* is the best indicator of severity of ARS:
 - $<1,000/\text{mm}^3$: Moderate exposure, 200–600 rad
 - $<500/\text{mm}^3$: Severe exposure, >600 rad

DIAGNOSIS TESTS & NTERPRETATION

Lab

- CBC with differential every 4–6 hr (for 24 hr or until stable)
- Swab both nares and survey swab for inhaled contaminants.
- Type and cross-match blood.
- 24-hr stool for radioassay if GI contamination suspected
- 24-hr urine for radioassay if any internal contamination is suspected

Imaging

- Diagnostic imaging as clinically indicated
- Whole-body gamma camera (without collimator) is best for ruling out internal contamination with low levels of radioisotopes, if suspicion is high and survey with Geiger counter is negative.

Diagnostic Procedures/Surgery

Cytogenetics allows more accurate dose assessment:

- 10 mL blood in lithium-heparin tube (ethylenediaminetetraacetic acid also acceptable)
- Draw 24 hr postexposure.
- Refrigerate (4°C) and ship cold to Radiation Emergency Assistance Center/Training Site (REAC/TS).
- Only limited number of samples can be processed due to resources required.

DIFFERENTIAL DIAGNOSIS

- Systemic illness: Lymphopenia, weakness, nausea:
 - Psychological effects are common in both exposed and unexposed patients and may mimic ARS:
 - Radiation casualty with vomiting from ARS should have falling ALC; if ALC normal and stable, consider psychological stress reaction or other type of illness.
 - Hematologic malignancy
 - Chemical warfare agents (blister/mustard)
 - HIV disease, immunosuppression
- Skin injuries:
 - Ischemic ulcer
 - Brown recluse spider bite
 - Pyoderma gangrenosum



TREATMENT

Personal protective equipment (PPE):

- Must provide protection from dust (particulate respirator; e.g., N-95, gown, gloves, hair, and shoe covers)
- Radiography aprons are of no value—they do not protect against most γ radiation.

PRE HOSPITAL

- Treat life threats (airway, breathing, and circulation management [ABCs]).
- Assess any bombing scene for radioactive contamination (RDD).
- Removing clothing will eliminate about 80% of external contamination.
- Survey for residual contamination:
 - No contamination: Patient may be cared for as usual.

- If contamination is present, assess medical condition:
 - Stable: Proceed with decontamination.
 - Unstable: Provide necessary care and transport; use sheets to control contamination.

INITIAL STABILIZATION/THERAPY

- ABCs
- Assess for contamination.
- If patient condition permits, perform decontamination before patient enters (and contaminates) facility.
- Minimize staff exposure:
 - *Time*: Limit time in contaminated area, remove contaminated material often.
 - *Distance*: Use long-handled instruments to handle contaminated material.
 - *Shielding*: Place contaminated material in a lead container (available in nuclear medicine department); radiography lead aprons are not effective.

ED TREATMENT/PROCEDURES

- Hospital issues:
 - Activate hospital disaster plan, if indicated, to mobilize resources.
 - Designate contaminated and “clean” treatment areas.
 - Appoint a temporary radiation safety officer (RSO) for incident to survey all patients and staff and all materials leaving treatment area
 - Any staff member who is trained to use Geiger counter and dosimeters may fill RSO role initially if necessary.
 - Patients and materials that are not contaminated do not need decontamination or containment.
 - Call for expert assistance: Hospital RSO, local department of nuclear safety, health department, or REAC/TS.
- Staff issues:
 - Provide PPE and psychological support as described above.
 - Assign pregnant personnel to “clean” areas only.
- Decontamination:
 - *Priorities*: Wounds > mucous membranes > intact skin
 - Use fenestrated drapes to shield adjacent skin.
 - Use soap and water; no harsh chemicals.
 - Diaper wipes work well for intact skin; wipe from edges of area to center, then lift away.
 - Irrigate wounds—collect and survey runoff, avoid splashing.
 - Resurvey frequently to assess effectiveness of decontamination.
 - Do not abrade skin.
 - If contamination cannot be removed, cover area to prevent spread and move on—residual contamination can be controlled.

- RDD:
 - Necessary surgery must be done immediately (36–48 hr), or else delayed 1–2 mo, with exposure > 200 rad.
 - Any bombing victim must be assessed for radioactive contamination until RDD is ruled out by assessment of scene.
 - Preserve evidence for criminal investigation.
- Treat vomiting and dehydration:
 - Antiemetics (ondansetron)
 - IV fluids
- Decorporation agents for internal decontamination are specific to each radionuclide:
 - Contact REAC/TS for guidance (see below).
- Cytokines and transfusions may be needed with doses > 200 rad.
- Potassium iodide:
 - Useful only to prevent thyroid uptake of radioactive iodine (found in nuclear reactors), and only if given within 4 hr after contamination. See www.remm.nlm.gov/potassiumiodide.htm for more information.

MEDICATION

- Ondansetron 8 mg IV (or equivalent 5-HT₃ serotonin antagonist)
- Potassium iodide:
 - Adults: 130 mg PO per day
 - Children:
 - 3–18 yr: 65 mg PO per day
 - 1 mo to 3 yr: 32 mg PO per day
 - <1 mo: 16 mg PO per day



FOLLOW-UP

DISPOSITION

Admission Criteria

- Lymphocyte count < 1,000 at 24–48 hr postexposure
- Lymphocyte count decreased 50% at 24–48 hr
- Suspect acute exposure > 200 rad
- Significant trauma or other illness
- Uncontrolled vomiting
- When in doubt, admit for serial CBC and obtain consultation

Discharge Criteria

- No residual contamination
- No evidence of acute exposure > 100 rad

- Tolerating oral fluids

Issues for Referral

- Internal contamination: Contact REAC/TS for guidance.
- 24-hr emergency number: 865-576-1005
- External contamination that cannot be removed
- Any patient with radiation exposure requires dose assessment and risk counseling.

PEARLS AND PITFALLS

- Emergency medical care takes precedence over decontamination:
 - _ No known case where a live, contaminated patient was so radioactive as to be an immediate hazard to emergency personnel
- Do not underestimate psychological impact of any incident involving “radiation”
- ALC can help differentiate ARS from psychosomatic illness: If vomiting is due to ARS, ALC should be low and falling over 4–8 hr

ADDITIONAL READING

- Centers for Disease Control and Prevention. Emergency Preparedness and Response: Radiation Emergencies. Available at <http://www.bt.cdc.gov/radiation/>.
- National Library of Medicine’s Radiation Emergency Medical Management (REMM) website Online, downloadable, and mobile apps. Available at <http://www.remm.nlm.gov/>.
- Oak Ridge Institute for Science and Education. Radiation Emergency Assistance Center/Training Site (REAC/TS). Available at <http://orise.orau.gov/reacts/>.
- 24-hr emergency radiation injury response line: (865) 576-1005 (ask for REAC/TS).



See Also (Topic, Algorithm, Electronic Media Element)

Chemical Weapons Poisoning



CODES

ICD9

- 508.0 Acute pulmonary manifestations due to radiation
- 990 Effects of radiation, unspecified
- V87.39 Contact with and (suspected) exposure to other potentially hazardous substances

ICD10

- J70.0 Acute pulmonary manifestations due to radiation

- T66.XXXA Radiation sickness, unspecified, initial encounter
- Z77.123 Cntct w & expsr to radon & oth naturally occuring radiation

BASICS

DESCRIPTION

- Morphology, distribution, associated systemic symptoms, and the evolution of a rash are important clinical considerations in identifying a dermatologic emergency
- Presentations of erythroderma, blistering/desquamation, purpura, and skin pain with systemic symptoms are warning signs of a potential emergency
- Abnormal skin lesions due to an inflammatory reaction that can be classified into patterns with distinctive clinical features
- Vesiculobullous lesions:
 - Fluid-filled swelling of the skin or sloughing due to disruption of epidermal/dermal integrity
- Purpura and petechiae:
 - Failure of normal vascular integrity/hemostatic mechanisms
 - Do not blanch on palpation
- Erythema:
 - Erythroderma when covering $\geq 90\%$ of the skin surface
 - Vascular dilatation of the superficial vessels leading to red macular lesions
 - Blanches on palpation
 - Figurate erythema:
 - Erythema classified by its particular annular or arcuate shape
- Papulosquamous:
 - Papules and scaly desquamation of the skin
 - Lesions may also be red and macular
 - Classified into psoriasiform, pityriasiform, lichenoid, annular, and eczematous
- Nodules:
 - Secondary to prolonged inflammatory response, cyst, or infiltrative process
 - Granulomatous lesions:
 - “Apple jelly” appearance when pressed with glass slide

ETIOLOGY/DIFFERENTIAL DIAGNOSES

- Vesiculobullous lesions:
 - Toxic epidermal necrolysis (mucosal and $> 30\%$ body surface area involvement)
 - Stevens–Johnson syndrome (mucosal and $\leq 10\%$ body surface area involvement)
 - Pemphigus vulgaris

- Bullous pemphigoid
- Disseminated herpes simplex
- Herpes zoster
- Varicella
- Smallpox
- Vaccinia
- Allergic contact dermatitis
- Purpura and petechiae:
 - Meningococemia
 - Gonococemia
 - Purpura fulminans/disseminated intravascular coagulopathy (DIC)
 - Rocky Mountain spotted fever (RMSF):
 - Pronounced prodrome of fever, headache, myalgia, rash, peripheral moves to palms/soles
 - Ecthyma gangrenosum:
 - *Pseudomonas* infections in critically ill and immunocompromised patients
 - Babesiosis: Similar to RMSF, rash less often, frequent coinfection with Lyme
 - Vasculitis
 - Multiple systemic illnesses (see chapter on Purpura)
- Erythroderma:
 - Toxic shock syndrome
 - Drug-induced
 - Psoriasis
 - Seborrheic dermatitis
 - Mycosis fungoides
 - Lymphoma of the skin
- Erythematous rashes:
 - Localized:
 - Cellulitis
 - Early necrotizing fasciitis with concomitant skin pain
 - Diffuse:
 - Staphylococcal scalded skin syndrome
 - Toxic shock syndrome
 - Drug-induced, including drug reaction with eosinophilia and systemic symptoms (DRESS)
 - Viral exanthema
- Figurate erythema:
 - Erythema chronicum migrans (large red ring that arises around a tick bite):
 - Lyme disease
 - Erythema multiforme:
 - *Mycoplasma pneumoniae*
 - Herpes simplex

- Drug reaction leading to Steven–Johnson syndrome
- Urticaria:
 - Allergic reaction from drugs, food, infection, pressure, heat, or cold
- Papulosquamous:
 - Psoriasiform:
 - Psoriasis
 - Seborrheic dermatitis
 - Drug-induced
 - Pityriasiform:
 - Pityriasis rosea
 - Secondary syphilis
 - Tinea versicolor
 - Lichenoid:
 - Lichen planus
 - Drug-induced
 - Annular:
 - Tinea
 - Figurate erythema (see below)
 - Eczematous:
 - Atopic dermatitis
 - Allergic contact dermatitis
 - Irritant dermatitis
- Nodules:
 - Granulomatous disease:
 - Sarcoid
 - Granuloma annulare
 - Infectious: Leprosy, tuberculosis, deep fungal infection
 - Panniculitis:
 - Erythema nodosum
 - Lymphoma of the skin
 - Cysts
 - Tumors and metastatic disease

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Age of patient
- Immune status (HIV, chemotherapy, diabetes, steroids)
- Chronologic and physical evolution
- Previous episodes/prior history of lesions/reactions

- Associated symptoms:
 - Pruritus
 - Fever
 - Abdominal pain
 - Myalgias/arthralgias
- Prodromal symptoms:
 - Fever
 - Headache
 - Cough
 - Odynophagia
 - Rhinorrhea
- Environmental exposure:
 - Tick bite
 - Unusual flora
 - Diet
 - Travel
 - Physical trauma (cold, heat, sun)
- Sick contacts
- Recent change in medication
- Family history

Physical-Exam

- Associated signs/symptoms:
 - Fever with infection/drug reaction/systemic inflammatory response
 - Skin pain out of proportion to the clinical picture is a worrisome sign of possible impending skin necrosis
 - Lymphadenopathy may be a symptom of DRESS
 - Pruritus associated with allergic reactions, systemic and contact
- Assess severity of systemic signs:
 - Abnormal vital signs, respiratory distress, hemodynamic instability
- Primary lesion appearance:
 - Vesicles:
 - Small, raised, clear fluid-filled lesions (<5 mm)
 - Bullae:
 - Large, raised, clear fluid-filled lesions (>5 mm)
 - Macule:
 - Nonraised areas of distinct coloration
 - Papule:
 - Raised, palpable lesions <5 mm in diameter, not fluid-filled
 - Pustules:
 - As vesicles and bullae, but containing purulent fluid
 - Nodule:
 - Solid, raised lesion >5 mm seated in deeper layer of skin and tissue

- Distribution of the rash:
 - Characterized as central/peripheral, confluent/scattered, mucosal/nonmucosal, presence of palm/sole involvement
- Secondary changes:
 - Scaling, lichenification, excoriation, fissuring all result from manipulation/scratching or proliferation/shedding of epidermal cells.
 - Erosions/ulcers from varying degrees of tissue loss due to loss of vascular supply/tissue integrity

ESSENTIAL WORKUP

- Identify systemic illness.
- Signs/symptoms of local infectious source
- Categorize the lesion morphology and distribution

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Presence of fever, systemic symptoms, or possible infection warrants blood work:
 - CBC with differential, electrolytes, BUN/creatinine
 - Blood cultures, viral cultures
 - Gram stain and culture of purulent lesions
 - Polymerase chain reaction (PCR) or Direct fluorescent antibody (DFA) of suspected viral lesions
- Rapid plasma reagin (RPR) or fluorescent treponemal antibody (FTA) for suspected syphilis
- Suspected autoimmune disorders:
 - CBC
 - ESR, CRP
 - Particular assays in consultation with a rheumatologist (ANA, antineutrophil cytoplasmic antibody)
- Petechiae/purpura:
 - CBC with platelets
 - Partial thromboplastin time, prothrombin time, INR
 - DIC screen: Fibrinogen, fibrin split products, haptoglobin, LDH
 - Urinalysis for suspected renal involvement in vasculitis

Diagnostic Procedures/Surgery

- In febrile and seriously ill patients, suspected septic lesions may be incised and drained and sent for cultures.
- Nikolsky test: Expansion of bullous lesion with lateral stress at margin indicates epidermal/dermal disruptive process
- Scrapings: Indicated to rule out topical fungal infections and parasites:
 - Potassium hydroxide preparation from edge of lesion reveal hyphae

- Plain mineral oil to rule out scabies in pruritic linear lesions of hands
- Biopsy under dermatologic consultation to differentiate allergic/autoimmune/infectious processes

DIFFERENTIAL DIAGNOSIS

See Etiology.



TREATMENT

PRE HOSPITAL

Universal precautions, masks if infectious etiology suspected

INITIAL STABILIZATION/THERAPY

Aggressive, presumptive management of potentially lethal presentations:

- Petechial lesions
- Disseminated erythematous or vesicobullous lesions
- Purpura with systemic symptoms
- Erythroderma with systemic symptoms

ED TREATMENT/PROCEDURES

- Treatment directed by underlying cause
- Immediate empiric antibiotics targeted toward meningococemia and RMSF in unstable patients with fever and purpura
- Treat disseminated bullous or exfoliative disease as a severe thermal burn.
- Symptomatic treatment of pruritus (diphenhydramine or hydroxyzine)
- Steroid therapy reserved for clear allergic reactions, relapse of known steroid responsive disease, or in consultation with dermatologist
- Allergic reactions:
 - H₁-blocker
 - H₂-blocker
 - Steroids
 - Epinephrine if respiratory compromise

MEDICATION

- Prednisone: 1 mg/kg (max. 60 mg/d)
- Diphenhydramine: 25–50 mg PO/IM/IV q6h
- Hydroxyzine: 25–100 mg PO q6h
- Methylprednisolone: 125 mg IV q24h
- Topical steroids: Classes 3–5 depending on location and severity



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients with significant bullous/exfoliative disorders
- Associated systemic symptoms

Discharge Criteria

- Limited lesions
- Viral exanthems
- Absence of systemic signs or symptoms
- Stable, chronic presentation

Issues for Referral

Discharge to follow up with primary care physician or dermatologist

FOLLOW-UP RECOMMENDATIONS

- Reassure patients that rashes that cannot be diagnosed in the ED are often due to a mild viral illnesses or allergic reactions
- Stress the importance, however, of a follow-up visit with their physician or a dermatologist to obtain the best possible outcome.
- The patient should see his doctor quickly or return to the ED if the condition worsens:
 - _ Spreading redness from the rash
 - _ Increasing pain from the rash
 - _ Joint pain
 - _ Spreading of the rash with crusting
 - _ Fever
 - _ Severe headache
 - _ Confusion
 - _ Signs of a life-threatening allergic reaction:
 - Feeling dizzy or faint
 - Trouble breathing or swallowing
 - Swelling of the tongue

PEARLS AND PITFALLS

- Rapid evolution of a rash with systemic symptoms can indicate a dermatologic emergency
- Treat rapidly with empiric antibiotics in patients with purpura and fever to cover for meningococemia and RMSF.
- Treat rapidly with empiric antibiotics in patients with erythroderma, fever, and hypotension to cover toxic shock syndrome.
- Hyperpigmented scaly papules on the palms and soles require that secondary

syphilis be ruled out.

ADDITIONAL READING

- Brady WJ, DeBehnke D, Crosby DL. Dermatological emergencies. *Am J Emerg Med.* 1994;12(2):217–237.
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- Usatine RP, Sandy N. Dermatologic emergencies. *Am Fam Physician.* 2010;82(7):773–780.



See Also (Topic, Algorithm, Electronic Media Element)

Purpura



CODES

ICD9

782.1 Rash and other nonspecific skin eruption

ICD10

R21 Rash and other nonspecific skin eruption



BASICS

DESCRIPTION

- Lesion morphology:
 - Macule:
 - Localized nonpalpable changes in skin color
 - Purpura or petechiae (nonblanching with pressure)
 - Maculopapule:
 - Slightly elevated lesions with localized changes in skin
 - Papule:
 - Solid, elevated lesions < 5 mm in diameter
 - Keratotic (rough-surfaced lesion)
 - Nonkeratotic (smooth lesion)
 - Palpable purpura (nonblanching with pressure)
 - Plaque:
 - Solid, elevated lesions > 5 mm in diameter
 - Often results from a confluence of papules
 - Nodule:
 - Solid, elevated lesions extending deep into the dermis or SC tissue > 5 mm in diameter
 - Wheal:
 - Circular, irregular lesions varying from red to pale
 - Vesicle:
 - Clear, fluid-filled lesions < 5 mm in diameter
 - Bullae:
 - Clear, fluid-filled lesions > 5 mm in diameter
 - Pustules:
 - Pus-filled lesions
- Secondary lesions:
 - Scales:
 - Thin plates of dried cornified epithelium partially separated from the epidermis
 - Lichenification:
 - Dried plaques resulting in skin furrowing
 - Erosion:
 - Moist surface uncovered by rupture of vesicles or bullae
 - Excoriation:
 - Linear loss of the skin due to trauma

- Ulcer:
 - Deep loss of the skin involving the epidermis and a variable amount of the dermis and SC tissue
- Configuration:
 - Circles or arcs
 - Serpiginous (creeping or worm like)
 - Iris grouping (bull's eye appearance)
 - Irregular grouping
 - Zosteriform grouping
 - Linear grouping
 - Retiform grouping
- The color of a lesion or the entire skin may be due to a number of substances:
 - Red or red-brown lesions result from oxyhemoglobin found in RBCs.
 - The macular erythematous lesions seen in viral exanthema usually represent dilated superficial cutaneous vessels.
 - Purpura and petechiae result from leakage of RBCs out of the vascular space.
 - Hypopigmentation or hyperpigmentation represent postinflammatory change from either increases or decreases in melanin production.
 - Depigmentation refers to the total loss of pigment secondary to autoimmune effect (vitiligo) or congenital disorders (albinism).
- Scales represent a proliferative disorder of epidermal cell turnover.

ETIOLOGY

- Papulosquamous:
 - Infections:
 - Viral or bacterial
 - Rickettsial or fungal
 - Allergic reactions
 - Autoimmune disorders
- Purpura and petechiae:
 - Clotting or platelet disorder
 - Vascular fragility disease
 - Vasculitis
 - Overwhelming infection
- Vesicobullous:
 - Infection
 - Drug reaction
 - Autoimmune disorder
- Ulcer:
 - Infection
 - Vascular insufficiency

DIAGNOSIS

SIGNS AND SYMPTOMS

- Fever (consider infectious exanthemas)
- Pruritus
- Joint pain
- Abdominal pain
- Heart murmur

History

Obtain a detailed history:

- Age group: Conditions, distribution, and appearance may vary with age
- Development, progression, pattern, and duration of the rash
- Lesions synchronous or asynchronous
- Associated symptoms
- Prodromes—cough, rhinorrhea, pharyngitis, fever, meningismal symptoms, pruritus
- Family history, exposures, immunizations
- Recent travel; insect or arthropod bites
- Medications especially new medications
- Generic dermatoses
- Atopic dermatitis; psoriasis

Physical-Exam

- Cardiac:
 - Murmurs/rubs
- Pulmonary:
 - Crackles/wheezing
- Abdominal:
 - Tenderness
 - Hepatosplenomegaly
- Skin: See Essential Workup.

ESSENTIAL WORKUP

Classify the rash based on the primary lesions:

- Papulosquamous
- Vesicobullous
- Purpuric

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Indicated if the rash is purpuric:
 - CBC with platelet count
 - Bleeding screen (prothrombin test, partial thromboplastin time, bleeding time, disseminated intravascular coagulation [DIC] screen)
- Indicated if fever present:
 - CBC
 - Electrolytes, BUN, creatinine to evaluate dehydration and scarlatiniform rash (exclude glomerulonephritis)
 - Viral culture and titers for suspected exanthems
 - Lactate and blood cultures for suspected sepsis/bacteremia
- Lumbar puncture if *meningococcus* or other meningitides or encephalitis suspected

Imaging

Chest radiograph for suspected pulmonary involvement

Diagnostic Procedures/Surgery

- Potassium hydroxide (KOH) preparations:
 - Indicated with scaling lesions to differentiate dermatophytosis from nummular eczema and pityriasis rosea
 - Superficial scale sample from active border of lesion removed from the skin with a scalpel or the edge of a glass slide
 - Place on a slide and add 1 drop of 10% KOH.
 - Place a coverslip and heat slowly without boiling. Allow to set for a few minutes and scan for hyphae.
- Wood lamp:
 - Useful in dermatophytosis and erythrasma
- Scabies preparations:
 - Most of the mite population resides on the hands and feet.
 - Place a drop of mineral oil on the lesion. Scrape with a no. 15 blade to produce speck of blood.
 - Examine under low power for the mite, ova, larva, or fecal matter.

DIFFERENTIAL DIAGNOSIS

Maculopapular Rash

- Solid, skin colored, or yellow:
 - Keratotic
 - Wart
 - Corn or callus
 - Nonkeratotic
 - *Molluscum contagiosum*
 - Sebaceous cyst

- Basal and squamous cell carcinoma
- Nevi
- Solid, brown:
 - Café au lait patch
 - Nevi
 - Freckle
 - Melanoma
 - Photoallergic/phototoxic drug eruption
 - Tinea nigra palmaris hypopigmentation
- Solid, red, nonscaling:
 - Nonpurpuric
 - Exanthems
 - Rubeola, rubella, or roseola
 - Scarlet fever
 - Toxin-producing staphylococcal or streptococcal disease
 - Erythema infectiosum (“fifth disease”)
 - Rubella-like rash (echoviruses, Coxsackie A viruses)
 - Varicella (early manifestations)
 - Variola (smallpox: Early manifestations)
 - Epstein–Barr virus
 - Enterovirus or adenovirus
 - *Mycoplasma*
 - Kawasaki disease
 - Erythema multiforme
 - Localized, pruriginous
 - Insect bites, scabies
 - Allergic or irritant contact dermatitis
 - Purpuric
 - Bacteremia sepsis
 - Meningococemia, pneumococemia, gonococemia, *Haemophilus influenzae*
 - Endocarditis
 - Plague
 - DIC
 - Rocky Mountain spotted fever (RMSF)
 - Henoch–Schönlein purpura
 - Idiopathic thrombocytopenic purpura
 - Leukemia
 - Underlying bleeding disorder
 - Ecthyma gangrenosum
 - Rarely, pityriasis rosea
- Solid, red, scaling:
 - Without epithelial disruption:
 - Tinea corporis, capitis, pedis, or cruris

- Pityriasis rosea
- Secondary syphilis
- Lupus erythematosus
- With epithelial disruption:
 - Papular urticaria
 - Eczema
 - Seborrheic, diaper, contact, or stasis dermatitis
 - Impetigo
 - Candidiasis
 - Tinea corporis, capitis, pedis, or cruris
 - Vesiculobullous rash
 - Herpes virus: Varicella, *variola* (smallpox)
 - Herpes simplex/zoster
 - Hand-foot-and-mouth syndrome
 - Scabies
 - Drug hypersensitivity, toxic epidermal necrolysis
 - Staphylococcal scalded skin syndrome
 - Impetigo, bullous impetigo
 - Cat scratch disease
 - Dermatitis herpetiformis
 - Eczema
 - Erythema multiforme
 - Lichen planus

Pustular

- Acne
- Folliculitis
- Candidiasis
- Gonococemia
- Meningococemia
- Fever present, consider:
 - Infection
 - Drug reaction
 - Systemic inflammatory disease (juvenile rheumatoid arthritis, systemic lupus erythematosus, etc.)



TREATMENT

PRE HOSPITAL

Field management is indicated when there are signs of systemic instability:

- Airway management using precautions to avoid exposure to respiratory secretions; IV access

- Identify rashes with a potentially life-threatening illness or need for special isolation.

INITIAL STABILIZATION/THERAPY

- Aggressive, empiric management of children with a purpuric rash associated with fever or unstable vital signs:
 - Airway support, IV access, fluid resuscitation, pressors if cardiovascular collapse
 - IV antibiotics should be administered for suspected etiologies

ED TREATMENT/PROCEDURES

- Specific ED treatment should be directed to the underlying etiology.
- Diphenhydramine should be used when an allergic reaction is suspected.

MEDICATION

- Acetaminophen: 10–15 mg/kg PO/PR q4–6h; do not exceed 5 doses/24 h
- Cefotaxime: 50 mg/kg IV q6h; max. dose, 12 g/24 h
- Ceftriaxone: 50 mg/kg IV q12h; max. dose, 4 g/24 h
- Diphenhydramine: 1.25 mg/kg PO/IM/IV q6h



FOLLOW-UP

DISPOSITION

Admission Criteria

- Hospital admission is determined by the underlying disorder.
- Other illnesses associated with systemic illness or potential deterioration, SSS, rubeola, and varicella, as well as others, may require inpatient care.

Discharge Criteria

Discharge instructions should be based on the underlying disorder.

Issues for Referral

- Exanthems associated with self-limited entities in stable children.
- Follow-up with primary care physician or dermatologist should be arranged.

FOLLOW-UP RECOMMENDATIONS

Patient should return for re-evaluation for any rapidly spreading rash, changes in rash morphology, petechiae or hemorrhage, new onset fever or neck stiffness.

PEARLS AND PITFALLS

- Note where rash 1st appeared and how it is spreading.

- Note associated signs and symptoms. They are often key for critical illness.
- Keep meningococemia in mind in any rash with fever.

ADDITIONAL READING

- Dermatology atlas: <http://www.dermatlas.org/>.
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- O'Connor NR, McLaughlin MR, Ham, P. Newborn skin: Part I. Common rashes. *Am Fam Physician*. 2008;77:47–52.
- Stocker JT. Clinical and pathologic differential diagnosis of selected potential bioterrorism agents of interest to pediatric health care providers. *Clin Lab Med*. 2006;26:329–344.



See Also (Topic, Algorithm, Electronic Media Element)

- Specific Condition for Management Guidelines
- Resuscitation, Pediatric



CODES

ICD9

- 691.0 Diaper or napkin rash
- 782.1 Rash and other nonspecific skin eruption
- 782.7 Spontaneous ecchymoses

ICD10

- L22 Diaper dermatitis
- R21 Rash and other nonspecific skin eruption
- R23.3 Spontaneous ecchymoses

REACTIVE ARTHRITIS

Christopher M. Fischer

BASICS

DESCRIPTION

- Syndrome classically includes triad of conjunctivitis, urethritis, arthritis
- Also known as “Reiter’s syndrome,” although the eponym has fallen out of favor:
 - Typically taught as the syndrome of “can’t see, can’t pee, can’t climb a tree”

ETIOLOGY

- Exact incidence difficult to determine because of lack of standardized diagnostic criteria
- 2 main types:
 - Postdysentery:
 - Salmonella, Shigella, Campylobacter, Yersinia, *Clostridium difficile*
 - Venereal:
 - *Chlamydia trachomatis*, *Neisseria gonorrhoeae*
- Also described after upper respiratory infections, UTIs, BCG treatment for bladder carcinoma
- M > F (~5:1)
- Peak onset during 3rd decade

DIAGNOSIS

SIGNS AND SYMPTOMS

- Urogenital: Occur in >90% of cases, seen in both forms of disease
- Arthritis, tendonitis:
 - Typically polyarticular, asymmetric
 - Knees and ankles most commonly affected
 - May also affect fingers, back, sacroiliac joints
 - Achilles tendonitis present in 40% of cases
- Ophthalmologic: Occur in 30–60% of cases:
 - Conjunctivitis is most common:
 - Usually bilateral
 - Uveitis, keratitis is less common:
 - Usually unilateral
 - Usually preceded by 1–2 days of eye discomfort
- Mucocutaneous:
 - More common in patients with HLA-B27 positivity

History

- Symptoms generally within 4 wk of infection, although may be delayed up to 1 yr
- Diagnosis made by history and physical exam findings
- Only 1/3 have the complete triad of conjunctivitis, urethritis, arthritis
- Postdysentery: Usually preceded by symptomatic GI infection, especially in children
- Venereal: Often follows asymptomatic infection

Physical-Exam

- General:
 - May include fever, fatigue, weight loss, malaise
- Urogenital:
 - Urethritis
 - Cervicitis
 - Prostatitis
- Extremities:
 - Swelling, painful range of motion, erythema may all be present.
 - Sausage digit (diffuse swelling of an entire digit) present in ~15% of cases
- Ophthalmologic:
 - Conjunctivitis:
 - Often with mucopurulent discharge
 - Symptoms range from mild irritation to severe inflammation.
 - Uveitis:
 - Eye pain, redness, photophobia, miosis, blepharospasm
- Skin/mucosa:
 - Keratoderma blennorrhagicum:
 - Begins as erythematous macules and vesicles on palms and soles, progresses to pustules and dark plaques
 - Similar in appearance to pustular psoriasis
 - Circinate balanitis: Present in >50% of males:
 - Plaques, vesicles or papules on glans penis
 - Ulcerative vulvitis may be associated with vaginal discharge
 - Nail changes, including nail dystrophy, periungual pustules
 - Oral lesions, include ulcerations, geographic tongue, palatal erosions, usually painless

ESSENTIAL WORKUP

- Clinical diagnosis is based on characteristic physical exam findings and a history of GI illness, sexually transmitted infection or upper respiratory infection.
- Must exclude other serious time-sensitive diagnoses that require prompt treatment

DIAGNOSIS TESTS & INTERPRETATION

Lab

No lab tests can confirm the diagnosis:

- CBC may show leukocytosis and mild anemia
- ESR and CRP are usually elevated
- Urinalysis may show sterile pyuria

Imaging

- No radiology exams can confirm the diagnosis
- Plain x-ray can be considered of affected extremities to exclude other diagnoses:
 - _ May show swelling around affected joint, indicating joint effusion

Diagnostic Procedures/Surgery

Arthrocentesis:

- Should be performed if septic arthritis is considered
- Synovial fluid analysis may show leukocytosis, PMN predominance:
 - _ Crystals not present, and indicate other pathologies (gout, pseudogout)

DIFFERENTIAL DIAGNOSIS

- Gonococcal urethritis
- Chlamydial urethritis
- Syphilis
- Gout
- Gonococcal arthritis
- Septic arthritis
- Rheumatoid arthritis
- Pustular psoriasis
- Behçet disease
- Contact dermatitis
- Psoriasis
- Kawasaki disease (in children)



TREATMENT

PRE HOSPITAL

No specific pre-hospital considerations

ED TREATMENT/PROCEDURES

- Once other serious infections have been excluded, treatment is symptomatic
- No consensus about the role of antibiotics
- Rationale for antibiotic treatment is that reactive arthritis is caused by bacterial infection, which may have long-term viability in synovium (especially Chlamydia):
 - _ Studies have demonstrated no long-term benefit with doxycycline,

ciprofloxacin, azithromycin

- Short course of systemic corticosteroids may be helpful in severe or prolonged disease
- Arthritis:
 - Rest, ice, elevation
 - NSAIDs
- Conjunctivitis:
 - Topical antibiotics may provide symptomatic relief
- Urethritis:
 - Should be treated if initial infection not recognized or treated

MEDICATION

No definite role for medication



FOLLOW-UP

DISPOSITION

Admission Criteria

Treatment is generally outpatient, once syndrome is recognized and other diagnoses have been excluded.

Discharge Criteria

Most patients with reactive arthritis can be discharged with follow-up with their primary care provider.

Issues for Referral

Severe uveitis should be referred to ophthalmology for close follow-up.

FOLLOW-UP RECOMMENDATIONS

With primary care provider. Most cases have a prolonged course (3–12 mo), and ~25% may have recurrent episodes.

PEARLS AND PITFALLS

Failing to diagnose serious life- or limb-threatening diseases is a pitfall:

- Septic arthritis
- Gonococcal arthritis
- Kawasaki disease

ADDITIONAL READING

- Carter JD, Hudson AP. Reactive arthritis: Clinical aspects and medical

management. *Rheum Dis Clin North Am.* 2009;35(1):21–44.

- Wu IB, Schwartz RA. Reiter's syndrome: The classic triad and more. *J Am Acad Dermatol.* 2008;59(1):113–121.



See Also (Topic, Algorithm, Electronic Media Element)

- Conjunctivitis
- Iritis/Uveitis
- Kawasaki Disease
- Septic Arthritis
- Urethritis



CODES

ICD9

- 099.3 Reiter's disease
- 372.33 Conjunctivitis in mucocutaneous disease
- 711.10 Arthropathy associated with Reiter's disease and nonspecific urethritis, site unspecified

ICD10

M02.30 Reiter's disease, unspecified site

RECTAL PROLAPSE

Marilyn M. Hallock



BASICS

DESCRIPTION

- Full-thickness evagination of the rectal wall outside the anal opening
- 3 types of rectal prolapse:
 - _ Full-thickness prolapse:
 - Protrusion of the rectal wall through the anal canal; the most common
 - _ Partial thickness or mucosal prolapse:
 - Only mucosal layer protrudes through anus
 - _ Occult (internal) prolapse or rectal intussusception:
 - Rectal wall prolapse without protrusion through the anus
 - May be difficult to diagnose

ETIOLOGY

- Cause unclear and multifactorial:
 - _ Chronic constipation/excessive straining
 - _ Laxity of sphincter:
 - Pelvic floor trauma/weakness; childbearing
 - Neurologic disease
- More common in women, peak in 7th decade

Pediatric Considerations

- Very rare after age 4 yr
- True rectal prolapse unusual in children; more likely partial or intussusception
- Consider chronic diarrhea, parasites, cystic fibrosis (CF), malnutrition as contributing causes



DIAGNOSIS

SIGNS AND SYMPTOMS

- Dark red mass protrudes from the rectum
- Possible mucous or bloody discharge
- Sensation of rectal mass
- Tenesmus
- Constipation or incontinence

History

- History with emphasis on bowel obstruction and duration of prolapse

- Often progressive symptoms over time with self-reducing prolapse initially

Physical-Exam

- Rectal exam must differentiate prolapse from polyps, hemorrhoids, and intussusception.
- True prolapse shows dark red mass at the anal verge with or without mucus; circumferential circular folds in beefy mucosa of protruding rectum.
- Mucosal prolapse rarely greater than a few centimeters of protrusion; will not contain circular folds of muscular layer
- Internal hemorrhoids identified by folds of mucosa radiating out like spokes in wheel
- Prolapsed polyps and hemorrhoids do not involve the entire rectal mucosa and do not have a hole in the center.
- Intussusception identified by complaints of intermittent, severe abdominal pain; may appear more ill:
 - Examiner's finger can be passed between the apex of the prolapsed bowel and the anal sphincter; whereas, in rectal prolapse the protruding mucosa is continuous with the perianal skin

ESSENTIAL WORKUP

Careful physical exam

DIAGNOSIS TESTS & INTERPRETATION

Lab

- No lab test necessary for uncomplicated prolapse
- Preoperative testing for incarcerated rectal prolapse, going to OR

Imaging

No imaging is necessary for uncomplicated prolapse

DIFFERENTIAL DIAGNOSIS

- Prolapsed internal hemorrhoids
- Prolapsed rectal polyp
- Intussusception
- Other rectal mass



TREATMENT

PRE HOSPITAL

- Position of comfort
- Prevent mucosal desiccation with moist gauze

- Avoid trauma to mucosa

INITIAL STABILIZATION/THERAPY

- Stabilization generally not needed in simple prolapse
- Incarcerated or ischemic prolapse:
 - _ NPO
 - _ IV fluids
 - _ Prepare for surgery

ED TREATMENT/PROCEDURES

Manual reduction of rectal prolapse:

- Place in knee-chest position
- Apply gentle steady pressure for 5–15 min
- Invert mucosa through lumen from distal
- Sedation as needed to relax sphincter
- Finger may be placed in rectum to guide reversal of prolapse
- Prolapse very large or difficult to reduce:
 - _ Apply 1/2–1 cup sugar to reduce swelling and assist manual reduction
- Prolapse recurs immediately after reduction:
 - _ Apply pressure dressing with lubricant, gauze, tape; buttock may be taped together for several hours
- If prolapse incarcerated or ischemic, or if manual reduction fails or prolapse frequently recurs:
 - _ Admission for emergent surgical correction

ALERT

- Constriction of blood flow to rectum by anal sphincter can lead to ischemia, venous obstruction and thrombosis, full-thickness necrosis, possible loss of gut
- Timely reduction decreases risk
- Surgical intervention required for ischemic mucosa
- Most common complication of spontaneous or manual reduction:
 - _ Localized pain
 - _ Self-limited mucosal bleeding

MEDICATION

Sedation and pain medication only as needed



FOLLOW-UP

DISPOSITION

Admission Criteria

- Necrotic or ischemic mucosa

- Inability to reduce acute prolapse or frequently recurs

Discharge Criteria

- Reduced rectal prolapse
- Stable and tolerating PO
- Instructions to treat the presumed underlying cause:
 - _ Correct constipation:
 - Stool softeners
 - Increase fluid intake
 - Increase dietary fiber
- Avoid prolonged sitting or straining

Discharge Criteria

Refer for workup including:

- Search for leading lesion
- Refer for definitive surgical repair of recurrent prolapse
- Testing for CF in children

FOLLOW-UP RECOMMENDATIONS

Colorectal follow-up

PEARLS AND PITFALLS

- Perform careful physical exam to differential rectal prolapse from polyps, hemorrhoids, and intussuscepted bowel
- For large or difficult to reduce rectal prolapse, apply sugar to reduce swelling and assist in manual reduction

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Hemorrhoid



CODES

ICD9

569.1 Rectal prolapse

ICD10

K62.3 Rectal prolapse

RECTAL TRAUMA

Stephen R. Hayden

BASICS

DESCRIPTION

- Injury to rectal mucosa
- Simple contusion to full-thickness laceration with extension into peritoneum or perineum
- 2/3 of rectum is extraperitoneal.

ETIOLOGY

- Penetrating trauma:
 - Gunshot wounds: 80% penetrating rectal trauma
 - Knife wounds
 - Impalement injuries
- Blunt trauma:
 - Motor vehicle accidents
 - Waterskiing and watercraft accidents:
 - Hydrostatic pressure injury
 - Pelvic fractures:
 - Bony fragments penetrate rectum
- Foreign body:
 - Autoeroticism
 - Anal intercourse
 - Assault
 - Ingestion of sharp objects
- Iatrogenic trauma: Most common cause of rectal injury:
 - Barium enema:
 - Perforation occurs in 0.04% patients
 - 50% mortality
 - Colonoscopy:
 - 0.2% perforation rate
 - Increased risk with polypectomy
 - Hemorrhoidectomy
 - Urologic and Ob-Gyn procedures:
 - Episiotomy

Pediatric Considerations

- Rectal injury may result from thermometer insertion.
- Any rectal trauma in young children should raise the suspicion of nonaccidental

trauma.



DIAGNOSIS

SIGNS AND SYMPTOMS

- Perineal, anal, or lower abdominal pain
- Signs of perforation or peritonitis:
 - Guarding
 - Rebound tenderness
 - Fever
- Rectal bleeding
- Obstipation
- Presence of pelvic fracture
- History of anal manipulation, foreign-body insertion, sexual abuse

History

- Time and mechanism of injury
- Suspect rectal injury in all patients with gunshot wound, stab wound, or impalement injury to trunk, buttocks, perineum, or upper thigh.
- Consider in any patient with history of anal manipulation complaining of lower abdominal or pelvic pain.

Physical-Exam

- Inspect and palpate thoroughly buttocks, anus, and perineum.
- Identify entrance and exit wounds if penetrating trauma.
- Perform digital rectal exam:
 - Assess for gross blood or guaiac-positive stool
 - Note position of prostate
- Assess perineal integrity:
 - Speculum and bimanual exam in all female patients
 - Thorough genitourinary exam in all male patients, including prostate exam

ESSENTIAL WORKUP

- Labs: CBC, urinalysis
- Acute abdominal series
- CT abdomen and pelvis if blunt trauma
- Sigmoidoscopy: Following extraction of foreign body
- Evidentiary exam: Required in cases of sexual assault

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC:
 - Blood loss
 - Leukocytosis/bandemia suggesting peritonitis
- Type and screen:
 - If evidence of hemorrhage
- Urinalysis:
 - Evaluate for fecal matter

Imaging

- Supine/upright abdominal films, pelvic radiographs:
 - Evaluate for pneumoperitoneum or extraperitoneal and extrarectal densities suggesting perforation.
 - Identify location, size, and shape of foreign body.
 - Identify pelvic fracture or diastasis of symphysis pubis, which may accompany rectal injury.
- CT abdomen and pelvis
 - IV, PO, or PR contrast (gastrografin) per the clinical situation

Diagnostic Procedures/Surgery

- Retrograde urethrogram if high-riding prostate noted on rectal exam
- Contrast enema helpful only in situations where perforation is unclear:
 - Water-soluble contrast (e.g., gastrografin)

DIFFERENTIAL DIAGNOSIS

- Colon injuries
- Genitourinary injuries



TREATMENT

PRE HOSPITAL

- Airway, breathing, and circulation
- Spinal precautions if blunt trauma
- Fluid resuscitation if blood loss, hypotension
- Do not attempt removal of rectal foreign body
- Control bleeding

INITIAL STABILIZATION/THERAPY

Penetrating or blunt abdominal trauma, follow trauma protocols:

- Primary survey
- Resuscitation
- Secondary survey
- Treatment

ED TREATMENT/PROCEDURES

- Tetanus prophylaxis if needed
- Broad-spectrum antibiotics if significant mucosal disruption or signs of peritonitis are present
- Foley catheter (after excluding urethral injury)
- Rectal foreign body removal in ED:
 - _ Determine location and type of foreign object
 - _ Sedation:
 - Avoid sedation if possible; ideally, patient can aid extraction by bearing down during procedure
 - _ With patient in lithotomy position:
 - Local anesthesia to maximize anal sphincter dilation
 - Gentle digital sphincter dilation
 - Obstetric, ring, or biopsy forceps, tenaculum, or suctioning device to aid extraction
 - Suprapubic pressure
 - Patient Valsalva
 - _ Foley catheter:
 - Pass above foreign body, inflate balloon, and apply gentle traction to release suction and permit extraction
 - Using 3 catheters, pass each alongside of foreign body, inflate, and gently pull (helpful for smooth objects or if unable to pass Foley above object)
 - _ Sigmoidoscopy to evaluate mucosal injury following extraction
- Surgical consultation:
 - _ Peritonitis
 - _ All traumatic rectal mucosal lacerations
 - _ Objects > 10 cm from anal verge
 - _ Sharp objects whose removal may provoke mucosal injury
 - _ Inability to extract foreign body in ED

MEDICATION

- Antibiotics with coverage against gram-negative and anaerobic organisms:
 - _ Ampicillin/sulbactam:
 - Adults: 3 g q6h IV (peds: 50 mg/kg IV)
 - _ Cefotetan:
 - Adults: 2 g q12h IV (peds: 40 mg/kg IV)
 - _ Cefoxitin:
 - Adults: 2 g q6h IV (peds: 80 mg/kg q6h IV)
 - _ Piperacillin/tazobactam:
 - Adults: 3.375 g IV (peds: 75 mg/kg IV)
 - _ Ticarcillin/clavulanate:
 - Adults: 3.1 g IV (peds: 75 mg/kg IV)

- Additional anaerobic coverage:
 - Clindamycin:
 - Adults: 600–900 mg IV (peds: 10 mg/kg IV)
 - Metronidazole:
 - Adults: 1 g IV (peds: 15 mg/kg IV)
- Combination therapy:
 - Adults: Ampicillin 500 mg IV q6h, gentamicin 1–1.7 mg/kg IV, and metronidazole 1 g IV
 - Peds: Ampicillin 50 mg/kg IV q6h, gentamicin 1–1.7 mg/kg IV, and metronidazole 15 mg/kg IV
- Sedation and analgesia:
 - Fentanyl: 2–3 µg/kg IV (peds and adults)
 - Midazolam: 0.01–0.2 mg/kg IV (peds and adults)
 - Lidocaine: Topical or injectable

SURGERY/OTHER PROCEDURES

- Perforation
- Torn sphincter
- Foreign body:
 - General anesthesia required to remove high-riding or sharp object
 - Laparotomy is last resort



FOLLOW-UP

DISPOSITION

Admission Criteria

- Perforation
- Significant bleeding
- Unstable vital signs
- Abdominal pain
- Torn anal sphincter
- Foreign body that requires extraction in operating room

Discharge Criteria

- Stable vital signs
- No abdominal pain
- Normal sigmoidoscopy/anoscopy exam

FOLLOW-UP RECOMMENDATIONS

- Repeat abdominal exam 12–24 hr
- Return to ED:

- Abdominal pain
- Vomiting
- Fever

PEARLS AND PITFALLS

- Consider rectal injury in all patients presenting with abdominal pain following lower GI or genitourinary procedure.
- 60% of foreign bodies can be removed in ED.
- Failure to recognize perforation following extraction of foreign body
- Creativity and imagination can aid successful extraction of foreign body in ED.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Abdominal Trauma, Blunt
- Abdominal Trauma, Imaging
- Abdominal Trauma, Penetrating
- Colon Trauma



CODES

ICD9

- 664.30 Fourth-degree perineal laceration, unspecified as to episode of care or not applicable
- 863.45 Injury to rectum, without mention of open wound into cavity
- 863.55 Injury to rectum, with open wound into cavity

ICD10

- O70.3 Fourth degree perineal laceration during delivery
- S36.60XA Unspecified injury of rectum, initial encounter
- S36.63XA Laceration of rectum, initial encounter

RED EYE

Franklin D. Friedman

BASICS

DESCRIPTION

- May be caused by almost any eye disorder
- Often benign; but may represent systemic disease
- Due to vascular engorgement of conjunctiva
- Main causes include inflammatory, allergic, infection, or trauma
- Conjunctivitis is the most common etiology

ETIOLOGY

- Inflammatory:
 - Uveitis:
 - Anterior and posterior
 - Iritis (perilimbal injection)
 - Episcleritis (70% are idiopathic)
 - Scleritis (50% associated with systemic disease)
 - Systemic inflammatory reactions
- Allergic:
 - Due to histamine release and increased vascular permeability, resulting in swelling of conjunctiva (chemosis), watery discharge, and pruritus; usually bilateral
- Infectious:
 - Bacterial (purulent mucous discharge), viral (watery or no discharge), or fungal
 - Orbital cellulitis
 - Dacryocystitis
 - Canaliculitis
 - Endophthalmitis
- Traumatic:
 - Corneal abrasion
 - Subconjunctival hemorrhage (SCH)
 - Foreign body
 - Occult perforation
- Other:
 - Pingueculitis and pterygium, hemorrhage, blepharitis, dry eye syndrome, acute angle-closure glaucoma, ophthalmia neonatorum, conjunctival tumor

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Age (especially neonatal and age > 50 yr)
- Time of onset, duration of symptoms
- Exposures (i.e., chemicals, allergens)
- Patient's occupation (i.e., metal worker)
- Associated signs and symptoms (headache, systemic symptoms, other infections)
- Ocular symptoms:
 - Pain
 - Foreign-body sensation
 - Change in vision
 - Discharge
 - Pruritus
- Contact lens use
- Other comorbidities

Physical-Exam

- Thorough physical exam:
 - Preauricular or submandibular adenopathy
 - Rosacea (may cause blepharitis)
 - Facial or skin lesions (herpes)
- Ophthalmologic:
 - Visual acuity
 - General appearance:
 - Universal eye redness or locally
 - Conjunctival injection
 - Lid involvement
 - Purulent or clear discharge
 - Obvious foreign body
 - Proptosis
 - Photophobia
 - Eyelash against globe (trichiasis)
 - Pupil exam
 - Confrontational visual field exam
 - Extraocular muscle function
 - Slit-lamp exam with fluorescein:
 - Anterior chamber cell or flare
 - Pinpoint or dendritic lesions in HSV
 - Corneal abrasion
 - Foreign body
 - Lid eversion

- Fundoscopy and tonometry

ESSENTIAL WORKUP

- Consider systemic causes of red eye
- Physical exam as described above

DIAGNOSIS TESTS & INTERPRETATION

Tests should be directed toward the suspected etiology of red eye:

- Dacryocystitis: Culture discharge
- Corneal ulcers: Scrape cornea for culture (often is performed by ophthalmologist)
- Bacterial conjunctivitis:
 - Moderate discharge: Obtain conjunctival swab for routine culture and sensitivity (usually *Staphylococcus aureus*, *Streptococcus*, and *Haemophilus influenzae* in unvaccinated children); however, not always needed, as conjunctivitis is often treated presumptively
 - Severe discharge: *Neisseria gonorrhoeae*
 - Note special culture media and procedures depending on suspected etiology (i.e., Thayer–Martin plate for GC)

Pediatric Considerations

- *Chlamydia trachomatis* is the most common neonatal infectious cause of conjunctivitis (monocular or bilateral, purulent or mucopurulent discharge)
- *N. gonorrhoeae* is the other neonatal infectious etiology; typically presents within 2–4 days after birth; marked purulent discharge, chemosis, and lid edema
- Complications may be severe

Lab

- Often not indicated
- Useful if etiology is thought to be systemic disease
- If bilateral, recurrent, granulomatous uveitis is suspected, send CBC, ESR, antinuclear antibody, VDRL, fluorescent treponemal antibody–absorption, purified protein derivative, ACE level, chest x-ray (sarcoidosis and tuberculosis), Lyme titer, and HLA-B27, *Toxoplasma*, and cytomegalovirus (CMV) titers

Imaging

Obtain plain films and/or CT scan of the orbits if suspect foreign body, orbital disease, or trauma

Diagnostic Procedures/Surgery

- Tonometry if glaucoma considered
- Slit-lamp exam with cobalt blue light and fluorescein:
 - Wood lamp exam with fluorescein in young children
- Removal of simple corneal foreign bodies

DIFFERENTIAL DIAGNOSIS

- Local: Infection, allergy, trauma (also see Etiology)
- Acute angle-closure glaucoma
- Systemic (generally an inflammatory reaction):
 - Arthritic disease
 - Ankylosing spondylosis
 - Ulcerative colitis
 - Reiter syndrome
 - TB
 - Herpes
 - Syphilis
 - Sarcoidosis
 - Toxoplasma
 - CMV



TREATMENT

PRE HOSPITAL

- Analgesic and comfort measures
- Initiate irrigation for a chemical exposure

INITIAL STABILIZATION/THERAPY

- Removal of contact lenses if applicable
- Irrigation for chemical insult
- Treat systemic illness if applicable

ED TREATMENT/PROCEDURES

- Direct therapy toward specific etiology
- Medication as indicated
- Special reminders:
 - Differentiate between a corneal abrasion and a corneal ulcer
 - Eye patching is no longer recommended and often contraindicated for abrasions
 - Update tetanus immunization for injury
 - Refrain from contact lens use
 - Do not spread infection to the unaffected eye or to unaffected individuals
 - Diagnosis of conjunctivitis caused by *N. gonorrhoeae* or *C. trachomatis* requires treatment of systemic infection for the individual and the source individual(s)
 - Always include workup and treatment of systemic disease if this is suspected

Special Topics

Corneal Abrasion

- Noncontact lens wearer:
 - Ointment or drops:
 - Erythromycin ointment every 4 hr
 - Polytrim drops 4 times/d
- Contact lens wearers need pseudomonal coverage:
 - Tobramycin, ofloxacin, or ciprofloxacin drops 4 times/d
- Dilate eyes with cyclopentolate 1–2%, 2–4 gtt daily to prevent pain from iritis
- Abrasions will heal without patching
- Systemic analgesics, opiate, or nonopiate
- Re-evaluation if symptomatic at 48 hr

Corneal Ulcer

- Noncontact lens wearer:
 - Polytrim ointment 4 times/d
 - Ofloxacin, ciprofloxacin drops q2–4h
- Contact lens wearers need pseudomonal coverage (see above)

Severe or Vision-threatening Corneal Ulcers

- Central > 1.5 mm or with significant anterior chamber reaction
- Treat as aforementioned and add increased frequency of antibiotic drops such as 1–2 gtt every 15 min for 6 hr, then every 30 min around the clock
- Ophthalmology consult for further recommendations, which may include ciprofloxacin 500 mg PO BID or fortified antibiotic drops made by pharmacist
- Hospitalization is often recommended in consultation with ophthalmologist

Acute Angle-closure Glaucoma

- Symptoms typically include rapid onset, severe eye pain, redness, decreased vision, and pupil in mid-dilation and unreactive
- Other symptoms may include:
 - Nausea and vomiting
 - Headache
 - Blurred vision and/or seeing halos around light
 - Increased tearing
- Diagnosis is further suspected when tonometry detects elevated eye pressure (> 21 mm Hg)

Subconjunctival Hemorrhage

- If large and in the setting of trauma exclude penetrating injury to the globe
- For minor SCH reassure, comfort measures and lubricating drops may speed recovery

Herpes Simplex or Zoster

- Add trifluridine (viroptic) 1%, 2 gtt 9 times/d or vidarabine 3% ointment 5 times/d (ointment preferred for children)
- Ophthalmology consultation

Pediatric Considerations

Herpes infections:

- Usually associated with HSV2 infections
- May be associated with encephalitis or as an isolated lesion
- Neonate onset occurs 1–2 wk after birth
- Presentation: Generally monocular, serous discharge, moderate conjunctival injection

ALERT

Ocular HSV infection carries significant risk of vision loss

Trauma or Uveitis

Rule out foreign body

MEDICATION

- Antibiotic drops:
 - Ciprofloxacin 0.3%: 1–2 gtt q1–6h
 - Gentamicin 0.3%: 1–2 gtt q4h
 - Ofloxacin 0.3%: 1–2 gtt q1–6h
 - Polytrim: 1 gtt q3–6h
 - Sulfacetamide 10%: 0.3% 1–2 gtt q2–6h
 - Tobramycin 0.3%: 1–2 gtt q1–4h
 - Trifluridine 1%: 1 gtt q2–4h
- Antibiotic ointments (ophthalmic):
 - Bacitracin: 500 U/g ½ in ribbon q3–6h
 - Ciprofloxacin 0.3%: ½ in ribbon q6–8h
 - Erythromycin 0.5%: ½ in ribbon q3–6h
 - Gentamicin 0.3%: ½ in ribbon q3–4h
 - Neosporin: ½ in ribbon of ointment q3–4h
 - Polysporin: ½ in ribbon of ointment q3–4h
 - Sulfacetamide 10%: ½ in ribbon of q3–8h
 - Tobramycin 0.3%: ½ in ribbon q3–4h
 - Vidarabine: ½ in ribbon 5 times/d
- Mydriatics and cycloplegics:
 - Atropine 1%, 2%: 1–2 gtt/d to QID
 - Cyclopentolate 0.5%, 1%, 2%: 1–2 gtt PRN
 - Homatropine 2%: 1–2 gtt
 - Phenylephrine 0.12%, 2.5%, 10%: 1–2 gtt BID–TID

- Tropicamide 0.5%, 1%: 1–2 gtt PRN
- Corticosteroid antibiotic combination drops (use only with ophthalmology consultation):
 - Blephamide: 1–2 gtt q1–8h
 - Cortisporin: 1–2 gtt q3–4h
 - Maxitrol: 1–2 gtt q1–8h
 - Pred G: 1–2 gtt q1–8h
 - Tobradex: 1–2 gtt q2–6h
- Glaucoma agents (always use with ophthalmology consultation):
 - Acetazolamide: 250–500 mg PO QD–QID
 - Betaxolol 0.25%, 0.5%: 1–2 gtt BID
 - Carteolol 1%: 1 gtt BID
 - Levobunolol 0.25%, 0.5%: 1 gtt QD–BID
 - Dipivefrin 1%: 1 gtt BID
 - Mannitol: 1–2 g/kg IV over 45 min
 - Pilocarpine 0.25%, 0.5%, 1%, 2%, 3%, 4%, 6%, 8%, 10%: 1–2 gtt TID–QID (use only if mechanical closure is ruled out)
 - Timolol 0.25%, 0.5%: 1 gtt BID



FOLLOW-UP

DISPOSITION

Admission Criteria

- Endophthalmitis
- Perforated corneal ulcers
- Orbital cellulitis
- Concurrent injuries (e.g., trauma)
- If indicated for systemic disease

Pediatric Considerations

Neonates with conjunctivitis suspected to be due to *N. gonorrhoeae* should be hospitalized for IV antibiotics (cefotaxime), and consideration should be given to septic workup

Discharge Criteria

Ability to follow outpatient instructions

Issues for Urgent Referral

- Dacryocystitis
- Corneal ulcer
- Scleritis
- Angle-closure glaucoma

- Uveitis
- Proptosis
- Orbital cellulitis
- Vision loss
- Uncertain diagnosis
- Gonorrheal or chlamydial conjunctivitis

FOLLOW-UP RECOMMENDATIONS

- Prompt re-evaluation if symptoms not resolving over expected time course
- Avoid use of contact lenses until approved by ocular specialist.

PEARLS AND PITFALLS

- Failure to recognize and treat ulcers, herpetic infections, neonatal bacterial infections, angle-closure glaucoma, and penetrating trauma
- Steroids should only be used with ophthalmology consultation

ADDITIONAL READING

- Gerstenblith AT, Rabinowitz MP. *The Wills Eye Manual: Office and Emergency Room Diagnosis and Treatment of Eye Disease*. 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2012.
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See Also (Topic, Algorithm, Electronic Media Element)

- Conjunctivitis
- Corneal Abrasion
- Corneal Burn
- Corneal Foreign Body
- Dacryocystitis
- Glaucoma
- Globe Rupture
- Hordeolum and Chalazion
- Hyphema
- Iritis
- Optic Artery Occlusion

- Optic Neuritis
- Periorbital and Orbital Cellulitis
- Ultraviolet Keratitis
- Visual Loss
- Vitreous Hemorrhage



CODES

ICD9

- 364.3 Unspecified iridocyclitis
- 372.30 Conjunctivitis, unspecified
- 379.93 Redness or discharge of eye

ICD10

- H11.829 Conjunctivochalasis, unspecified eye
- H20.9 Unspecified iridocyclitis
- H57.9 Unspecified disorder of eye and adnexa

RENAL CALCULUS

Matthew A. Wheatley • Ryan A. Stroder



BASICS

DESCRIPTION

- Urinary tract obstruction
- Intermittent distention of the renal pelvis of proximal ureter produces pain
- Kidney stones:
 - Most common cause of renal colic
 - Stone composition:
 - 80%: Calcium stones (calcium oxalate > calcium phosphate)
 - 5% uric acid
 - Others: Magnesium ammonium phosphate (struvite), cystine
 - Associated with infections caused by urea-splitting organisms (e.g., *Pseudomonas*, *Proteus*, *Klebsiella*) along with an alkalotic urine
 - 90% of urinary calculi are radiopaque

ETIOLOGY

- 6–12% lifetime risk in the general population
- Twice as common in men as women
- Peak incidence between 40 and 60 yr old
- Theories on stone formation:
 - Urinary supersaturation of solute followed by crystal precipitation
 - Decrease in the normal urinary proteins inhibiting crystal growth
 - Urinary stasis from a physical anomaly, catheter placement, neurogenic bladder, or the presence of a foreign body
- Recurrence rate of 40% at 5 yr and 75% at 20 yr
- Associated with chronic kidney disease, hypertension, type 2 diabetes mellitus, metabolic syndrome, and an increased risk of coronary artery disease

Pediatric Considerations

- Rare in children
- When present, often is an indication of a metabolic or genetic disorder
- 60% present with flank or abdominal pain though up to 30% only present with hematuria
- Pediatric patients < 16 yr comprise ~7% of all cases of renal stones.
- 1:1 sex distribution
- Causes of stone formation:
 - Metabolic abnormalities (50%)
 - Urologic abnormalities (20%)

- Infection (15%)
- Immobilization syndrome (5%)

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Sudden onset of severe pain in the costovertebral angle, flank, and/or lateral abdomen
- Colicky or constant pain:
 - Patient cannot find a comfortable position
- Hematuria:
 - Gross hematuria in 1/3 of patients
- Nausea/vomiting
- Diaphoresis
- History of prior stone formation

Physical-Exam

- Vital signs:
 - Fever suggests an occult infection.
 - Hypotension with an altered mental status suggests urosepsis
- Abdominal exam:
 - Tenderness to palpation, rebound tenderness, or guarding suggests a more serious intra-abdominal process
 - Palpate the abdominal aorta for tenderness or pulsatile enlargement suggestive of an aneurysm
- Genitourinary exam:
 - Examine the genitalia for evidence of hernia, epididymitis, torsion, or testicular masses

ESSENTIAL WORKUP

- Urinalysis
 - Microscopic hematuria present in > 80%
 - Gross hematuria
 - Absent urinary blood in 10–30%
 - WBC/bacteria suggests infection
 - No correlation between the amount of hematuria and the degree of urinary obstruction

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC:
 - WBC > 15,000 suggests concomitant infection
- Urine culture
- Electrolytes, glucose, BUN, creatinine
- Pregnancy test when suggestive

Imaging

- CT:
 - Helical CT has replaced IV pyelogram (IVP) as test of choice
 - Detects calculi as small as 1 mm in diameter
 - Directly visualizes complications, such as hydroureter, hydronephrosis, and ureteral edema
 - Advantages over IVP:
 - Performed rapidly
 - Does not require IV contrast media
 - Detects other nonurologic causes of symptoms, such as abdominal aortic aneurysms (AAAs)
 - Disadvantages:
 - Does not evaluate flow or renal function
 - Nonenhanced helical CT in the evaluation of renal colic:
 - Sensitivity 95%
 - Specificity 98%
 - Accuracy 97%
 - Indications:
 - 1st-time diagnosis
 - Persistent pain
 - Clinical confusion with pyelonephritis
- IVP:
 - Establishes diagnosis in 95%
 - Demonstrates the severity of obstruction
 - Scout film prior may localize stones that would otherwise be obscured by the dye.
 - Postvoiding film
 - Useful to identify stones at the ureteral vesicular junction or distal ureter that are obscured by a full bladder
- Kidney, ureter, and bladder (KUB) radiograph:
 - Indicated when allergy to IVP dye and when renal scanning and US not available
 - Distinguishes calcium-bearing stones (radiopaque) from noncalcium stones
 - Assists in locating radiopaque stones and the exclusion of other pathologies in nonpregnant patients
 - Difficult to distinguish radiopaque body:

- Phlebolith
- Bowel contents
- Obstruction within the urinary tract on the KUB
- Oblique films assist in localizing suspicious calcifications.
- US:
 - _ Useful in the detection of larger stones and hydronephrosis
 - _ Provides anatomic information only
 - _ Helpful in diagnosing obstruction and localizing stones in the proximal and distal portions of the ureter
 - _ Ability to detect hydronephrosis:
 - Sensitivity 85–94%
 - Specificity 100%
 - _ Limitations:
 - May miss stones <5 mm in size
 - May miss an obstruction in the early phase of renal colic
 - Time delay until the onset of pyelocaliectasis even after total obstruction

Pregnancy Considerations

- Every effort should be made to minimize ionizing radiation exposure to the fetus
- US is the imaging modality of choice

Diagnostic Procedures/Surgery

Ureteroscopy, shock-wave lithotripsy, percutaneous nephrolithotomy

DIFFERENTIAL DIAGNOSIS

- Dissecting or rupturing AAA
- Pyelonephritis
- Papillary necrosis (sickle cell disease, NSAID analgesic abuse, diabetes, or infection)
- Renal infarction (vascular dissection or arterial embolus)
- Ectopic pregnancy
- Ovarian cyst/torsion
- Appendicitis
- Intestinal obstruction
- Biliary tract disease
- Musculoskeletal strain
- Lower lobe pneumonia
- Malingering or narcotic dependence (diagnosis of exclusion)



TREATMENT

PRE HOSPITAL

Parenteral opiates may be required for pain control with long transport times

INITIAL STABILIZATION/THERAPY

- Rapid dipstick urine test for blood:
 - Positive test in conjunction with clinical findings sufficient to begin analgesic therapy
- Provide adequate analgesia when diagnosis suspected on clinical and lab findings

ED TREATMENT/PROCEDURES

- Hydration:
 - Initiate IV crystalloid infusion with 1 L of normal saline infused over 30–60 min followed by 200–500 mL/h
 - Bolus volume compromised patients with 500 mL increments until urine output adequate
- Analgesics (morphine, ketorolac):
 - Combination of IV NSAIDs and opioids decrease ED stay and provide better pain control than either alone
- Antiemetics (prochlorperazine, ondansetron, droperidol, hydroxyzine)
- α -Blockers (tamsulosin) or calcium-channel blockers (nifedipine) have been shown to decrease time to spontaneous stone passage:
 - Most efficacious for stones < 5 mm in diameter
 - Tamsulosin and nifedipine equally effective
 - Prescribe on discharge

Pregnancy Considerations

Avoid NSAIDs in pregnancy, particularly in 3rd trimester

MEDICATION

- Hydromorphone (Dilaudid): 1–4 mg (peds: 0.015 mg/kg/dose) IM/IV/SC q4–6h PRN. Reduce dose in opiate-naive patients.
- Hydroxyzine hydrochloride (Vistaril): 25–50 mg (peds: 0.5–1 mg/kg/dose) IM (not IV) q4–6h
- Ketorolac (Toradol): 30–60 mg IM or 30 mg (peds: 0.5 mg/kg/dose up to 1 mg/kg/24–48 h) IV (alone or with opiates); reduce dose to 30 mg IM or 15 mg IV if > 65 yr or < 50 kg.
- Morphine sulfate: 2–10 mg (peds: 0.1–0.2 mg/kg/dose q2–4h) IM/IV/SC q2–6h PRN; may redose more frequently if needed
- Nifedipine 30 mg PO daily.
- Ondansetron (Zofran): 4 mg (peds: 0.1 mg/kg \times 1) IM/IV, not to exceed 8 mg/dose IV.
- Prochlorperazine (Compazine): 5–10 mg IM/IV q4–6h; 25 mg suppository PR
- Promethazine (Phenergan): 12.5–25 mg (peds: 0.25–1 mg/kg not to exceed 25 mg)

IM/IV/PR q4–6h

- Tamsulosin (Flomax) 0.4 mg PO daily for 4 wk



FOLLOW-UP

DISPOSITION

Admission Criteria

- Obstruction in the presence of infection mandates immediate urologic intervention.
- Intractable pain with refractory nausea and vomiting
- Severe volume depletion
- Urinary extravasation
- Hypercalcemic crisis
- Solitary kidney and complete obstruction
- Relative admission indications (discuss with urologist):
 - High-grade obstruction
 - Renal insufficiency
 - Intrinsic renal disease
 - Stones of size <5 mm usually pass spontaneously; those >8 mm rarely do.

Discharge Criteria

- Normal vital signs
- No evidence of concomitant urinary tract infection
- Adequate analgesia
- Able to tolerate PO fluids to maintain hydration status
- Reliable patient with an adequate home situation
- Appropriate outpatient follow-up arranged
- Normal renal function
- Provide a urine strainer to collect the stone for possible future stone analysis
- Arrange urologic follow-up

Issues for Referral

Imaging if pain persists and diagnosis not established in ED

FOLLOW-UP RECOMMENDATIONS

All patients should have urology follow-up, especially:

- 1st episode of renal stone
- Large stone >5 mm
- Patients who fail to pass a stone after 4 wk of conservative therapy

PEARLS AND PITFALLS

- Do not miss a vascular catastrophe mimicking as renal colic
- Aggressive pain management and hydration promote passage of stones
- The absence of hematuria does not exclude the diagnosis of acute renal colic

ADDITIONAL READING

- Hollingsworth JM, Rogers MA, Kaufman SR, et al. Medical therapy to facilitate stone passage: A meta-analysis. *Lancet*. 2006;368:1171–1179.
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- Teichman JM. Clinical practice. Acute renal colic from ureteral calculus. *N Engl J Med*. 2004;350:684–693.
- Worcester EM, Coe FL. Clinical practice. Calcium kidney stones. *N Engl J Med*. 2010;363(10):954–963.



CODES

ICD9

- 592.0 Calculus of kidney
- 592.1 Calculus of ureter
- 788.0 Renal colic

ICD10

- N20.0 Calculus of kidney
- N20.2 Calculus of kidney with calculus of ureter
- N23 Unspecified renal colic

RENAL FAILURE (ACUTE KIDNEY INJURY)

Michael D. Burg • Matthew N. Graber



BASICS

DESCRIPTION

- The disorder is now known as acute kidney injury (AKI); the term renal failure is outdated.
- Changes in glomerular filtration rate (GFR) and urine output (UO) encompassing a spectrum ranging from normal physiologic response to end-stage renal disease (ESRD) and measured by accumulation of nitrogenous by-products.
- Defined by the RIFLE criteria:
 - 3 stages of renal injury:
 - **Risk**: Increased creatinine (Cr) $\times 1.5$ or GFR decrease $> 25\%$, UO < 0.5 mL/kg/h $\times > 6$ h
 - **Injury**: Increased Cr $\times 2$ or GFR decrease $> 50\%$, UO < 0.5 mL/kg/h $\times > 12$ h
 - **Failure**: Increased Cr $\times 3$ or GFR decrease $> 75\%$ or Cr ≥ 4 mg/dL (acute rise of ≥ 0.5 mg/dL), UO < 0.3 mL/kg/h $\times 24$ h or anuria $\times 12$ h
 - 2 stages of outcome:
 - **Loss**: Loss of renal function > 4 wk
 - **ESRD**: Loss of renal function > 3 mo
- The most severe marker defines stage.
- AKI based upon changes within last 48h; however, must often base on most recent data.
- Higher RIFLE stages correlate with higher 1 and 6 mo mortality rates for hospitalized patients.

ETIOLOGY

- Prerenal AKI:
 - Caused by renal hypoperfusion
 - Renal tissue remains normal unless severe/prolonged hypoperfusion.
- Intrarenal AKI:
 - Caused by diseases of the renal parenchyma
- Iatrogenic AKI causes include:
 - Aminoglycoside antibiotics
 - Radiocontrast material administration
 - NSAIDs
 - ACE inhibitors
 - Angiotensin receptor blockers

- Postrenal AKI:
 - Due to urinary tract obstruction (e.g., prostatic hypertrophy, prostatitis)

DIAGNOSIS

SIGNS AND SYMPTOMS

Acute Kidney Injury

- Often asymptomatic and commonly diagnosed with incidental lab findings
- Oliguria (< 400 mL/d urine production)
- Fluid overload:
 - Dyspnea
 - Hypertension
 - Jugular venous distention
 - Pulmonary and peripheral edema
 - Ascites
 - Pericardial and pleural effusion
- Nausea/vomiting
- Pruritus/skin changes
- Confusion/mental status changes

Prerenal AKI

- Absolute or relative volume deficit
- Dry mucous membranes
- Hypotension
- Tachycardia
- Low cardiac output
- Congestive heart failure
- Systemic vasodilation (e.g., sepsis, anaphylaxis)

Intrinsic AKI

- Allergic Interstitial Nephritis:
 - Fever
 - Rash
 - Recent myocardial infarction
- Renal vein thrombosis:
 - Nephrotic syndrome
 - Can be associated with pulmonary embolus
 - Flank or abdominal pain
- Glomerulonephritis, vasculitis
- Hemolytic uremic syndrome (HUS)
- Thrombotic thrombocytopenic purpura (TTP):

- Mild elevation of BUN/Cr
- Fever
- Altered mental status
- Anemia & thrombocytopenia
- Neurologic: Coma, seizure, headache, altered mental status
- Allergic interstitial nephritis fever:
 - Rash
 - Arthralgias

Postrenal AKI

- Abdominal or flank pain
- Distended bladder
- Oliguria or anuria

Complications of AKI

- Uremic syndrome:
 - Altered mental status
 - Asterixis
 - Reflex abnormalities
 - Focal neurologic abnormality
 - Seizures
 - Restless leg syndrome
 - Pericarditis
 - Pericardial effusion/cardiac tamponade
 - Ileus
 - Platelet dysfunction
 - Pruritus
- Hematologic disorders:
 - Anemia
 - Increased bleeding time & platelet dysfunction
 - Leukocytosis

History

- Prior history of AKI
- Medication history including nephrotoxins
- Weight change

Physical-Exam

- Mental status changes/confusion
- Eyes: Fundoscopy
- CV exam: Jugular venous distention, S3
- Lungs: Rales, crackles

- Abdomen: Flank tenderness, palpable kidneys
- Edema
- Skin changes

Geriatric Considerations

- Prone to prerenal AKI
- Cr will vary by body mass index, so a “normal” range in elderly may represent an elevation.
- Increased risk of contrast- and medication-induced AKI

Pediatric Considerations

- Prerenal AKI a concern in neonates
- Anatomic abnormalities

Pregnancy Considerations

- Intrinsic renal azotemia
- Pre-eclampsia/eclampsia
- Ischemia: Postpartum hemorrhage, abruptio placentae, amniotic fluid embolus
- Direct toxicity of illegal abortifacients
- Postpartum TTP, HUS

ESSENTIAL WORKUP

- Electrolytes including Ca, Mg, PO₄
- BUN/Cr
- Urinalysis (UA):
 - Centrifuged specimen helps to distinguish different etiologies of AKI.
 - Exam for casts, blood, WBCs, and crystals
- Fractional excretion (FE) of Na and/or urea
- CBC: Anemia common with chronic disease
- Postvoid residual volume (> 100 mL suggests obstruction) OR
- Ultrasound to rule out obstruction—especially in older men (e.g., prostatic hypertrophy, prostatitis)
- ECG

DIAGNOSIS TESTS & INTERPRETATION

Lab

Prerenal

UA:

- Specific gravity > 1.018
- Osmolality > 500 mmol/kg
- Sodium < 10 mmol/L

- Hyaline casts
- BUN/Cr ratio > 20
- $FE_{NA} < 1\%$
- Rapid recovery of renal function when renal perfusion normalized

Intrarenal

- BUN/Cr ratio < 10–15
- $FE_{NA} > 2\%$
- Glomerulonephritis, vasculitis:
 - _ UA with red cell or granular casts
 - _ Complement and autoimmune antibodies
- HUS or TTP:
 - _ UA normal
 - _ Anemia
 - _ Thrombocytopenia
 - _ Schistocytes on blood smear
- Nephrotoxic acute tubular necrosis (ATN):
 - _ UA:
 - Brown granular or epithelial cell casts
 - Specific gravity = 1.010
 - Urine osmolality < 350 mmol/kg
 - Urine Na > 20 mmol/L
- Ethylene glycol ingestion:
 - _ UA: Calcium oxalate crystals
 - _ Anion gap metabolic acidosis
 - _ Osmolar gap
- Rhabdomyolysis:
 - _ Elevated serum K^+ , PO_4 , myoglobin, creatine phosphokinase, uric acid
 - _ Decreased serum Ca^{2+}
- Tubulointerstitial disease
- Allergic interstitial nephritis:
 - _ UA with WBC casts, WBCs, RBCs, and proteinuria
 - _ Peripheral eosinophilia

Postrenal

UA:

- Usually normal
- May have some hematuria but no casts or protein
- FE_{NA} often > 4%
- Urine osmolality usually < 350 mmol/kg

Imaging

- US:

- 98% sensitive for excluding obstruction
- Helical CT scan:
 - Without contrast sensitive for obstruction
 - May detect intrarenal changes
- Duplex scan for:
 - Renal artery or vein thrombosis
- Renal arteriogram:
 - Definitive diagnosis of renal artery thrombosis
- Inferior vena cava and renal vessel venogram for renal vein thrombosis
- IV pyelogram

Diagnostic Procedures/Surgery

ECG:

- Hypertension secondary to volume overload may cause ischemia.
- Sensitive for significant, acute electrolyte changes



TREATMENT

PRE HOSPITAL

- Airway, breathing, and circulation (ABCs):
 - Supplemental oxygen for hypoxia
- IV NS for volume depletion

INITIAL STABILIZATION/THERAPY

- ABCs:
- Supplemental oxygen for hypoxia
- IV NS for volume depletion
- Correct electrolyte disturbances
- Indications for emergent dialysis:
 - Intractable hypertension
 - Intractable volume overload
 - Uremic encephalopathy, bleeding, or pericarditis
 - BUN > 100 mg/dL
 - Intractable metabolic acidosis (pH < 7.2)
- Avoid nephrotoxic drugs.
- Monitor UO.

ED TREATMENT/PROCEDURES

Prerenal AKI

- Treat hypoperfusion with IV NS
- Packed RBC for blood loss or anemia after lack of response after 2 boluses

- Invasive cardiac monitoring if unable to assess cardiac failure vs. hypovolemia
- Response to NS good indicator of the degree to which hypovolemia is a factor

ALERT

Administer NS fluid challenge cautiously to avoid fluid overload in liver failure with ascites.

Intrarenal AKI

- Glomerulonephritis:
 - Glucocorticoids or plasma exchange
- ATN:
 - Volume replacement
- Hyponatremia: Free water restriction
- Hyperkalemia:
 - Sodium polystyrene sulfonate (SPS) or calcium polystyrene sulfonate (CPS) for asymptomatic patient with $K^+ > 5.5$ mEq/L
 - For $K^+ > 6.5$ mEq/L or ECG abnormalities consistent with hyperkalemia:
 - Albuterol via nebulizer
 - Dextrose and insulin
 - Furosemide if patient not anuric
 - Calcium stabilizes myocardium in severe hyperkalemia
 - Calcium gluconate for awake patient
 - Calcium chloride for patient without pulse
 - Dialysis for intractable hyperkalemia
- Metabolic acidosis:
 - Consider sodium bicarbonate for $pH < 7.2$ or $HCO_3^- < 15$ mEq/L in *chronic disease*
 - Hyperphosphatemia:
 - Calcium carbonate
 - Aluminum hydroxide
 - Myoglobinuria—aggressive fluid resuscitation with NS

ALERT

- Calcium is only indicated by ECG for widened PR, QT, or QRS intervals. Peaked T waves alone are *not* an indication.
- Sodium bicarbonate is a considerable sodium load; use caution in anuric/oliguric patients.

MEDICATION

- Albuterol: 10–20 mg via nebulizer
- Aluminum hydroxide (amphojel): 0.5–1.5 g PO
- Calcium carbonate (Os-Cal): 0.250–3 g PO

- Calcium gluconate: 10 mL of 10% solution over 5 min IV (may repeat q5min)
- Calcium chloride: 10 mL of 10% solution
- Dextrose: D₅₀W 1 amp (50 mL or 25 g) (peds: D₂₅W 2 mL/kg) IV
- Furosemide: 20–400 mg IV push
- Insulin: 0.1 U/kg regular IV with dextrose (decrease dose by 50% for severe renal and/or liver disease)
- Sodium bicarbonate: 1–2 mEq/kg IV
- SPS (Kayexalate) or CPS: 1 g/kg up to 15–60 g PO or 30–50 g retention enema in sorbitol q6h

ALERT

Diuretics (in the absence of volume overload) and dopamine are not recommended in AKI.



FOLLOW-UP

DISPOSITION

Admission Criteria

- New-onset AKI
- Hyperkalemia/significant electrolyte abnormalities
- Fluid overload with hypoxia/congestive heart failure
- Uremia
- Altered mental status

Discharge Criteria

- Stable
- Normal electrolytes

Issues for Referral

Refer to primary physician for progressive AKI in an otherwise stable patient.

PEARLS AND PITFALLS

- Insulin dose for hyperkalemia should be reduced for significant liver or renal disease so as to avoid hypoglycemia.
- NSAIDs to be avoided with any degree of AKI
- SPS has a considerable sodium load; CPS is preferred when volume overload is a concern.
- Avoid contrast if possible in AKI, as it may worsen renal function.

ADDITIONAL READING

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- Rahman M, Shad F, Smith MC. Acute kidney injury: A guide to diagnosis and management. *Amer Fam Physician.* 2012;86(7):631–639.



See Also (Topic, Algorithm, Electronic Media Element)

- Hyperkalemia
- Renal Injury



CODES

ICD9

- 584.5 Acute kidney failure with lesion of tubular necrosis
- 584.9 Acute kidney failure, unspecified
- 997.5 Urinary complications, not elsewhere classified

ICD10

- N17.0 Acute kidney failure with tubular necrosis
- N17.9 Acute kidney failure, unspecified
- N99.0 Postprocedural (acute) (chronic) kidney failure

RENAL INJURY

Albert S. Jin

BASICS

DESCRIPTION

- Kidneys are located in the retroperitoneal space and are surrounded by adipose tissue and loose areolar connective tissue.
- Kidneys lie along the lower 2 thoracic vertebrae and 1st 4 lumbar vertebrae.
- Left kidney is positioned slightly higher than the right.
- Kidneys are not fixed:
 - Shift with the diaphragm and are supported by the renal arteries, veins, and adipose tissue to the renal (Gerota) fascia

ETIOLOGY

- Most common of all urologic injuries
- Occurs in ~8–10% of all abdominal trauma
- Blunt renal trauma accounts for 80–85% of all renal injuries and is 5 times more common than penetrating injury:
 - Mechanisms include motor vehicle accidents, falls, domestic violence, and contact sports.
 - Pathophysiology includes rapid deceleration and displacement mechanisms.
 - ~20% of cases are associated with intraperitoneal injury.
- Mechanisms responsible for significant renal injury almost never affect the kidney alone:
 - Most often disrupt and injure other vital organs that can be responsible for patient mortality
- Renal injuries are graded by type and severity of injury (Association for the Surgery of Trauma [AAST] criteria)
 - Grade I
 - Contusion: Microscopic or gross hematuria, urologic studies normal
 - Hematoma: Subcapsular, nonexpanding without parenchymal laceration
 - Grade II:
 - Hematoma: Nonexpanding, perirenal hematoma confined to retroperitoneum
 - Laceration: <1 cm parenchymal depth of renal cortex without urinary extravasation
 - Grade III
 - Laceration: >1 cm parenchymal depth of renal cortex without collecting system rupture or urinary extravasation

- Grade IV:
 - Laceration: Parenchymal laceration extending through renal cortex, medulla, and collecting system
 - Vascular: Main renal artery or vein injury with contained hemorrhage
- Grade V:
 - Laceration: Completely shattered kidney
 - Vascular: Avulsion of renal hilum, devascularizing the kidney

Pediatric Considerations

- The kidney is the organ most commonly damaged by blunt abdominal trauma.
- Contributing factors:
 - Relatively larger size of kidneys compared with adults
 - 10th and 11th ribs are not completely ossified until the 3rd decade of life.
- Significant abdominal injury occurs in about 5% of nonaccidental trauma cases but is the 2nd most common cause of death after head injury.



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Mechanism of injury and kinematics are important factors.
- Majority of renal injuries are associated with injury of other abdominal organs.
- In blunt trauma, note the type and direction (horizontal or vertical) of any deceleration or compressive forces.
- In penetrating trauma, note the characteristic of the weapon (type and caliber), distance from the weapon, or the type and length of knife or impaling object:
 - Injuries result from a combination of kinetic energy and shear forces of penetrating object.

Physical-Exam

- Hematuria is the best indicator of traumatic urinary system injury:
 - Severity of renal trauma does not correlate with the degree of hematuria.
 - Absence of hematuria does not exclude renal injury
- Microscopic hematuria with a systolic BP < 90 mm Hg
- Flank mass or ecchymosis
- Tenderness in the flank, abdomen, or back
- Fracture of the inferior ribs or spinal transverse processes
- Nausea and vomiting

ESSENTIAL WORKUP

- In 1989, Mee et al. published the hallmark article (10-yr prospective study) that

established guidelines for the evaluation and treatment of blunt renal trauma:

- Major renal lacerations represent significant reparable renal injuries.
- Adult patients at risk for having sustained major lacerations:
 - Gross hematuria, *or*
 - Microhematuria ($\geq 3\text{--}5$ RBCs/HPF) with shock (systolic BP ≤ 90 mm Hg) in the field or on arrival in the ED, *or*
 - History of sudden deceleration without hematuria or shock
- IV contrast-enhanced CT scan is the procedure of choice in identifying urologic injury.
- Guidelines are not applicable in cases of penetrating renal trauma or in children.
- Adults with blunt renal trauma and gross hematuria, or microhematuria in the presence of shock, require renal imaging for further evaluation of renal injury.
- In adults with penetrating renal trauma, significant injuries to the kidney and ureter can occur without hematuria:
 - Location of penetrating wound in relation to urinary tract is the most important factor in deciding need for radiographic imaging.
 - Penetrating injuries with any degree of hematuria should be imaged.
- Important to rule out coexisting injuries

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Urinalysis: Gross hematuria or > 50 RBCs/HPF in adults and > 20 RBC/HPF in children is suggestive of renal injury.
- Baseline lab values including hematocrit and BUN/creatinine should be obtained.

Imaging

- Plain abdominal films:
 - May show fractured inferior ribs or transverse processes, a unilateral enlarged kidney shadow, or obscuring of the psoas margin
- IV pyelogram (IVP):
 - Bolus infusion IVP with nephrotomography study of choice in institutions without 24-hr availability of CT
 - Rapid injection of 1.5–2 mL of contrast material per kilogram of body weight to a maximum of 150 mL after obtaining a preliminary kidney, ureter, and bladder image
 - Postinfusion supine film is obtained followed by 1-, 2-, and 3-min supine films.
 - Allows evaluation for renal viability and function
 - Extravasation reflects injury to the collecting system.
 - Nonvisualization of a kidney may indicate renal pedicle injury or parenchymal shattering.
 - Abnormal findings are often nonspecific and require more definitive

studies.

- **Ultrasound:**
 - Role in evaluation of renal injury is controversial
 - Routinely performed at bedside in trauma patients as part of focused assessment with sonography in trauma (FAST)
 - May show size of perirenal hematoma and whether it is expanding or resolving
 - Low sensitivity for identification of retroperitoneal free fluid
 - Otherwise, exam is nonspecific and does not provide enough information
- **CT scan:**
 - An IV contrast-enhanced helical CT scan is the diagnostic procedure of choice.
 - Superior anatomic detail and diagnostic accuracy of 98% for renal injury
 - Sensitive indicator of minor extravasation, parenchymal laceration, vascular injury, and nonrenal injuries

Pediatric Considerations

- Major blunt renal trauma can occur in the absence of gross hematuria or shock (as children have a high catecholamine output after trauma, which maintains BP until ~ 50% of blood volume has been lost).
- Meta-analysis has defined 50 RBC/HPF as the microscopic quantity below which imaging can be omitted and no significant injuries missed.
- CT scan is the imaging modality of choice.

Diagnostic Procedures/Surgery

- Renal parenchymal injury
- Renal vascular injury
- Ureteral injury
- Bladder or urethral injury



TREATMENT

PRE HOSPITAL

- Obtain details of injury from pre-hospital providers.
- IV access
- Penetrating wounds or evisceration should be covered with sterile dressings.

INITIAL STABILIZATION/THERAPY

- Airway management (including C-spine immobilization)
- Standard Advanced Trauma Life Support (ATLS) resuscitation measures:
 - Adequate IV access, including central lines and cutdowns, as dictated by the patient's hemodynamic status

- Fluid resuscitation, initially with 2 L of crystalloid (NS or lactated Ringer solution), followed by blood products as needed
- Rule out potential life-threatening injuries 1st.

ED TREATMENT/PROCEDURES

- Immediate laparotomy in the acutely injured patient who is hemodynamically unstable with presumed hemoperitoneum and renal injury
- Significant injuries (grades II–V) are found in only 5.4% of renal trauma cases.
- 98% of blunt renal injuries can be managed nonoperatively.
- ~80–90% of renal injuries have major associated organ injury that can affect the choice of renal injury management.
- Angiography and selective renal embolization has an increasing role and is an alternative treatment to laparotomy in patients not requiring immediate surgery.
- Penetrating renal trauma:
 - Previously exploratory laparotomy was recommended for all patients with penetrating renal injuries.
 - Nonoperative management has become more accepted for grades I–III with penetrating renal injuries in the absence of associated intra-abdominal injury or hemodynamic instability
- Blunt renal trauma:
 - Isolated renal injury without significant associated injuries occurs more commonly from blunt trauma, and in most circumstances, can be managed nonoperatively.
 - Classes I and II: Contusions and minor lacerations with stable vital signs and urographically normal renal function can be managed nonoperatively.
 - Class III: Renal lacerations with urinary extravasation:
 - Controversy between operative vs. nonoperative management
 - Management should be based on degree of injury using CT scanning.
 - Classes IV and V: Shattered kidney or renal pedicle injuries and hemodynamically unstable patients require emergent laparotomy.
 - All ureteral injuries require operative repair.



FOLLOW-UP

DISPOSITION

Admission Criteria

Patients with significant renal injury require hospitalization for definitive laparotomy or observation.

Discharge Criteria

- Adult trauma patients without hematuria, shock, or no renal injury confirmed

radiographically

- Adult blunt trauma patient with microhematuria ($\geq 3\text{--}5$ RBCs/HPF) but no shock (systolic BP ≤ 90 mm Hg)
- Pediatric blunt trauma patient with ≤ 50 RBC/HPF and no other coexisting major organ injuries

Issues for Referral

- Outpatient referral to urologist should be made for microhematuria to ensure that it does not represent a more serious underlying condition.
- Urinoma formation is the most common complication (1–7%) of patients with renal trauma:
 - Urinary extravasation resolves spontaneously in 76–87% of cases

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- Mee SL, McAninch JW, Robinson AL, et al. Radiographic assessment of renal trauma: A 10-year prospective study of patient selection. *J Urol*. 1989;141:1095–1098.
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- Tinkoff G, Esposito TJ, Reed J, et al. American Association for the Surgery of Trauma Organ Injury Scale I: Spleen, liver, and kidney, validation based on the National Trauma Data Bank. *J Am Coll Surg*. 2008;207(5):646–655.



CODES

ICD9

- 866.00 Injury to kidney without mention of open wound into cavity, unspecified injury
- 866.01 Injury to kidney without mention of open wound into cavity, hematoma without rupture of capsule
- 866.02 Injury to kidney without mention of open wound into cavity, laceration

ICD10

- S37.009A Unspecified injury of unspecified kidney, initial encounter
- S37.019A Minor contusion of unspecified kidney, initial encounter
- S37.049A Minor laceration of unspecified kidney, initial encounter

REPERFUSION THERAPY, CARDIAC

Shamai A. Grossman • Joshua J. Solano



BASICS

DESCRIPTION

- Cardiac reperfusion therapy is required on patients that present with ST-segment elevation myocardial infarction (STEMI)
- Early percutaneous coronary intervention (PCI), but not fibrinolytics may be considered in those with unstable angina (UA)/non-ST-segment elevation MI (NSTEMI)
- Fibrinolytic therapy:
 - Reduces morbidity and mortality in STEMI in cases that PCI is not available in < 120 min
 - The earlier fibrinolytics are started, the more myocardium is salvaged
 - Goal of fibrinolytic therapy is a door-to-needle time of 30 min if PCI is not planned or delayed > 120 min
- PCI:
 - Balloon inflation, stent placement, and thrombus removal are possible options in the cath lab and result in overstretching of vessel wall and partial disruption of intima, media, and adventitia, resulting in enlargement of lumen and outer diameter of diseased vessel and restoration of epicardial coronary arterial flow
 - Goal of primary PCI is a door-to-balloon time of 90 min from 1st medical contact for STEMI or < 120 min if at a non-PCI center
 - Stent placement decreases early and late loss in luminal diameter seen with percutaneous transluminal coronary angioplasty (PTCA).
 - PCI provides greater coronary patency and thrombolysis in MI flow than do fibrinolytics and decreased mortality and morbidity
 - Lower risk of bleeding than with fibrinolytics
 - Immediate knowledge of extent of disease
 - PCI should be strongly considered within 1st 48 hr after NSTEMI in discussion with a cardiologist
- Glycoprotein IIb/IIIa inhibitors:
 - Antiplatelet agents that bind to platelet receptor glycoprotein IIb/IIIa and inhibit platelet aggregation
 - Reduce mortality and reinfarction rate in patients in whom PCI is planned; reasonable to administer at time of primary PCI
 - Not indicated for patients with STEMI, unless also undergoing PCI
- Unfractionated heparin (UFH) and low-molecular-weight heparin (LMWH):
 - Adjuncts in treatment with aspirin, clopidogrel, fibrinolytics, glycoprotein

Iib/IIIa inhibitors, and PCI

- Anticoagulant therapy with either UFH or LMWH is indicated in patients with either STEMI (with PCI or fibrinolytics) or UA/NSTEMI
- Clopidogrel or Prasugrel should be added to standard therapy regardless of whether PCI or reperfusion therapy is planned.
- Statin therapy reduces clinical events in patients with stable coronary artery disease. This may also extend to patients experiencing an acute ischemic coronary event
- Post arrest patients may have therapeutic hypothermia initiated in the ED prior to PCI or during PCI

ETIOLOGY

- STEMI is caused by occlusion of an epicardial coronary artery, usually as a result of a thrombotic event
- UA/NSTEMI is caused by a partial occlusion of coronary artery, also due to thrombus.



DIAGNOSIS

SIGNS AND SYMPTOMS

- Chest pain, heaviness, or pressure feeling
- Shortness of breath
- Arm, neck, or back pain
- Weakness or fatigue
- Nausea, vomiting
- Diaphoresis
- Palpitations
- Dizziness or syncope
- STEMI ECG

ESSENTIAL WORKUP

- History is critical in assessing window for use of both fibrinolytics and PCI.
- ECG:
 - Will be normal ~ 50% of time
 - Must be compared with prior tracings if available and may evolve in short period of time, consider repeat ECGs
 - ST elevation in the absence of left ventricular hypertrophy or left bundle branch block (LBBB) with new ST elevation at the J point in at least 2 contiguous leads of ≥ 2 mm in men or ≥ 1.5 mm in women in leads V_2 – V_3 and/or of ≥ 1 mm (0.1 mV) in other contiguous chest leads or the limb leads (7)
 - New or presumably new LBBB has been considered a STEMI equivalent.

Most cases of LBBB at time of presentation; however, are “not known to be old” because prior ECG is not available for comparison.

- New or presumably new LBBB at presentation occurs infrequently, may interfere with ST-elevation analysis, and should not be considered diagnostic of acute myocardial infarction (MI) isolation without symptoms of ACS; use of Sgarbossa criteria is recommended for definitive diagnosis
- Baseline ECG abnormalities other than LBBB (e.g., paced rhythm, LV hypertrophy, Brugada syndrome) may obscure interpretation
- New ST-segment changes or T-wave inversions are suspicious for UA or non-Q-wave infarct
- 1-mm depression of the ST segment below the baseline, 80 ms from the J point, is characteristic of UA or non-Q-wave infarct
- Chest radiograph: May be helpful if aortic dissection is being considered
- Heme stool test: Helpful in establishing baseline, especially in setting of anticipated anticoagulation

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Cardiac enzymes, troponin preferred
- Baseline creatinine, hematocrit, and coagulation profile are all appropriate in initial workup.

DIFFERENTIAL DIAGNOSIS

- Aortic dissection
- Anxiety
- Biliary colic
- Coronary aneurysm
- Costochondritis
- Esophageal spasm
- Esophageal reflux
- Herpes zoster
- Hiatal hernia
- Hyperkalemia
- Mitral valve prolapse
- Peptic ulcer disease
- Psychogenic symptoms
- Panic disorder
- Pericarditis
- Pneumonia
- Pulmonary embolus
- Ventricular aneurysm



TREATMENT

PRE HOSPITAL

- IV access
- Oxygen
- Cardiac monitoring
- Sublingual nitroglycerin for symptom relief, unless use of phosphodiesterase inhibitor in the last 24 hr
- Aspirin 162 or 325 nonenteric coated
- Local EMS system and hospital system should preferentially transport STEMI to PCI-capable hospital
- Controversies:
 - _ Whether to allow EMS activation of cardiac catheterization labs and administration of fibrinolytics.

ALERT

- All chest pain should be treated and transported as a possible life-threatening emergency.
- Therapy with fibrinolytics and glycoprotein IIb/IIIa inhibitors in the field is not currently standard of care.

INITIAL STABILIZATION/THERAPY

- IV access
- Oxygen
- Cardiac monitoring
- Oxygen saturation
- Continuous BP monitoring and pulse oximetry
- Nitrates
- Therapeutic hypothermia if indicated post arrest

ED TREATMENT/PROCEDURES

- Aspirin
- Clopidogrel
- Fibrinolytics for STEMI
 - _ Unless contraindicated
 - _ If PCI is not readily available within 120 min
- PCI is preferred for both diagnostic and therapeutic options for STEMI and UA/NSTEMI
- PCI and fibrinolytics therapy must be used with either UFH or an LMWH, such as enoxaparin or bivalirudin
- LMWH:
 - _ Kinetics more predictable
 - _ Requires no monitoring

- Less potential for platelet activation
- Lower bleeding rate
- Is at least as effective as UFH in treatment of acute coronary syndromes
- Glycoprotein IIb/IIIa inhibitors
- Direct thrombin inhibitors—bivalirudin if history of heparin-induced thrombocytopenia

MEDICATION

- Aspirin: 162–325 mg PO nonenteric coated
- Enoxaparin (Lovenox): 1 mg/kg SC q12h
- Clopidogrel (Plavix): 300–600 mg PO load, 75 mg PO per day
- Prasugrel 60 mg PO load, 10 mg PO per day
- Not to be used in patients with history of stroke
- Ticagrelor 180 mg PO load, 90 mg PO BID
- Glycoprotein IIb/IIIa inhibitor:
 - Abciximab (ReoPro): For use before PCI only; 0.25 mg/kg IV bolus; 0.125 µg/kg/min to a max. of 10 µg/kg/min for 12 hr
 - Eptifibatide (Integrilin): 180 µg/kg IV over 1–2 min, followed by continuous IV infusion of 2 µg/kg/min up to 72 hr
 - Tirofiban (Aggrastat): 0.4 µg/kg/min for 30 min, then 0.1 µg/kg/min for 48–108 hr
- Heparin 60 U/kg IV bolus (max. 4,000 U), then 12 U/kg/h (max. 1,000 U/h)
- Bivalirudin 0.1 mg/kg bolus, followed by 0.25 mg/kg/h for UA/NSTEMI and 0.75 mg/kg bolus, followed by 1.75 mg/kg/h in STEMI
- Metoprolol: 5 mg IV q2min for 3 doses followed by 25–50 mg PO starting dose as tolerated (note: β-blockers contraindicated in cocaine chest pain)
- Fibrinolytics:
 - Recombinant tissue plasminogen activator (Retepase): 10 U IV bolus, repeat dose after 30 min; patients should also receive heparin 5,000 IU IV bolus, then infuse 1,000 IU/h for 48 hr, keeping activated partial thromboplastin time (aPTT) 1.5–2.5.
 - Streptokinase: 1.5 million U over 60 min; patients should also receive methylprednisolone 250 mg IV.
 - Tissue plasminogen activator: 15 mg IV bolus, then 0.75 mg/kg (max. 50 mg) over 30 min, then 0.5 mg/kg (max. 35 mg) over 60 min; patients should also receive heparin 5,000 IU IV bolus, then infuse 1,000 IU/h for 48 hr keeping a PTT 1.5–2.5
 - Tenecteplase: Weight-based dosing with max. single dose of 30–50 mg given over 5 sec; IV bolus over 5 sec
 - Contraindications:
 - Active internal bleeding
 - History of cerebrovascular accident in last 6 mo
 - History of a hemorrhagic cerebrovascular accident

- Recent (within 2 mo) intracranial or intraspinal surgery or trauma
- Intracranial neoplasm, arteriovenous malformation, or aneurysm
- Known bleeding diathesis
- Severe, uncontrolled hypertension
- Pregnancy
- Head trauma within last month
- Trauma or surgery within last 2 wk that may result in closed-space bleed



FOLLOW-UP

DISPOSITION

Admission Criteria

All patients being considered for reperfusion therapy should be admitted to a cath lab or transferred to a PCI center or admitted to tele bed or an ICU setting

Discharge Criteria

No patient being considered for reperfusion therapy should be discharged home from ED

PEARLS AND PITFALLS

- Goal of reperfusion therapy is primary PCI within 90 min of 1st medical contact. Transfer to a PCI-capable facility when this window can be accomplished or assess for fibrinolytics if > 120 min for transfer
- Goal of fibrinolytics therapy is a 30 min door-to-needle time if PCI not possible or will be delayed
- Goal of reperfusion in STEMI patients by either fibrinolytics or PCI is the major goal
- PCI should be considered in all post arrest patients along with hypothermia

ADDITIONAL READING

- American College of Emergency Physicians; Society for Cardiovascular Angiography and Interventions, O’Gara PT, et al. 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction: A report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol*. 2013;61:e78–e140. doi:10.1016/j.jacc.2012.11.019.
- Wright RS, Anderson JL, Adams CD, et al. 2011 ACCF/AHA focused update of the Guidelines for the Management of Patients with UA/Non-ST-Elevation Myocardial Infarction (updating the 2007 guideline): A report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice

Guidelines developed in collaboration with the American College of Emergency Physicians, Society for Cardiovascular Angiography and Interventions, and Society of Thoracic Surgeons. *J Am Coll Cardiol.* 2011;57(19):1920–1959.
doi:10.1016/j.jacc.2011.02.009.



See Also (Topic, Algorithm, Electronic Media Element)

Acute Coronary Syndrome: Myocardial Infarction



CODES

ICD9

410.90 Acute myocardial infarction, unspecified site, episode of care unspecified

ICD10

[I21.3 ST elevation \(STEMI\) myocardial infarction of unspecified site](#)

REPERFUSION THERAPY, CEREBRAL

Kama Guluma



BASICS

DESCRIPTION

- An ischemic cerebrovascular accident (CVA), or stroke, is an acute, sudden or gradual, interruption of regional cerebral blood supply
- Cerebral reperfusion therapy involves:
 - Administration of an IV thrombolytic agent to rapidly dissolve a thromboembolic occlusion
 - Site-specific endovascular intra-arterial thrombolysis
 - Mechanical clot removal

ETIOLOGY

- Thrombotic CVA is from an in situ thrombosis:
 - At an ulcerated atherosclerotic plaque or other prothrombotic endothelial abnormality
 - From hypercoagulable states:
 - Antithrombin III, protein C or S deficiency
 - From sludging:
 - Sickle cell disease
 - Polycythemia vera
- Embolic CVA is caused by acute obstruction by an embolus from:
 - Cardiac mural thrombus formed in:
 - Atrial fibrillation
 - Hypokinetic ventricle (MI, cardiomyopathy)
 - Ventricular aneurysm
 - An abnormal or prosthetic cardiac valve
 - Aortic, carotid, or cerebrovascular atherosclerotic plaques
- Other occlusive events include:
 - Vascular dissection in aorta, cerebral, vertebral, carotid, or innominate arteries
 - Cerebral vasospasm induced by:
 - Subarachnoid hemorrhage (SAH)
 - Vasoconstrictive agents (e.g., cocaine)



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Acute focal neurologic symptoms presenting within 4–5 hr of onset
- Time of symptom onset is critical:
 - If time of onset cannot be firmly established, the time the patient was last known normal should be used as a surrogate
- Historical elements that may suggest an etiology other than routine thromboembolic stroke:
 - Neck injury in carotid or vertebral dissection
 - Tearing back pain in aortic dissection
 - Drug abuse in vasospastic occlusions

Physical-Exam

- Consider reperfusion therapy for symptoms and signs consistent with a distinct vascular supply territory
- Middle cerebral artery:
 - Contralateral hemiplegia and hemisensory deficits (upper > lower)
 - Contralateral homonymous hemianopsia
 - Expressive or receptive aphasia (if in dominant hemisphere)
 - Contralateral neglect
- Posterior cerebral artery:
 - Cortical blindness in half the visual field
 - Visual agnosia (inability to recognize and identify persons and objects)
 - Thalamic syndromes:
 - Abnormal movements (chorea or hemiballismus)
 - Hemisensory deficit
- Vertebrobasilar system:
 - Impaired vision, visual field defects
 - Nystagmus, vertigo, dizziness
 - Facial paresthesia, dysarthria
 - Cranial nerve palsies
 - Contralateral sensory deficits (pain and temperature)
 - Limb ataxia, abnormal gait
- Anterior cerebral artery:
 - Contralateral hemiplegia and hemisensory deficits (lower > upper)
 - Apraxia
 - Confusion, impaired judgment
- Lacunar (deep subcortical):
 - Pure motor hemiplegia (most common), or pure sensory hemiplegia
 - Dysarthria with hand ataxia (clumsy hand), or dysarthria with facial weakness
 - Ataxic hemiparesis
- The National Institutes of Health Stroke Scale (NIHSS) can be used to delineate severity of a CVA as follows (total of subcategory scores):

- 1a. Level of consciousness (LOC): Alert = 0; drowsy = 1; stuporous = 2; coma = 3
- 1b. LOC questions: Answers both correctly = 0; 1 correctly = 1; none correct = 2
- 1c. LOC commands: Obeys both correctly = 0; 1 correctly = 1; none correctly = 2
- 2. Best gaze: Normal = 0; partial gaze palsy = 1; forced deviation = 2
- 3. Visual: No visual loss = 0; partial hemianopia = 1; complete hemianopia = 2; bilateral hemianopia = 3
- 4. Facial palsy: Normal, symmetric = 0; minor paralysis = 1; partial paralysis = 2; complete paralysis = 3
- 5 to 8. Best motor (computed for each arm and leg): No drift = 0; drift = 1; some effort against gravity = 2; no effort against gravity = 3; no movement = 4
- 9. Limb ataxia: Absent = 0; present in 1 limb = 1; present in 2 or more limbs = 2
- 10. Sensory (pinprick): Normal = 0; partial loss = 1; dense loss = 2
- 11. Best language: No aphasia = 0; mild to moderate aphasia = 1; severe aphasia = 2; mute = 3
- 12. Dysarthria: Normal articulation = 0; mild to moderate dysarthria = 1; unintelligible = 2
- 13. Neglect/inattention: No neglect = 0; partial neglect = 1; complete neglect = 2

ESSENTIAL WORKUP

Essential Labs

- Stat bedside blood glucose testing
- CBC, prothrombin time (PT)/partial thromboplastin time (PTT)
 - To assess thrombolytic therapy risk in patients at risk of coagulopathy

Essential Imaging

- Immediate noncontrast head CT scan:
 - Can be part of a multimodal imaging protocol
 - Can reveal other etiologies of symptoms (such as hemorrhage, tumor)
 - Very likely normal in the hours after symptom onset:
 - Early signs of ischemia (e.g., edema) should prompt a re-evaluation of time of onset

DIAGNOSIS TESTS & INTERPRETATION

EKG to assess for dysrhythmia, pericarditis, MI

Additional Labs

- Serum electrolytes, BUN, creatinine
- Urine pregnancy test
- Urine toxicology screen
- Liver function tests in patients prone to liver dysfunction

Additional Imaging

- Multimodal MRI (with perfusion- and diffusion-weighted protocols):
 - Can detect ischemic CVA almost immediately after onset
- Perfusion brain CT can reveal a perfusion deficit immediately after onset
- MR angiography or CT angiography can provide anatomical information
- Carotid US
- CXR

DIFFERENTIAL DIAGNOSIS

- Intracranial hemorrhage (ICH) or SAH
- Seizure
- Complex migraine
- Bell palsy or other focal neuropathies
- Hypoglycemia and other metabolic abnormalities
- Cerebral venous sinus thrombosis
- Intracranial neoplasm
- Intracranial trauma
- Meningitis, encephalitis, or brain abscess
- Vasculitis
- Air embolism or decompression illness
- Spinal cord lesion
- Psychogenic



TREATMENT

PRE HOSPITAL

- Assess for deficits:
 - Dysarthria, facial weakness
 - Arm or leg weakness
- Notify and mobilize ED and hospital resources
- Test blood glucose:
 - Hypoglycemia can mimic a CVA
 - Treat hypoglycemia with dextrose

INITIAL STABILIZATION/THERAPY

- Supplemental oxygen to correct hypoxia (pulse ox < 94%)
- RSI for airway protection or ventilatory insufficiency if needed

- IV access and NS bolus to correct hypotension
- Cardiac monitoring and pulse oximetry

ED TREATMENT/PROCEDURES

- Exclude other diagnoses in the differential
- Thrombolytic therapy should be reserved for thromboembolic ischemic strokes
- Inclusion criteria for IV thrombolytic therapy:
 - _ Age \geq 18 yr of age
 - _ Defined onset of symptoms within 4.5 hr
 - _ No hemorrhage on noncontrast head CT
- Absolute contraindications to IV thrombolytic therapy:
 - _ CVA, serious brain injury, or intracranial surgery within previous 3 mo
 - _ Prior ICH
 - _ Clinical presentation consistent with SAH
 - _ Arterial puncture at noncompressible site in previous 7 days
 - _ Active bleeding on exam
 - _ Uncontrollable HTN $>$ 185/110 mm Hg
 - _ Known bleeding diathesis such as:
 - Platelet count $<$ 100,000/mm³ (if no history of thrombocytopenia, tissue plasminogen activator [tPA] can be initiated before platelet count, but should be discontinued if it is low)
 - Heparin within 48 hr, with elevated aPTT
 - Current anticoagulant use with an INR $>$ 1.7, or PT $>$ 15 sec
 - _ Blood glucose $<$ 50 mg/dL
 - _ Hypodensity in $>$ 1/3 cerebral hemisphere on CT
- Relative contraindications to IV thrombolytics (weigh risk against benefit):
 - _ Major surgery or trauma within previous 14 days
 - _ Mild or resolving neurologic symptoms
 - _ GI or GU bleeding within 21 days
 - _ Seizure at the time stroke was observed
 - _ Acute MI within previous 3 mo
- Treat BP $>$ 185/110 mm Hg with 1–2 doses of labetalol, nicardipine, or other appropriate agent:
 - _ Do not aggressively normalize BP
 - _ Stroke patient may be dependent on an elevated mean arterial pressure for cerebral perfusion
 - _ Avoid thrombolytic therapy if BP cannot be reduced to \leq 180/110 mm Hg with minimal intervention
- Administer IV tPA; alteplase
- Avoid antiplatelet agents and anticoagulants for 24 hr
- Monitor arterial BP during the 1st 24 hr after treatment with tPA and aggressively treat an SBP $>$ 180 mm Hg or a DBP $>$ 105 mm Hg:
 - _ Check BP every 15 min for 2 hr, then every 30 min for 6 hr, then every hour

- for 24 hr
- Keep BP < 180/105 mm Hg using medication such as labetalol or nicardipine
- Consider nitroprusside for HTN unresponsive to labetalol or nicardipine, or for a DBP > 140 mm Hg
- Monitor for signs of ICH:
 - Decreased LOC
 - Increased weakness
 - Headache
 - Acute HTN or tachycardia
 - Nausea or vomiting
- If ICH suspected, obtain an emergent head CT to confirm diagnosis:
 - If present, treat as follows:
 - Discontinue tPA
 - Obtain blood samples for PT, PTT, platelet count, fibrinogen level
 - Prepare cryoprecipitate, fibrinogen, and platelets, and infuse as needed
 - Obtain neurosurgical consultation
- Intra-arterial or mechanical recanalization may be considered for selected patients
 - Though not as well studied as IV tPA, they may be administered out to 6 hr from onset

ALERT

- For patients presenting between 3 and 4.5 hr of onset; there are additional exclusion criteria for IV tPA:
 - Age > 80 yr
 - Oral anticoagulant use (regardless of INR)
 - NIHSS > 25 or > 1/3 MCA territory involved
 - History of previous stroke and diabetes
- There is up to a 6% risk of ICH with tPA that goes up significantly in patients with NIHSS > 20

MEDICATION

First Line

- Alteplase (tPA): 0.9 mg/kg IV, max. 90 mg, over 1 hr:
 - Give 10% of dose as a bolus over 1 min.
 - Immediately follow with the remainder, infused over the subsequent 59 min
- Labetalol: 10 mg IV over 1–2 min; then, if needed:
 - Repeat or double dose q10–20min up to a max. of 300 mg, or
 - Start a drip at 2–8 mg/min
- Nicardipine: 5 mg/h as a drip; titrate upward in 2.5 mg/h increments every 5 min, up to a max. of 15 mg/h

Second Line

- Nitroprusside: 0.5–1 µg/kg/min, continuous IV drip, titrated to BP parameters
- Cryoprecipitate and fibrinogen: 6–8 U IV
- Platelets: 6–8 U IV



FOLLOW-UP

DISPOSITION

Admission Criteria

All patients given reperfusion therapy for a CVA should be admitted to an intensive care setting for frequent neurologic checks and vital sign assessments.

Issues for Referral

Not applicable

PEARLS AND PITFALLS

- Be specific in eliciting time of onset; patient or family may note “time of onset” as the time the stroke was 1st recognized (e.g., upon awakening from sleep)
- tPA has a plasma half-life of <5 min; a delay between bolus and infusion, or pause in the infusion, may result in a decrease in plasma levels and effectiveness
- “Time is brain” (and hemorrhage); initiate treatment as quickly as possible, even if the patient presents early

ADDITIONAL READING

- Jauch EC, Cucchiara B, Adeoye O, et al. Part 11: Adult stroke: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation*. 2010;122:S818–S828.
- Lansberg MG, O’Donnell MJ, Khatri P, et al. Antithrombotic and thrombolytic therapy for ischemic stroke: Antithrombotic Therapy and Prevention of Thrombosis, 9th ed: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines. *Chest*. 2012;141:e601S–e636S.
- Lees KR, Bluhmki E, von Kummer R, et al. Time to treatment with intravenous alteplase and outcome in stroke: An updated pooled analysis of ECASS, ATLANTIS, NINDS, and EPITHET trials. *Lancet*. 2010;375:1695–1703.



See Also (Topic, Algorithm, Electronic Media Element)

- Cerebral Vascular Accident
- Transient Ischemic Attack



CODES

ICD9

- 434.01 Cerebral thrombosis with cerebral infarction
- 434.11 Cerebral embolism with cerebral infarction
- 434.91 Cerebral artery occlusion, unspecified with cerebral infarction

ICD10

- I63.9 Cerebral infarction, unspecified
- I63.30 Cerebral infarction due to thrombosis of cerebral artery
- I63.40 Cerebral infarction due to embolism of cerebral artery

RESPIRATORY DISTRESS

Erik D. Barton • Joy English

BASICS

DESCRIPTION

Respiratory distress, shortness of breath, or dyspnea is a common complaint for patients presenting to the ED.

ETIOLOGY

- Upper airway obstruction:
 - Epiglottitis
 - Croup syndromes
 - Laryngotracheobronchitis
 - Foreign body
 - Angioedema
 - Retropharyngeal abscess
- Cardiovascular:
 - Pulmonary edema/CHF
 - Dysrhythmias
 - Cardiac ischemia
 - Pulmonary embolus
 - Pericarditis
 - Tamponade
 - Air embolism
- Pulmonary:
 - Asthma
 - Chronic obstructive pulmonary disease (COPD)/emphysema
 - Pneumonia
 - Influenza
 - Bronchiolitis
 - Aspiration
 - Adult respiratory distress syndrome (ARDS)
 - Pulmonary edema
 - Pleural effusion
 - Toxic inhalation injury
- Trauma:
 - Pneumothorax
 - Tension pneumothorax
 - Rib fractures
 - Pulmonary contusion

- Fat embolism with long-bone fractures
- Neuromuscular:
 - Guillain–Barré syndrome
 - Myasthenia gravis
- Metabolic/systemic/toxic:
 - Anaphylaxis
 - Anemia
 - Acidosis
 - Hyperthyroidism
 - Sepsis
 - Septic emboli from IV drug use or infected indwelling lines
 - Salicylate intoxication
 - Drug overdose
 - Amphetamines
 - Cocaine
 - Sympathomimetic
 - Obesity
- Psychogenic:
 - Anxiety disorder
 - Hyperventilation syndrome
- Bioterrorist threats:
 - Anthrax
 - Pneumonic plague
 - Tularemia
 - Viral hemorrhagic fevers

Pediatric Considerations

- Respiratory failure is the most common cause of cardiac arrest in infants.
- Croup syndromes include:
 - Viral
 - Spasmodic
 - Bacterial
 - Congenital defects
 - Noninflammatory causes (foreign body, gastroesophageal reflux, trauma, tumors)
- Most common cause of upper airway obstruction:
 - <6 mo: Congenital laryngomalacia
 - >6 mo: Viral croup
- Epiglottitis:
 - Highest incidence at ages 2–4 yr
 - Abrupt onset
 - Fever
 - Respiratory distress and stridor

- Difficulty swallowing oral secretions
- Restlessness and anxiety

Pediatric Considerations

- Amniotic fluid embolism during or after delivery
- Septic embolism from septic abortion or postpartum uterine infection



DIAGNOSIS

SIGNS AND SYMPTOMS

- Tachypnea
- Dyspnea
- Tachycardia
- Anxiety
- Diaphoresis
- Cough (“barking,” productive)
- Stridor
- Hoarse voice
- Difficulty swallowing or handling oral secretions
- Upper airway rhonchi (wheezes)
- Lower airway crackles (rales)
- Increased work of breathing
- Accessory and intercostal muscle use
- Hypoxemia
- Hypocapnia or hypercapnia if severe
- Respiratory acidosis
- Cyanosis
- Lethargy, then obtundation

History

- Previous history of asthma, COPD, cardiac disease, or dysrhythmia, CHF, foreign-body aspiration, or toxic exposure
- Recent fever or upper respiratory tract infection, cough, sputum production, sore throat, systemic disease, anxiety disorder
- Recent chest or long-bone trauma
- IV drug use or indwelling catheters
- Recurrent fevers, night sweats, weight loss

Physical-Exam

- Observe: Mental status, level of distress, work of breathing, jugular venous pressure, skin color
- Feel/palpate: Distal pulses, heart perioperative MI, chest wall, peripheral edema

- Percuss: Lungs for dullness or resonance, abdominal distention, or hepatomegaly
- Auscultate: Heart sounds, murmurs, lung wheezes or crackles, neck for upper airway stridor, abdomen bowel sounds

Pediatric Considerations

- Evaluate retractions, behavior, respiratory rate, breath sounds, and skin color.
- Weak cry, expiratory grunting, nasal flaring, tachypnea and tachycardia, retractions, and cyanosis in neonates

ESSENTIAL WORKUP

- Pulse oximetry
- Cardiac and BP monitoring
- EKG if suspected cardiac etiology

DIAGNOSIS TESTS & INTERPRETATION

Lab

- ABG for severity and acid–base determination
- CBC
- Electrolytes, BUN/creatinine, glucose
- Sputum cultures, smears, and Gram stain
- Blood cultures for fever or sepsis
- B-type natriuretic peptide (BNP) for undifferentiated shortness of breath or CHF severity
- Venous thromboembolus test (VTE) for low-risk PE
- HIV
- Seasonal and “novel” flu testing
- Urinary output monitoring for CHF
- Toxicology screen or salicylate level if suspected

Imaging

- CXR for:
 - Pneumonia
 - Pneumothorax
 - Hyperinflation
 - Atelectasis
 - CHF/pulmonary edema
 - Abscess/cavitary lesions/other infiltrates
 - Tuberculosis
- Ultrasound for:
 - Lung and rib evaluation using *linear transducer*
 - Pneumothorax
 - Hemothorax/pleural effusion

- CHF
- Rib fractures
- Echocardiography using *phased array transducer*:
 - Cardiac effusion/tamponade
 - CHF/cardiac dilatation
 - RV dilatation for PE
- Spirometry (peak expiratory flow rates) for asthma, COPD
- Neck CT or radiographs to assess epiglottis and soft-tissue spaces, foreign body
- CT angiography or ventilation/perfusion scan for pulmonary embolus

Pediatric Considerations

- Chest/neck radiograph may show foreign body or “steeple sign” in croup syndromes.
- Chest fluoroscopy may be used to assess inspiratory and expiratory excursions if foreign body is suspected.

Diagnostic Procedures/Surgery

- Fiberoptic laryngoscopy to assess epiglottis, vocal cords, and pharyngeal space
- Bronchoscopy for foreign body in trachea or bronchus
- Pulmonary artery (Swan-Ganz) catheter for severe CHF, ARDS, pulmonary edema

DIFFERENTIAL DIAGNOSIS

See Etiology.



TREATMENT

PRE HOSPITAL

- Assume a position of comfort for patient.
- 100% oxygen:
 - Assisted bag-valve mask (BMV) ventilation if obtunded
- Airway adjunct devices (oral or nasal) to maintain patency if tolerated
- Intubation for severe respiratory distress
- Needle aspiration of suspected tension pneumothorax

INITIAL STABILIZATION/THERAPY

- ABCs
- Ensure patent airway; BVM assist or intubate for severe distress or arrest
- IV fluids if hypotensive
- 100% oxygen by face mask:
 - Use cautiously in patients with severe COPD or chronic CO₂ retention.
- Monitor BP, heart rate, respirations, pulse oximetry
- Advanced cardiac life support for dysrhythmias or arrest

ED TREATMENT/PROCEDURES

- Treat underlying etiology as appropriate.
- CHF or pulmonary edema:
 - Diuretics
 - Nitroglycerin
 - Nitroprusside if hypertensive
 - Pulmonary artery catheter if severe
 - Noninvasive positive-pressure ventilation (NPPV/BiPAP) or intubation if severe
- Asthma, bronchiolitis, COPD:
 - Bronchodilators
 - Steroids
 - Antibiotics for infection
 - Antivirals for influenza
 - NPPV or intubation if severe
- ARDS, aspiration, toxic lung injury:
 - Mechanical ventilation as needed
 - Steroids controversial
- Pneumonia:
 - Antibiotics
 - Respiratory isolation for TB
- Pneumothorax:
 - Immediate decompression if suspected tension pneumothorax
 - Aspiration or tube thoracostomy (see Pneumothorax)
- Pleural effusion:
 - Determine etiology
 - Diagnostic and symptomatic thoracentesis
- Croup:
 - Cool, misted air or oxygen
 - Steroids
 - Racemic epinephrine
 - Antibiotics for bacterial infection
- Epiglottitis:
 - Immediate airway stabilization with intubation or tracheostomy in OR if possible
 - Antibiotics for *Haemophilus influenzae*
- Anaphylaxis, angioedema:
 - IV steroids
 - H₁/H₂-blockers
 - SQ or IV epinephrine
 - Early intubation
- Retropharyngeal abscess:

- Drainage
- IV antibiotics
- ENT consult
- Cardiac:
 - Treat dysrhythmias or ischemia
 - Anticoagulation or thrombolysis for PE
 - Pericardiocentesis for tamponade
 - NSAIDs or aspirin for pericarditis
- Neuromuscular:
 - Support ventilation
 - Pyridostigmine bromide or neostigmine for myasthenia gravis
- Metabolic/toxic:
 - Treat underlying cause
- Psychogenic:
 - Anxiolytics

Pediatric Considerations

- Transtracheal jet ventilation if unable to intubate (cricothyrotomy not recommended in children < 10 yr)
- Bronchiolitis:
 - Bronchodilators
 - Antivirals for respiratory syncytial virus
 - Antibiotics for infection
- Spasmodic croup:
 - Very sensitive to misted air
- Bacterial croup (membranous laryngotracheobronchitis):
 - Treat *Staphylococcus aureus*.

Pregnancy Considerations

- Supportive oxygen therapy and heparin for PE or amniotic fluid embolism
- IV antibiotics for septic embolism

MEDICATION

Refer to specific etiologies



FOLLOW-UP

DISPOSITION

Admission Criteria

- Continued supplemental oxygen requirement
- Cardiac or hemodynamic instability:

- Requiring IV therapy or hydration
- Requiring close airway observation or repeated treatments
- Respiratory isolation
- As required by underlying cause or significant comorbid disease

Discharge Criteria

- Correction of underlying disease
- Stable airway
- Acute supplemental oxygen not required

Issues for Referral

Refer to specific etiologies

PEARLS AND PITFALLS

- Consider immune-compromised state.
- Consider “novel” flu strains (H1N1).
- Start antibiotic treatment within 6 hr of ED arrival (JCAHO Quality Measure).

ADDITIONAL READING

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- Sigillito RJ, DeBlieux PM. Evaluation and initial management of the patient in respiratory distress. *Emerg Med Clin North Am*. 2003;21(2):239–258.
- Williams SA, Hutson HR, Speals HL. Dyspnea. In: *Emergency Medicine: Concepts and Clinical Practice*. 4th ed. St. Louis, MO: Mosby; 1998:1460–1469.



CODES

ICD9

- 786.00 Respiratory abnormality, unspecified
- 786.05 Shortness of breath
- 786.09 Other respiratory abnormalities

ICD10

- R06.00 Dyspnea, unspecified
- R06.02 Shortness of breath
- R06.09 Other forms of dyspnea

RESUSCITATION, NEONATE

Roger M. Barkin



BASICS

DESCRIPTION

- Annually, almost 1 million deaths worldwide are related to birth asphyxia.
- 10% of newborns require some assistance at birth.
- 1% of newborns require extensive resuscitation.
- Consider NOT initiating resuscitation if:
 - Newborns confirmed to be <23-wk gestation or 400 g
 - Anencephaly
 - Babies with confirmed trisomy 13 or 18
 - Ideally, discuss with family and health care team prior to delivery.
- Activity, pulse, grimace, appearance, respiration (APGAR) scores do not guide resuscitation:
 - Do not wait to assign APGAR scores before starting resuscitation.
 - APGAR scores should NOT guide resuscitative efforts. It is a measure of an infant's status and response to resuscitation.
 - APGAR score: 5 categories with score of 0, 1, or 2 in each at 1 and 5 min
- Heart rate (HR): 0 = absent; 1 = <100 bpm; 2 = >100 bpm
- Respirations: 0 = absent; 1 = slow, irregular; 2 = good, crying
- Muscle tone: 0 = limp; 1 = some flexion; 2 = active motion
- Reflex irritability: 0 = no response; 1 = grimace; 2 = cough, sneeze, cry
- Color: 0 = blue or pale; 1 = pink body and blue extremities; 2 = all pink

ETIOLOGY

- Newborn's transition from dependence on the placenta to dependence on the lungs for oxygen.
- Hypoxia initially causes tachypnea followed by primary apnea.
- Stimulation may cause resumption of breathing during primary apnea.
- Continued hypoxia leads to secondary apnea.
- Secondary apnea requires assisted ventilation.
- Antepartum risk factors associated with need for resuscitation include:
 - Maternal diabetes
 - Pregnancy-induced hypertension
 - Chronic hypertension
 - Anemia
 - Previous fetal or neonatal death
 - Bleeding in 2nd or 3rd trimester
 - Maternal infection

- Maternal cardiac, renal pulmonary, thyroid or neurologic disease
- Polyhydramnios
- Oligohydramnios
- Premature rupture of membranes
- Post-term gestation
- Multiple gestation
- Size–dates discrepancy
- Drug therapy
- Maternal substance abuse
- Fetal malformation
- Diminished fetal activity
- No prenatal care
- Maternal age <16 yr or >35 yr
- Intrapartum risk factors associated with need for resuscitation include:
 - Emergency C-section
 - Forceps or vacuum assist
 - Breech or other abnormal presentation
 - Premature labor
 - Precipitous labor
 - Chorioamnionitis
 - Prolonged rupture of membranes
 - Prolonged 2nd stage of labor
 - Fetal bradycardia
 - Nonreassuring fetal heart tracing
 - General anesthesia
 - Uterine tetany
- Narcotics administered to mother within 4 hr:
 - Meconium-stained amniotic fluid
 - Prolapsed cord
 - Abruption placenta
 - Placenta previa

DIAGNOSIS

SIGNS AND SYMPTOMS

Compromised infants requiring resuscitation may exhibit 1 or more of:

- Decreased muscle tone
- Depressed respiratory drive
- Bradycardia
- Hypotension
- Tachypnea
- Cyanosis

History

Risk factors as above predict the need for resuscitation

Physical-Exam

- Respirations—rate and effectiveness
- HR—by auscultation or palpation of umbilical cord
- Color

ESSENTIAL WORKUP

ABCs:

- Airway
- Breathing
- Circulation
- Drying and warming child

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Bedside blood glucose measurement
- Blood gas

Imaging

Chest radiograph

Diagnostic Procedures/Surgery

- Endotracheal intubation:
 - Straight blades Miller 1 for full term, Miller 0 for preterm
 - Endotracheal tubes (ETTs):
 - 2.5 for <1,000 g or <28 wk
 - 3 for 1,000–2,000 g or 28–34 wk
 - 3.5 for 2,000–3,000 g or 34–38 wk
 - 4 for >3,000 g or >38 wk
 - Have stylet, end-tidal CO₂ detector, suction, tape, meconium aspirator available.
- Umbilical vein catheterization:
 - Tie umbilical tape around base of cord.
 - Prefill syringe attached to umbilical catheter (3.5 or 5F).
 - Cut cord on clean edge below clamp.
 - Identify umbilical vein (large, thin walled, and single).
 - Insert catheter into umbilical vein directed cephalad.
 - Advance 2–4 cm until blood flows freely into syringe.
 - Check position with plain film.

- Inject drugs/fluids as appropriate.



TREATMENT

PRE HOSPITAL

- Resuscitation should be started by pre-hospital personnel.
- Neonatal resuscitation equipment should be available. Anticipation and preparation required.
- Pay particular attention to heat retention and warming.

INITIAL STABILIZATION/THERAPY

- ABCs
- Provide warmth, clear airway, stimulate
- If meconium, poor respiratory effort, poor muscle tone, cyanosis, or prematurity are present, proceed with resuscitation.
- Initial steps include:
 - Warm the baby.
 - Position (neck slightly extended, sniffing position) and clear the airway (meconium may necessitate intubation—see below).
 - Dry thoroughly; stimulate (flick feet, rub trunk or extremities).
 - Provide oxygen:
 - In term infant, room air resuscitation may be advantageous to avoid hyperoxia.
 - In premature infants, blended oxygen with close monitoring of oximetry is appropriate.
- Meconium:
 - Meconium present and baby is NOT vigorous:
 - Insert ETT.
 - Suction with ETT meconium aspiration device.
 - Slowly withdraw tube.
 - Repeat as necessary until little meconium is recovered or HR is maintained.
 - Meconium present and baby is vigorous:
 - Suction mouth then nose with bulb or suction catheter.
- If re-evaluation within 30 sec reveals apnea or HR < 100 bpm, proceed with:
 - Positive-pressure ventilation with 100% oxygen
 - Self-inflating or flow-inflating (anesthesia type) bag
 - Proper-fitting mask
 - 1st breath may require high pressure, necessitating occlusion of “pop-off” valve.
 - Rate of 40–60 breaths/min
 - Pressure of 30–40 cm H₂O

- If prolonged, place nasogastric (NG) tube.
- If re-evaluation after 30 sec of positive-pressure ventilation with 100% oxygen reveals HR < 60 bpm, proceed with:
 - Continued positive-pressure ventilation and chest compressions
 - 2-thumb technique: Hands encircle torso
 - 2-finger technique:
 - Compress ~ 1/3 of the anterior–posterior diameter of chest and release.
- 3 compressions followed by 1 ventilation
- 120 events/min (90 compressions and 30 breaths)
- If after 30 sec HR is > 60 bpm, stop compressions.
- If after 30 sec HR is > 100 bpm, stop positive-pressure ventilator.
- If after 30 sec HR still < 60 bpm, administer epinephrine (IV or via ET tube).

ED TREATMENT/PROCEDURES

- If evidence of blood loss or poor response to resuscitation, administer volume expander.
- NS, lactated Ringer, O-negative blood (cross-matched if time permitting)
- If severe metabolic acidosis is suspected or proven:
 - Ensure adequate ventilation.
 - Administer sodium bicarbonate.
- If hypoglycemia is proven or suspected, treat with IV dextrose.
- If HR and color improve but respiratory effort and tone are poor and mother received narcotics within 4 hr, treat with naloxone hydrochloride:
 - Contraindicated in mothers addicted to narcotics or receiving methadone: Can precipitate seizures.
- Persistent distress may indicate pneumothorax.
- Known or suspected diaphragmatic hernias should be treated with immediate endotracheal intubation and placement of NG tube.
- Consider discontinuation of resuscitation if 10 min of asystole.

MEDICATION

- Dextrose: 2–4 mL/kg of D₁₀W given IV (umbilical vein)
- Epinephrine: 0.1–0.3 mL/kg of 1:10,000 solution, may be given IV or via ETT (0.3–1 mL/kg if giving via ETT)
- Naloxone hydrochloride: 0.1 mg/kg. Administer IV or via ETT; can administer IM or SC, but onset of action is delayed.
- Sodium bicarbonate: 2 mEq/kg (4 mL/kg of 4.2% solution) (0.5 mEq/mL). Administer slowly via IV route (umbilical vein).
- Volume expanders: NS, lactated Ringer, blood. Initial dose 10 mL/kg, may be repeated, all given IV (umbilical vein).
- Other agents as specifically indicated by newborn's underlying condition



FOLLOW-UP

DISPOSITION

Admission Criteria

- All newborns require admission.
- If significant resuscitation is necessary, admit to NCIU.

PEARLS AND PITFALLS

- Resuscitation and care of low-birth-weight infants may lead to the following complications:
 - Difficulty with thermoregulation
 - Intraventricular hemorrhage
 - Chronic lung disease
 - Retinopathy of prematurity
- Oxygen and the very low-birth-weight (VLBW) infant:
 - VLBW infant defined as birth weight < 1,500 g
 - VLBW infants are at increased risk of oxidative stress and damage including retinopathy of prematurity.
 - Some studies suggest resuscitating with < 100% oxygen in this group, possibly even 21% (room air), to avoid oxidative stress and damage.

ADDITIONAL READING

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- Kattwinkel J, Perlman JM, Aziz K, et al. 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation*. 2010;122:S909–S919.
- Kubicka ZJ, Limauro J, Darnall, RA. Heated, humidified high-flow nasal cannula therapy: Yet another way to deliver continuous positive airway pressure? *Pediatrics*. 2008;121:82–88.
- Vaucher YE, Peralta-Carcelen M, Finer NN, et al. Neurodevelopmental outcome in the early CPAP and pulse oximetry trial. *N Engl J Med*. 2012;36:2495–2504.



See Also (Topic, Algorithm, Electronic Media Element)

- Skills may be enhanced with education and practice at a simulation center.
- Resuscitation, Pediatric



CODES

ICD9

- 768.5 Severe birth asphyxia
- 768.6 Mild or moderate birth asphyxia
- 768.9 Unspecified severity of birth asphyxia in liveborn infant

ICD10

P84 Other problems with newborn

RESUSCITATION, PEDIATRIC

Brian Clyne • Seth Gemme



BASICS

DESCRIPTION

Emergent treatment of pediatric patients with imminent or ongoing respiratory or circulatory failure

ETIOLOGY

- Respiratory failure
- Early shock (compensated)
- Late shock (uncompensated)
- Cardiopulmonary arrest
- Respiratory and/or circulatory failure leads to tissue hypoxia, acidosis, and cell death.
- Multisystem organ failure subsequently develops.



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- History from caregivers/parents of onset, progression, inciting, contributing, or predisposing trauma/exposure/conditions, associated findings, past medical history, family history, medications, ingestions
- History of preceding events from pre-hospital personnel
- Respiratory failure:
 - Tachypnea
 - Slow, irregular breathing pattern prearrest
 - Decreased or absent breath sounds; inadequate ventilation
 - Retractions, accessory muscle use, expiratory grunting, nasal flaring
 - Mottled skin, cyanosis
 - Altered level of consciousness: Irritability, agitation, lethargy, weak or absent cry, decreased response to pain
 - Weak or absent cough or gag reflex
 - Most common presenting condition
- Early shock (compensated):
 - Vital signs initially compensated
 - Orthostatic changes or isolated tachycardia
 - Slightly delayed cap refill (> 2 sec)

- Warm, dry skin in early septic shock
- Late shock (uncompensated):
 - Tachycardia, tachypnea, prearrest bradycardia
 - Hypotension, weak peripheral pulses
 - Mottled, pale, cool extremities with markedly decreased capillary refill
 - Poor muscle tone
 - Decreased urine output progressing to anuria
 - Decreased LOC, seizures, coma
 - Fever or hypothermia in septic shock
- Cardiopulmonary arrest:
 - Final common pathway of progressive deterioration of respiratory and circulatory function

Physical-Exam

- Airway assessment:
 - Look, listen, feel for air movement, breath sounds, and chest movement. Observe for stridor or signs of obstruction.
- Breathing assessment:
 - Respiratory rate: Tachypnea or slow/irregular pattern (more ominous)
 - Respiratory effort: Note grunting, nasal flaring, head bobbing, retractions, stridor.
 - Pulse oximetry reflects hemoglobin oxygen saturation, not necessarily oxygen delivery.
 - Auscultation: Assess for wheezing, rales, diminished breath sounds.
- Circulatory assessment:
 - Pulse: Tachycardia or bradycardia (more ominous); orthostatic changes noted easily.
 - BP: Typical SBP in children is 90mm Hg plus twice the age (yrs). Hypotension is a late finding; widened pulse pressure in early septic shock.
 - Peripheral pulse presence and strength (correlates better than BP)
 - Capillary refill: Delayed > 2 sec with poor perfusion
 - Skin: Mottled, pale, or cyanotic
- Mental status assessment:
 - Decreased responsiveness, irritability, confusion, agitation, poor muscle tone, sluggish pupillary response, posturing.
- Complete set of vital signs including rectal temperature, oximetry, and orthostatics when appropriate

ESSENTIAL WORKUP

- ABCDE evaluation:
 - Airway: Assess ability to speak/cry; assess for air movement. Assess for stridor or trauma.
 - Breathing: Observe for nasal flaring, grunting, head bobbing, retractions,

- tracheal deviation, chest injury or pneumothorax; auscultate, apply oxygen.
- Circulation: Evaluate for pulses, capillary refill, mottling, cyanosis.
- Disability: Determine mental status with alert/verbal/painful/unresponsive (AVPU) scale or Glasgow Coma Scale. Assess for neurologic deficits; check stat glucose.
- Exposure/environment: Fully expose for skeletal survey. Prevent hypothermia.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Workup directed by history, assessment of (ABCs), and differential diagnosis
- Arterial blood gas with oximetry to assess oxygenation, ventilation, acid–base status
- Glucose, electrolytes
- Other metabolic/toxicology tests as indicated
- Sepsis evaluation including lumbar puncture, urine and blood cultures as indicated

Imaging

- CXR to evaluate pulmonary or cardiac sources
- Lateral decubitus, inspiratory/expiratory film, or laryngoscopy/bronchoscopy if foreign body (FB) suspected
- ECG
- Echocardiogram
- Cervical spine, other trauma films as indicated
- CT brain for trauma or abnormal neuro exam
- US as indicated

DIFFERENTIAL DIAGNOSIS

- Respiratory:
 - Upper airway obstruction: Croup, epiglottitis, peritonsillar or retropharyngeal abscess, FB, tracheitis, congenital abnormalities
 - Lower airway obstruction: Asthma, pneumonia, bronchiolitis, FB, cystic fibrosis
 - Thoracic trauma, near drowning
- Hypovolemia: Trauma/hemorrhage, diarrhea/vomiting, burns
- Cardiovascular: Congenital/acquired heart disease, myocarditis, pericarditis, CHF, dysrhythmias
- Infectious: Sepsis, meningitis, gastroenteritis, peritonitis, pyelonephritis
- CNS: Status epilepticus, epidural/subdural hematoma
- Metabolic: DKA, hypoglycemia, hyponatremia, hypo/hyperkalemia, acidosis
- Toxicologic: CO poisoning, cardiotoxic agents
- Near sudden infant death syndrome/apparent life-threatening event

- Consider child abuse when history is inconsistent with the illness or pattern of injury.



TREATMENT

PRE HOSPITAL

- Stabilize ABCs; monitor.
- Avoid prolonged on-scene times
- Gather pertinent history from family/bystanders
- Recognize respiratory or circulatory failure; intervene early.
- Recognize impending arrest; support ABCs
- Automated external defibrillator for ventricular fibrillation (VF) and pulseless ventricular tachycardia (VT) in children ≥ 1 yr.
- Early ED notification to allow preparation

INITIAL STABILIZATION/THERAPY

- Early recognition and stabilization of shock
- Glucose, IV, oxygen, cardiac monitoring
- Diagnose and treat immediate life-threats
- Employ Broselow Pediatric Emergency Tape for appropriate drug doses and equipment.

ED TREATMENT/PROCEDURES

- Airway:
 - Secure 1st in every resuscitation.
 - Employ head tilt/chin lift or modified jaw thrust (if trauma suspected).
 - Clear secretions and blood with suction.
 - Temporary stabilization with oral or nasal airway, bag-valve mask assistance
 - Intubation as necessary using appropriate tube size ($[16 + \text{age in years}]/4$) or size similar to patient's little finger or nares
- Rapid-sequence intubation:
 - Preoxygenate
 - Pretreatment: Atropine to prevent bradycardia, lidocaine if head injury
 - Induction agents: Midazolam, thiopental, etomidate (avoid in septic shock), ketamine
 - Paralytics: Rocuronium, vecuronium, pancuronium, succinylcholine
 - Position of endotracheal tube (ETT) at lips (cm) = 3 times diameter of tube (mm)
 - Postintubation: Confirm placement with continuous end-tidal CO₂ monitoring
- Breathing:

- Oxygenate with supplemental O₂, nonrebreather mask; assist ventilation with bag-valve mask or control ventilation if intubation performed.
- Treat conditions that limit ability to oxygenate/ventilate: Pneumothorax, hemothorax, cardiac tamponade, circumferential burns.
- Circulation:
 - Obtain IV, intraosseous (IO), or central access
 - Resuscitate with 0.9% NS or LR bolus at 20 mL/kg; repeat if necessary
 - Control obvious bleeding sources: Apply direct pressure; elevate.
 - Consider transfusion of packed RBCs after crystalloid replacement in trauma.
 - Use pressors early; peripheral use OK
 - Dopamine preferred 1st line; if refractory, norepinephrine (warm shock) or epinephrine (cold shock)
- Cardiopulmonary resuscitation:
 - Provide blood flow to vital organs while restoring spontaneous circulation
 - Infant < 1 yr: Check brachial/femoral pulse
 - Child 1–8 yr: Check carotid pulse
- Cardiac dysrhythmias:
 - Often due to respiratory/metabolic process
 - Treat dysrhythmias per PALS algorithms.
 - Unstable tachydysrhythmias may require adenosine, amiodarone, procainamide, cardioversion, or defibrillation.
 - Unstable bradydysrhythmias may require atropine, epinephrine, or pacing.
 - Pulseless rhythms: VF, pulseless VT, pulseless electrical activity, asystole may require defibrillation, epinephrine, amiodarone, lidocaine.

MEDICATION

- 1st or loading dose unless otherwise noted
- All IV doses may be given IO if necessary
- LEAN (lidocaine, epinephrine, atropine, naloxone) may be given by endotracheal route
- Epinephrine: Multiple uses:
 - Pulseless arrest/symptomatic bradycardia: 0.01 mg/kg 1:10,000 IV q3–5min (max. 1 mg) or 0.1 mg/kg 1:1,000 ETT q3–5min
 - Asthma: 0.01 mg/kg 1:1,000 SC q15min
 - Anaphylaxis: 0.01 mg/kg 1:1,000 IM in thigh q15min (max. 0.3 mg); if hypotensive, 0.01 mg/kg 1:10,000 IV q3–5min (max. 1 mg)
 - Shock/hypotension: 0.1–1 mcg/kg/min IV
 - Toxins/overdose: 0.01 mg/kg 1:10,000 IV; may repeat to max. 0.1 mg/kg 1:1,000 IV.
- Rapid-sequence intubation
 - Pretreatment:
 - Atropine: 0.02 mg/kg IV (min. 0.1 mg)

- Lidocaine: 1–2 mg/kg IV
- Induction:
 - Etomidate: 0.3 mg/kg IV
 - Ketamine: 1–1.5 mg/kg IV; 4–5 mg/kg IM
 - Midazolam: 0.1–0.2 mg/kg IV
 - Thiopental: 3–5 mg/kg IV
- Paralytics:
 - Succinylcholine: 1–2 mg/kg IV
 - Rocuronium: 0.6–1.2 mg/kg IV
 - Vecuronium: 0.1–0.2 mg/kg IV
 - Pancuronium: 0.1 mg/kg IV
- Antiarrhythmic agents:
 - Adenosine: 0.1 mg/kg (max. 6 mg) IV rapid push; 2nd dose 0.2 mg/kg (max. 12 mg).
 - Amiodarone: 5 mg/kg IV, max. dose 300 mg. Give as bolus for pulseless VF/VT, load over 20–60 min for SVT/VT.
 - Lidocaine: For VF or pulseless VT: 1 mg/kg IV bolus, 20–50 ug/kg/min IV infusion
 - Magnesium sulfate: 25–50 mg/kg (max. 2 g) for pulseless VT with torsades de pointes
 - Procainamide: 15 mg/kg IV over 30–60 min
- Inotropes and pressors:
 - Dobutamine: 2–20 ug/kg/min IV
 - Dopamine: 2–20 ug/kg/min IV
 - Inamrinone: Load 0.75–1 mg/kg IV over 5 min; maintenance 5–10 mcg/kg/min
 - Milrinone: Load 50 ug/kg IV over 10–60 min; maintenance 0.25–0.75 ug/kg/min
 - Norepinephrine: 0.1–2 ug/kg/min IV
- Other agents:
 - Albuterol: For asthma or anaphylaxis, multidose inhaler 4–8 puffs q20min or nebulizer 2.5 mg/dose (5 mg/dose if >20 kg) q20min; severe symptoms: 0.5 mg/kg/h by nebulizer (max. 20 mg/h)
 - Alprostadil: 0.05–0.1 ug/kg/min IV for ductal-dependent congenital heart disease
 - Calcium chloride: 20 mg/kg slow IV push in hypocalcemia, hyperkalemia, Ca channel blocker overdose
 - Dexamethasone: 0.6 mg/kg IV (max. 16 mg) for severe croup or asthma
 - Dextrose: 0.5–1 g/kg IV. D₂₅W 2–4 mL/kg or D₁₀W 5–10 mL/kg.
 - Diphenhydramine: 1–2 mg/kg IV q4–6 hr
 - Ipratropium: 250–500 mcg q20min × 3
 - Naloxone: 0.1 mg/kg IV q2min (max. 2 mg)

- Sodium bicarbonate: 1 mEq/kg IV
- Terbutaline: 10 mcg/kg SC q10–15min or 0.1–10 mg/kg/min IV for status asthmaticus
- Cardioversion: 0.5–1 J/kg, increase to 2 J/kg
- Defibrillation: 2 J/kg, increase to 4 J/kg



FOLLOW-UP

DISPOSITION

Admission Criteria

- All patients with impending or ongoing respiratory or cardiovascular compromise
- Survivors of cardiopulmonary arrest require continuous monitoring for decompensation postresuscitation in an ICU setting.
- Consider transfer to pediatric critical care center.

Discharge Criteria

Patients with mild dehydration who respond to fluid resuscitation without signs of hemodynamic instability may be considered for discharge.

Discharge Criteria

- Consultation as appropriate depending on specific etiology
- Involve authorities if abuse is suspected.

FOLLOW-UP RECOMMENDATIONS

- Educate patients, parents, and caregivers regarding household products and toxins
- Educate patients about self-administration of epinephrine in anaphylaxis (if age appropriate).

PEARLS AND PITFALLS

- Empiric treatment is often necessary.
- Be vigilant for signs of early sepsis in children.
- Consider abuse if history contradicts exam
- Early recognition and stabilization

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- Ralston M, Hazinski MF, Zaritsky AL, et al. *Pediatric Advanced Life Support*. Dallas, TX: American Heart Association; 2006.



CODES

ICD9

- 427.5 Cardiac arrest
- 518.81 Acute respiratory failure
- 785.50 Shock, unspecified

ICD10

- I46.9 Cardiac arrest, cause unspecified
- J96.00 Acute respiratory failure, unsp w hypoxia or hypercapnia
- R57.9 Shock, unspecified

RETINAL DETACHMENT

Carl G. Skinner

BASICS

DESCRIPTION

- 3 types of retinal detachments with common final pathway:
 - Rhegmatogenous retinal detachments (RRD)
 - Tractional retinal detachments (TRD)
 - Exudative retinal detachments (ERD)
- RRD:
 - Most common
 - Break or tear of sensory retina allows vitreous fluid to separate the sensory and pigmented parts of retina from each other.
 - Acute event, flashes secondary to tearing of nerve fibers, floaters secondary to bleeding
- TRD:
 - Contraction of fibrous vitreous bands, as a result of previous insult, pulls the sensory retina off the pigmented retina.
 - Chronic and progressive
 - Asymptomatic unless hemorrhage or retinal tear occurs
- ERD:
 - Subretinal fluid accumulates and separate retinal layers without violating either layer.
 - Do not usually require surgery
 - Usually secondary systemic disease such as severe acute hypertension, sarcoid, cancer

ETIOLOGY

- RRD:
 - Myopia
 - Cataract surgery
 - Marfan syndrome
 - Structural degeneration of underlying anatomy of vitreous body, sensory or pigmented retina
 - Trauma
- TRD:
 - Proliferative diabetic retinopathy
 - Vasculopathy
 - Perforating injury
 - Chorioretinitis:

- Retinopathy of prematurity, sickle cell disease, or toxocariasis
- Trauma
- ERD:
 - Malignant hypertension, preeclampsia
 - Tumors of the choroid or retina (melanoma, retinoblastoma)
 - Inflammatory disorders (Coats or Harada disease, posterior scleritis)



DIAGNOSIS

SIGNS AND SYMPTOMS

- Flashes of light
- Floaters
- Curtain-like vision loss
- Peripheral/central vision loss or other visual field defects
- Asymptomatic

History

- Symptoms onset, course, description:
 - May progress over hours or weeks
 - Dark curtain or veil
 - Usually begins peripherally
- Associated symptoms: Flashing lights, floaters, painless
- Ophthalmologic history:
 - Baseline eyesight, myopia, surgery, eye disease, trauma
- Systemic disease

Physical-Exam

- Visual acuity, visual fields by confrontation—prior to dilation:
 - May have normal visual acuity if macula spared
 - Detachment is on opposite side of field defect
- May have afferent pupillary defect
- May have loss of red reflex
- Fundoscopy:
 - Pale, opaque, wrinkled retina
 - Cannot rule out detachment on fundoscopy alone
- Slit-lamp exam: Anterior vitreous pigment granules (“tobacco dust”) suggest retinal tear.

ESSENTIAL WORKUP

- Complete ophthalmologic exam
- Thorough neurologic exam to exclude cerebrovascular accident/transient ischemic attack

DIAGNOSIS TESTS & INTERPRETATION

Lab

As needed to work up underlying diseases

Imaging

Ocular US: ~97% sensitive by trained EM physicians

Diagnostic Procedures/Surgery

- Intraocular pressure (IOP) measurement: IOP usually lower in the affected eye
- Dilating pupil with short-acting mydriatic carries very low risk of acute angle-closure glaucoma.

DIFFERENTIAL DIAGNOSIS

- Central retinal artery or vein occlusion
- Vitreous hemorrhage
- Migraine with or without aura
- Choroidal detachment
- Methanol poisoning
- Other retinal or CNS disease



TREATMENT

PRE HOSPITAL

- Bed rest
- Consider transport to hospital with neurology and ophthalmology availability.

INITIAL STABILIZATION/THERAPY

If suspected ERD, treat systemic disease.

ED TREATMENT/PROCEDURES

- Bed rest:
 - Rest head on pillow with side of detachment down, side opposite of field defect
- Emergent ophthalmologic consultation



FOLLOW-UP

DISPOSITION

Admission Criteria

Need for surgical repair

Discharge Criteria

- Any patient with retinal detachment seen by an ophthalmologist and deemed safe to go home
- Chronic retinal detachments are repaired over the same time course as it took to create them.
- ERD resolves with treatment of the underlying problem.

Issues for Referral

Detachments with macula involvement require repair within 1 day.

FOLLOW-UP RECOMMENDATIONS

Per ophthalmologist

PEARLS AND PITFALLS

- Fundoscopy alone does not provide sufficient visualization to rule out detachment.
- Early recognition of retinal tears allows possible prophylactic:
 - 90% risk of retinal tear with “tobacco dust”
- Do not fail to recognize central retinal artery occlusion (CRAO):
 - Increased risk of stroke for patient with CRAO in setting of carotid disease or cardioembolic disease

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Visual Loss
- Vitreous Hemorrhage



CODES

ICD9

- 361.00 Retinal detachment with retinal defect, unspecified

- 361.81 Traction detachment of retina
- 361.9 Unspecified retinal detachment

ICD10

- H33.009 Unsp retinal detachment with retinal break, unspecified eye
- H33.20 Serous retinal detachment, unspecified eye
- H33.40 Traction detachment of retina, unspecified eye

RETRO-ORBITAL HEMATOMA

Chao Annie Yuan • Michael J. Holman



BASICS

DESCRIPTION

- Also known as retrobulbar hematoma
- Rare complication of orbital trauma and facial surgery
- Collection of blood behind the globe causing increased retro-orbital pressure leading to tissue ischemia
- Vision loss can occur within 90 min if not diagnosed and treated with irreversible damage at 120 min
- A sight-saving procedure called lateral canthotomy is often needed to be performed in the emergency department
- A thorough exam is needed as many patients with ROH may be unconscious
- Frequent repeat exams are mandatory due to hematoma progression

EPIDEMIOLOGY

- Incidence is difficult to estimate because ROH can be from multiple causes, both traumatic and iatrogenic.
 - _ 0.45–3% of blunt or penetrating trauma
 - _ 0.45–0.6% coexist with orbital wall fractures
 - _ 0.0052% of blepharoplasty
 - _ 0.3% of surgical facial fracture repair
 - _ 0.006% of endoscopic sinus surgery
- True incidence has been debated as only slightly more than half of diagnosed retro-orbital hemorrhage has been confirmed either with a preceding CT scan or with the presence of an evacuated hematoma.

ETIOLOGY

- Trauma to the globe or orbital walls and the orbital plexus
- Rapid increasing pressure behind the orbit secondary to hematoma formation impedes venous outflow and arterial inflow to the retina and the optic nerve to cause orbital compartment syndrome
- There may also be a stretching to the optic nerve as the patient develops proptosis which contributes to the decrease in visual acuity



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Penetrating or blunt trauma to the orbit
- Recent facial/orbital surgery
- Eye pain
- Vision loss

Physical-Exam

- Decreased visual acuity
- Increased IOP
- Proptosis
- Diplopia
- Pain
- Decreased EOM
- Relative afferent papillary defect, preserved consensual reflex

ESSENTIAL WORKUP

- Obtain history of injury
- High degree of suspicion
- Thorough physical exam
- Evaluate for immediate surgical decompression
- STAT ophthalmology consult
- Imaging

DIAGNOSIS TESTS & INTERPRETATION

Lab

None diagnostic or suggestive of this diagnosis

Imaging

- CT scan is gold standard but do not delay sight-saving intervention pending imaging
- Ultrasound (bedside if available): Sensitivity/specificity not studied. “Guitar-pick” sign.

DIFFERENTIAL DIAGNOSIS

The patient may present after trauma to the face with any of the following:

- Decreased vision
- Blurry vision
- Eye pain
- Eye discharge
- Photophobia
- Eye pressure
- Nausea and vomiting

The patient may present after having the following procedures:

- Reduction of facial fracture
- Eyelid surgery
- Endoscopic sinus surgery
- Regional anesthesia via retrobulbar injection
- Dacryocystectomy

One must consider as their differential:

- Orbital fracture
- Retro-orbital edema
- Retro-orbital emphysema
- Blow-in fractures
- Orbital roof fractures with brain herniation
- Intracranial bleeds
- Other major trauma associated with injury



TREATMENT

PRE HOSPITAL

- ABCs
- Pre-hospital lateral canthotomy very controversial

INITIAL STABILIZATION/THERAPY

- ABCs
- Immediate transfer to Level 1 Trauma Center
- If past window of 90–120 min, lateral canthotomy & inferior cantholysis may be attempted by competent physician provider

ED TREATMENT/PROCEDURES

Surgical therapy:

- Indication: IOP > 40, proptosis in unconscious patient
- Contraindication: Ruptured globe.
- The only definitive treatment

Lateral canthotomy and inferior cantholysis:

- Prep site with 5% Betadine
- Local anesthesia of cutaneous and deep tissues lateral to angle of the eye. Take caution to avoid the globe and orbit
- Clamp across the lateral canthus with hemostats for ~ 1 min
- With blunt scissors cut in lateral fashion along clamp marks from lateral angle of eyelid to the orbital rim
- Expose the inferior and superior crus of the lateral canthal tendon by pulling down the lateral aspect of the lower lid
- Ligate the inferior crus at its insertion into the lower lid with blunt scissors. The

lower lid should relax downward

MEDICATION

- Methylprednisolone
 - 30 mg/kg loading dose
 - 15 mg/kg q6h
- Mannitol
 - 1.5–2 g/kg over 30 min, with the 1st 12.5 g over 3 min
- Acetazolamide: 500 mg intravenously (do not use if allergic to sulfa or sickle cell pts)
- Hyperbaric oxygen



FOLLOW-UP

DISPOSITION

Admission Criteria

- All patients with suspected ROH should be admitted for definitive treatment in the OR and observation
- All patients need to be followed by an ophthalmologist
- All patients need to be worked up for other significant trauma

Discharge Criteria

Patients should not be discharged

Issues for Referral

- STAT ophthalmology consultation in the ED
- Do not delay decompression procedure due to consultation delay
- Emergency lateral canthotomy is within the scope of practice for emergency physicians

PEARLS AND PITFALLS

- Delayed diagnosis of retro-orbital hematoma due to:
 - Poor physical exam
- Lack of suspicion
- Lack of equipment such as a Tono-Pen:
 - Unconscious patient
- Waiting for CT/imaging thereby delays sight saving procedure
- Delayed consultation arrival

ADDITIONAL READING

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CODES

ICD9

376.89 Other orbital disorders

ICD10

- H05.239 Hemorrhage of unspecified orbit
- S05.10XA Contusion of eyeball and orbital tissues, unsp eye, init
- S05.11XA Contusion of eyeball and orbital tissues, right eye, init

RETROPHARYNGEAL ABSCESS

Jasmeet S. Dhaliwal • Maria E. Moreira

BASICS

DESCRIPTION

- Deep tissue infection of the retropharyngeal space:
 - Potential space bound anteriorly by buccopharyngeal fascia, posteriorly by alar fascia, superiorly by skull base, inferiorly by fusion of fascial layers at T2
 - Space fused by raphe at midline with chains of lymph nodes extending down each side
 - Alar fascia is poor barrier and allows retropharyngeal infections to spread into “danger” space and posterior mediastinum
- Primarily a disease of children, but increasing frequency in adults:
 - Peak incidence at 3–5 yr when retropharyngeal nodes most prominent
- Prognosis is good when promptly diagnosed and aggressively managed with IV antibiotics and/or surgical drainage
- Complications due to mass effect, rupture, or spread are the major source of morbidity and include:
 - Airway compromise (most common)
 - Aspiration pneumonia due to rupture
 - Sepsis
 - Spontaneous perforation
 - Necrotizing fasciitis
 - Mediastinitis
 - Thrombosis of the internal jugular vein
 - Jugular vein suppurative thrombophlebitis (Lemierre syndrome)
 - Erosion into carotid artery (primarily adults)
 - Atlantoaxial dislocation from erosion of ligaments
 - Cranial nerve palsies (typically IX–XII)
 - Epidural abscess
 - Recurrent abscess formation (1–5%)

ETIOLOGY

- Causes:
 - Most often arises from infection of nasopharynx, paranasal sinuses, or middle ear
 - Infection then spreads to lymph nodes between posterior pharyngeal wall and alar fascia
 - Trauma, foreign bodies, and iatrogenic introduction of infection from

instrumentation also common cause, especially in adults

- Diabetes and other immunosuppressed states may predispose to this infection
- Bacteriology: Predominately polymicrobial with anaerobes and aerobes
- Most common organisms are:
 - *Streptococcus pyogenes* and *Streptococcus viridans*
 - *Staphylococcus aureus* (including MRSA)
 - Respiratory anaerobes (including *Prevotella*, *Fusobacterium*, and *Veillonella*)
- Less common organisms are:
 - *Haemophilus* species
 - Acid-fast bacilli
 - *Klebsiella pneumoniae*
 - *Escherichia coli*
 - *Mycobacterium tuberculosis*
 - *Aspergillus* and *Candida* species

DIAGNOSIS

SIGNS AND SYMPTOMS

May differ between adults and children

History

- Most common:
 - Sore throat
 - Neck pain/stiffness
 - Odynophagia
 - Dysphagia
 - Fever
- Additional presenting symptoms:
 - Stridor, dyspnea
 - Muffled voice
 - Trismus

Pediatric Considerations

Young children may present with only:

- Poor oral intake
- Lethargy or irritability
- Cough

Physical-Exam

- Adults:
 - Posterior pharyngeal edema

- Nuchal rigidity
- Cervical adenopathy
- Fever (67%)
- Drooling
- Stridor
- Dysphonia (cri du canard)
- Tracheal “rock” sign: Tenderness on moving the larynx and trachea side to side
- Children and infants:
 - Cervical adenopathy
 - Fever
 - Neck stiffness with extension most frequently limited
 - Retropharyngeal bulge
 - Trismus
 - Torticollis
 - Drooling
 - Agitation
 - Respiratory distress

ESSENTIAL WORKUP

Rapid assessment of airway and respiratory status:

- Normal exam does not rule out diagnosis
- No lab tests make the diagnosis
- When suspicious, obtain lateral neck x-ray or CT of neck with IV contrast

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC (WBC >12,000 in 91% of children):
 - Nonspecific
- Blood cultures (both aerobic and anaerobic)
- Throat cultures

Imaging

- Portable films appropriate if concern for airway compromise
- Lateral neck radiographs:
 - Film taken in inspiration with neck slightly extended
 - May not get good exposure of soft tissue if cannot adequately extend neck due to pain or difficulty cooperating in young age
 - Increased suspicion if:
 - Retropharyngeal space anterior to C2 >7 mm or 2× the diameter of the vertebral body (sensitivity 90%)
 - Space anterior to C6 >14 mm in preschool children or 22 mm in

adults

- Loss of normal cervical lordosis

- Chest radiograph:
 - Indicated if abscess identified to assess for inferior spread of infection and/or aspiration of ruptured abscess contents
 - Mediastinal widening is suggestive of mediastinitis and possible rupture
- US of neck:
 - Low sensitivity
 - Not recommended
- CT of neck with IV contrast:
 - Now preferred imaging modality
 - Obtain when x-rays nondiagnostic or to determine exact size and location of abscess noted on x-ray
 - Abscess appears as hypodense lesion with peripheral ring enhancement in retropharyngeal space
 - Sensitivity: 64–100%
 - Specificity: 45–88%
 - Can aid in operative planning, revealing extent of invasion into retro/parapharyngeal spaces
 - Unclear if it reliably can distinguish abscess from cellulitis and lymphadenitis
 - Due to radiation exposure and need for sedation, CT should only be obtained in young children if x-rays are nondiagnostic
- MRI:
 - More sensitive than CT
 - Also useful for imaging vascular lesions such as jugular thrombophlebitis

Diagnostic Procedures/Surgery

- Surgical drainage/needle aspiration should be performed in OR:
 - Presence of pus is gold standard for making diagnosis
 - Abscess should be completely evacuated
 - Pus should be sent for Gram stain and culture
- No role for nasopharyngolaryngoscopy

DIFFERENTIAL DIAGNOSIS

- Tonsillopharyngitis
- Epiglottitis
- Peritonsillar abscess
- Croup
- Foreign body
- Tracheitis
- Meningitis
- Retropharyngeal hemorrhage

- Dystonic reactions
- Cervical osteomyelitis
- Dental infections
- Mononucleosis
- Epidural abscess
- Other deep space infection of the neck



TREATMENT

PRE HOSPITAL

- Keep child in position of comfort:
 - _ Forcing child to sit up or flex neck may occlude airway
- Pulse oximetry, cardiac monitor
- Supplemental oxygen
- Adequate hydration
- Suction, endotracheal tube, tracheostomy equipment ready for potential emergent intubation
- Airway control will be required for:
 - _ Airway compromise
 - _ Prior to long transport

INITIAL STABILIZATION/THERAPY

- Assess and control airway
- Provide supplemental oxygen
- IV access:
 - _ Avoid if signs of airway compromise

ED TREATMENT/PROCEDURES

- Early endotracheal intubation or tracheostomy for patients with respiratory distress or impending obstruction:
 - _ Caution must be used with induction, as sedation medications may lead to relaxation of airway muscles causing complete obstruction
 - _ Rescue airway equipment such as a laryngeal mask airway available, as pharyngeal swelling may make intubation difficult
 - _ Cricothyrotomy may be required if upper airway is obstructed
- Surgical consultation (ear/nose/throat if available)
- Early administration of IV antibiotics

MEDICATION

Empiric IV antibiotic therapy to cover group A streptococci, *S. aureus* (including MRSA), and respiratory anaerobes:

- Antibiotic tailored to local preferences and susceptibilities
- Coverage is narrowed when culture results and sensitivities return

- Use of corticosteroids is controversial and recommended only after consultation with ear/nose/throat
- Immunocompromised, diabetics, IV drug users, institutionalized patients, and young children (<1 yr) at high risk for MRSA

First Line

Several antibiotic regimens are available:

- Clindamycin: 600–900 mg (peds: 25–40 mg/kg/24 h) IV q8h (max. 4.8 g/d)
- Clindamycin + Metronidazole (loading dose 15 mg/kg IV not to exceed 4 g/d followed by 7.5 mg/kg PO/IV)
- Penicillin G + Metronidazole
- Cefoxitin 1 g IV q6–8h/3–4 g/d max.
- Ticarcillin/Clavulanate 3.1 g IV q4–6h
- Piperacillin/Tazobactam 3.375 g IV q6h

Second Line

If patients do not respond or there is concern for MRSA:

- Vancomycin: 15–20 mg/kg (peds: 40–60 mg/kg/24 h IV q6–8h) IV q12h
- Linezolid: 600 mg (peds: 0–11 yr: 30 mg/kg/24 h q8h; >12 yr: Adult dose) IV/PO q12h



FOLLOW-UP

DISPOSITION

Admission Criteria

- All patients with retropharyngeal abscess should be admitted to the hospital for IV antibiotics and possible surgical drainage
- Criteria for surgical drainage:
 - Airway compromise or other life-threatening complications
 - Large (>2 cm hypodense area on CT)
 - Failure to respond to parenteral antibiotic therapy
- ICU admission for patients with:
 - Airway compromise
 - Sepsis
 - Altered mental status
 - Hemodynamic instability
 - Infants and toxic-appearing children
 - Major comorbidities

Discharge Criteria

Patients with retropharyngeal abscesses should not be discharged

Issues for Referral

Transfer should be considered if facility does not have the ability to drain infection:

- Airway should be stabilized prior to transfer

PEARLS AND PITFALLS

- Diagnosis should be considered in all children who present with fever, stiff neck, or dysphagia:
 - High clinical suspicion is required in children, as they present with nonspecific signs and symptoms
- Adult cases most often present in the setting of underlying illness, recent intraoral procedures, neck trauma, or head and neck infections
- When imaging is nondiagnostic and clinical suspicion remains high, surgery should be consulted
- Early surgical consultation and administration of IV antibiotics is essential to prevent complications such as airway compromise and extension into mediastinal structures

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See Also (Topic, Algorithm, Electronic Media Element)

- Epiglottitis
- Peritonsillar Abscess



CODES

ICD9

478.24 Retropharyngeal abscess

ICD10

J39.0 Retropharyngeal and parapharyngeal abscess

REYE SYNDROME

Brian D. Euerle



BASICS

DESCRIPTION

- Reversible clinicopathologic syndrome of unknown etiology
- Primary mitochondrial injury
- Decreased enzyme activity:
 - Krebs cycle
 - Gluconeogenesis
 - Urea biosynthesis
- Fatty infiltration:
 - Liver:
 - Hyperammonemia due to decreased conversion from ammonia to urea
 - Hepatorenal syndrome may be the end result.
 - Rapid recovery of liver function in survivors
 - Brain:
 - Encephalopathy of unclear etiology
 - Cytotoxic edema
 - Deteriorating level of consciousness reflects increasing intracranial pressure (ICP).
 - Herniation is the most common cause of death.
 - Normal recovery of neurologic function in survivors
 - Skeletal and myocardial muscle
 - Fatty infiltration and distorted mitochondria
- <10% of cases occur before the age of 1 yr:
 - Average age is 7 yr
 - Peak age is 4–11 yr
 - Extremely rare in age >18 yr.
- Regional differences:
 - Highest incidence in the Midwestern states
 - Lower incidence in the states of the Southeast and far West
- More common in whites than in blacks
- Peak incidence in winter and early spring
- Reye-like syndrome:
 - Describes conditions resulting in defects in urea and fatty acid metabolism, toxicologic injury, and impaired gluconeogenesis

ETIOLOGY

- Not known with certainty
- Multifactorial causes have been epidemiologically implicated:
 - Antecedent viral syndrome
 - Influenza A or B
 - Varicella
 - Diarrhea illness
 - Genetic predisposition
 - Exposure to salicylates
 - Other undefined factors

DIAGNOSIS

SIGNS AND SYMPTOMS

- Usually the patient is afebrile.
- Tachycardia
- Hyperventilation

History

- Biphasic history marked by an infectious phase (viral illness or prodrome) followed by an encephalopathic stage
- Profuse and repeated vomiting:
 - Typically 4–5 days after the start of the viral illness
- Marked behavioral changes, including delirium and combativeness, disorientation, and hallucination

Physical-Exam

- No focal neurologic signs
- Hepatomegaly in 40% of cases
- Pancreatitis
- Clinical staging of Reye syndrome with Lovejoy classification:
 - Stage 0:
 - Wakeful
 - Stage I:
 - Vomiting
 - Lethargy
 - Sleepiness
 - Stage II:
 - Disorientation
 - Delirium
 - Combative/stuporous
 - Hyperventilation
 - Hyperreflexia

- Appropriate response to noxious stimuli
- Stage III:
 - Obtunded
 - Coma
 - Hyperventilation
 - Inappropriate response to noxious stimuli
 - Decorticate posturing
 - Preservation of pupillary light reflexes
 - Preservation of oculovestibular light reflexes
- Stage IV:
 - Deeper coma
 - Decerebrate rigidity
 - Loss of oculovestibular reflexes
 - Dilated, fixed pupils
 - Disconjugate eye movements in response to caloric stimulation
- Stage V:
 - Seizures
 - Absent deep tendon reflexes
 - Respiratory arrest
 - Flaccid paralysis
 - No pupillary response
- Infants: Atypical presentation:
 - Tachypnea
 - Apnea
 - Irritability
 - Seizures
 - Hypoglycemia

ESSENTIAL WORKUP

- Establish the presence of encephalopathy and liver abnormalities.
- Lab testing to assess for characteristic biochemical abnormalities
- Liver biopsy confirms the diagnosis.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Liver function tests:
 - $\geq 3 \times$ rise in aspartate aminotransferase, alanine aminotransferase
 - Serum ammonia level $> 1.5\text{--}3 \times$ normal:
 - Transient 24–48 hr after mental status changes
 - Level $> 300 \mu\text{g/dL}$ is associated with poor prognosis.
 - Serum bilirubin should be normal or slightly elevated.
- Hypoglycemia may be present, especially in infants.

- Elevated BUN
- Ketonuria
- The prothrombin time may be prolonged due to decreased liver-dependent clotting factors (II, VII, IX, X).
- Normal platelet count and blood smear
- Negative toxicology screen

Imaging

Head CT scan:

- May show diffuse cerebral edema
- Edema is diffuse, and lumbar puncture is not contraindicated.

Diagnostic Procedures/Surgery

- Lumbar puncture:
 - Perform after head CT
 - Measure opening pressure
 - < 8 leukocytes/mm³
- Percutaneous liver biopsy:
 - Useful in patients with atypical presentation (1 yr old, recurrent, familial)

DIFFERENTIAL DIAGNOSIS

- Inborn errors of metabolism:
 - Disorders of the urea cycle
 - Disorders of fatty acid oxidation
 - Systemic carnitine deficiency
 - Organic acidemias
 - Disorders of the electron transport chain
- Hypoglycemia
- Toxin exposure:
 - Toxic encephalopathy without liver dysfunction (Gall syndrome)
 - Lead
 - Hydrocarbons
- Drug intoxication:
 - Acetaminophen
 - Salicylates
 - Ethanol
- Infection:
 - Sepsis
 - Meningitis
 - Encephalitis
 - Varicella hepatitis
- Trauma, head



TREATMENT

PRE HOSPITAL

- Decreased mental status:
 - Glucose
 - Narcan
- Coma:
 - Assist respirations with bag-valve mask.

INITIAL STABILIZATION/THERAPY

- Place on a cardiorespiratory monitor.
- Supplemental oxygen
- Rapid-sequence intubation if airway management required
- Glucose if mental status is altered:
 - 10% glucose solution IV
 - Rate of 2/3 maintenance requirement after dehydration is corrected
 - Follow serum glucose hourly; maintain glucose 125–175 mg/dL.
- Avoid early overhydration.

ED TREATMENT/PROCEDURES

- Institute treatment before the liver biopsy.
- Vitamin K:
 - Indicated if prothrombin time is elevated.
- Fresh-frozen plasma:
 - To control bleeding
 - To correct a severe coagulopathy
- Interventions aimed at lowering ICP:
 - Stage III or greater
 - Stage II with serum ammonia $> 300 \mu\text{g/L}$:
 - Intubation using rapid-sequence protocol
 - Hyperventilation
 - Fluid restriction
 - Barbiturate coma
- Osmotically active agents:
 - Mannitol
 - Furosemide
- Monitor ICP:
 - Subarachnoid bolt
 - Intraventricular cannula

MEDICATION

- D₅₀W: 1–2 mL/kg/dose (0.5–1 g/kg) IV for age > 3 yr

- D₂₅W: 2–4 mL/kg/dose (0.5–1 mg/kg) IV for age of < 3 yr; maintenance infusion 10% dextrose solution at a rate of 2/3 maintenance
- Fresh-frozen plasma: 10 mL/kg/dose q12–24h IV or PRN
- Lasix: 1 mg/kg IV
- Mannitol: 0.25–1 g/kg IV q4–6h
- Pentobarbital: 3–5 mg/kg IV slowly while monitoring BP; maintenance infusion 1–2 mg/kg/h; maintain level at 2–5 mg/L
- Vitamin K: 1–2 mg/dose IV slowly (infants and children); 2–10 mg/dose IV (adolescents)



FOLLOW-UP

DISPOSITION

Admission Criteria

- All children with suspected Reye syndrome should be admitted to the ICU.
- Hospital capable of ICP monitoring

Discharge Criteria

Hospital discharge criteria are individualized for each case:

- Mental status and lab values have improved and stabilized.

Issues for Referral

Close follow-up with specialists in gastroenterology (hepatology) and neurology

FOLLOW-UP RECOMMENDATIONS

Long-term psychological and neuropsychological testing

PEARLS AND PITFALLS

- Aspirin and salicylates are found in many medications and combination products.
- All efforts must be directed at identifying other possible causes of illness in the patient with suspected Reye syndrome.
- Monitoring and control of intracranial pressure is a key component of treatment.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Altered Mental Status
- Coma
- Influenza
- Varicella
- Salicylates



CODES

ICD9

331.81 Reye's syndrome

ICD10

G93.7 Reye's syndrome

RHABDOMYOLYSIS

Stephen R. Hayden

BASICS

DESCRIPTION

Abnormal systemic release of muscle contents—creatinine phosphokinase (CPK), myoglobin, potassium, phosphate, urate—caused by trauma, poisoning, infection, primary muscle disorders, and many other disease states. Complications include:

- Myoglobin-induced renal failure in 15–50% adults, only 5% in children
- Hyperkalemia may lead to sudden death
- Hypocalcemia and acidosis
- Volume loss—fluid sequestration in injured muscle or result of underlying illness
- Compartment syndrome of muscles in crush, worsened by IV fluid sequestration in damaged tissue
- Hepatic dysfunction in 25%
- DIC (Disseminated intravascular coagulation)

EPIDEMIOLOGY

Incidence

- 26,000 per year in US
- Disaster situations lead to 100s of cases of renal failure.

RISK FACTORS

- Inherited myopathy
- Alcohol or drug use
- Medications as listed below
- Overexertion with or without risk factors

PATHOPHYSIOLOGY

- Sarcolemma keeps intracellular calcium low.
- Etiologies disrupt cell membrane and lead to following cascade.
- Breakdown of sarcolemma Na–Ca pumps allows calcium to enter cell.
- Calcium-dependent proteases cause destruction.
- Ischemia and neutrophils cause damage.
- Escape of cell contents: Myoglobin, potassium, phosphate, CPK, lactate, etc.
- Myoglobin causes renal damage by direct toxicity in acidic urine.
- Myoglobin precipitates with other proteins to obstruct renal tubular flow.
- Volume depletion also leads to renal vasoconstriction and failure.
- Hyperkalemia can lead to arrhythmias.
- Calcium precipitates with phosphate, leading to systemic hypocalcemia.

ETIOLOGY

Cause usually obvious, but not always.

Adults: Trauma, toxicity, infection

Children: Viral myositis, trauma

- Muscle injury—due to trauma/crush, burn, electrical shock—most common cause overall.
- Muscle exertion: Strenuous exercise; marathon running; exercise in hot, humid conditions; exercise in individuals with an inherited myopathy or with poor physical training; status epilepticus; delirium tremens; tetanus; psychotic agitation
- Muscle ischemia: Extensive thrombosis, multiple embolism, generalized shock, sickle cell crisis
- Surgery: Immobilization, hypotension, ischemia due to vessel clamping
- Massive blood transfusion
- Hypothermia, hyperthermia
- Prolonged immobile state without trauma
- Drugs/toxins: Alcohols, cocaine, amphetamines, and analogs (methamphetamine and ecstasy), toluene, opiates, LSD, phencyclidine (PCP), caffeine, carbon monoxide, snake venom, bee/hornet venom, hemlock, buffalo fish, tetanus toxin, mushroom poisoning (*Tricholoma equestre*)
- Medications: Most common—haloperidol, phenothiazines, HMG-CoA reductase inhibitors (statins) and other cholesterol-lowering agents, antihistamines, selective serotonin receptor inhibitors (SSRIs).
- Sports supplements including ephedra, caffeine, androgenic steroids, creatine, diuretics
- Neuroleptic malignant syndrome (idiosyncratic and not dose-related)
- Metabolic disorders: Hypokalemia, hypophosphatemia, hypocalcemia, hyper- and hyponatremia, diabetic ketoacidosis, hyperosmolar state, hypoxia, hyperthyroid state (rare), pheochromocytoma (rare)
- Infections:
 - Viral: Coxsackievirus, herpesviruses, HIV, influenza B, cytomegalovirus, Epstein–Barr virus, adeno/echovirus
 - Bacterial: Legionnaires' disease, pyomyositis, salmonellosis, shigellosis, *Staphylococcus*, *Streptococcus*, *Listeria*, tetanus, toxic shock syndrome, tularemia, gas gangrene, *Bacillus cereus*
 - Parasitic (*Plasmodium falciparum*), protozoan (leptospirosis), rickettsial
 - Inherited myopathic disorders: McArdle disease, Tarui disease, CPT deficiency.
- Immunologic disorders: Dermatomyositis, polymyositis
- Idiopathic

COMMONLY ASSOCIATED CONDITIONS

- Crush syndrome
- Compartment syndrome

- Alcohol and drug abuse
- Elderly and acutely immobile (found on floor)



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Can vary dramatically, reflecting underlying disease process.
- Trauma or crush usually obvious.
- Consider nonaccidental trauma with unclear details of history.
- If no trauma, consider in drug toxicity, heat illness, immobilization, or overexertion states.
- Ask about reddish brown urine and decreased urine output
- Most nontraumatic cases in children <9 yr old are due to viral illness with myositis

Physical-Exam

- Hypothermia/hyperthermia
- Alert/obtunded
- Muscle pain (only 40–50%)
- Neurovascular status of involved muscle groups if compartment syndrome is suspected.
- Hypovolemic state, dry mucous membranes, poor skin turgor, tachycardia, hypotension
- Decreased urine output
- Urine color (tea-colored) is early sign
- Children more often have absent physical findings

DIAGNOSIS TESTS & INTERPRETATION

Lab

Initial lab tests

- History and physical exam are insensitive in making the diagnosis
- Serum and urine myoglobin levels often normal due to rapid metabolism and excretion.
- Serum CPK level > 1,000 (standard) considered positive
- CPK level not always predictive of renal failure but most often associated with level > 15,000
- Urine dipstick test positive for heme but absent for RBCs suggests rhabdomyolysis
- Microscopic urinalysis to look for pigmented tubular casts
- Because of rapid urinary excretion of myoglobin, some patients with

rhabdomyolysis have negative urine dipstick test.

- In children, heme <2+ on urine dip correlates with reduced risk of acute renal failure (ARF)
- Serum electrolytes (potassium, calcium, magnesium, phosphorus, BUN, creatinine, uric acid, bicarbonate)
- In addition to above consider:
 - Arterial and venous blood gases (ABG/VBG) (baseline pH if considering bicarbonate therapy).
 - Urine/serum myoglobin, but may be too transient to be useful
 - Serum glucose
 - LFTs including GGTP, LDH, albumin
 - Toxicology screen in absence of physical injury
 - PT/PTT, platelet count, fibrinogen, fibrin split products if DIC is suspected

Imaging

- Renal US to rule out long-standing renal failure (small, shrunken kidneys) or renal obstruction (hydronephrosis)
- MRI is 90–95% sensitive in visualizing muscle injury but does not change initial ED treatment.
- Other imaging as indicated

Diagnostic Procedures/Surgery

- Early ECG: Hyperkalemia or hypocalcemia before serum levels available
- Measure compartment pressure if compartment syndrome is suspected.

DIFFERENTIAL DIAGNOSIS

Conditions that may present with elevated serum CPK but are not rhabdomyolysis:

- Nontraumatic myopathies including muscular dystrophies and inherited myopathies
- Chronic renal failure
- IM injections
- Myocardial injury
- Stroke



TREATMENT

PRE HOSPITAL

- Rapid extrication in case of crush injury
- Early IV saline before extrication to prevent complications of restored blood flow to injured limb (hypovolemia, hyperkalemia, etc.)
- “Crush injury cocktail” during extrication is 1.5 L 0.9% NS per hour; consider adding 1 amp (50 mEq) bicarbonate and 10 g of mannitol to each liter

(controversial)

- Pediatric recommendation: 10–15 mL/kg/h saline initially, then switch to hypotonic (0.45%) saline upon arrival to hospital. Add 50 mEq bicarbonate to each 2nd or 3rd liter to alkalinize urine

INITIAL STABILIZATION/THERAPY

- Manage ABCs
- Immobilization of trauma/crush injuries
- Adult crush injury treatment literature extrapolated to children
- IV saline for hypovolemia at rate of 1–1.5 L/h (10–20 mL/kg/h). Volume restored within 6 hr helps prevent renal failure

ED TREATMENT/PROCEDURES

- May need 12 L/d, 4–6 of which should include bicarbonate. Use CVP, urine output
- Diuretics only after patient's volume restored to keep urine output 200–300 mL/h (3–5 mL/kg/h)
- Mannitol: Diuretic, free radical scavenger. May help compartment syndrome
- Furosemide and other loop diuretics if indicated in management of oliguric (<500 mL/d) renal failure; controversial
- Bicarbonate: Alkalinize urine (pH >6.5) most studied in crush/trauma. Most authorities recommend its use as long as urine pH and calcium are monitored.
- Monitor for hyperkalemia frequently with serum levels and ECG. Higher potassium correlates with more severe injury
- Treat hyperkalemia as usual but do not use calcium unless it is severe
- Hypocalcemia: Treat only if symptomatic (tetany or seizures) or arrhythmias present. Calcium infusion can lead to hypercalcemia later as precipitated calcium mobilizes
- Bicarbonate can trigger symptoms by increasing free calcium binding to albumin



FOLLOW-UP

DISPOSITION

Admission Criteria

All but the most trivial elevations in CPK (<1,000) should be admitted, since complications can occur at any level and are difficult to predict. Children seem to be less susceptible to renal complications:

- Critical care admission criteria:
 - Hyperkalemia or CPK levels > 15,000–30,000 due to worse prognosis
 - Underlying severe illness

Discharge Criteria

Levels decreased to <1,000 after therapy

MEDICATION

First Line

- Bicarbonate; add 50 mEq bicarbonate to each 2nd or 3rd liter to keep urine pH >6.5. Discontinue if urine pH fails to rise after 6 hr or if symptomatic hypocalcemia develops
- Albuterol, insulin/dextrose, polystyrene resin (kayexalate), for hyperkalemia treatment. Avoid calcium if possible.

Second Line

- Mannitol 20%: 50 mL (10 g added to each liter up to 120–200 g/d (1–2 g/kg/d)
- Discontinue if fail to achieve diuresis and osmolal gap >55

SURGERY/OTHER PROCEDURES

- Hemodialysis for refractory hyperkalemia, fluid overload, anuria, acidosis
- Consider central venous monitoring of volume
- Fasciotomy for compartment syndrome

PROGNOSIS

- No renal failure—almost no mortality
- Renal failure—3.4–30% mortality
- ICU—59% if renal failure, 22% without

COMPLICATIONS

- ARF
- Hyperkalemia
- Compartment syndrome
- Hypocalcemia
- Acidosis

PEARLS AND PITFALLS

Suspect in unexplained renal failure.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Compartment Syndrome
- Hyperkalemia



CODES

ICD9

- 728.88 Rhabdomyolysis
- 958.90 Compartment syndrome, unspecified

ICD10

- M62.82 Rhabdomyolysis
- T79.6XXA Traumatic ischemia of muscle, initial encounter

RHEUMATIC FEVER

Jon D. Mason



BASICS

DESCRIPTION

- Constellation of symptoms and signs (Jones criteria)
- Follows group A streptococcal infection (GAS) also known as *Streptococcus pyogenes*; usually pharyngitis
- Uncommon in US; most cases are in developing nations
- Remains a major cause of cardiac morbidity and mortality worldwide with over 230,000 deaths per year
- Most common in 5- to 15-yr-olds

ETIOLOGY

- GAS infection
- Inflammatory, autoimmune response following GAS infection



DIAGNOSIS

2 major or 1 major and 2 minor elements of the **Jones criteria** plus evidence of a recent GAS infection

SIGNS AND SYMPTOMS

Jones Criteria

- Major manifestations:
 - *Migratory polyarthriti*s in 60–75% of initial attacks:
 - Involves larger joints: Knees, hips, ankles, elbows, and wrists
 - Lower extremity joints more commonly involved
 - Rheumatic arthritis generally responds to salicylates
 - *Carditis* occurs in 1/3 to 1/2 of new cases:
 - Pericardium, myocardium, and endocardium may be affected (pancarditis)
 - Myocarditis may lead to heart failure but is frequently asymptomatic
 - Valvular disease and endocarditis are most serious sequelae of acute rheumatic fever (ARF)
 - Carditis heralded by a new murmur, tachycardia, gallop rhythm, pericardial friction rub, or CHF
 - Echocardiogram aids in diagnosis
 - *Chorea* occurs in 10% of cases:
 - Sydenham chorea predominantly affects teenage girls
 - Purposeless, uncoordinated movements of the extremities sometimes

- called St. Vitas dance
- Movements are more apparent during periods of anxiety and disappear with sleep
- Chorea may be the sole manifestation of ARF
- Other neuropsychiatric symptoms of emotional lability or obsessive compulsive disorder may also occur
- *Erythema marginatum* occurs in <5% of cases:
 - Nonpruritic pink eruptions with central clearing and well-demarcated irregular borders
 - Usually seen on the trunk and the extremities
- SC nodules in small percentage of patients:
 - Crops of small SC, painless nodules located most commonly on extensor surfaces
- Minor manifestations:
 - Clinical:
 - Fever (>38°C)
 - Arthralgia
 - Lab:
 - Elevated acute phase reactants
 - Prolonged P-R interval
- Supporting evidence of recent GAS throat infection:
 - Positive throat culture or rapid antigen test
 - Elevated or increasing antibody test: Antistreptolysin O (ASO) titer

History

- Fever
- Sore throat (often 2–4 wk prior)
- Rash
- Joint pains
- Unusual movements of extremities
- Dyspnea
- Lower extremity edema

Physical-Exam

- Pharyngeal erythema
- Rash consistent with erythema marginatum
- SC nodules
- New heart murmur consistent with mitral or aortic disease
- Evidence of fluid overload/CHF

ESSENTIAL WORKUP

- Careful exam to look for skin lesions/joint swelling
- Careful heart and lung exam

- Throat swab for rapid strep test or culture
- ECG
- Chest x-ray
- Echocardiogram
- See other labs below

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Rapid antigen strep test
- Throat culture
- ASO titer
- CBC
- ESR or C-reactive protein
- Other serology tests to rule out other rheumatologic diseases

Imaging

- Chest radiograph
- Echocardiogram

Diagnostic Procedures/Surgery

- ECG
- Diagnosis is based on clinical picture and meeting Jones criteria

DIFFERENTIAL DIAGNOSIS

- Juvenile idiopathic arthritis
- Infective endocarditis
- Reiter syndrome
- Systemic lupus erythematosus
- Postgonococcal arthritis
- Other infectious causes of arthritis and carditis:
 - _ Coxsackie B virus and parvovirus

Pediatric Considerations

Rheumatic fever is primarily a pediatric disease but can occur in young adults. Testing for strep throat is not recommended under 3 yr of age in US due to low incidence of strep throat and rare ARF

Pregnancy Considerations

Prenatal counseling recommended if woman has a history of rheumatic fever due to increased cardiac risks



TREATMENT

PRE HOSPITAL

- Oxygen as needed
- Monitors if in distress
- IV access may be prudent

INITIAL STABILIZATION/THERAPY

Some patients in CHF will need airway management

ED TREATMENT/PROCEDURES

- Pericardial effusions may need drainage
- In severe carditis, start prednisone
- In case of severe chorea, start haloperidol
- Penicillin IM, IV, or PO
- Aspirin for arthritis/arthralgia

MEDICATION

- Aspirin: 4–8 g/d (peds: 100 mg/kg/d) PO q4–6h; do not exceed 4 g/24h
- Azithromycin 500 mg day 1, then 250 mg PO for 4 more days. (peds: 10 mg/kg day 1 then 5 mg/kg daily PO for 4 more days)
- Digoxin: 0.25–0.5 mg (peds: 0.04 mg/kg) IV
- Erythromycin: 250 mg (peds: 30–50 mg/kg/d) q6h PO for 10 days
- Furosemide: 20–80 mg (peds: 1 mg/kg/dose) IV
- Haloperidol: 2–10 mg (peds: 0.01–0.03 mg/kg/d; use only > 2 yr and > 15 kg) q6h IM or PO
- Penicillin (benzathine benzylpenicillin): 1.2 million U (peds: 600,000 U for < 27 kg) IM acutely and monthly thereafter (prophylaxis)
- Penicillin VK: 500 mg (peds: 250 mg) PO q8h for 10 days (acute treatment)
- Prednisone: 1–2 mg/kg/d for 14 days with taper for the next 2 wk

First Line

- Aspirin (carditis patients)
- Penicillin
- Haloperidol (for chorea)

Second Line

Corticosteroids



FOLLOW-UP

DISPOSITION

Most patients with a new diagnosis should be admitted for stabilization and further evaluation of the severity of the heart disease.

Admission Criteria

- CHF
- New diagnosis
- Uncontrolled chorea
- Uncontrolled pain
- Pericardial effusion

Discharge Criteria

- Pain is controlled
- Stable cardiovascular status
- Education regarding prolonged treatment and endocarditis prophylaxis
- Patient has reliable follow-up option

Issues for Referral

- All patients need close follow-up with their primary physician and cardiologist
- Consider referral to infectious disease specialist and rheumatologist

FOLLOW-UP RECOMMENDATIONS

- Cardiology for echocardiogram and advice on subacute bacterial endocarditis prophylaxis
- Infectious disease specialist to advise on prolonged use of penicillin to prevent recurrence
- Rheumatology if needed for chronic joint problems (uncommon)

PEARLS AND PITFALLS

- Rheumatic fever is uncommon in US, but must be vigilant to treat strep infections to prevent resurgence of disease
- More common in patients living in poor and crowded conditions
- No need to do throat cultures in children under age 3

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Pharyngitis



CODES

ICD9

- 390 Rheumatic fever without mention of heart involvement
- 391.9 Acute rheumatic heart disease, unspecified
- 714.0 Rheumatoid arthritis

ICD10

- I00 Rheumatic fever without heart involvement
- I01.9 Acute rheumatic heart disease, unspecified
- M06.9 Rheumatoid arthritis, unspecified

RIB FRACTURE

Charles W. O'Connell



BASICS

DESCRIPTION

- Result of major or minor thoracic trauma
- Can be classified as traumatic or pathologic

ETIOLOGY

- Blunt thoracic trauma:
 - Simple fall, fall from height
 - Motor vehicle crash
 - Assault
 - Missile
 - CPR-related
- Penetrating trauma is a less likely cause.
 - Ribs usually break at the point of impact or the posterior angle, the structurally weakest region
- Stress fractures in upper and middle ribs can occur with recurrent, high force movements:
 - Athletic activities: Golf, rowing, throwing
 - Severe cough
- Pathologic fractures associated with minor trauma or significant underlying disease:
 - Advanced age
 - Osteoporosis
 - Neoplasm

Pediatric Considerations

- Relatively elastic chest wall makes rib fractures less common in children.
- Consider nonaccidental trauma for infants and toddlers without appropriate mechanism.
- Obtain a skeletal survey to assess for other fractures in infants suspected of being abused

Geriatric Considerations

- Elderly are more prone to rib fractures as well as atelectasis, pneumonia, respiratory failure, and other associated complications.
- Morbidity and mortality are twice that found in younger populations.

SIGNS AND SYMPTOMS***History***

- Blunt thoracic trauma by any mechanism
- Mechanism as described by patient, parent, or pre-hospital personnel:
 - Seat belt usage
 - Steering wheel damage
 - Air bag deployment
- Localized chest wall pain that increases with deep inspiration, coughing, movement
- Pleuritic chest pain
- Dyspnea, shortness of breath

Physical-Exam

- Point tenderness
- Pain referred to fracture site with palpation of the involved rib elsewhere
- Bony step-off
- Crepitus
- Localized edema
- Erythema
- Ecchymosis:
 - Impact from seat belt, aka “seat belt sign” or steering wheel associated with motor vehicle accidents
- Intercostal muscle spasm
- Splinting respirations
- Hypoxia, tachypnea, respiratory distress
- Auscultation shows normal or diminished breath sounds, occasionally an audible click over fracture site.
- Segmental paradoxical movement of chest suggests flail chest indicating multiple, unattached fractured ribs.

ESSENTIAL WORKUP

- Diagnosis is initially made on clinical grounds and confirmed on imaging studies.
- Evaluate for injury to underlying structures

ALERT

- The 1st 3 ribs are relatively protected and require significant impact to fracture, may indicate intrathoracic injury.
- Ribs 9–12 are relatively mobile; their fracture suggests possible intra-abdominal injury.

- Multiple rib fractures may be associated with flail chest and pulmonary contusion.
- Morbidity correlates with degree of injury to underlying structures, number of ribs fractured, and age.

DIAGNOSIS TESTS & INTERPRETATION

Lab

ABGs may reveal hypoxemia or elevated alveolar–arterial gradient:

- Not indicated for simple, uncomplicated rib fractures
- May consider in patients with multiple rib fractures or pre-existing pulmonary disease

Imaging

- Anteroposterior (AP) and lateral chest films are used routinely to diagnose rib fractures
- Chest radiography is indicated to rule out associated intrathoracic injury but can miss up to 50% of rib fractures:
 - _ May reveal associated intrathoracic pathology:
 - Pneumothorax
 - Hemothorax
 - Pneumomediastinum
 - Pulmonary contusion
 - Atelectasis
 - Widened mediastinal silhouette
 - _ Pulmonary contusion appears within 6–12 hr after injury:
 - Ranges from patchy alveolar infiltrates to frank consolidation
- Rib radiograph series offer higher sensitivity but are controversial and are often low yield
- CT is more sensitive for detecting rib fractures and internal injuries.
- CT of the chest may be required to rule out intrathoracic injuries.
- CT or US of the abdomen may be required to rule out associated intra-abdominal injuries.
- Angiography can be used for the detection of vascular injury if signs and symptoms of neurovascular compromise are present:
 - _ Injury to the 1st and 2nd ribs can be associated with vascular injury, particularly with posterior displacement.
- Ultrasound is a promising diagnostic tool for evaluating rib fractures, even for cartilaginous injury

DIFFERENTIAL DIAGNOSIS

- Rib contusion or intercostal muscle strain
- Pneumothorax
- Costochondral separation

- Sternal fracture and dislocation
- Nontraumatic causes of chest pain:
 - Cardiovascular:
 - Myocardial ischemia or infarction
 - Pericarditis
 - Aortic dissection
 - Pulmonary embolism
 - Pulmonary:
 - Embolism
 - Infections
 - Inflammation
 - Barotrauma
 - Musculoskeletal:
 - Costochondritis
 - Cervical or thoracic spine disease
 - GI:
 - Esophageal reflux or spasm
 - Mallory–Weiss tear
 - Biliary or renal colic
 - Peptic ulcer disease
 - Gastritis, pancreatitis, hepatitis
 - Dermatologic:
 - Herpes zoster
 - Chest wall tumor



TREATMENT

PRE HOSPITAL

Focus on airway maintenance, analgesia, and supplemental oxygen

INITIAL STABILIZATION/THERAPY

- For simple fractures, generally no significant stabilization is required.
- Multiple fractures, elderly patients, or significant underlying lung disease:
 - Manage airway and resuscitate as indicated.
 - Endotracheal intubation indicated for patients with severe hypoxemia ($\text{PaO}_2 < 60$ mm Hg on room air, < 80 mm Hg on 100% O_2) or impending respiratory failure

ED TREATMENT/PROCEDURES

- Simple fractures:
 - Pain control:
 - Key to maintaining adequate pulmonary function, avoiding

atelectasis and subsequent pneumonia

- Intercostal nerve blocks with 0.5% bupivacaine are safe and effective:
 - Provides 6–12 hr of pain relief
 - Intercostal nerve block should be performed posteriorly, 2–3 fingerbreadths from the vertebral midline.
 - Inject 0.5–1 mL just under the inferior surface of the rib where the neurovascular bundle is located.
 - Aspirate 1st to be certain the intercostal vessels have not been punctured.
- Deep breathing or incentive spirometry should be encouraged with adequate pain control.
- Avoid binders or banding of the chest wall because these restrict ventilation and promote atelectasis.
- Multiple fractures, elderly patients, or significant underlying lung disease:
 - Pain control and pulmonary toilet
 - Search for associated injuries; treat exacerbation of underlying lung disease.
 - Intercostal nerve blocks for multiple fractures are safe and effective providing 6–12 hr of pain relief.
 - For the admitted patient, thoracic epidural analgesia or patient-controlled analgesia (PCA) is effective, with minimal inhibition of respiratory drive.

MEDICATION

- 1st Line: NSAIDs with or without opioids
 - Ibuprofen: 600 mg PO q6h (peds: 5–10 mg/kg PO q6–8h)
 - Naproxen: 250–500 mg PO q12h (peds: 10–20 mg/kg/d PO div. q12h)
- Opioid analgesics
- Multiple acetaminophen/opioid analgesic combinations are available; see “Alert” below.
 - Acetaminophen: 300 mg/codeine 30 mg (peds: 0.5–1 mg/kg codeine) PO q4–6h
 - Acetaminophen: 325 mg/hydrocodone 2.5–10 mg PO q4–6h
 - Acetaminophen: 325 mg/oxycodone 2.5–10 mg PO q4–6h
- 2nd line: For PO intolerance or more severe pain
 - Hydromorphone: 2–8 mg PO q3–4h (peds: 0.03–0.08 mg/kg PO q4–6h)
 - Hydromorphone: 0.5–4 mg IV/IM/SC q4–6h (peds: 0.03–0.08 mg/kg)
 - Morphine sulfate: 2.5–10 mg IV/IM/SC q2–6h (peds: 0.1–0.2 mg/kg)
 - PCA using hydromorphone or morphine sulfate is effective.
 - Bupivacaine 0.5%: 0.5–1 mL per injection for intercostal nerve blocks

ALERT

- Consider thoracic epidural analgesia:
 - Patients with intractable pain
 - Oversedation

- Hypoventilation from narcotic analgesics
- Avoid NSAIDs when contraindicated due to renal insufficiency or GI bleed
- The dose of acetaminophen/narcotic analgesic combinations is limited by acetaminophen's potential for causing hepatic toxicity.
 - Do not exceed 4 g/24h acetaminophen in adults, 5 doses of 10–15 mg/kg/24 h acetaminophen in children.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Intractable pain
- Inability to cough and clear secretions
- Compromised pulmonary function
- Multiple fractures, fractures of the 1st 3 ribs
- Displaced rib fractures
- Associated pneumothorax, pneumomediastinum, pulmonary contusion, intra-abdominal or intrathoracic pathology
- Elderly patients and patients with significant underlying lung disease:
 - Chronic COPD, CHF, pulmonary fibrosis, asthma
- Inadequate pain control on oral analgesics
- ICU care for elderly patients with 6 or more rib fractures

Discharge Criteria

- Patients with normal pulmonary function, no underlying pulmonary injury, and adequate pain control on oral analgesics
- Strict return criteria should be discussed with the patient prior to discharge:
 - Shortness of breath
 - Increased pain
 - Inadequate pain control
 - Fever
 - Cough

FOLLOW-UP RECOMMENDATIONS

- Most rib fractures heal within 6 wk, but patients should be able to return to regular daily activities much sooner.
- Routine follow-up chest x-ray are not recommended

PEARLS AND PITFALLS

- Be vigilant for the underlying intrathoracic and intra-abdominal pathology that

can be associated with rib fractures.

- Ensuring adequate pain control and ventilation are paramount in the treatment
- Each successive rib fracture carries added morbidity and mortality
- Pediatric rib fractures imply significant force and should raise suspicion for nonaccidental trauma

ADDITIONAL READING

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CODES

ICD9

- 733.19 Pathologic fracture of other specified site
- 807.00 Closed fracture of rib(s), unspecified
- 807.09 Closed fracture of multiple ribs, unspecified

ICD10

- M84.48XA Pathological fracture, other site, init encntr for fracture
- S22.39XA Fracture of one rib, unsp side, init for clos fx
- S22.49XA Multiple fractures of ribs, unsp side, init for clos fx

RING/CONSTRICTING BAND REMOVAL

Carl K. Hsu • Bradley Peckler

BASICS

DESCRIPTION

- *Primary constricting band*: A band tightened around an appendage causes swelling and pain (e.g., a hair knotted around a toddler's toe).
- *Secondary constricting band*: Injury or disease process that causes swelling and edema as a result of tightness against the band (e.g., impacted ring with an underlying fracture of the finger)
- Untreated, the constricting band may become *embedded* and interrupt skin integrity.
- Tourniquet syndrome occurs when anything causes a constriction and there is distal tissue effect.

Pediatric Considerations

In the preverbal child, a constricting band may be a manifestation of child abuse or neglect. It should also be considered as a cause of inconsolable crying.

Geriatric Considerations

The cognitively impaired nursing home resident or Alzheimer patient may be unable to give an indication of injury or pain.

ETIOLOGY

Tourniquet syndrome may result from allergic, dermatologic, iatrogenic, endocrinologic, infectious, malignant, metabolic, physiologic, or traumatic conditions, or it may be related to pregnancy.

DIAGNOSIS

SIGNS AND SYMPTOMS

- A constricting band with swollen tissue and skin of an appendage, most commonly involving a finger
- Other locations include wrist, ankle, toe, umbilicus, earlobe, nipple, septum or nares of nose, penis, scrotum, vagina, labia, uvula, or tongue.
- Pain on manipulation of the appendage or constricting band

History

Usually straightforward but in nonverbal populations it can be a cause of unidentified pain. An inconsolable crying infant may be having pain due to a hair tourniquet.

Physical-Exam

- Evaluate area of concern.
- If evaluating an inconsolable infant or agitated nonverbal adult, assess fingers, toes, and genitalia.

ESSENTIAL WORKUP

- Primary constricting band: Diagnosis made by history and physical exam with special attention to neurovascular status.
- Secondary constricting band: Diagnosis of underlying pathology may depend on results of imaging and lab test results.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Usually not indicated for acute treatment
- Measurement of electrolytes, BUN, and creatinine; thyroid function tests; and Tzanck smear of vesicular lesions may be useful in identifying the underlying diagnosis.

Imaging

Plain films for evaluation of underlying fracture or residual foreign body *after* band removal

DIFFERENTIAL DIAGNOSIS

Any condition causing marked swelling and edema predisposing to the tourniquet syndrome



TREATMENT

PRE HOSPITAL

Remove rings and other potential constricting bands before development of tourniquet syndrome:

- Particularly in regions of extremity trauma

INITIAL STABILIZATION/THERAPY

Pain management or procedural sedation as needed

ED TREATMENT/PROCEDURES

- Removal of the constricting band either by advancing the band distally or by division
- These adjuvant methods may be used alone or in combination:
 - Elevation of the affected extremity may decrease vascular congestion.
 - *Cooling* the extremity with ice or cold water may reduce edema and

erythema.

- *Lubrication* with soap or mineral oil may allow slippage over an inflamed or edematous area.
- *Digital block* with 1–2% lidocaine *without epinephrine* decreases the discomfort of removal and manipulation of an underlying injury.
- A digital block may; however, increase local swelling. Consider regional blocks.
- *Gauze* or a *needle holder* may be used to manipulate the band.
- The distal swollen finger, especially the proximal interphalangeal joint, is an important obstacle in constricting band removal.
- Distal to proximal edema reduction by sequential compression:
 - *Self-adherent tape* is wrapped from distal to proximal to form a smooth and decompressed area over which the band is advanced.
 - A *Penrose surgical drain* or a finger cut from a small glove is stretched to fit over the distal swelling before attempted removal.
 - With lubrication, the proximal end of the drain is pulled under the ring to form a cuff around the ring; the cuff with distal traction applied advances the band over the decompressed area.
 - *Suture material* (no. 0 silk, dental floss, or umbilical tape) is wrapped under tension in a tight layer advancing over the edema in a distal-to-proximal direction; the proximal tail of the suture material or floss is tucked under the ring; with lubrication, the tail under tension is pulled distally and unwound, forcing the ring over the layered suture material and decompressed area.
- Constricting band removal by division:
 - *Scissors* may be used to 1st lift and then cut the offending fibrous band constricting a toddler's toe or penis.
 - A *no. 11 scalpel* blade with cutting edge up may be sufficient to cut constricting bands formed by hair, fibers, or plastic ties.
 - A topical commercially available depilatory agent may be used to divide a tourniquet formed by a suspected hair obscured by local edema.
 - A *handheld wire cutter/stripper* may divide small-girth metallic rings with minimal discomfort to the underlying injury; this type of removal may; however, impart a crush defect to the ring, making repair difficult.
 - A *long-handled bolt cutter*, available in most operating rooms or hospital engineering departments, may be used to divide large-girth or broad-sized rings:
 - Long handles provide the significant mechanical advantage needed to cut large rings.
 - The reinforced cutting blades may not easily fit through a constricting band with adjacent swollen tissue and skin.
 - A *standard hand-powered, medically approved ring cutter* (Steinmann pin cutter with a MacDonald elevator) may be used to divide small-girth metallic constricting bands made of soft metals (gold/silver)

- This method has the advantage of a cleaner cut for subsequent repair of the ring.
- The disadvantage is that the handheld ring cutter is labor-intensive and may aggravate the pain of an underlying injury.
- A *motorized high-RPM cutting device* may be used to rapidly divide constricting bands irrespective of girth and size of the ring; it may be DC- or AC-powered or pneumatically driven in the operating suite.
- Cutting procedure:
 - The initial cut is made on the band on the volar aspect of the extremity.
 - A tenaculum may be used to spread the band in softer metals.
 - For a 2nd cut, the band should be rotated 180° on the extremity, allowing the 2nd cut on the band over the volar aspect of the extremity.
- Motorized cutting:
 - Remove *flammable solvents* from the work area.
 - *Protective eyewear* should be worn by everyone present, including the patient.
 - Place a thin *aluminum splint* (shaped to the curvature of the ring) between the patient's skin and the ring as a shield to protect underlying tissue.
 - Cool splint and cutting surface with ice water irrigations before and during the cutting procedure.
 - Limit cutting with motorized device to 5 sec with max. intervals of 60–90 sec between ice water irrigations to avoid producing local excessive heat.
- Depilatory cream:
 - Can be used if suspected constriction is caused by hair in place of “unwinding or excising” the hair.
 - Swelling of tissues heaped up around the hair may obscure the hair tourniquet leaving only a visible crease with the underlying hair buried below.
 - Depilatory cream applied to the crease may release the hair tourniquet within 10 min.
- Postdivision care:
 - Underlying injuries should be irrigated thoroughly to remove metallic dust and avoid foreign-body reaction and granuloma formation.
 - Tetanus prophylaxis should be provided if indicated.

MEDICATION

- Tetanus prophylaxis: Tetanus toxoid 0.5 mL IM
- No medications are typically required unless evidence of or at risk for infection

First Line

- Cefazolin: 1 g IV/IM (peds: 20–40 mg/kg IV/IM single dose in ED) and
- Cephalexin: 500 mg PO (peds: 25–50 mg/kg/d) QID for 7 days.
- Amoxicillin/clavulanate: 875/125 mg PO (peds: 25 mg/kg/d) BID for 7 days

- Erythromycin: 333 mg PO TID (peds: 40 mg/kg/d q6h for 7 days)

Second Line

- If patient is penicillin allergic:
- EES: 800 mg PO, then 400 mg PO q6h for 7 days *or*
- Clindamycin: 300 mg PO q6h for 7 days



FOLLOW-UP

DISPOSITION

Admission Criteria

- Neurovascular compromise or injury requiring surgical repair
- Concomitant infection or necrosis
- Investigation of abuse and or neglect

Discharge Criteria

Successful band removal with restoration of circulation.

Issues for Referral

Wounds at high risk for infection should have close follow-up in 1–2 days.

FOLLOW-UP RECOMMENDATIONS

Return to the ED for increasing pain, numbness, tingling, redness, swelling drainage, fevers, or other changes in clinical presentation.

PEARLS AND PITFALLS

- Failure to completely examine the fingers, toes, and genitalia of the irritable infant
- The hair causing a hair tourniquet may be obscured by edema and heaped up tissue and skin.
- Rings must be removed early after trauma to the distal extremity.

ADDITIONAL READING

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- Sung S, Hsu CK, O'Rourke K. Resident training in constricting band removal: Motorized cutting. *Ann Emerg Med*. 2007;50(3):Sup:S76.



CODES

ICD9

- 959.5 Finger injury
- 959.7 Knee, leg, ankle, and foot injury

ICD10

- S60.448A External constriction of other finger, initial encounter
- S60.449A External constriction of unspecified finger, initial encounter
- S90.446A External constriction, unspecified lesser toe(s), initial encounter

ROCKY MOUNTAIN SPOTTED FEVER

Roger M. Barkin

BASICS

DESCRIPTION

Rickettsial invasion of small blood vessels:

- Causes direct vascular damage
- Superimposed additional vascular damage/vasculitis due to immunologic phenomena

ETIOLOGY

- Acute infection by *Rickettsia rickettsii* via tick vector:
 - _ *Dermacentor andersoni* (wood tick) in the western states
 - _ *Dermacentor variabilis* (dog tick) in the eastern states
- Reported in all states; 1/2 of cases occur in 5 states (NC, SC, TN, OK, AR), as well as parts of Central America and South America
- More common April–September, but can occur any month
- More common in males and in individuals 40–64 yr of age

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Tick bite reported within 14 days of rash in 60% of patients
- Incubation varies 2–14 days with median 7 days
- Exposure to ticks, often in rural environment

Physical-Exam

- Rash:
 - _ Initial rash (3–5 days)
 - Macular, red, and flat
 - Blanches under pressure
 - 1–4 mm diameter
 - _ In hours to days:
 - Becomes darker, papular, dusky, and palpable
 - _ In 2–3 days:
 - Petechial or purpuric
 - Positive Rumpel–Leede test
 - May coalesce or ulcerate

- In severe disease, necrosis of dependent peripheral parts may occur.
- Location:
 - Begins in flexor surfaces of wrist and ankles, rapidly spreading to palms and soles
 - Spreads centripetally involving extremities; may involve trunk and face
 - 15% with centrifugal spread to palms and soles
 - 10% of patients do not have rash
 - Often not identified when patient initially presents for care
- Pulmonary:
 - Nonproductive cough
 - Chest pain
 - Dyspnea
 - Rales
- GI:
 - Often associated with fatal Rocky Mountain spotted fever
 - Secondary to vasculitis
 - Nausea/vomiting
 - Abdominal pain/distention
 - Ileus
 - Hepatosplenomegaly
- Neurologic:
 - Focal or generalized neurologic manifestation in 2/3
 - Meningismus
 - Severe, unremitting headache
 - Encephalitis
- Other:
 - Generalized edema
 - Dehydration
 - Malaise
 - Myalgia
 - Retinal hemorrhage and conjunctivitis
- Complications:
 - Disseminated intravascular coagulation (DIC)
 - Noncardiogenic pulmonary edema
 - Acute renal failure
 - Severe or fatal in advanced age, male sex, African American, chronic alcohol abuse, glucose-6-phosphate dehydrogenase deficiency

ESSENTIAL WORKUP

Clinical diagnosis supplemented by confirmatory lab findings such as hyponatremia, anemia, and thrombocytopenia

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Serology:
 - Diagnose by single titer $> 1:64$ or 4-fold increase. Antibody may not be detected in the 1st few days of symptoms
 - Methods:
 - Immunofluorescent antibody (sensitivity of 95%)
 - Complement fixation
 - Indirect hemagglutination test
 - Indirect immunofluorescence assay is reference standard.
- CBC:
 - Normal WBC count
 - Thrombocytopenia
 - Anemia
- Electrolytes, BUN/creatinine, glucose:
 - Hyponatremia < 130 mEq/L
- Liver profile:
 - Elevated aspartate aminotransferase
 - Lactate dehydrogenase
- Arterial blood gas for:
 - Hypoxia
 - Respiratory alkalosis
- Coagulation profile if DIC suspected
- Microbiology:
 - Immunohistologic antibody stain of skin biopsy
 - Isolation of *R. rickettsii* (time-consuming/ expensive)
 - Polymerase chain reaction assay
- CSF:
 - Pleocytosis and increased protein

Imaging

- Chest radiograph for pulmonary edema, pneumonia
- Echocardiography:
 - Decreased left ventricular contractility

Diagnostic Procedures/Surgery

Skin biopsy may be confirmatory if immunohistologic antibody studies available.

DIFFERENTIAL DIAGNOSIS

- Other tick-borne diseases:
 - Ehrlichiosis: Older adults

- Relapsing fever
- Lyme disease: Erythema chronicum migrans
- Tularemia
- Babesiosis
- Colorado tick fever
- Infectious diseases:
 - Meningococemia—late winter, early spring; maculopapular or petechial rash
 - Measles—late winter, early spring; severe prodrome
 - Rubella—palms and soles spared
 - Varicella—does not have rash in extremities
 - Viral exanthem
 - Infectious mononucleosis—palms and soles spared
 - Disseminated gonococcal infection—pustular lesions
 - Typhus—rash starts at trunk with centrifugal spread
 - Secondary syphilis
 - Scarlet fever
 - Kawasaki disease—red, cracked lips
 - Toxic shock syndrome
 - Gastroenteritis
 - Staphylococcal sepsis
- Inflammatory causes:
 - Allergic vasculitis
 - Thrombotic thrombocytopenic purpura
 - Collagen vascular disease
 - Juvenile rheumatoid arthritis
- Heat illness



TREATMENT

PRE HOSPITAL

Stabilize as appropriate

INITIAL STABILIZATION/THERAPY

- ABC management
- 0.9% NS IV fluid bolus for dehydration
- Oxygen for hypoxia

ED TREATMENT/PROCEDURES

- Correct fluid and electrolyte deficits.
- Initiate antibiotic therapy immediately based on clinical and epidemiologic findings. Should not be delayed until lab confirmation is obtained:

- Doxycycline—drug of choice
- Chloramphenicol in pregnant and allergic patients
- Sulfonamides make infection worse.
- Administer acetaminophen for fever.
- Consider high-dose steroids for severe cases complicated by extensive vasculitis, encephalitis, or cerebral edema (controversial).
- Better outcome in children if treatment begins before day 5 of illness
- Treat complications:
 - DIC
 - Adult respiratory distress syndrome
 - CHF
- Medication

Pediatric Considerations

- Highest incidence in 5–9 yr olds
- 2/3 of cases occur in children < 15 yr.
- Doxycycline is used in children due to potential for fatal cases, the relatively low risk of significant dental discoloration with a short course, and adverse effects of chloramphenicol

Pregnancy Considerations

Use chloramphenicol in pregnant patients.

MEDICATION

First Line

Doxycycline: 100 mg (peds: 2 mg/kg for <45 kg) PO or IV BID for 5–7 days. Patient should generally be treated 2–3 days beyond becoming afebrile.

Second Line

- Acetaminophen: 500 mg (peds: 10–15 mg/kg/dose) PO q4h; do not exceed 5 doses/24 h or 4 g/24 h
- Chloramphenicol: 75 mg/kg/24 h PO or IV q6h for 5–7 days and 48 hr after defervescence
- Solu-Medrol: 125 mg (peds: 1–2 mg/kg) IV



FOLLOW-UP

DISPOSITION

Admission Criteria

Moderate to severe symptoms

Discharge Criteria

- Mild, early disease with early treatment
- Notify family because of clustering and potential exposures.

Issues for Referral

Reflective of defined complications

FOLLOW-UP RECOMMENDATIONS

Reflective of ongoing complications

PEARLS AND PITFALLS

Early treatment based on the clinical presentation and epidemiology is indicated.

ADDITIONAL READING

- Buckingham SC, Marshall GS, Schutze GE, et al. Clinical and laboratory features, hospital course, and outcome of Rocky Mountain spotted fever in children. *J Pediatr*. 2007;150:180–184.
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- Masters EJ, Olson GS, Weiner SJ, et al. Rocky Mountain spotted fever: A clinician's dilemma. *Arch Intern Med*. 2003;163:769–774.



CODES

ICD9

082.0 Spotted fevers

ICD10

A77.0 Spotted fever due to *Rickettsia rickettsii*

ROSEOLA

Moses S. Lee

BASICS

DESCRIPTION

- Exanthem subitum
- Incubation period of 5–15 days
- Mode of acquisition unknown:
 - Horizontal spread by oral shedding suggested
 - It is spread person to person but is not very contagious.
 - Human is the only host.
- Pathophysiology:
 - Complex immune response (cytokines, antibody responses, T-cell reactivity)

ETIOLOGY

- Human herpesvirus 6 (HHV-6):
 - Large, double-stranded DNA
 - Closely related to human cytomegalovirus
- Peak incidence at 6–12 mo; 90% occurrence within 1st 2 yr
- Highest incidence in late spring and early summer

DIAGNOSIS

SIGNS AND SYMPTOMS

- Usually self-limited
- Diarrhea
- Irritability
- Rarely causes severe or fatal disseminating diseases:
 - Infectious mononucleosis syndrome of hepatitis
- Complications
 - Febrile seizures in 5–35%
 - Aseptic meningitis/encephalopathy
 - Thrombocytopenic purpura
- Reactivation in immunocompromised individuals. Manifestations are fever, rash, hepatitis, bone marrow suppression, pneumonia, and encephalitis

Pediatric Considerations

- Most newborns are seropositive for HHV-6 due to transplacental antibodies.
- By age 1–2 yr, >90% of infants are seropositive.

History

- Classic history is the onset of sudden, high fever 39.4–41.2°C (103–106°F) commonly followed by defervescence and the appearance of rash
- Absence of physical findings:
 - Child looks well
 - Temperature normalizes in 3–4 days
 - Irritability and anorexia may be present
 - Bulging fontanelle may be noted

Physical-Exam

- Enlarged lymph nodes
- Maculopapular eruption from trunk to arms and neck after temperature normalizes
- Rash fades within 3 days.
- Erythematous papules in pharynx (Nagayama spots)
- Otitis media is common
- Cervical and postoccipital lymphadenopathy

ESSENTIAL WORKUP

Clinical diagnosis:

- High fever in well-appearing child

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC:
 - Initial increase in WBC, then normalization with lymphocytosis; WBC may decrease 3–5 days after onset of illness
 - Platelets may be decreased
- HHV-6 DNA:
 - Detected by polymerase chain reaction
 - Available at research level
 - IgM appears early and declines as IgG is produced
 - May be done on blood and CSF
- CSF if concern about meningitis

DIFFERENTIAL DIAGNOSIS

- Fever of unknown origin
- Scarlet fever:
 - “Sandpaper” rash, Pastia lines, and strawberry tongue
- Measles (rubeola):
 - Koplik spots, cough, coryza, conjunctivitis, and fever
- Rocky Mountain spotted fever:

- Rash begins at ankles and wrists.
- Rubella:
 - Fever after rash
- “Fifth disease” (erythema infectiosum)
- Dengue fever
- Pneumococcal bacteremia
- Meningitis, especially with bulging fontanelle



TREATMENT

PRE HOSPITAL

None

INITIAL STABILIZATION/THERAPY

ABC management

ED TREATMENT/PROCEDURES

- Supportive
- Antipyretics:
 - Acetaminophen
 - Ibuprofen

MEDICATION

- Acetaminophen: 500 mg (peds: 15 mg/kg/dose) PO q4h; do not exceed 5 doses/24 h or 4 g/24 h
- Ibuprofen: 200–600 mg (peds: 5–10 mg/kg PO q6–8h); suspension 100 mg/5 mL; oral drops 40 mg/mL



FOLLOW-UP

DISPOSITION

Admission Criteria

Fever in child who is toxic and does not respond to initial supportive care

Discharge Criteria

Usually, all patients may be discharged. Usually may not return to daycare until rash has resolved

FOLLOW-UP RECOMMENDATIONS

Re-evaluate if persistent fever after 3–4 days

PEARLS AND PITFALLS

- Child looks well
- Antivirals are not recommended in the immunocompetent child.
- Febrile seizures need appropriate evaluation.

ADDITIONAL READING

- American Academy of Pediatrics. *Report of the Committee on Infectious Diseases*. 29th ed. Elk Grove, IL: American Academy of Pediatrics; 2012.
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- Prober CG. Human herpesvirus 6. In: *Hot Topics in Infection and Immunity in Children VII. Advances in Experimental Medicine and Biology*. New York, NY: Springer; 2011:87–90.



See Also (Topic, Algorithm, Electronic Media Element)

- Fever, Pediatric
- Rash, Pediatric
- Seizures, Febrile



CODES

ICD9

- 058.10 Roseola infantum, unspecified
- 058.11 Roseola infantum due to human herpesvirus 6

ICD10

- B08.20 Exanthema subitum [sixth disease], unspecified
- B08.21 Exanthema subitum [sixth disease] due to human herpesvirus 6

RUBELLA

Moses S. Lee

BASICS

DESCRIPTION

- Also known as German measles or 3-day measles
- Transmission via droplets from respiratory secretions
- Moderately contagious:
 - Especially during rash eruption and infants with congenital rubella syndrome (CRS)
- Up to 50% may be subclinical.
- Infants with congenital rubella shed large quantities of virus for several months.
- Infectious period 7 days before to 5 days after appearance of rash
- Incubation period: 14–21 days

ETIOLOGY

- Rubella virus (family: *Togaviridae*, genus: *Rubivirus*)
- Live, attenuated virus vaccine indications:
 - All children > 12 mo and entering school
 - All women of childbearing age

DIAGNOSIS

SIGNS AND SYMPTOMS

- Acute viral disease
- Complications:
 - Uncommon, tend to occur more in adults
 - CRS: Infected women in 1st trimester (hearing loss, mental retardation, cardiovascular defect, ocular defect)
 - Arthritis:
 - More common in women (up to 79%)
 - Chronic arthritis is rare.
 - Begins after 2–3 days of illness
 - Knees, wrists, fingers affected
 - Hemorrhagic manifestations:
 - Secondary to thrombocytopenia
 - More common in children
 - Neurologic sequelae:
 - Encephalitis most common in adults; prognosis usually good
 - No causal relationship to autism

History

- Low-grade fever
- Malaise
- Headache
- Upper respiratory tract symptoms

Physical-Exam

- Rash:
 - Rash is fainter than measles rash and does not coalesce.
 - Red macular rash evolving to pink-red maculopapules with occasional pruritus
 - Begins in face with rapid caudal spread
 - Completed in 1st day and disappears in 3 days
 - May have hemorrhagic manifestations
- Lymphadenopathy:
 - Postauricular
 - Occipital
 - Posterior cervical

ESSENTIAL WORKUP

Generally clinical diagnosis

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC:
 - Decreased WBC, platelets (more common in children)
- Urinalysis:
 - Hematuria
- Reverse transcriptase–polymerase chain reaction
- ELISA to detect rubella IgM
- Rubella antibody titer:
 - Acute and convalescent serum specimens
 - Hemagglutination-inhibition test most common
 - Rubella specific IgM antibodies using enzyme immunoassay (EIA) commercially available. Detectable 4 days after onset of rash
 - Definitive diagnosis in acute infection
 - Compare infant with maternal sera for CRS.
 - False positives in parvovirus, infectious mononucleosis, rheumatoid factor
 - May be useful to check for immunity of pregnant patients with potential exposure.
- Pharynx:
 - Virus may be isolated from pharynx 1 wk before and until 2 wk after rash

onset (valuable epidemiologic tool).

- CSF:
 - Few WBCs (monocytes) in encephalitis

Diagnostic Procedures/Surgery

- Lumbar puncture if suspected encephalitis
- Arthrocentesis in unexplained arthritis.

DIFFERENTIAL DIAGNOSIS

- Scarlet fever:
 - “Sandpaper” rash, Pastia lines, and strawberry tongue
- Measles (rubeola):
 - Koplik spots, cough, coryza, conjunctivitis, and fever
- Roseola infantum:
 - Spring and fall
- Rocky Mountain spotted fever:
 - Rash begins at ankles and wrists.
- Rheumatoid arthritis



TREATMENT

PRE HOSPITAL

Use N95 filter mask for potential respiratory transmission.

INITIAL STABILIZATION/THERAPY

ABC management

ED TREATMENT/PROCEDURES

- Symptomatic therapy
- Antipyretics and anti-inflammatory agents:
 - Acetaminophen
 - Ibuprofen
- Isolate rubella patients from susceptible persons (e.g., pregnancy).
- Vaccine:
 - Measles, mumps, and rubella vaccine
 - Rubella vaccine is live attenuated virus.
 - Indications:
 - > 12 mo and entry to school
 - Susceptible postpubertal females
 - High-risk groups (colleges, military, places of employment)
 - Unimmunized contacts
 - Healthcare workers and women of childbearing age born after 1957

- Nonpregnant women may have arthralgia in up to 25%
- Contraindicated in pregnant women
- Avoid pregnancy for 3 mo after vaccination.
- 1 dose confers probable lifelong protection.
- Common complaints are fever, lymphadenopathy, and arthralgia.
- Immunoglobulin:
 - Will not prevent viremia but may modify symptoms

MEDICATION

- Acetaminophen: 500 mg (peds: 15 mg/kg/dose) PO q4h; do not exceed 5 doses/24 h or 4 g/24 h
- Ibuprofen: 200–600 mg (peds: 5–10 mg/kg PO q6–8h); suspension 100 mg/5 mL; oral drops 40 mg/mL
- Immunoglobulin: 0.5 mL reconstituted vial SC (0.25–0.50 mL/kg)



FOLLOW-UP

DISPOSITION

Admission Criteria

- CRS
- Encephalitis

Discharge Criteria

- Most patients may go home.
- Inquire regarding vaccination status of family members.

Issues for Referral

- Potential exposure or disease in pregnant women
- Complications
- CRS-suspected child will need comprehensive evaluation.

FOLLOW-UP RECOMMENDATIONS

Pregnant women with suspected rubella or exposure must be followed with titers and counseling should have obstetric consult.

PEARLS AND PITFALLS

- Current literature does not support a causal relationship between childhood vaccination with thimerosal-containing vaccines and development of autism-spectrum disorders.
- Infected individual should be isolated from susceptible (pregnancy,

immunocompromised) individual for 7 days.

ADDITIONAL READING

- American Academy of Pediatrics. *Report of the Committee on Infectious Diseases*. 29th ed. Elk Grove, IL: American Academy of Pediatrics; 2012.
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- Mason WH. Rubella. In: Kliegman RM, Behrman RE, Jenson HB, et al., eds. *Nelson Textbook of Pediatrics*. 18th ed. Philadelphia, PA: WB Saunders; 2007:1337–1340.



CODES

ICD9

- 056.9 Rubella without mention of complication
- 647.50 Rubella in the mother, unspecified as to episode of care or not applicable
- 771.0 Congenital rubella

ICD10

- B06.9 Rubella without complication
- O35.3XX0 Maternal care for (suspected) damage to fetus from viral disease in mother, not applicable or unspecified
- P35.0 Congenital rubella syndrome

SACRAL FRACTURE

Allan V. Hansen • Jaime B. Rivas

BASICS

DESCRIPTION

- They occur in 45% of all pelvic fractures and are rarely isolated
- They are defined by the orientation of the fracture line.
- Mechanism:
 - Axial compression
 - Direct posterior trauma
 - Massive crush injury
 - Insufficiency fractures in elderly and osteoporotic patients

Fracture Classification

Transverse

- Above S4:
 - Neurologic injury common
 - Can see cauda equina syndrome (CES)
- Below S4:
 - Associated rectal tears
 - Neurologic injury is rare

Vertical

- *Lateral to sacral foramina:*
 - Sciatica
 - L5 root injury
 - Neurologic deficit infrequent
- *Foraminal (zone 2):*
 - Bowel/bladder dysfunction
 - L5, S1, S2 root injury
 - Neurologic deficit frequent
- *Canal (zone 3):*
 - Bowel/bladder dysfunction
 - Sexual dysfunction
 - L5, S1 root injury
 - Neurologic deficit often present (> 50%)

ETIOLOGY

- Transverse: Fall from height, flexion injuries, direct blow
- Vertical: Usually high-energy mechanism

Geriatric Considerations

Sacral insufficiency fractures should be considered in elderly patients with severe back pain

DIAGNOSIS

SIGNS AND SYMPTOMS

- Pain in buttocks, perirectal area, and posterior thigh
- Swelling and ecchymosis over the sacral prominence
- Possible sacral nerve dysfunction:
 - Absent or diminished anal sphincter tone is an important finding.
 - Bowel or bladder incontinence

ESSENTIAL WORKUP

- History and physical exam with attention to loss of anal sphincter tone, sensation in the perineum, and bowel and bladder sphincter control.
- Sacral fractures rarely occur in isolation; look for associated injuries.
- Rectal exam will elicit pain in the sacrum; blood in the rectum suggests an open fracture.

DIAGNOSIS TESTS & INTERPRETATION

Imaging

- Only 30% of sacral fractures are detected on plain radiograph.
- CT provides optimal imaging to identify sacral fractures.
- MRI is indicated when neurologic dysfunction is present.

DIFFERENTIAL DIAGNOSIS

- Contusion
- Lumbar spine fracture
- Pelvic fractures

TREATMENT

PRE HOSPITAL

- Sacral fractures are frequently associated with other spinal and intra-abdominal injuries.
- Immobilize with backboard and C-spine collar.

INITIAL STABILIZATION/THERAPY

- Manage ABCs as needed.
- Early immobilization in unstable pelvis or spine fractures

- Pain control with NSAIDs or narcotic analgesics

ED TREATMENT/PROCEDURES

- Vertical unstable fractures require a rapid and thorough assessment for life-threatening injuries as well as orthopedic consultation (see “Pelvic Fracture”).
- Nondisplaced isolated transverse sacral fractures are treated symptomatically with touch-down weight bearing on affected side and early orthopedic referral.
- Surgery is often required for fractures associated with neurologic injury.

MEDICATION

First Line

Analgesia as indicated



FOLLOW-UP

DISPOSITION

Admission Criteria

- Critically injured trauma patient with unstable pelvic fracture
- Neurologic impairment requires orthopedic consultation.

Discharge Criteria

- Isolated nondisplaced sacral fractures
- Consider intermediate or assisted-care setting for elderly patients.

FOLLOW-UP RECOMMENDATIONS

- Only nondisplaced, transverse fractures are appropriate for outpatient follow-up
- Prompt surgical evaluation is indicated for displaced fractures.

PEARLS AND PITFALLS

- Sacral fractures are rarely isolated; consider associated pelvic fractures.
- Detailed neurologic exam, including rectal sphincter tone and perianal sensation, is indicated to assess for associated sacral nerve root injury.
- Foley catheter in a trauma patient may mask voiding problems from sacral nerve root injury.

ADDITIONAL READING

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overlooked cause of back pain in the ED. *Am J Emerg Med.* 2011;29(3):359.e5–e6.

- Hak DJ, Baran S, Stahel P. Sacral fractures: Current strategies in diagnosis and management. *Orthopedics.* 2009;32:752–757.



See Also (Topic, Algorithm, Electronic Media Element)

Pelvic Fracture



CODES

ICD9

- 733.13 Pathologic fracture of vertebrae
- 805.6 Closed fracture of sacrum and coccyx without mention of spinal cord injury
- 806.62 Closed fracture of sacrum and coccyx with other cauda equina injury

ICD10

- M84.48XA Pathological fracture, other site, init encntr for fracture
- S32.10XA Unsp fracture of sacrum, init encntr for closed fracture
- S32.14XA Type 1 fracture of sacrum, init encntr for closed fracture

SALICYLATE POISONING

Michele Zell-Kanter



BASICS

DESCRIPTION

- Respiratory alkalosis and metabolic acidosis:
 - Secondary to inhibition of Krebs cycle and uncoupling of oxidative phosphorylation
- Dehydration, hyponatremia or hypernatremia, hypokalemia, hypocalcemia:
 - Owing to increased sweating, vomiting, tachypnea
- Noncardiogenic pulmonary edema:
 - Because of toxic effect of salicylate on pulmonary endothelium resulting in extravasation of fluids
- Salicylate pharmacokinetics change from first order to zero order in overdose setting; i.e., a small dosage increment results in a large increase in salicylate concentration.

Geriatric Considerations

- Greater morbidity
- Respiratory distress/altered mental status indicative of severe toxicity
- Diagnosis of salicylate intoxication delayed because underlying disease states mask signs and symptoms; e.g., CHF

Pediatric Considerations

- Children exhibit faster onset and more severe signs and symptoms than adults:
 - Results from salicylate being distributed more quickly into target organs such as brain, kidney, and liver
- Respiratory alkalosis (hallmark of salicylate poisoning in adults) may not occur in children.
- Metabolic acidosis occurs more quickly in children than in adults.
- Hypoglycemia more common than hyperglycemia
- Ingestion of more than “a taste” of oil of wintergreen (98% methyl salicylate) by children < 6 yr or > 4 mL of oil of wintergreen by patients > 6 yr warrants ED assessment.

ETIOLOGY

Sources of salicylate:

- Aspirin:
 - Ingestion of > 150 mg/kg can cause serious toxicity
- Oil of wintergreen:

- Any exposure should be considered dangerous.
- Bismuth subsalicylate
- Salicylsalicylic acid (salsalate)



DIAGNOSIS

SIGNS AND SYMPTOMS

- GI:
 - Nausea
 - Vomiting
 - Epigastric pain
 - Hematemesis
- Pulmonary:
 - Tachypnea
 - Noncardiogenic pulmonary edema
- CNS:
 - Tinnitus
 - Deafness
 - Delirium
 - Seizures
 - Coma

History

- Ask if taking aspirin or aspirin products:
 - Many patients do not list aspirin among their regular medications, may not consider aspirin a medication.
- Patients may not know the difference between aspirin, acetaminophen, and the OTC NSAIDs

ESSENTIAL WORKUP

- Salicylate level:
 - At presentation and then q2h until level begins to decline
 - Verify that units are correct, generally mg/dL.
- Watch for recurrence of signs of salicylate toxicity and increasing levels even after levels have declined due to intestinal absorption of enteric-coated products and salsalate

Guidelines for Assessing Severity of Salicylate Poisoning

- Acute ingestion of:
 - < 150 mg/kg or < 6.5 g of aspirin equivalent—considered nontoxic
 - 150–300 mg/kg—mild to moderately toxic
 - > 300 mg/kg—potentially lethal
- In the chronic overdose setting:

- Manage patient on clinical findings and not solely on levels
- Clinical findings are better indication of severity than plasma salicylate levels
- No valid nomogram exists for salicylate level interpretation
- Salicylate levels needed to achieve anti-inflammatory effect (20–25 mg/dL) approach toxic levels
- Enteric-coated aspirin absorbed in intestine; peak level delayed

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Arterial blood gas (ABG):
 - Respiratory alkalosis
 - Metabolic acidosis
- CBC
- Electrolytes, BUN/creatinine, glucose:
 - Anion-gap metabolic acidosis
 - Hypokalemia
 - Baseline renal function
- Urinalysis:
 - Urine pH
- PT/PTT with significant ingestions
- Ferric chloride test:
 - Purple if salicylate present
 - Positive 30 min postingestion
- In the presence of salicylate, Phenistix turn brown-purple; may detect concentrations as low as 20 mg/dL

Imaging

- Abdominal flat-plate radiograph for concretions
- Chest radiograph for pulmonary edema

DIFFERENTIAL DIAGNOSIS

- Acute salicylate poisoning:
 - Consider with change in mental status, unexplained noncardiogenic pulmonary edema, mixed acid–base disorder.
 - Methanol
 - Ethylene glycol
 - Conditions causing noncardiogenic pulmonary edema
- Chronic salicylate poisoning:
 - Impending myocardial infarction
 - Alcohol withdrawal
 - Organic psychoses

- Sepsis
- Dementia



TREATMENT

PRE HOSPITAL

In suspected overdose settings, medication bottles must be brought in for review

INITIAL STABILIZATION/THERAPY

- Management of airway, breathing, and circulation (ABCs)
- Naloxone, thiamine, glucose (or Accu-Chek) for altered mental status
- IV rehydration with 0.9% normal saline (NS) for hypotension

ED TREATMENT/PROCEDURES

- Morbidity from chronic salicylate poisoning may be greater than from acute poisoning.
- Aggressively manage all salicylate intoxication.

Gastric Decontamination

- Administer activated charcoal in alert patients.
- Whole-bowel irrigation of theoretical benefit:
 - For concretions visible on abdominal radiograph
 - For ingestion of sustained-release preparation
 - If salicylate levels continue to increase despite appropriate management
 - Do not use in patients who may develop altered mental status

Enhanced Elimination

- Alkalinization:
 - Enhances elimination of ionized salicylate
 - Indications:
 - Acidosis
 - Presence of symptoms
 - Elevated salicylate levels
 - 1 or 2 ampules of sodium bicarbonate followed by IV D₅W 1L with 3 ampules of sodium bicarbonate:
 - Goal: Urine pH of 7.5–8 at the rate of 3–6 mL/kg/h
 - Add 20–40 mEq KCl per liter to avoid hypokalemia
 - Avoid fluid overload with CHF or CAD
 - Closely monitor serum potassium
- Indications for hemodialysis include:
 - CHF
 - Noncardiogenic pulmonary edema

- CNS depression
- Seizures
- Unstable vital signs
- Severe acid–base disorder
- Hepatic compromise
- Coagulopathy
- Underlying disease state compromising elimination of salicylate
- Absolute salicylate level should not be used as sole criterion for deciding to dialyze without considering patient’s clinical status unless level is $> 80\text{--}100$ mg/dL in acute ingestion.
- Threshold to dialyze is lower in patients with chronic overdose.

MEDICATION

- Activated charcoal slurry: 1–2 g/kg up to 90 g PO
- Dextrose: D₅₀W 1 amp (50 mL or 25 g) (peds: D₂₅W 2–4 mL/kg) IV
- Naloxone (Narcan): 2 mg (peds: 0.1 mg/kg) IV or IM initial dose
- Thiamine (vitamin B₁): 100 mg (peds: 50 mg) IV or IM



FOLLOW-UP

DISPOSITION

Admission Criteria

- Monitor patients with salicylate levels > 25 mg/dL until level drops < 25 mg/dL and symptoms abate.
- Salicylate levels increasing after having trended downward to nontoxic levels:
 - In patients who ingest sustained-release aspirin, enteric-coated aspirin, and any aspirin product with delayed absorption
- ICU admission for altered mental status, metabolic acidosis, pulmonary edema

Discharge Criteria

Repetitive salicylate levels < 25 mg/dL and resolution of symptoms

FOLLOW-UP RECOMMENDATIONS

- Psychiatric referral for intentional ingestions
- Close primary care follow-up for chronic ingestions

PEARLS AND PITFALLS

- Patients need to maintain their respiratory drive to reverse acidemia, respiratory acidosis:
 - Do not intubate prematurely.

- It is extremely difficult to achieve and maintain mechanical hyperventilation in these patients.
- Salicylate poisoning may result from topical exposure to salicylate-containing lotions or creams, rectal suppositories, oral antidiarrheal preparations.
- Salicylate levels may trend downward only to begin increasing again due to absorption of product from the intestine or from a salicylate bezoar in the gut.

ADDITIONAL READING

- Kent K, Ganetsky M, Cohen J, et al. Non-fatal ventricular dysrhythmias associated with severe salicylate toxicity. *Clin Toxicol (Phila)*. 2008;46:297–299.
- Stolbach AI, Hoffman RS, Nelson LS. Mechanical ventilation was associated with acidemia in a case series of salicylate-poisoned patients. *Acad Emerg Med*. 2008;15:866–869.
- West PL, Horowitz BZ. Delayed recrudescence to toxic salicylate concentrations after salicylate overdose. *J Med Toxicol*. 2010;6:150–154.



CODES

ICD9

- 276.2 Acidosis
- 276.3 Alkalosis
- 965.1 Poisoning by salicylates

ICD10

- E87.2 Acidosis
- E87.3 Alkalosis
- T39.011A Poisoning by aspirin, accidental (unintentional), init

SARCOIDOSIS

Maureen L. Joyner • Jesse B. Cannon

BASICS

DESCRIPTION

- Chronic, multisystem disorder characterized by local accumulation of T lymphocytes and mononuclearphagocytes forming noncaseating epithelioid granulomas
- Symptoms mainly due to organ dysfunction due to disruption of local tissue architecture:
 - Predominance of lung symptoms
- ACE and Vitamin D levels may be increased due to secretion from granulomatous tissue
- Prevalence 10–20/100,000 in US and Europe
- Affects almost all races and geographic locations
- Symptoms typically begin in patients 10–40 yr of age
- 2.4% lifetime risk to blacks in US, relative to whites at 0.85%

ETIOLOGY

Unclear, but appears to be an overly robust cell-mediated immune response to unidentified self- or nonself antigen(s)

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Constitutional:
 - Fatigue, general weakness
 - Fever
- Skin (25% patients):
 - Rash, lesions
- Cardiac/respiratory (most patients):
 - Dyspnea
 - Chest pain
 - Palpitations
 - Cough
 - Hemoptysis
- Neurologic:
 - Nerve palsy (usually CN VII)

- Seizure
- Altered mental status
- Ocular (20% patients):
 - Eye pain
 - Blurred vision
- Renal:
 - Flank pain
- Musculoskeletal:
 - Arthralgias

Physical-Exam

- Constitutional:
 - Fever
 - Lethargy
- Skin:
 - Erythema nodosum
 - Subcutaneous nodules
 - Maculopapules
 - Plaques
 - Infiltrative scars
 - Lupus pernio
- EENT:
 - Uveitis
 - Keratoconjunctivitis
 - Parotid gland enlargement
- Neurologic:
 - Nerve palsy (usually CN VII)
- Respiratory:
 - Rales
 - Rarely wheezing
- Cardiac (~ 5% patients):
 - Dysrhythmias, conduction abnormalities, AV block
 - CHF (due to restrictive cardiomyopathy)
 - Murmurs (due to papillary muscle dysfunction)
- Renal:
 - Nephrolithiasis
- Musculoskeletal:
 - Polyarthralgias
- Löfgren syndrome:
 - Bilateral hilar adenopathy
 - Erythema nodosum
 - ± Polyarthralgias
- Heerfordt–Waldenström syndrome:

- Fever
- Uveitis
- Parotid gland enlargement
- ± CN VII palsy

Pediatric Considerations

- Children < 4 yr old classically present with triad of rash, uveitis, and arthritis.
- Children ≥ 4 yr old present similarly to adults.

ESSENTIAL WORKUP

- Physical exam with emphasis on lung, skin, eye, heart, and musculoskeletal
- Pulse oximetry/ABG
- ECG (dysrhythmias, conduction delays)
- Slit-lamp eye exam

DIAGNOSIS TESTS & NTERPRETATION

Lab

- Serum ACE elevated in 75% cases
- Basic chemistry panel
- LFTs: Mild, usually asymptomatic, mainly elevated alk phos but possible mild elevation transaminases
- Serum calcium: Hypercalcemia due to excessive vitamin D
- UA: Hypercalciuria
- Hypergammaglobulinemia
- CSF analysis: Lymphocyte predominance, elevated ACE level

Imaging

Chest radiograph (abnormal in 90% sarcoid patients)—reason for frequent incidental diagnosis:

- Type 1: Bilateral hilar lymphadenopathy
- Type 2: Lymphadenopathy and parenchymal lung changes (reticular opacities)
- Type 3: Parenchymal lung changes without hilar lymphadenopathy
- Type 4: Reticular opacities, pulmonary fibrosis; particularly in upper lobes
- Radiotracer scans may identify granulomatous disease but is nonspecific

Diagnostic Procedures/Surgery

- Biopsy:
 - Bronchoscopy and bronchoalveolar lavage
 - Skin lesions if feasible
- Kveim–Siltzbach test:
 - Subcutaneous injection of antigen with subsequent spleen biopsy
 - Rarely used

DIFFERENTIAL DIAGNOSIS

- HIV
- Interstitial lung disease
- Lymphoma
- Mycobacterial infection
- Parathyroid disease



TREATMENT

PRE HOSPITAL

Provide supplemental oxygen.

INITIAL STABILIZATION/THERAPY

- Provide supplemental oxygen.
- Monitor for dysrhythmias.

ED TREATMENT/PROCEDURES

- Patients should be observed without therapy, if possible, since disease resolves spontaneously in 50% patients.
- Initiate steroids in patients demonstrating 1 of the following:
 - Symptomatic or progressive stage II pulmonary disease
 - Stage III pulmonary disease
 - Malignant hypercalcemia
 - Severe ocular disease
 - Neurologic sequelae
 - Nasopharyngeal/laryngeal involvement
- Consider topical corticosteroids and cycloplegic agents for anterior uveitis or dermatologic manifestations.

MEDICATION

- Prednisone: 10–80 mg (peds: 0.5–2 mg/kg) PO QD
- Lower doses for hypercalcemic nephropathy and mild to moderate disease
- Higher doses for neurosarcoïdosis



FOLLOW-UP

DISPOSITION

Admission Criteria

- Hypoxia
- Patients with moderate to severe respiratory symptoms
- Significant cardiac conduction delays

- Severe thrombocytopenia

Discharge Criteria

Follow-up is established.

Issues for Referral

- Cardiology:
 - For any conduction disturbances or CHF
- Rheumatology:
 - For routine care and follow-up:
 - ~q2mo for patients with active disease on steroids, q3–4mo for asymptomatic patients
- Pulmonary:
 - For formal pulmonary function testing (to monitor for progression of restrictive lung disease) with spirometry and DLCO
- Ophthalmology:
 - Within 48 hr for acute uveitis

FOLLOW-UP RECOMMENDATIONS

- Restrict excess calcium from the diet.
- Monitor for complications related to chronic steroid therapy

PEARLS AND PITFALLS

- Evaluate patients with chest radiographs to determine stage and progression of disease.
- Prednisone is treatment of choice for exacerbations of disease.
- Monitor for signs of hypercalcemia and related complications.
- Be aware of acute neurologic and ocular sequelae.

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- Iannuzzi MC, Rybicki BA, Teirstein AS. *Sarcoidosis*. *N Engl J Med*. 2007;357:2153–2165.
- King TE. Sarcoidosis. Updated Apr 18, 2011. Available at www.UpToDate.com. Accessed on January 2013.



See Also (Topic, Algorithm, Electronic Media Element)

- Dyspnea
- HIV/AIDS

- Hyperparathyroidism
- Tuberculosis



CODES

ICD9

- 135 Sarcoidosis
- 517.8 Lung involvement in other diseases classified elsewhere

ICD10

- D86.0 Sarcoidosis of lung
- D86.3 Sarcoidosis of skin
- D86.9 Sarcoidosis, unspecified

SCABIES

James Q. Hwang

BASICS

DESCRIPTION

- Mites mate on skin surface and gravid female burrows into stratum corneum to lay eggs:
 - Animal scabies can burrow but cannot reproduce on human hosts
- Symptoms result from delayed type IV hypersensitivity reaction to mite, eggs, saliva, and feces:
 - Inflammatory reaction leads to intense pruritus, which is the hallmark of the disease
 - Crusted Norwegian scabies is characterized by large numbers of mites and is seen in the immunocompromised, disabled, and institutionalized:
 - More infectious than ordinary scabies due to high mite count
- Despite > 2,500-yr existence, an effective way to prevent scabies is still not known
- Secondary infection is common and, as such, the morbidity associated with scabies may be underestimated
- Scabies is a major global health problem in many crowded, resource-poor communities
- Infestations become secondarily infected and epidemic acute poststreptococcal glomerulonephritis and rheumatic heart disease are often associated with endemic scabies

Pediatric Considerations

- Scabies manifests itself in various forms in children and differs from that in adults:
 - More inflammatory (vesicular or bullous)
 - Involvement of face, scalp, palms, or soles
- Highest prevalence is in children < 2 yr old

ETIOLOGY

- Epidemiology:
 - Over the past 2 decades, the number of patients with scabies is increasing
 - Up to 300 million cases yearly
 - Burden of disease is highest in tropical countries
- Produced by the human scabies mite, *Sarcoptes scabiei* var. *hominis*, or from animal mites
- Transmitted by prolonged (15–20 min) direct skin-to-skin contact or, less commonly, by infested bedding or clothing:
 - It is a disease of overcrowding and poverty, rather than a reflection of poor

hygiene

- Probability of being infected is related to number of mites on infected person and length of contact
- Family members, sexual contacts, and institutional settings are at high risk for transmission
- Schools do not ordinarily provide the level of contact necessary for transmission
- Mites subsist on a diet of dissolved human tissue (do not feed on blood) and can live up to 3 days off a host's body
- On average, the number of mites on a host at any time is ~5–15:
 - Main difference between crusted Norwegian scabies and ordinary scabies is the number of mites present on the host
 - Patients with crusted Norwegian scabies are infected with thousands or up to a million mites



DIAGNOSIS

SIGNS AND SYMPTOMS

Generalized and intense itching that is worse at night and usually spares the head and face

History

- Site, severity, duration, and timing of itch
- History should include family members and close contacts
- Generalized, intensely pruritic eruption:
 - Pruritus is intensified at night
- Onset 10–30 days after exposure and infestation; reinfestation provokes immediate (within 1–3 days) pruritus:
 - Patients with crusted Norwegian scabies are usually immunocompromised, have a decreased inflammatory response, and have less pruritus

Physical-Exam

- Often minimal cutaneous findings
- Primary lesion: Linear, elevated, white-gray burrow (up to 1 cm long, width of a human hair) with small vesicle containing black dot at the end (mites barely visible to naked eye):
 - Found symmetrically in web spaces of fingers, flexor surfaces of wrists and elbows, waistline, periumbilical skin, axillary folds, buttocks, penis, scrotum, vulva, and areola
 - Head and neck rarely affected in adults but more commonly in infants and children
- Secondary lesions: Inflammatory papules, nodules, excoriations, or secondary

impetigo or folliculitis seen on back, shoulders, axilla, waist, buttocks, and flexor aspects of elbows:

- Secondary lesions are usually more numerous and prominent than burrows but also may be few if topical steroids used
- Longstanding infestation results in chronic excoriation, eczematization, and hyperpigmented and lichenified skin
- Crusted Norwegian scabies produces gross scaling with hyperkeratotic plaques on hands, feet, scalp, and pressure-bearing areas:
 - Scales can become warty
 - Fissures may appear
 - Nail involvement is common
- Genitalia should be examined in all instances of suspected scabies

Pediatric Considerations

- Eruption may be seen from head to toe
- Vesicles are often found in infants due to their predisposition for vesicle formation
- Neonatal scabies is associated with poor feeding, poor weight gain, and super infection

ESSENTIAL WORKUP

- Careful history and skin exam for characteristic lesions
- The diagnosis is easily missed and should be considered in any patient with persistent generalized pruritus
- Factors related to missed diagnosis in patients admitted through the ED:
 - Overcrowding, time constraints, and lower patient illness severity scores

DIAGNOSIS TESTS & INTERPRETATION

Lab

- May be indicated in immunocompromised patients or in patients with systemic infection:
 - Elevated IgE and IgG and peripheral eosinophilia can be seen in crusted scabies
- New diagnostic lab studies are being developed (circulating IgE levels, PCR, ELISA, and DNA finger printing)
- When endemic, empiric treatment may be more cost effective than lab testing
- Consider screening for other STDs

Imaging

Epiluminescence microscopy and noncomputed dermoscopy are noninvasive, simple, accurate, and rapid imaging techniques

Diagnostic Procedures/Surgery

- Scrape skin at burrows or under fingernails with no. 15 blade and mineral oil (adheres scraped material to blade) and observe under low-power microscope for mites, eggs, or fecal material; may be operator dependent
- A negative scraping does not exclude infestation due to low number of mites in classic scabies:
 - Sensitivity < 50% and is affected by number of sites sampled and sampler's experience
- Skin biopsy may confirm diagnosis but findings may also be absent and reveal only a delayed hypersensitivity reaction

DIFFERENTIAL DIAGNOSIS

- Atopic dermatitis
- Eczema
- Dermatitis herpetiformis
- Papular urticaria
- Folliculitis
- Lichen planus
- Pruritic urticarial papules and plaques of pregnancy
- Adult linear IgA bullous dermatosis
- Syphilis
- Pediculosis
- Pityriasis rosea
- Impetigo
- Seborrheic dermatitis
- Flea bites and bedbugs



TREATMENT

PRE HOSPITAL

Maintain universal precautions.

INITIAL STABILIZATION/THERAPY

- No specific stabilization necessary
- ED is an important route for admission to the hospital and detecting infested patients early can be achieved by screening for high risk patients

ED TREATMENT/PROCEDURES

- Treatment should not be empiric for patients with generalized itching but reserved for patients with a history of exposure, a typical eruption in a characteristic distribution, or both
- Treat patient and all persons in immediate contact with topical scabicide:
 - Treat all contacts at the same time, regardless of the presence of symptoms
- Permethrin 5% is 89–92% effective, and is well tolerated (category B pregnancy):

- < 2% of permethrin is absorbed into the skin, making its potential toxicity low:
 - For children ≥ 2 mo older
 - Massage from head to toe (avoid eyes and mouth) and remove in shower 8–14 hr later
 - Repeat 2nd application in 1–2 wk time
- Crotamiton 10% is 50–60% effective and used when other scabicides are not tolerated
- Ivermectin administered orally for 2 doses 7–14 days apart has shown similar efficacy as permethrin (but not used in pregnant or lactating women or children < 15 kg):
 - Effective in patients unable to tolerate topical scabietics or in patients with resistant or crusted Norwegian scabies
 - May not be effective against all stages of life cycle (may not sterilize scabies eggs)
- Lindane 1% may be slightly less effective and is potentially toxic to the CNS:
 - Lindane absorption (through skin, lung or intestinal mucosa, or mucous membranes) is about 10%
 - Side effects include nausea, headache, vertigo, amblyopia, irritability, and seizure
 - Do not use in pediatric patients or patients with extensive excoriations or dermatitis
- Sulfur is the oldest known treatment of scabies, and is the drug of choice for infants < 2 mo and for pregnant or lactating women
- Crusted Norwegian scabies 1st requires removal of hyperkeratotic scale with keratolytic to facilitate entry of the scabicide
- Treatment failures:
 - Treatment failures are frequent in crusted Norwegian scabies, and use of multiple agents including oral medications is often necessary
 - Machine wash and dry in hot cycles (60°C) or dry clean all clothes and bedding worn within 2 days of treatment or place items in plastic bags for 3 days
 - Vacuum household floors, carpets, mattresses, and furniture
 - Autoclaving, bleaching, or fumigation are not indicated
 - Emphasize that itching may continue for 1–4 wk after mites are killed due to skin inflammatory reaction
 - Topical steroids and oral antihistamines can reduce pruritic symptoms
 - Relapses can occur from untreated areas such as the scalp and subungual regions
 - Treatment failures tend to arise from poor patient understanding and inadequate patient education

MEDICATION

- Scabicides:
 - Crotamiton 10% lotion or cream: Apply topically from neck down in adults and entire skin surface in children QHS for 2 nights, then rinse off 48 hr after last application
 - Ivermectin 3 mg tablets: 1st PO dose of 200 µg/kg should be followed by 2nd PO dose of 200 µg/kg 7–14 days later (pregnancy category C). Take with food
 - Lindane 1% lotion or cream: Apply topically from neck down and rinse off after 8–12 hr; contraindicated in infants, pregnancy, lactation, excessive excoriations, or seizure disorder
 - Permethrin 5% cream (Elimite): Apply topically from neck down in adults and entire skin in children QHS; rinse off after 8–14 hr (pregnancy class B, unknown safety in breast-feeding)
 - Sulfur 5–10% precipitated in petrolatum: Apply topically nightly for 3 consecutive nights and then wash off 24 hr later
- Antipruritics:
 - Low sedating/selective antihistamines:
 - Cetirizine (Zyrtec): Adults and peds >6 yr: 5–10 mg/d PO; 6–12 mo: 2.5 mg/d PO; 12–24 mo: 2.5 mg/d PO to BID; 2–6 yr: 2.5–5 mg/d PO
 - Fexofenadine (Allegra): Adult and peds >12 yr: 180 mg/d PO or 60 mg PO BID; 6 mo–5 yr: 15–30 mg PO BID; 6–11 yr: 30 mg PO BID
 - Loratadine (Claritin): Adults and peds >6 yr: 10 mg/d PO; 2–5 yr: 5 mg/d PO
 - Sedating/nonselective antihistamines:
 - Diphenhydramine (Benadryl): Adults and peds >12 yr: 25–50 mg PO q4–6h; 2–6 yr: 6.25 mg PO q4–6h; 6–12 yr: 12.5–25 mg PO q4–6h
 - Doxepin: 25–50 mg PO BID, peds: Dosing currently unavailable
 - Hydroxyzine HCl (Atarax): Adults and peds >12 yr: 25–100 mg PO q6–8h; <6 yr: 2 mg/kg/d PO div. q6–8h; 6–12 yr: 12.5–25 mg PO q6–8h

First Line

Permethrin 5% cream

Second Line

PO Ivermectin or Crotamiton 10% lotion or cream



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients with severe topical or systemic super infection
- Refractory or relapsing cases

Discharge Criteria

Nontoxic appearing patients with routine symptoms

FOLLOW-UP RECOMMENDATIONS

Re-evaluate after 1–4 wk for recurrence:

- Itching may persist for up to 4 wk after correctly applied therapy
- Treatment failure is often due to incorrect application of topical agents or due to failure to treat all contacts
- Retreat if live mites are found

PEARLS AND PITFALLS

- Scabies is a common parasitic infection that is transmitted by prolonged direct skin-to-skin contact
- Scabies in children can differ from that in adults
- Crusted Norwegian scabies is characterized by a large number of mites, and is seen in immunocompromised or institutionalized patients
- Treatment failure is common:
 - Proper patient education can decrease treatment failures.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Pediculosis
- Pityriasis rosea

ICD9
133.0 Scabies

ICD10

B86 Scabies

SCAPHOID FRACTURE

Davut J. Savaser • Robyn Heister Girard



BASICS

DESCRIPTION

- The scaphoid is the most commonly fractured carpal bone.
- This bone is the stabilizer between the distal and proximal carpal rows.
- Injury may result in arthritis, avascular necrosis, or malunion.
- Classified as:
 - Proximal 3rd (10–20%)
 - Middle 3rd (the waist, 70–80%)
 - Distal 3rd (the tuberosity)
 - Tubercle fractures
- Fractures are missed on initial radiographs 10–15% of the time, and delayed diagnosis greatly increases risk of complications.
- The blood supply to the scaphoid enters distally
- The more proximal the fracture, the higher the likelihood for avascular necrosis
- As the wrist is forcibly hyperextended, the volar aspect of the scaphoid fails in tension and the dorsal aspect fails in compression resulting in a fracture.

ETIOLOGY

Generally results from a fall on an outstretched (dorsiflexed) hand (FOOSH injury).



DIAGNOSIS

SIGNS AND SYMPTOMS

History

FOOSH injury

Physical-Exam

- Maximal pain and tenderness in the anatomic snuffbox (may be elicited with direct palpation or axial loading of the thumb); 90% sensitive, 40% specificity.
- Dorsal wrist pain distal to the radial styloid and decreased range of motion of the wrist and thumb
- Rarely, incidental damage to the superficial branches of the radial nerve results in sensory changes.
- Palpate the scaphoid tubercle for tenderness by radially deviating the wrist and palpating over the palmar aspect of the scaphoid; 87% sensitivity, 57% specificity.

Pediatric Considerations

- Carpal fractures are rare in children (and the elderly), as the distal radius usually fails 1st.
- If present, carefully evaluate mechanism.

DIAGNOSIS TESTS & INTERPRETATION

Imaging

- Radiographic imaging should include 3 views of the wrist: PA, lateral, oblique, and scaphoid views (wrist prone and in ulnar deviation).
- Pay special attention to the middle 3rd, or waist, of the bone: 70% of injuries occur here.
- Fracture may be identified by subtle findings such as a displaced fat pad.
- 10–15% of all fractures are not visible on radiographs at the time of injury.
- Bone scintigraphy or MRI as early as 3 days postinjury can rule out fracture and allow for earlier rehabilitation:
 - CT is not as reliable.

Diagnostic Procedures/Surgery

- If fracture is open or associated injuries are identified, urgent surgical intervention may be indicated.
- Associated injuries with scaphoid fracture:
 - Scapholunate dissociation
 - Distal radial fracture
 - Lunate fracture/dislocation
 - Bennett fracture of thumb
 - Radiocarpal joint dislocation
 - Proximal and distal carpal bone joint dislocations

DIFFERENTIAL DIAGNOSIS

- Bennett fracture
- Rolando fracture
- Extra-articular fracture at the base of the thumb metacarpal
- Gamekeeper thumb
- De Quervain tenosynovitis
- Perilunate dislocation
- Scapholunate dissociation
- Lunate fracture or dislocation



TREATMENT

PRE HOSPITAL

Splint or immobilize as appropriate.

INITIAL STABILIZATION/THERAPY

- Evaluate patient for other injuries.
- Dress open wounds.
- Immobilize with thumb in neutral position, ice, and elevate.

ED TREATMENT/PROCEDURES

- Assess mechanism of injury and point of maximal tenderness.
- Exam with special attention to skin integrity and neurovascular status.
- If snuffbox tenderness is present, place in thumb spica splint.
- Counsel patient regarding risk of malunion (10%) and avascular necrosis.
- Clinically suspected scaphoid fractures without radiographic evidence:
 - Should be treated as a nondisplaced scaphoid fracture
 - Spica splint thumb in a position as if the patient was embracing a wine glass.
 - Repeat physical/radiographic exam in 7–10 days.
- Nondisplaced scaphoid fractures:
 - Thumb spica splint
- Displaced scaphoid fractures:
 - Nonunion rate of 50%
 - Often an indication for internal fixation

MEDICATION

Pain control with NSAIDs or narcotics as needed



FOLLOW-UP

DISPOSITION

Admission Criteria

Open fracture or presence of other more serious injuries

Discharge Criteria

- Closed injuries, with 72-hr orthopedic follow-up
- Patients with splints for nondisplaced fractures may be allowed to return to full work or activity of work/sport if the cast does not interfere with the exercises of work or specific sport activities.

Issues for Referral

- If fracture is angulated or displaced > 1 mm, immediate orthopedic referral is indicated.

- All scaphoid or suspected scaphoid injuries must be referred to orthopedics.
- If no radiographic abnormalities found on initial radiograph, after placing in thumb spica splint, refer to orthopedics or primary care in 7–10 days with repeat radiographs at that time.

PEARLS AND PITFALLS

- Perfusion enters scaphoid bone distally.
- Avascular necrosis (especially with proximal 3rd fractures), occurs with inadequately reduced or immobilized fractures.
- Patients presenting with symptoms of a sprained wrist must have the diagnosis of acute scaphoid fracture ruled out.

ADDITIONAL READING

- Chudnofsky CR, Byers SE. *Clinical Procedures in Emergency Medicine: Splinting Techniques*. 5th ed. Philadelphia, PA: Saunders Elsevier; 2010.
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See Also (Topic, Algorithm, Electronic Media Element)

Lunate Fracture and Dislocations



CODES

ICD9

814.01 Closed fracture of navicular [scaphoid] bone of wrist

ICD10

- S62.009A Unsp fracture of navicular bone of unsp wrist, init
- S62.026A Nondisp fx of middle third of navic bone of unsp wrist, init
- S62.036A Nondisp fx of prox third of navic bone of unsp wrist, init

SCHIZOPHRENIA

Celeste N. Nadal • Melissa P. Bui

BASICS

DESCRIPTION

- A chronic psychotic disorder characterized by delusions, hallucinations, disorganization, negative symptoms, and cognitive deficits:
 - Premorbid phase:
 - Development of negative symptoms with deterioration of personal, social, and intellectual functioning
 - Active phase:
 - Development of active delusions, hallucinations, and bizarre behavior
 - May be precipitated by a stressful event
 - Residual phase:
 - Patients are left with impaired social and cognitive abilities
 - Psychotic symptoms may persist
 - Subtypes: Catatonic, disorganized, paranoid, residual, undifferentiated
- Onset typically early in adulthood (age < 30)
- Comorbid substance abuse (alcohol, cannabis, tobacco, and stimulants) is common
- Violence may result from impaired judgment, paranoia, and command hallucinations
- Life expectancy 12–25 yr less than general population likely because:
 - 41% of patients have metabolic syndrome with increased risk of death due to cardiovascular events
 - 5–10% of patients commit suicide
 - Patients have decreased access to medical care
- Disorganized thinking, abnormal behavior, and delusions may obscure the detection of medical illness
- Medication noncompliance is a key reason for psychiatric decompensation and presentation to the ED

ETIOLOGY

- Pathophysiology unclear but dopamine pathway strongly implicated
- Genetic component (concordance rate of 50% in monozygotic twins)
- Specific genes uncertain:
 - Higher risk in patients with DiGeorge syndrome (22q11.2 deletion)
- Perinatal risk factors:
 - Influenza during 2nd trimester
 - Maternal and postnatal infections
 - Advanced paternal age

- Use of cannabis may unmask psychosis in predisposed individuals



DIAGNOSIS

SIGNS AND SYMPTOMS

Criteria of the *Diagnostic and Statistical Manual of Mental Disorders IV (DSM-IV)* require the presence of at least 2 of the following symptoms for more than 6 mo:

- Delusions (fixed, false beliefs):
 - Bizarre, paranoid, or grandiose
 - Often persecutory, religious, or somatic content
- Hallucinations:
 - Commonly auditory or visual but may involve any sensory modality
- Thought disorder:
 - Disorganized speech ranging from odd, idiosyncratic logic to incoherence
- Grossly disorganized or catatonic behavior
- Negative symptoms:
 - Apathy and amotivation
 - Flat affect
 - Social isolation
 - Anhedonia

ESSENTIAL WORKUP

- Complete general and neurologic exam including vital signs and mental status exam
- Screen for psychosis:
 - Delusions:
 - “Do you feel anyone is trying to harm you or that you are being followed?”
 - “Is anyone trying to send you messages, steal, control, or block your thinking?”
 - Hallucinations:
 - “Do you ever see or hear things that other people cannot see or hear?”
 - “Do you ever hear voices telling you to do things such as to harm yourself or others?”
- Evaluate potential dangerousness to self or others:
 - Screen for past violence or self-injury
 - Content of psychotic symptoms should be explored to assess safety
- Patient history and medication compliance may be unreliable. Obtain collateral history from additional sources:
 - Friends and family
 - Treaters (PCP, therapist, psychiatrist)
 - Pharmacy

- Evaluate for affective psychosis (bipolar, major depression, or schizoaffective disorder)
- Evaluate for delirium or dementia
 - Schizophrenia does not affect orientation.
- Assess for drug-induced psychosis (see “Psychosis, Acute”)
- Psychosis due to medical etiology should be ruled out

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Toxicology screen
- Electrolytes, BUN, creatinine, glucose, calcium
- CBC with differential
- TSH
- Urinalysis

Imaging

Consider head imaging for new onset psychosis of undetermined etiology or new onset neurologic symptoms

Diagnostic Procedures/Surgery

EKG to monitor QT

DIFFERENTIAL DIAGNOSIS

- Delirium
- Drug-induced psychosis
- Psychosis secondary to general medical conditions such as TLE, MS, LBD
- Bipolar disorder
- Major depression with psychotic features
- Schizoaffective disorder:
 - Schizophrenia with prominent depressive and/or manic symptoms during psychosis
- Delusional disorder
- Schizotypal personality
- Brief psychotic episode:
 - Similar symptoms, duration of < 1 mo
- Schizophreniform disorder:
 - Similar symptoms, duration between 1 and 6 mo



TREATMENT

PRE HOSPITAL

- Patients can display unpredictable and violent behavior toward themselves and others
- Patients may require police presence and/or restraints to maintain safety
- Local laws vary as they apply to involuntary restraint

INITIAL STABILIZATION/THERAPY

- Safety of healthcare workers and patient is paramount; security presence may be required
- Behavioral interventions should be 1st line:
 - _ Provide a calm, containing environment
 - _ Potentially dangerous items should be removed from the patient's room
 - _ Use a reassuring voice and calm demeanor to set boundaries and verbally redirect the patient
- If safety is a concern, patient needs to be under constant observation and physical or chemical restraints may be necessary
- Acute agitation may be treated with haloperidol PO/IV/IM which can be augmented with lorazepam PO/IV/IM:
 - _ Encourage voluntary PO meds prior to IM administration
 - _ Other IM antipsychotics include olanzapine, chlorpromazine (monitor orthostatics), ziprasidone (monitor QT), and aripiprazole
 - _ IM olanzapine should not be combined with IV benzodiazepines as this increases risk of cardiopulmonary collapse

ED TREATMENT/PROCEDURES

- Psychiatric consultation in cases of decompensated schizophrenia
- Antipsychotic medications are the mainstay of treatment
- High-potency typical antipsychotic agents:
 - _ Associated with less QT prolongation
 - _ Higher propensity for extrapyramidal symptoms:
 - Dystonia
 - Parkinsonism
 - Akathisia
 - Tardive dyskinesia
 - _ IV haloperidol associated with fewer extrapyramidal symptoms than PO/IM
- Low-potency typical antipsychotics:
 - _ Higher risk of QT prolongation
 - _ Fewer extrapyramidal symptoms
 - _ More sedating
 - _ Orthostatic hypotension (must monitor)
 - _ Anticholinergic side effects
 - _ Lower seizure threshold
- Atypical antipsychotic agents:
 - _ Better tolerated with less EPS

- Associated with metabolic syndrome and weight gain
- Can cause orthostatic hypotension
- Nearly all antipsychotics increase QT:
 - More likely (ziprasidone)
 - Less likely (aripiprazole)
- Clozapine is the only antipsychotic that is clearly more effective for reducing psychotic symptoms and suicide risk:
 - Requires close monitoring of WBCs due to agranulocytosis
 - Highly sedating, hypotensive, lowers seizure threshold
 - Can cause QT prolongation
- Long-acting antipsychotic preparations (given q2–6wk) include:
 - Fluphenazine decanoate
 - Haloperidol decanoate
 - Olanzapine depot (Relprevv)
 - Paliperidone palmitate (Sustenna)
 - Risperidone microspheres (Consta)
- If a high-potency conventional antipsychotic agent is initiated, patients younger than age 40 can be started on benztropine (Cogentin) 2 mg BID for 10 days to reduce the risk of dystonic reactions

MEDICATION

- Typical antipsychotics (1st generation):
 - High potency:
 - Haloperidol 0.5–100 mg/d. Acute agitation 2.5–10 mg PO/IV/IM. Repeat q20–60min as needed
 - Fluphenazine 10 mg/d
 - Thiothixene 1–30 mg/d
 - Medium potency:
 - Perphenazine 2–24 mg/d
 - Trifluoperazine 1–20 mg/d
 - Low potency:
 - Chlorpromazine 0–200 mg/d in 3 div. doses
 - Loxapine 5–100 mg/d
 - Thioridazine 50–800 mg/d in 2–3 div. doses
- Atypical antipsychotics (2nd generation):
 - Aripiprazole 5–30 mg/d
 - Asenapine 5–20 mg/d (SL)
 - Clozapine 12.5–900 mg/d
 - Iloperidone 1–24 mg/d
 - Lurasidone 20–160 mg/d
 - Olanzapine 5–20 mg/d
 - Paliperidone 6–12 mg/d
 - Quetiapine 25–800 mg/d

- Risperidone 1–16 mg/d
- Ziprasidone 20–160 mg/d
- Benzodiazepines:
 - Lorazepam (Ativan) 0.5–2 mg per dose augments antipsychotic for acute agitation

Geriatric Considerations

Black box warning: Elderly patients with dementia-related psychoses treated with antipsychotic drugs are at increased risk of death.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Admit to inpatient psychiatric hospital, if patient is medically stable and:
 - Is a danger to self or others
 - Is gravely disabled and unable to care for himself due to psychosis
 - Has new-onset psychosis and medical etiology has been ruled out
- Prior to transfer to psychiatric facility, patient must have acute medical and surgical issues addressed
- Criteria for involuntary psychiatric hospitalization vary by state

Discharge Criteria

- Patient is not a danger to self or others and is able to perform activities of daily living
- Psychiatric follow-up is arranged
- Psychotic symptoms may persist at time of discharge

FOLLOW-UP RECOMMENDATIONS

- Outpatient psychopharmacologic follow-up should occur within 1 wk of discharge
- Patients taking antipsychotics (especially atypicals) should be monitored for QT prolongation and for obesity and related metabolic syndromes
- Adjunctive cognitive behavioral therapy and other psychosocial treatments can help patients manage psychotic symptoms and improve medication compliance
- Discuss smoking cessation and referral:
 - 50–80% of patients with schizophrenia smoke tobacco

PEARLS AND PITFALLS

- Visual, olfactory, gustatory, or tactile hallucinations should prompt medical workup for secondary causes of psychosis, as should atypical age of onset (> 30 yr)

old)

- Early treatment with antipsychotic medications and social interventions have consistently been associated with better outcomes in schizophrenia
- Avoid using IM olanzapine with IV benzodiazepines as this increases risk for cardiopulmonary collapse
- Patients who recently started antipsychotics who present with fever, rigidity, autonomic instability, and mental status changes should be assessed for neuroleptic malignant syndrome

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Delirium
- Dystonic Reaction
- Neuroleptic Malignant Syndrome
- Psychosis, Acute
- Psychosis, Medical vs. Psychiatric
- Violence, Management



CODES

ICD9

- 295.10 Disorganized type schizophrenia, unspecified
- 295.20 Catatonic type schizophrenia, unspecified state
- 295.90 Unspecified schizophrenia, unspecified state

ICD10

- F20.1 Disorganized schizophrenia
- F20.2 Catatonic schizophrenia
- F20.9 Schizophrenia, unspecified

SCIATICA/HERNIATED DISC

Nas N. Rafi



BASICS

DESCRIPTION

- Pain that radiates from the back into buttocks and lower extremity distal to knee, with or without sensory or motor deficits:
 - 95% sensitive, 88% specific for herniated disc (HD)
 - 3–5% lifetime prevalence
 - Peaks 4th to 5th decade
 - 2–10% of low back pain
 - 95% L5 or S1 nerve root
 - 90% improve with conservative management
 - Radicular symptoms usually resolve within 6 wk
 - 5–10% require surgery

ETIOLOGY

- Protrusion of colloidal gel (*nucleus pulposus*) through weakened surrounding fibrous capsule (*annulus fibrosis*)
- Risk factors:
 - Smoking
 - Repetitive lifting/twisting
 - Vehicular/machinery vibration
 - Obesity
 - Sedentary lifestyle



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Low back pain precedes onset of leg pain
- Leg pain predominates with time
- Sharp, well localized, radiates distal to knee
- Exacerbated by activities that increase intradiscal pressure:
 - Valsalva maneuver
 - Cough
 - Nerve-root tension (sitting, straight leg raise)
- Relieved by decreasing pressure/tension:
 - Lying supine

- Walking
- Paresthesia is the most common sensory symptom

Physical-Exam

- Neurologic exam (motor, sensory, deep tendon reflexes)
- L4 root/L3–L4 disc:
 - Knee extension/hip adduction
 - Anteromedial leg/knee/medial malleolus
 - Patellar reflex
- L5 root/L4–L5 disc:
 - Great toe and foot dorsiflexion
 - Dorsomedial foot/1st web space
 - No reflex
- S1 root/L5–S1 disc:
 - Foot plantarflexion
 - Posterior leg/lateral malleolus/dorsolateral foot
 - Achilles reflex
 - Rectal exam (tone, sensation)
- Straight leg raise:
 - Elevate ipsilateral leg by heel 30–60° with or without dorsiflexing foot
 - Reproduces radicular pain past knee
 - 80% sensitive for HD
- Crossed straight leg raise test (pathognomonic):
 - Elevate contralateral leg
 - Pain in involved leg
 - Less sensitive but very specific for HD

ESSENTIAL WORKUP

- Complete history and physical exam
- See below for test indications

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Indicated if clinical suspicion for differential diagnoses (DDX), not limited to:
 - CBC
 - ESR/CRP
 - UA

Imaging

PA/Lateral of LS spine

- Helps to rule out some DDX
- Indications:

- Extremes of age (< 20, > 55 yr)
- Unresolved back pain (> 4–6 wk) despite conservative treatment
- Red flags on history and physical exam:
 - Trauma
 - Constitutional symptoms (fever, unexplained weight loss, malaise)
 - History of cancer
 - Immunocompromised
 - IV drug abuse
 - Recent bacterial infection
 - Worse at night/wakes patient from sleep
 - Fever
 - Midline point tenderness
 - Neurologic deficits

MRI (Criterion Standard)

- Indications:
 - Acute, severe neurologic deficits (order from ED)
 - Suspicion of infectious etiology of back pain:
 - Epidural abscess
 - Osteomyelitis
 - Discitis
 - 6 wk failed conservative therapy (order on outpatient basis)
 - Disc disease (> 25%):
 - Incidental finding on MRI in asymptomatic patients
 - No relationship between extent of protrusion and degree of symptoms

CT Myelogram

- Rarely used alternative for MRI
- CT better at bone details

Diagnostic Procedures/Surgery

- Postvoid residual (PVR):
 - Overflow incontinence = PVR > 100 mL, suspect cauda equina syndrome

DIFFERENTIAL DIAGNOSIS

- Lumbosacral strain
- Degenerative joint disease
- Spondylolisthesis
- Hip/sacroiliac joint (infection, fracture, bursitis)
- Pneumonia, pulmonary embolus
- Pyelonephritis, renal calculi
- Ectopic pregnancy, pelvic inflammatory disease
- Abdominal aortic aneurysm (AAA)
- Peripheral vascular disease (claudication)
- Herpes zoster

- Psychological: Functional or secondary gain (drug seeking, disability)
- Irritating lesion affecting a lumbosacral nerve anywhere along its route:
 - Brain:
 - Thalamic or spinothalamic tumor, hemorrhage
 - Spinal cord (*myelopathy*):
 - Spinal stenosis, tumor, hematoma, infection (epidural abscess, discitis, osteomyelitis)
 - Root (*radiculopathy*):
 - Intradural: Tumor, infection
 - Extradural: HD, lumbar spine/foraminal stenosis (pseudoclaudication), spondylolisthesis, cyst, tumor, infection
 - Plexus (*plexopathy*):
 - Tumor, AAA, infection (iliopsoas abscess), hematoma (retroperitoneal)
 - Peripheral nerve (*neuropathy*):
 - Toxic/metabolic/nutritional, infection, trauma, ischemia, infiltration, compression, entrapment

Pediatric Considerations

- Usually secondary to trauma or serious underlying medical disease (e.g., leukemia); consider complete workup
- < 10 yr:
 - Infection
 - Tumor
 - Arteriovenous malformation
- ≥ 10 yr:
 - Traumatic HD
 - Spondylolisthesis
 - Scheuermann disease
 - Tumor

Pregnancy Considerations

- Ectopic pregnancy
- Labor
- Pyelonephritis
- Musculoskeletal



TREATMENT

PRE HOSPITAL

Full spine precautions for trauma victims

INITIAL STABILIZATION/THERAPY

Evaluate for neurosurgical emergency

ED TREATMENT/PROCEDURES

Pain relief:

- NSAIDs 1st line
- Muscle relaxants, opioids as needed in acute phase

MEDICATION

- NSAIDs:
 - Ibuprofen (Motrin, Advil): 600–800 mg (peds: 5–10 mg/kg/dose) PO TID--QID
 - Naproxen (Naprosyn, Aleve): 500 mg PO BID
- Muscle relaxants (short term):
 - Cyclobenzaprine (Flexeril): 5--10 mg TID
 - Diazepam (Valium): 2–10 mg (peds: 0.1 mg/kg/dose) PO TID--QID
 - Methocarbamol (Robaxin): 1,000–1,500 mg PO QID
- Opioids (short term):
 - Hydromorphone (Dilaudid): 2–4 mg PO/0.5–2 mg IM/IV q4–6h PRN
 - Morphine sulfate: 2–10 mg (peds: 0.1 mg/kg/dose) IM/IV q2–4h PRN
 - Codeine 30 mg + acetaminophen 300 mg; do not exceed acetaminophen 4 g/24 h
 - Hydrocodone 5 mg + acetaminophen 300 mg; do not exceed acetaminophen 4 g/24 h



FOLLOW-UP

DISPOSITION

Admission Criteria

- Severe neurologic deficit (cauda equina syndrome, inability to walk)
- Progressive neurologic deficit
- Multiple root involvement
- Unstable fracture, infection, neoplasm
- Inability to manage as outpatient (social situation/pain)

Discharge Criteria

Patient able to ambulate, follow instructions, has reliable home situation and planned follow-up

Issues for Referral

Abnormal workup that does not warrant immediate admission. Where and when depend

on results (large DDX)

FOLLOW-UP RECOMMENDATIONS

- Consultant (orthopedic spine surgeon or neurosurgeon) or PCP within 1 wk
- Conservative treatment (4–6 wk):
 - Medication as noted
 - Avoid complete bed rest, 2 days at most
 - Limited activity in acute phase but gradually increase activity/exercise as tolerated
 - Avoid movements that load lower back or exacerbate pain:
 - Heavy lifting, twisting, bending, stooping, bodily vibration
- Therapies of unproven benefit:
 - Chiropractic care
 - Transcutaneous electrical nerve stimulation
 - Traction
 - Back brace/corset
 - Ultrasound
 - Diathermy
 - Acupuncture, acupressure
 - Massage
 - Systemic glucocorticoids

ADDITIONAL READING

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- Jegede KA, Ndu A, Grauer JN. Contemporary management of symptomatic lumbar disc herniations. *Orthop Clin North Am.* 2010;41(2):217–224.
- Schoenfeld AJ, Weiner BK. Treatment of lumbar disc herniation: Evidence-based practice. *Int J Gen Med.* 2010;3:209–214.
- Tarulli AW, Raynor EM. Lumbosacral radiculopathy. *Neurol Clin.* 2007;25:387–405.
- van der Windt DA, Simons E, Riphagen II, et al. Physical examination for lumbar radiculopathy due to disc herniation in patients with low-back pain. *Cochrane Database Syst Rev.* 2010;(2):CD007431.

CODES

ICD9

- 722.10 Displacement of lumbar intervertebral disc without myelopathy
- 724.3 Sciatica
- 724.4 Thoracic or lumbosacral neuritis or radiculitis, unspecified

ICD10

- G57.00 Lesion of sciatic nerve, unspecified lower limb
- M51.16 Intervertebral disc disorders w radiculopathy, lumbar region
- M54.30 Sciatica, unspecified side

SEBORRHEIC DERMATITIS

Ian Glen Ferguson • Eric Norman Chow

BASICS

DESCRIPTION

- A common and chronic papulosquamous inflammatory skin disorder
- Affects all age groups and varies from mild dandruff to extensive adherent scale
- Found in areas with high concentrations of sebaceous follicles and glands
- Sharply demarcated yellow to red to brown, greasy, scaling, crusting patches/plaques
- Periods of remission and exacerbation frequent in adults

ETIOLOGY

- Exact pathogenesis not fully understood
- Multifactorial with environmental, genetic, hormonal, immunologic, microbial, and nutritional influences
- Strong association with *Malassezia* yeasts
- Complex physiologic response:
 - Immunologic
 - Inflammatory
 - Hyperproliferation
- Disease flares are common with physical and emotional stresses or illness
- Factors predisposing patients to develop seborrheic dermatitis and more severe or refractory disease:
 - Parkinson disease
 - Paralysis
 - HIV/AIDS
 - Mood disorders including depression
 - Congestive heart failure
 - Immunosuppression in premature infants
- Medications known to induce or aggravate seborrheic dermatitis include:

Arsenic Interferon- α

Auranofin Lithium

Aurothioglucose Methoxsalen

Buspirone Methyl dopa

Carbamazepine Phenothiazines

Chlorpromazine Phenytoin

Cimetidine	Primidone
Ethionamide	Psoralen
Gold	Stanozolol
Griseofulvin	Thiothixene
Haloperidol	Trioxsalen

DIAGNOSIS

SIGNS AND SYMPTOMS

Infants

- Onset during 1st few weeks of life, is usually self-limited and resolves by 12 mo of age
- May present concurrently with atopic dermatitis
- Flexural fold involvement may appear as diaper dermatitis:
 - Frequently develops a bacterial or fungal superinfection
- Cradle cap:
 - Thick greasy, adherent scale concentrated on the vertex of the scalp
 - Affects up to 70% of newborns during the 1st 3 mo of life
 - May be accompanied by inflammation or secondary infection

Young Children

- Blepharitis:
 - White scale adherent to eyelashes and eyelid margins with erythema
 - Resistant to treatment and persistent
 - May result in blepharoconjunctivitis

Adolescents and Adults

- Classic seborrheic dermatitis:
 - Minor itching with greasy, fine, dry, white scaling overlying red, inflamed skin
- Exacerbated by avoidance of washing
- Usually bilateral, symmetrical, and favoring the following areas:
 - Scalp, forehead, eyebrows, eyelids
 - Areas of facial hair
 - External ear canals
 - Nasolabial and posterior auricular folds
 - Posterior neck
 - Presternal, navel, and body folds:
 - Axillary and inframammary regions

- Groin and anogenital regions
- May cause areas of hypopigmentation in dark-skinned individuals

ESSENTIAL WORKUP

Diagnosis is based on clinical history and physical exam

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Potassium hydroxide preparations of skin scrapings may suggest yeast involvement
- Fungal culture may help to exclude dermatophytosis as an alternate diagnosis

Imaging

None required

Diagnostic Procedures/Surgery

Skin biopsy (rarely required):

- May help to exclude other diagnoses
- Consider, if the diagnosis remains unclear or the condition fails to respond to treatment

DIFFERENTIAL DIAGNOSIS

- Atopic dermatitis:
 - Later onset in infants (usually > 3 mo)
 - Characteristically affects antecubital and popliteal fossa in adults
 - Pruritus, oozing, and weeping support the diagnosis of atopic dermatitis
 - Family history of atopy (asthma and allergic rhinitis) favors atopic dermatitis
 - Axillary involvement favors the diagnosis of seborrheic dermatitis
- Contact dermatitis:
 - Polymorphous with erythema, edema, and vesicles
 - Tends to spare skin folds
 - May complicate seborrheic dermatitis as an unwanted reaction to treatment agents
- Cutaneous candidiasis:
 - Primary or secondary infection of the skin by *Candida* fungus
 - May affect any body area
 - Pruritus, erythema, mild scaling, and occasional blistering
 - Often associated with diabetes, obesity, or other illness
 - Common in infants
 - Presence of pseudohyphae on cytologic exam with potassium hydroxide does not exclude seborrheic dermatitis

- Dermatophytosis:
 - Generally distributed asymmetrically
 - Tinea capitis (scalp), corporis (body), cruris (groin), barbae (facial hair), faciei (face)
 - Can be very difficult to distinguish from seborrheic dermatitis
 - Hyphae on cytologic exam with potassium hydroxide is suggestive of tinea
- Langerhans cell histiocytosis:
 - Systemic signs (e.g., fever and adenopathy)
 - Infants affected may display scaling
 - Reddish-brown papules or vesicles
 - Associated splenomegaly
 - Purpuric lesions
- Leiner disease:
 - Prevalent in infant females
 - Rapid onset in 2nd to 4th month of life
 - Deficiencies of complement C3, C5
 - Severe generalized, exfoliative, erythrodermic form of seborrheic dermatitis
 - Fever, anemia, diarrhea, vomiting, weight loss, and failure to thrive
- Lupus erythematosus:
 - Erythematous malar rash of the nose and malar eminences
 - Chronic or discoid lupus:
 - Discrete erythematous papules/plaques
 - Thick adherent scale
 - “Carpet tack” appearance if removed
- Psoriasis:
 - Thicker plaques with silvery white scales
 - Less likely confined to scalp
- Rosacea:
 - Usually with central facial erythema or forehead involvement
- Tinea versicolor (pityriasis versicolor):
 - Chronic superficial fungal disease usually located on the neck, upper arms, and trunk
 - Characterized by fine, scaly, coalescing, hypopigmented or hyperpigmented macules
 - Patient usually asymptomatic
 - Also associated with *Malassezia* yeast
 - Not a dermatophyte
 - Short, thick hyphae with spores (spaghetti-and-meatball pattern) seen on cytology with potassium hydroxide

Pediatric Considerations

Infants with seborrheic dermatitis and cradle cap may present with concurrent atopic dermatitis

ALERT

- Seborrheic dermatitis is 1 of many conditions that may cause erythroderma (generalized exfoliative dermatitis):
 - _ Severe scaling erythematous dermatitis involving 90% or more of the body



TREATMENT

PRE HOSPITAL

None required

INITIAL STABILIZATION/THERAPY

None required

ED TREATMENT/PROCEDURES

- Seborrheic dermatitis is a chronic condition:
 - _ Emergent treatment is not required unless secondary infection or erythroderma is present

MEDICATION

- Pharmacologic options are often utilized in a multifaceted approach
- Therapy is directed at decreasing the reservoir of lipophilic yeast and the sebum that supports its growth, thus reducing inflammation and improving hygiene
- Severe cases may require removing scales and cornified nonviable epithelium to facilitate further treatment
- Scales may be softened by applying mineral oil (overnight if necessary) prior to washing
- Gentle brushing with a soft brush (toothbrush) or fine-tooth comb after washing may help remove stubborn scales
- Patient education:
 - _ Early treatment when condition flares
 - _ Emphasize hygiene and demonstrate proper cleansing of scaly lesions
 - _ Moderate UV-A/UV-B sunlight exposure may be beneficial as it inhibits growth of *Malassezia* yeasts
 - _ Refrain from hair sprays and hair pomades
- Infantile seborrheic dermatitis:
 - _ Responds readily to shampoos, emollients, and mild topical steroids
 - _ Aggressive keratolytic or mechanical removal may cause further inflammation
- Adult seborrheic dermatitis:
 - _ Treatment aimed at controlling symptoms, rather than curing the condition
- Blepharitis:
 - _ Warm to hot compresses to affected areas
 - _ Gentle cleansing with baby shampoo and cotton tip debridement of thick

scale

- Cradle cap in infants:
 - Topical olive oil (as emollient)
 - Topical imidazoles
 - Low-potency topical corticosteroids
- Scalp findings in children & adults:
 - Topical shampoos:
 - Pyrithione zinc
 - Coal tar
 - Salicylic acid
 - Selenium sulfide
 - Ciclopirox
 - Ketoconazole
- Nonscalp findings in children & adults:
 - Topical antifungals ± corticosteroids
 - Topical calcineurin inhibitors

First Line

- Imidazoles:
 - Inhibits ergosterol synthesis of fungal cell membrane
 - Target *Malassezia* species:
 - Ketoconazole 2% topical
 - Nizoral, Extina, Xolegel
- Topical corticosteroids:
 - Skin atrophy, striae, hypopigmentation, and telangiectasia may occur with extended use
 - Higher-potency agents indicated only for refractory conditions to less-potent agents
 - Use only briefly, as frequent use may foster recurrence and rebound effect
 - Use low-potency agents on areas with thinner skin (e.g., skin folds, neck, face):
 - Hydrocortisone 0.5%, 1%, 2.5%
 - Consider high- to mid-potency agents only on areas of thicker skin (e.g., trunk, scalp):
 - Fluocinolone acetonide
 - Triamcinolone acetonide
 - Betamethasone dipropionate
 - Clobetasol propionate
- Pyrithione zinc*:
 - Reduces epidermal cell turnover
 - Antifungal & antibacterial properties
- Salicylic acid*:
 - Keratolytic properties

- Useful in areas where scaling and hyperkeratosis are prominent
- Selenium sulfide*:
 - Reduces epidermal and follicular corneocyte production
 - Antifungal properties
- Coal tar/liquor carbonis detergens (LCD)*:
 - Inhibits mitotic cell division
 - Antipruritic, antiseptic properties
 - Reduces epidermal thickness
 - Avoid on face, skin flexures, or genitalia
- Sulfur/sulfonamide combinations:
 - Prevents PABA to folic acid conversion via dihydropteroate synthase inhibition:
 - Carmol scalp treatment
 - Ovace

*These agents are contained alone or in combination in formulations of the following:

- Denorex
- Head & Shoulders
- Neutrogena T/Gel or T/Sal
- Selsun Blue

Second Line

- Ciclopirox:
 - Anti-fungal, -bacterial, -inflammatory effects
- Topical calcineurin inhibitors:
 - Anti-inflammatory & antifungal properties
 - Lack long-term effects of corticosteroids
 - Black box warning concerning malignancy:
 - Pimecrolimus 1%
 - Tacrolimus 0.1%



FOLLOW-UP

DISPOSITION

Admission Criteria

Admission unlikely to be required unless severe secondary infection or erythroderma is present

Discharge Criteria

Patients may be discharged with recommended medications and follow-up

Issues for Referral

- Refer patients to primary care physician when considering underlying illness or comorbidities
- Consider referral to a qualified dermatologist when the diagnosis remains elusive or the condition fails to respond to therapy

FOLLOW-UP RECOMMENDATIONS

- Symptoms should improve within 7–10 days, but may take months to resolve completely and may recur
- Adolescent and adult forms may persist as a chronic dermatitis
- Provide return precautions for signs of secondary bacterial or fungal infections:
 - Fever, erythema, tenderness, or ulcerations

PEARLS AND PITFALLS

- Severe and sudden attacks of seborrheic dermatitis may be the initial presentation of an immunocompromised patient (e.g., HIV/AIDS)
- Admission may be warranted for further evaluation of the underlying disease process

ADDITIONAL READING

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- Goldsmith LA, Katz SI, Gilchrest BA, et al. *Fitzpatrick's Dermatology in General Medicine*. 8th ed. New York, NY: McGraw-Hill; 2012.
- Hurwitz S. *Clinical Pediatric Dermatology*. 3rd ed. Philadelphia, PA: Elsevier Saunders; 2006.
- Naldi L, Rebora A. Clinical practice. Seborrheic dermatitis. *N Engl J Med*. 2009;360:387–396.



CODES

ICD9

- 690.10 Seborrheic dermatitis, unspecified
- 690.11 Seborrhea capitis
- 690.12 Seborrheic infantile dermatitis

ICD10

- L21.0 Seborrhea capitis
- L21.1 Seborrheic infantile dermatitis
- L21.9 Seborrheic dermatitis, unspecified

SEIZURE, ADULT

Atul Gupta • Rebecca Smith-Coggins



BASICS

DESCRIPTION

- Generalized seizures:
 - Classically tonic–clonic (grand mal)
 - Begin as myoclonic jerks followed by loss of consciousness
 - Sustained generalized skeletal muscle contractions
 - Nonconvulsive generalized seizures:
 - Absence seizures (petit mal); alteration in mental status without significant convulsions or motor activity
- Partial seizures:
 - Simple:
 - Brief sensory or motor symptoms without loss of consciousness (i.e., Jacksonian)
 - Complex:
 - Mental and psychological symptoms
 - Affect changes
 - Confusion
 - Automatism
 - Hallucinations
 - Associated with impaired consciousness
- Status epilepticus:
 - Variable definitions:
 - Seizure lasting longer than 5–10 min
 - Recurrent seizures without return to baseline mental status between events
 - Life-threatening emergency with mortality rate of 10–12%
 - Highest incidence in those <1 yr and >60 yr of age
- At least one-half of patients presenting to the ED in status do not have a history of seizures.
- Alcohol withdrawal seizures (“rum fits”):
 - Peak within 24 hr of last drink
 - Rarely progress to status epilepticus
- Patients with a single seizure have a 35% risk of recurrent seizure within 5 yr

Pediatric Considerations

Febrile seizures are generalized seizures occurring between 3 mo and 5 yr of age:

- Typically lasts <15 min

- Associated with a rapid rise in temperature
- Without evidence of CNS infection or other definitive cause

ETIOLOGY

- Hypoxia
- Hypertensive encephalopathy
- Eclampsia
- Infection:
 - Meningitis
 - Abscess
 - Encephalitis
- Vascular:
 - Ischemic stroke
 - Hemorrhagic stroke
 - Subdural hematoma
 - Epidural hematoma
 - Subarachnoid hemorrhage
 - Arteriovenous malformation
- Structural:
 - Primary or metastatic neoplasm
 - Degenerative disease (i.e., multiple sclerosis)
 - Scar from previous trauma
- Metabolic:
 - Electrolytes
 - Hyponatremia
 - Hyponatremia
 - Hypocalcemia
 - Hypo/hyperglycemia
 - Uremia
- Toxins/drugs:
 - Lidocaine
 - Tricyclic antidepressants
 - Salicylates
 - Isoniazid
 - Cocaine
 - Alcohol withdrawal
 - Benzodiazepine withdrawal
- Congenital abnormalities
- Idiopathic
- Trauma



DIAGNOSIS

SIGNS AND SYMPTOMS

- Altered level of consciousness
- Involuntary repetitive muscle movements:
 - Tonic posturing or clonic jerking
- Seizures of abrupt onset:
 - Aura may precede a focal seizure
- Duration usually 90–120 sec:
 - Impaired memory of the event
 - Postictal state is a brief period of confusion and somnolence following a seizure
- Evidence of recent seizure activity:
 - Confusion or somnolence
 - Acute intraoral injury
 - Urinary incontinence
 - Posterior shoulder dislocation
 - Temporary paralysis (Todd paralysis)
- Other findings may suggest etiology of seizure:
 - Fever and nuchal rigidity (CNS infection)
 - Needle tracks; stigmata of liver disease (drugs and alcohol)
 - Head trauma:
 - Papilledema (increased intracranial pressure)
 - Lateralized weakness, sensory loss, or asymmetric reflexes

History

- History of seizures:
 - Medication compliance
- Recent illness
- Head trauma
- Headaches
- Anticoagulation therapy
- Fever
- Neck stiffness

Physical-Exam

- Complete neurologic exam:
 - Todd paralysis
- Complete secondary and tertiary survey to evaluate for any trauma secondary to seizure or potential cause for seizure

ESSENTIAL WORKUP

- A thorough history is the most valuable part of the workup:
 - Witness accounts
 - History of prior seizures

- Presence of acute illness
- Past medical problems
- History of substance use
- Patients with chronic seizure disorder and typical seizure pattern may need to have only serum glucose and anticonvulsant levels checked
- New-onset seizure mandates workup:
 - Electrolytes including calcium, phosphorus
 - Head CT
 - Toxicology screen
 - Pregnancy test if woman is of childbearing age
 - Lumbar puncture indicated if:
 - New-onset seizure with fever
 - Severe headache
 - Immunocompromised state
- Persistently altered mental state:
 - Search for specific underlying cause
 - Patient's condition and resources for follow-up determine whether all these tests must be done in the ED

Pediatric Considerations

- A child with a 1st febrile seizure should receive fever workup as dictated by clinical condition
- Inquire about family history of febrile seizures
- Labs and radiographs as needed to determine source of fever
- Lumbar puncture for 1st febrile seizure:
 - Consider if age < 1 yr
 - Ill appearing
 - Lethargy or poor feeding
 - Exam difficult
 - Unreliable follow-up

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Serum anticonvulsant levels
- Blood–alcohol level
- Toxicology screen
- CBC:
 - WBC often elevated
- Chemistry panel:
 - Bicarbonate often low
- Lactate may be elevated
- CSF:

- May have transient increase in WBC to 20/ μ L

Imaging

- Noncontrast head CT:
 - Persistent or progressive alteration of mental status
 - Focal neurologic deficits
 - Seizure associated with trauma
- CT scan with contrast should be obtained in HIV-positive patients to rule out toxoplasmosis
- MRI is sensitive for low-grade tumors, small vascular lesions, early inflammation, and early cerebral infarcts:
 - Consider electively in new-onset seizures

Diagnostic Procedures/Surgery

- EEG may be arranged with neurology on an outpatient basis
- Bedside EEG may be performed in ED if there is suspicion of nonconvulsive status epilepticus or psychogenic seizures

DIFFERENTIAL DIAGNOSIS

- Syncope (may also have incontinence, twitching, and jerking)
- Hyperventilation syndrome
- Psychogenic seizures
- Transient ischemic attacks
- Sleep disorders
- Delirium tremens
- Hypoglycemia



TREATMENT

PRE HOSPITAL

Anticonvulsant as per local protocol

INITIAL STABILIZATION/THERAPY

- Airway management as indicated
- Pulse oximetry, oxygen with suction available:
 - C-spine precautions
 - Rapid-sequence intubation if patient cannot protect airway or with hypoxia or major head trauma
 - IV access, rapid determination of serum glucose:
 - If hypoglycemic, give IV dextrose 25 g
 - Lorazepam or diazepam for active seizures
 - Naloxone if concern for narcotic overdose

ED TREATMENT/PROCEDURES

- 1st-time seizure:
 - Normal head CT if performed
 - Return to baseline with normal neuro exam:
 - Discharge with close follow-up with PCP and/or neurologist
- 1st-time seizure:
 - Structural lesion on CT or MRI:
 - Start antiepileptic drug (AED) in consultation with PCP and/or neurologist
- Recurrent seizure not on AED:
 - Start AED in consultation with PCP and/or neurologist
- Recurrent seizure with subtherapeutic AED level:
 - IV and/or PO load current AED
- Recurrent seizure with therapeutic AED level:
 - Need careful evaluation for cause of seizures, new lesions, etc.:
 - Adjust and/or add AED in consultation with neurologist
- Seizure in a pregnant patient:
 - Evaluate as other seizure patients
 - Strongly consider eclampsia if >20-wk gestation
 - OB consultation, arrange for C-section
 - Magnesium
- Seizures related to alcohol:
 - Determine if seizure is caused by withdrawal (typically 6–48 hr after cessation of drinking) or another cause
 - Management of withdrawal seizures is benzodiazepines

Pediatric Considerations

- Fever control with acetaminophen and ibuprofen
- Anticonvulsants not needed for febrile seizures
- Anticonvulsants should be prescribed in conjunction with neurologist.

MEDICATION

- Acetaminophen: 500 mg PO/PR q4–6h; do not exceed 4 g/24 h
- Diazepam: 0.2 mg/kg IV per dose; 0.5 mg/kg PR
- Fosphenytoin: 15–20 mg/kg phenytoin equivalents (PE) at rate of 100–150 mg/min IV/IM
- Ibuprofen: 5–10 mg/kg PO
- Levetiracetam: Start 500 mg PO/IV q12h (peds: Start 20 mg/kg/d PO div. BID; age 4–15 yr)
- Lorazepam: 2–4 mg IV/IM (peds: 0.05–0.1 mg/kg IV per dose)
- Naloxone: 0.4–2 mg IV/IM/SQ (peds: 0.1 mg/kg IV/IM/SQ)
- Phenobarbital: 15–20 mg/kg IV at rate of 1 mg/kg/min (plan to protect airway)
- Phenytoin: 15–20 mg/kg IV at rate of 40–50 mg/min (peds: Use rate of 0.5–1

mg/kg/min)

- Propofol: 5–50 µg/kg/min IV, titrate to effect (plan to protect airway)
- Valproate sodium: 10–20 mg/kg/d

First Line

Benzodiazepines

Second Line

- Fosphenytoin
- Levetiracetam
- Phenobarbital
- Phenytoin
- Propofol
- Valproate sodium:
 - works as well as second line agent in status epilepticus and can be given faster



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients with status epilepticus should be admitted to the ICU
- Patients with seizures secondary to underlying disease (e.g., meningitis, intracranial lesion) must be admitted for appropriate treatment and monitoring
- Patients with poorly controlled repetitive seizures should be admitted for monitoring
- Delirium tremens

Discharge Criteria

- Patient with normal workup and appropriate neurology follow-up
- Uncomplicated seizure in patient with chronic seizure disorder
- Seizure secondary to reversible cause:
 - Hypoglycemia if blood sugar has stabilized
 - Alcohol withdrawal if baseline mental status and no further seizures
- Simple febrile seizure

Issues for Referral

- Consider early neurology follow-up
- Anticonvulsant drug level monitoring

FOLLOW-UP RECOMMENDATIONS

No driving until seizures are under control

PEARLS AND PITFALLS

- Most common cause of recurrent seizure is subtherapeutic anticonvulsant drug level
- Benzodiazepines are the 1st-line treatment to stop seizure activity
- Treat the underlying cause if identifiable
- Seizures lasting longer than 5–10 min should be treated as status epilepticus
- Valproate likely works as well as phenytoin/fosphenytoin as a second line agent in treating status epilepticus and can be administered more quickly with less chance of an adverse effect

ADDITIONAL READING

- ACEP Clinical Policies Subcommittee (Writing Committee) on Seizures; Huff JS, Melnick ER, Tomaszewski CA, et al. Clinical policy: Critical issues in the evaluation and management of adult patients presenting to the emergency department with seizures. *Ann Emerg Med.* 2014;63:437–447.
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See Also (Topic, Algorithm, Electronic Media Element)

- Headaches
- Hypertensive Emergencies
- Intracerebral Hemorrhage
- Preeclampsia/Eclampsia
- Seizure, Febrile
- Seizure, Pediatric



CODES

ICD9

- 345.00 Generalized nonconvulsive epilepsy, without mention of intractable epilepsy
- 345.90 Epilepsy, unspecified, without mention of intractable epilepsy
- 780.39 Other convulsions

ICD10

- G40.009 Local-rel idio epi w seiz of loc onst,not ntrct,w/o stat epi
- G40.409 Other generalized epilepsy and epileptic syndromes, not intractable, without status epilepticus
- R56.9 Unspecified convulsions

SEIZURE, FEBRILE

John P. Santamaria



BASICS

DESCRIPTION

- Occurs between 6 mo and 5 yr of age associated with fever:
 - No evidence of intracranial infection or other defined CNS primary cause
 - Average age of onset is 18–22 mo
 - Children with previous nonfebrile seizures excluded
- Most common pediatric convulsive disorder:
 - Affects 2–4% of young children in US
- Occurs in normal children with a systemic viral illness
- High-risk children:
 - History of febrile seizure in immediate family members
 - Delayed neurologic development
 - Males
- Subgroups:
 - Simple febrile seizures:
 - Brief, self-limited lasting < 10–15 min, resolve spontaneously
 - Generalized without any focal features
 - Complex febrile seizures:
 - Duration > 15 min
 - Focal features
 - More than 1 seizure within a 24-hr period
- Risk of recurrence:
 - One-third of cases
 - Early age of onset, history of febrile or afebrile seizures in 1st-degree relatives, and temperature < 40°C during initial seizure increase the likelihood of recurrence
- Risk of subsequent epilepsy:
 - Greatest for those with prior abnormal neurologic development, a complex (> 15 min) 1st febrile seizure, a focal seizure, or a family history of afebrile seizures
 - Only slightly greater than the general population if 1st febrile seizure is simple and neurologic development normal
 - Not affected by the use of prophylactic medications

ALERT

Because this is usually self-limited, intervention must be individualized in relation to airway, breathing, and seizure management

ETIOLOGY

Common childhood infections:

- Upper respiratory illnesses
- Otitis media
- Roseola
- GI infections
- *Shigella* gastroenteritis

DIAGNOSIS

SIGNS AND SYMPTOMS

- Fever
- Seizure may occur concurrent with recognition of the febrile illness
- Seizure
- Generalized tonic-clonic seizure most common:
 - _ Tonic phase:
 - Muscular rigidity
 - Apnea and incontinence
 - Self-limited and last only a few minutes
 - _ Other seizure types:
 - Staring with stiffness
 - Limpness
 - Jerking movements without prior stiffening

History

- Careful history and physical exam help confirm diagnosis and rule out other etiologies
- Symptoms/evidence of infectious illness
- Duration and pattern of fever
- Medication exposure/toxin
- Recent immunizations
- Trauma/occult trauma
- Growth pattern and developmental level
- Family history of seizures
- Complete description of seizure

Physical-Exam

- Reducing temperature may be useful in evaluation; give antipyretics early
- Evidence of infectious illness-rash, ear infection, respiratory infection, diarrhea, etc.
- Careful neurologic exam including mental status
- Presence of meningismus, bulging fontanelle, nuchal rigidity, etc.

- Evidence of focal deficit or increased ICP

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Routine lab studies not indicated
- Evaluate for a source of fever if serious bacterial infection is suspected:
 - WBC
 - UA
 - Blood and urine cultures
- Lumbar puncture:
 - Not routinely indicated
 - Indications 12–18 mo of age:
 - History or irritability, decreased feeding, lethargy
 - Consider if deficient in *Haemophilus influenzae* type b or *Streptococcus pneumoniae* immunizations
 - Physical signs of meningitis and/or history consistent with meningitis
 - Complex seizure
 - Prolonged postictal state
 - Prior antibiotics altering presentation
 - Abnormal mentation after postictal state
 - Indications >18 mo old:
 - Signs/symptoms of CNS infection present
 - Electrolytes and bedside glucose in infants and children with vomiting or diarrhea
- EEG:
 - Not helpful in the initial evaluation of febrile seizures
 - May be indicated if developmental delay, underlying neurologic abnormality, or focal seizure
 - Does not help predict recurrences or risk for later epilepsy
- Anticonvulsant levels
- Toxicology studies of blood and urine if history and physical exam suggestive

Imaging

- Chest radiograph only in patients with significant respiratory symptoms or pertinent findings on physical exam
- Head CT:
 - Indicated with traumatic injuries, focal neurologic findings, or inability to exclude elevated intracranial pressure

DIFFERENTIAL DIAGNOSIS

- Febrile delirium
- Febrile shivering with pallor and perioral cyanosis

- Breath-holding spell during febrile event
- Acute life-threatening event
- Other causes of seizure:
 - Afebrile seizure occurring during febrile event
 - Sudden discontinuance of anticonvulsants
 - Infection:
 - Meningitis/encephalitis
 - Acute gastroenteritis, often with dehydration
 - Head trauma
 - Toxicologic:
 - Anticholinergics
 - Sympathomimetics
 - Other
 - Hypoxia
 - Metabolic disease
 - Intracranial masses
 - CNS vascular lesions



TREATMENT

PRE HOSPITAL

- Protect the airway
- Oxygen
- Support breathing as needed
- Cautions:
 - Keep child from incurring injury while actively convulsing
 - Respiratory insufficiency and apnea occur secondary to overaggressive treatment with benzodiazepines
 - Simple febrile seizures are self-limited and generally require no anticonvulsant therapy or ventilatory support

INITIAL STABILIZATION/THERAPY

- Support the airway and breathing
- Benzodiazepines rarely needed:
 - Prolonged seizures or compromised patients
 - Lorazepam, diazepam, or midazolam
 - Rectal diazepam or nasal midazolam may be easily administered with good efficacy

ED TREATMENT/PROCEDURES

- Rarely is pharmacologic intervention required; usually self-limited
- Seizures refractory to benzodiazepines:

- Phenytoin or fosphenytoin
- Phenobarbital
- Workup to exclude other etiologies
- Administer antipyretics acutely and routinely for at least the next 24 hr:
 - Acetaminophen and/or ibuprofen (may use both)
- Appropriate antibiotic treatment for specific bacterial disease if identified
- Reassure and education of parents is essential

MEDICATION

- Acetaminophen: 10–15 mg/kg/dose PO, PR; do not exceed 5 doses/24 h
- Diazepam: 0.2 mg/kg IV (max. 10 mg); 0.2–0.5 mg/kg PR (max. 20 mg)
- Fosphenytoin: 20 mg/kg IV over 20 min
- Ibuprofen: 10 mg/kg PO
- Lorazepam: 0.1 mg/kg IV (max. 5 mg)
- Midazolam: 0.05–0.1 mg/kg IV; 0.2 mg/kg buccal/IN/IM (max. 7.5 mg)
- Phenobarbital: 15–20 mg/kg IV over 20 min or IM; monitor for respiratory depression
- Phenytoin: 15–20 mg/kg IV over 30–45 min



FOLLOW-UP

DISPOSITION

Admission Criteria

- Recurrent or prolonged seizures
- Fever with source not appropriately treated as outpatient

Discharge Criteria

- Simple febrile seizures:
 - Normal neurologic exam
 - Source of fever is appropriately treated as outpatient
- Reassurance to parents

FOLLOW-UP RECOMMENDATIONS

Schedule follow-up with primary care physician

PEARLS AND PITFALLS

- Although aggressive treatment of fever with antipyretics is often recommended, there is no evidence that this reduces seizure recurrence
- Oral diazepam during febrile illness may reduce risk of recurrence; prophylactic anticonvulsants with other anticonvulsants rarely indicated—such treatment is controversial and to be considered only after extensive discussion

ADDITIONAL READING

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- Steering Committee on Quality Improvement and Management; Subcommittee on Febrile Seizures American Academy of Pediatrics. Febrile seizures: Clinical practice guideline for the long-term management of the child with simple febrile seizures. *Pediatrics.* 2008;121(6):1281–1286.
- Strengell T, Uhari M, Tarkka R, et al. Antipyretic agents for preventing recurrences of febrile seizures: Randomized controlled trial. *Arch Pediatr Adolesc Med.* 2009;163(9):799–804.
- Subcommittee on Febrile Seizures; American Academy of Pediatrics. Neurodiagnostic evaluation of the child with a simple febrile seizure. *Pediatrics.* 2011;127(2):389–394.



See Also (Topic, Algorithm, Electronic Media Element)

- Anticholinergic Poisoning
- Seizures, Pediatric
- Fever, Pediatric



CODES

ICD9

- 780.31 Febrile convulsions (simple), unspecified
- 780.32 Complex febrile convulsions

ICD10

- R56.0 Febrile convulsions
- R56.00 Simple febrile convulsions
- R56.01 Complex febrile convulsions

SEIZURE, PEDIATRIC

John P. Santamaria



BASICS

DESCRIPTION

Sudden, abnormal discharges of neurons resulting in a change in behavior or function

ETIOLOGY

- Febrile seizures
- Infection
- Idiopathic
- Trauma
- Toxicologic:
 - Ingestion
 - Drug action
 - Drug withdrawal
- Metabolic:
 - Hypoglycemia
 - Hypocalcemia
 - Hypo/hyponatremia
 - Inborn errors of metabolism
- Perinatal hypoxia
- Intracranial hemorrhage
- CNS structural anomaly or malformation
- Degenerative disease
- Psychogenic



DIAGNOSIS

SIGNS AND SYMPTOMS

Neonates

- Subtle abnormal repetitive motor activity:
 - Facial movements
 - Eye deviations
 - Eyelid fluttering
 - Lip smacking/sucking
- Respiratory alterations
- Apnea
- Seizure activity:

- Focal or generalized tonic seizures
- Focal or multifocal clonic seizures
- Myoclonic movements
- Generalized problems (metabolic, infection, etc.) may present with focal seizures

Older Infants and Children

- Generalized seizures:
 - Tonic-clonic
 - Tonic
 - Clonic
 - Myoclonic
 - Atonic (“drop”)
 - Absence
- Partial or focal seizures:
 - Simple:
 - Consciousness maintained
 - Simple partial seizures:
 - Motor, sensory, and/or cognitive symptoms
 - Motor activity focal: 1 part or side
 - Paresthesias, metallic tastes, and visual or auditory hallucinations
 - Complex:
 - Consciousness impaired
 - Complex partial seizure
 - Simple partial seizure progresses with impaired consciousness:
 - Aura precedes altered consciousness; auditory, olfactory, or visual hallucination
 - May generalize
- Status epilepticus:
 - Generalized is most common
 - Sustained partial seizures
 - Absence seizures
 - Persistent confusion; postictal period

History

- Determine whether seizures are febrile or afebrile
- Determine type of seizure:
 - Partial vs. generalized
 - Presence of eye findings, aura, movements, cyanosis
 - Duration
 - State of consciousness, postictal state
 - Predisposing conditions/history/family history (syndromes with a genetic component)

Physical-Exam

- Vital signs, including temperature
- Careful neurologic exam, including state of consciousness
- Eye, including fundoscopic exam
- Skin exam to identify neurocutaneous diseases such as tuberous sclerosis

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Bedside glucose test
- Performed in young infants and those in status epilepticus
- Select studies in other children reflecting history and physical exam:
 - Electrolytes
 - BUN
 - Creatinine
 - Glucose
 - Calcium
 - Magnesium
 - CBC
 - Toxicology screen
- Patients on anticonvulsant therapy:
 - Drug levels
- Febrile seizure:
 - Lab studies to evaluate for a serious underlying bacterial infection if suspected

Imaging

- Head CT:
 - Focal seizure
 - New focal neurologic abnormality
 - Suspected intracranial hemorrhage or mass lesion
 - New-onset status epilepticus without identifiable cause
 - Not routinely indicated for 1st afebrile seizure
- Lumbar puncture:
 - Suspicion of meningitis or encephalitis
 - CT 1st if suspect increased intracranial pressure
- MRI:
 - Rarely urgently indicated for seizures
- EEG:
 - Generally indicated in children with an afebrile seizure as a predictor of risk of recurrence and to classify the seizure type/epilepsy syndrome
 - Postictal slowing seen within 24–48 hr of a seizure and may be transient; delay EEG if possible

- Rarely helpful in the acute setting

DIFFERENTIAL DIAGNOSIS

- Neonates:
 - Apnea due to other causes
 - Jitters or tremors
 - Gastroesophageal reflux
- Infants and toddlers:
 - Breath-holding spells
 - Night terrors
- Children and adolescents:
 - Migraine headache
 - Syncope
 - Tics
 - Pseudoseizures
 - Hysteria



TREATMENT

PRE HOSPITAL

Cautions:

- Many conditions may be mistaken for seizures (see “Differential Diagnosis,” below)
- Immobilize cervical spine if trauma suspected
- Check fingerstick glucose or administer dextrose as appropriate

INITIAL STABILIZATION/THERAPY

- ABC support if actively seizing
- Airway:
 - Oxygen/monitor pulse oximetry
 - Nasopharyngeal airway preferred over oral airway
 - Bag valve–mask support if hypoventilating or persistently hypoxic
 - Intubation if seizures are refractory and bag valve–mask support is unsuccessful
- IV access:
 - If hypoglycemic, give dextrose
- Maintain spine precautions if trauma suspected

ALERT

Airway and breathing must be stabilized concurrent with management of ongoing seizures if present

ALERT

Early treatment of long-lasting seizure is critical in reducing potential morbidity, including brain damage

ED TREATMENT/PROCEDURES

Status Epilepticus

- Benzodiazepine:
 - When treating IV lorazepam is preferred due to its longer duration of action
 - Valium is acceptable
 - If IV access is not available:
 - Buccal midazolam (most convenient)
 - Intranasal lorazepam
 - Per rectum diazepam
- Phenytoin:
 - If benzodiazepines fail
 - For longer-term control
 - Fosphenytoin easier to administer
- Phenobarbital:
 - Use if benzodiazepines and phenytoin fail to break the seizure
 - Risk of respiratory depression greatly increases if a benzodiazepine has also been given
- Alternative therapies in the event of refractory status epilepticus
- Consultation appropriate:
 - Paraldehyde (per rectum)
 - Barbiturate coma:
 - Barbiturate (pentobarbital) coma requires intubation and EEG monitoring to be sure the seizure is suppressed
 - Associated hypotension
 - General anesthesia:
 - A final resort
 - Continuous EEG is needed to be sure the seizure is abolished
- Neonates:
 - Phenobarbital is an acceptable 1st-line therapy
 - Preferred maintenance drug

ALERT

Note: Aggregate response to 2nd- and 3rd-line agents is <10%

MEDICATION

- D₁₀: 5 mL/kg IV for neonates
- D₂₅: 2 mL/kg IV for children
- Diazepam: 0.2 mg/kg IV (max. 10 mg); 0.2–0.5 mg/kg PR (max. 20 mg)

- Fosphenytoin: 20 mg/kg IV over 20 min
- Lorazepam: 0.1 mg/kg IV, IN (max. 5 mg)
- Midazolam: 0.05–0.1 mg/kg IV; 0.2 mg/kg buccal/IN/IM (max. 7.5 mg)
- Pentobarbital: 3–5 mg/kg IV over 1–2 hr; maintenance: 1–3 mg/kg/h IV; monitor for respiratory depression
- Phenobarbital: 15–20 mg/kg IV over 20 min; monitor for respiratory depression
- Phenytoin: 15–20 mg/kg IV slowly over 30–45 min



FOLLOW-UP

DISPOSITION

Admission Criteria

- ICU:
 - Active status epilepticus, intubated, or persistent mental status changes
 - Repetitive seizures in narrow time frame
- Inpatient unit:
 - Status epilepticus resolved in the ED
 - Underlying cause of seizure unresolved, uncontrolled, or poorly understood
 - Intracranial hemorrhage
 - Mass lesion
 - Meningitis/encephalitis
 - Drug
 - Toxin ingestions

Discharge Criteria

- The child is alert with normal mental status and neurologic exam
- No evidence of an underlying cause requiring hospitalization
- Reliable parent or caregiver
- Home telephone

Issues for Referral

Unresponsive or repetitive seizures

FOLLOW-UP RECOMMENDATIONS

- Provide seizure precautions and aftercare instructions
- Follow-up with PCP or pediatric neurologist

PEARLS AND PITFALLS

- Phenobarbital is the preferred treatment for theophylline-induced seizures, poor response to benzodiazepines and phenytoin

- Consider buccal or intranasal benzodiazepine if no IV access

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Seizures, Febrile



CODES

ICD9

- 780.31 Febrile convulsions (simple), unspecified
- 780.33 Post traumatic seizures
- 780.39 Other convulsions

ICD10

- R56.00 Simple febrile convulsions
- R56.1 Post traumatic seizures
- R56.9 Unspecified convulsions

SEPSIS

Daniel J. Henning • Nathan Shapiro



BASICS

DESCRIPTION

- Presence of an infection with an associated systemic inflammatory response
- The systemic inflammatory response syndrome (SIRS) is composed of 4 criteria:
 - _ Temperature $> 38^{\circ}\text{C}$ or $< 36^{\circ}\text{C}$
 - _ Heart rate > 90 bpm
 - _ Respiratory rate $> 20/\text{min}$ or $\text{PaCO}_2 < 32$ mm Hg
 - _ WBC $> 12,000/\text{mm}^3$, $< 4,000/\text{mm}^3$, or $> 10\%$ band forms
- Sepsis = infection with ≥ 2 SIRS criteria:
 - _ Release of chemical messengers by the inflammatory response
 - _ Macrocirculatory failure through decreased cardiac output or decreased perfusion pressure
 - _ Microcirculatory failure through impaired vascular autoregulatory mechanisms and functional shunting of oxygen
 - _ Cytopathic hypoxia and mitochondrial dysfunction
- Hemodynamic changes result from the inflammatory response:
 - _ Elevated cardiac output in response to vasodilatation
 - _ Later myocardial depression:
- Multiple organ dysfunction syndrome (MODS):
 - _ Adult respiratory distress syndrome (ARDS)
 - _ Acute tubular necrosis and kidney failure
 - _ Hepatic injury and failure
 - _ Disseminated intravascular coagulation
- Sepsis should be viewed as a continuum of severity from a proinflammatory response to organ dysfunction and tissue hypoperfusion:
 - _ Severe sepsis: Sepsis with at least 1 of the following organ dysfunctions:
 - Acidosis
 - Renal dysfunction
 - Acute change in mental status
 - Pulmonary dysfunction
 - Hypotension
 - Thrombocytopenia or coagulopathy
 - Liver dysfunction
 - _ Septic shock: Sepsis-induced hypotension despite fluid resuscitation:
 - Systolic BP < 90 mm Hg or reduction of > 40 mm Hg from baseline
- Sepsis is the 10th leading cause of death in US:
 - _ In-hospital mortality for septic shock is $\sim 30\%$

ETIOLOGY

- Gram-negative bacteria most common:
 - *Escherichia coli*
 - *Pseudomonas aeruginosa*
 - Rickettsiae
 - *Legionella* spp.
- Gram-positive bacteria:
 - *Enterococcus* spp.
 - *Staphylococcus aureus*
 - *Streptococcus pneumoniae*
- Fungi (*Candida* species)
- Viruses

Pediatric Considerations

- Children with a minor infection may have many of the findings of SIRS.
- Major causes of pediatric bacterial sepsis:
 - *Neisseria meningitidis*
 - *Streptococcal pneumonia*
 - *Haemophilus influenzae*

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Question for signs of infection and a systemic inflammatory response:
 - Fever
 - Dyspnea
 - Altered mental status:
 - Change in mental status
 - Confusion
 - Delirium
 - Nausea and vomiting
- Look for a source of the infection:
 - Cough, shortness of breath
 - Abdominal pain
 - Diarrhea
 - Dysuria/frequency
- Past history should highlight risk factors and immunosuppressive states:
 - Underlying terminal illness
 - Recent chemotherapy
 - Malignancy

- History of a splenectomy
- HIV
- Diabetes
- Nursing home resident

Physical-Exam

- An elevated respiratory rate is an early warning sign of sepsis and occurs without underlying pulmonary pathology or acidosis.
- BP is often normal early in sepsis.
- Hypotension when septic shock occurs
- Extremities are often warmed and flushed despite hypotension.
- Look for a source of the infection:
 - Abdominal exam
 - Rectal exam to assess for an abscess
 - Chest exam for signs of pneumonia
 - Any rash is important:
 - Localized erythema with lymphangitis (streptococcal or staphylococcal cellulitis)
 - Rash involving palms of hands and soles of feet (rickettsial infection)
 - Petechiae scattered on the torso and extremities (meningococemia)
 - Ecthyma gangrenosum (pseudomonas septicemia)
 - Round, indurated, painless lesion with surrounding erythema and central necrotic black eschar
 - Decubitus ulcers
 - Indwelling catheter:
- CNS infections:
 - Coma
 - Neck stiffness (meningitis)

ESSENTIAL WORKUP

- Serum lactate should be done early in the course to assess severity and need for goal-directed therapy
- Blood cultures prior to antibiotics:
 - Broad spectrum of lab tests and imaging studies to locate the source of the infection and assess for MOF.
 - Placement of a central line with an ScvO₂ catheter may be used to adjust therapy.

DIAGNOSIS TESTS & NTERPRETATION

Lab

- Serum lactate:

- > 4 mmol/L defines severe sepsis
- Normal lactate does not rule out septic shock
- CBC with differential:
 - Leukocytosis is insensitive and nonspecific
 - Neutrophil count < 500 cells/mm³ should prompt isolation and empiric IV antibiotics in chemotherapy patients.
 - > 5% bands on a peripheral smear is an imperfect indicator of infection.
 - Hematocrit:
 - Patients should be maintained with a hematocrit > 30% and hemoglobin > 10 g/dL.
 - Platelets:
 - May be elevated in the presence of infection or sepsis-induced volume depletion
 - Low platelet count is a significant predictor of bacteremia and death.
- Electrolytes, BUN, creatinine, glucose:
- Ca, Mg, pH
- C-reactive protein
- Cortisol level
- INR/prothrombin time/partial thromboplastin time
- Liver function tests
- ABG or VBG:
 - Mixed acid–base abnormalities: Respiratory alkalosis with metabolic acidosis
 - VBG correlates very closely with ABG, except for SaO₂
- Blood cultures:
 - From 2 different sites
 - 1 may be drawn through an indwelling central line (i.e., Broviac).
- Urine analysis and culture

Imaging

- CXR:
 - Determine whether pneumonia is the infectious source.
 - Fluffy, bilateral infiltrates may indicate that ARDS is already present.
 - Free air under the diaphragm indicates the source of the infection is intraperitoneal and a surgical intervention is mandatory.
- Soft tissue plain films:
 - Indicated if extremity erythema or severe pain
 - Air in the soft tissues associated with necrotizing or gas-forming infection
- Imaging studies to locate the source of the infection based on the presentation:
 - CT scan of the abdomen and pelvis
 - Abdominal US for gallbladder disease
 - Transthoracic or transesophageal echocardiogram

Diagnostic Procedures/Surgery

- Lumbar puncture:
 - For meningeal signs or altered mental status
- Central venous access:
 - Central venous pressure (CVP) and ongoing measurement of central venous oximetry.

DIFFERENTIAL DIAGNOSIS

- Pancreatitis
- Trauma
- Hemorrhage
- Cardiogenic shock
- Toxic shock syndrome
- Anaphylaxis
- Adrenal insufficiency
- Drug or toxin reactions
- Heavy metal poisoning
- Hepatic insufficiency
- Neurogenic shock



TREATMENT

PRE HOSPITAL

Aggressive fluid resuscitation for hypotension

INITIAL STABILIZATION/THERAPY

- ABCs
- Supplemental oxygen to maintain $\text{PaO}_2 > 60$ mm Hg
- Intubation and mechanical ventilation if shock or hypoxia are present
- Administer 0.9% NS IV.

ED TREATMENT/PROCEDURES

- Early goal-directed therapy:
 - 500 cc boluses of 0.9% saline up to 1–2 L empirically
 - Place central line.
 - Continue 500 cc saline boluses until $\text{CVP} > 8$ cm H_2O .
 - If the mean arterial pressure < 65 mm Hg and $\text{CVP} > 8$, then initiate pressors:
 - Norepinephrine or dopamine to raise BP
 - Norepinephrine is preferred if tachycardia or dysrhythmias are present.
 - Epinephrine for cases where shock is refractory to other pressors

- If the ScvO₂ < 70 and HCT < 30, transfuse 2 U PRBCs.
- If ScvO₂ > 70 and HCT > 30 and MAP > 60, then add dobutamine.
- Administer antibiotics early, based on the most likely organisms or site of infection.
- If source identified, or highly suspected, treat the most likely organisms:
 - _ Cover for MRSA, VRE, and *Pseudomonas* if there are risk factors
 - _ Pulmonary source:
 - 2nd- or 3rd-generation cephalosporin and gentamicin
 - _ Intra-abdominal source:
 - Ampicillin and metronidazole and gentamicin
 - Cefoxitin and gentamicin
 - _ Urinary tract source:
 - Ampicillin or piperacillin and gentamicin or levofloxacin
- Consider stress-dose hydrocortisone if recent steroid use or possible adrenal insufficiency

Pediatric Considerations

- Antibiotic therapy based on age:
 - _ < 3 mo (2 drugs): Ampicillin and gentamicin or cefotaxime (50–180 mg/kg/d div. q4–6h)
 - _ ≥ 3 mo: Cefotaxime or ceftriaxone (50–100 mg/kg/d div. q12–24 h)
- Initiate vasopressors after no response to 60 mL/kg IV fluid.
- Avoid hyponatremia and hypoglycemia.
- Dexamethasone for children with bacterial meningitis:
 - _ 0.15 mg/kg q6h for 4 days

MEDICATION

- Ampicillin: 1–2 g (peds: 50–200 mg/kg/24 h) IV q4–6h
- Cefoxitin: 1–2 g (peds: 100–160 mg/kg/24 h) IV q6–8h
- Ceftazidime: 1–2 g (peds: 100–150 mg/kg/24 h) IV q8–12h
- Dopamine: 1–5 µg/kg/min (renal dose); 5–10 µg/kg/min (pressor dose)
- Gentamicin: 1–1.5 mg/kg (peds: 2–2.5 mg/kg q8h) IV q8h
- Hydrocortisone: 100 mg IV q6–8h
- Metronidazole: Load with 1 g (peds: 15 mg/kg) IV, then 500 mg (peds: 7.5 mg/kg q6h)
- Nafcillin: 1–2 g IV q4h (peds: 50 mg/kg/24 h div. q4–6h)
- Norepinephrine: 2–8 µg/min
- Piperacillin: 3–4 g IV q4–6h
- Vancomycin: 500 mg (peds: 10 mg/kg) IV q6h

First Line

- Normal immune function without an identifiable source:

- 2nd- or 3rd-generation cephalosporin and gentamicin
- Nafcillin and gentamicin
- Add vancomycin if there is a history of methicillin-resistant *S. aureus*, or the patient resides in a nursing facility, or there is a history of recent hospitalizations.

Second Line

Immunocompromised host without an identifiable source:

- Piperacillin and gentamicin
- Ceftazidime and either nafcillin or vancomycin and gentamicin



FOLLOW-UP

DISPOSITION

Admission Criteria

Sepsis almost always requires inpatient care.

Discharge Criteria

Patients with less severe infections (e.g., streptococcal pharyngitis) meeting the criteria for sepsis with stabilized vital signs

Issues for Referral

Sepsis with toxicity, septicemia, or septic shock requires admission, generally to an ICU.

PEARLS AND PITFALLS

- Start antibiotics as soon as sepsis is suspected.
- Failure to recognize multiorgan failure and initiate aggressive fluid resuscitation in the initial presentation of sepsis is a pitfall.

ADDITIONAL READING

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CODES

ICD9

- 038.42 Septicemia due to escherichia coli [E. coli]
- 038.43 Septicemia due to pseudomonas
- 995.91 Sepsis

ICD10

- A41.9 Sepsis, unspecified organism
- A41.51 Sepsis due to Escherichia coli [E. coli]
- A41.52 Sepsis due to Pseudomonas

SEROTONIN SYNDROME (DRUG-INDUCED)

Andrew C. Kendall • Jenny J. Lu



BASICS

DESCRIPTION

- Constellation of signs and symptoms from excessive stimulation of central and peripheral serotonergic receptors
- Spectrum of symptoms may range from mild and subtle findings to severe and sometimes fatal toxicity
- Results from use of serotonergic agents, alone or in combination with other serotonergic agents (may be therapeutic, intentional overdose, recreational, drug interactions)
- Classic triad:
 - Autonomic dysfunction: Hyperthermia, diaphoresis, tachycardia, and hypertension
 - Cognitive changes: Confusion, agitation, hallucinations, decreased responsiveness
 - Neuromuscular excitability: Hyperreflexia, myoclonus, tremors

EPIDEMIOLOGY

Incidence and Prevalence Estimates

- SSRIs implicated most often, alone or in combination with other drugs
- Incidence higher in females but fatalities greater in males
- Highest incidence in ages 19–39
- Most fatalities from drug/drug interactions or recreational abuse

ETIOLOGY

- Serotonin produced by metabolism of L-tryptophan
- Exerts action on 5-hydroxytryptophan (5-HT) receptors of which there are 7 types located in central and peripheral nervous systems:
 - Influences sleep and temperature regulation, affective behavior, food intake, migraines, emesis, sexual behavior, nociception, motor tone, GI motility, and vascular tone
- Extensive list of serotonergic agents, with psychiatric meds most common (SSRIs, SNRIs):
 - Examples: Citalopram, desvenlafaxine, duloxetine, escitalopram, fluoxetine, fluvoxamine, paroxetine, sertraline, trazodone, venlafaxine
- Other serotonergic agents include (not exhaustive):
 - Buspirone, cocaine, dextromethorphan, fentanyl, lithium, MAOIs, MDMA

(ecstasy), meperidine, methadone, metoclopramide, ondansetron, selegiline, St. John's wort, TCAs, tramadol, triptans (controversial)

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- May be difficult to obtain:
 - Family, friends, EMS personnel, may provide additional information
- Patient medication list: Prescribed medications, over-the-counter medications, herbal supplements
- Medical history:
 - Seizures, migraines, attention deficit/hyperactivity disorder, Parkinson, recent illnesses
- Psychiatric history
- Illicit drug abuse history
- Onset of symptoms:
 - Mental status/behavioral changes, development of hyperthermia, muscular rigidity/clonus

Physical-Exam

- Vital signs:
 - Hyperthermia
 - Tachycardia
 - Hypertension or hypotension (may evolve into frank shock and cardiovascular collapse)
- Dermatologic:
 - Diaphoresis, normal color
- GI:
 - Hyperactive bowel sounds
 - Diarrhea
- Mental status/neurologic:
 - Agitation
 - Altered mental status
 - Ocular clonus
 - Hallucinations
 - Waxing/waning delirium
- Musculoskeletal:
 - Clonus: Most helpful finding in diagnosis, greater in lower extremities
 - Hypertonicity and rigidity, greater in lower extremities
 - Hyperreflexia, greater in lower extremities

ESSENTIAL WORKUP

- Careful history and physical exam as it is a clinical diagnosis
- Hunter criteria – most sensitive (84%) and specific (97%) criteria for diagnosis. Requires having taken/been on a serotonergic agent and 1 of the following:
 - Spontaneous clonus
 - Inducible clonus plus agitation or diaphoresis
 - Ocular clonus plus agitation or diaphoresis
 - Tremors plus hyperreflexia
 - Hypertonia plus temperature $> 38^{\circ}\text{C}$ plus ocular clonus or inducible clonus
- Consider other etiologies (sepsis, CVA, etc.)

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Blood chemistry/electrolytes, renal function
- Urine and serum tox screens may detect coingestants
- Lactate, pH
- Total CK
- Cell count, blood/urine cultures if infectious process suspected

Imaging

- Consider CT head if appropriate (trauma, infectious)
- EKG:
 - Evaluate QRS/QT intervals, dysrhythmias

DIFFERENTIAL DIAGNOSIS

- Other intoxications (cocaine, amphetamines, anticholinergic agents, ecstasy, PCP):
 - Neuroleptic malignant syndrome
 - Sympathomimetic toxicity
 - Malignant hyperthermia
 - Anticholinergic toxicity
 - Infectious process (meningitis, encephalitis)



TREATMENT

PRE HOSPITAL

- Stabilize airway
- Vital signs
- IV access
- Fingerstick glucose
- Oxygen administration if needed

INITIAL STABILIZATION/THERAPY

- Stabilize airway, establish IV access, continuous cardiac and temperature monitoring
- Conscientious avoidance of additional serotonergic agents while in-hospital (e.g., caution with ondansetron, fentanyl, linezolid, meperidine, dextromethorphan)
- Supportive care is cornerstone of treatment
 - Aggressive cooling measures particularly important if hyperthermia present
 - Fluid resuscitation

ED TREATMENT/PROCEDURES

- Benzodiazepines are 1st-line medications:
 - Lorazepam, diazepam
- Aggressive cooling measures for hyperthermia:
 - Ice packs, cooling blanket, cool mists/fans
 - Hyperthermia derives from muscular rigidity and is not usually responsive to antipyretic medications
- Severe symptoms (e.g., uncontrollable hyperthermia) may necessitate intubation:
 - Paralytics may be required to control muscular rigidity and hyperthermia
- Cyproheptadine:
 - Nonspecific antihistamine with 5-HT_{2A} antagonist activity may be considered for severe cases, but benefit has not been definitively established
 - Only PO available (must be crushed and given through oro- or nasogastric tube)
- Poison Control Center/Toxicology guidance (1-800-222-1222)



FOLLOW-UP

DISPOSITION

Admission Criteria

- All patients suspected to have serotonin toxicity, even mild-appearing cases, should be admitted for monitoring and treatment
- Severe symptoms including uncontrollable hypertension, altered mental status, cardiovascular instability, hyperthermia require ICU monitoring

Discharge Criteria

- Discharge may be considered when all symptoms have resolved
- Careful evaluation of discharge medications and patient education is essential
- Poison Control Center guidance is recommended

FOLLOW-UP RECOMMENDATIONS

Follow up with primary care after discharge

PEARLS AND PITFALLS

- Serotonin syndrome may be mild to severe in presentation; diagnosis in mild cases often elusive/missed
- Mental status changes, hyperthermia, muscular clonus in lower extremities are important findings
- Hyperthermia is due to muscular rigidity, should be aggressively controlled, and is not responsive to antipyretics
- Cyproheptadine has not been shown definitively to be beneficial but may be considered in severe cases
- Attentive supportive care and avoidance of serotonergic agents is the mainstay of care

ADDITIONAL READING

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CODES

ICD9

333.99 Other extrapyramidal diseases and abnormal movement disorders

ICD10

G25.89 Other specified extrapyramidal and movement disorders

SERUM SICKNESS

Anika Backster • Murtaza Akhter



BASICS

DESCRIPTION

- Type III hypersensitivity reaction
- When a foreign protein or drug (the antigen) is injected, the body's immune system responds by forming antibodies to the foreign material and subsequently forms complexes composed of the antigen, antibody, and complement.
- These complexes then deposit in tissue, inciting an inflammatory response:
 - C3a and C5a act as anaphylatoxins.
 - C5a is strongly chemotactic for neutrophils.
 - The neutrophils then infiltrate the vessel wall at the site of the immune complex deposition and release enzymes, such as collagenase and elastase, which damage vessel walls.
- Typically, symptoms arise 6–21 days after the primary exposure to the antigen.
- Symptoms can start 1–4 days after exposure if there has been an initial immunizing exposure.
- Symptoms typically last 1–2 wk before spontaneously resolving.

ETIOLOGY

- Serum sickness:
 - Vaccines containing foreign protein or serum such as pneumococcal vaccine or rabies.
 - Antivenom and tetanus inoculations made with horse or sheep protein
 - Monoclonal antibodies
- Serum sickness–like reaction:
 - Caused by nonprotein drugs, mostly antibiotics:
 - Penicillins, amoxicillin
 - Cephalosporins (i.e., Cefaclor)
 - Sulfonamides (i.e., Bactrim)
 - Thiazides
 - Gold
 - Thiouracils
 - Hydantoins
 - Phenylbutazone
 - Aspirin
 - Streptomycin



DIAGNOSIS

SIGNS AND SYMPTOMS

Classic presentation is fever, rash, arthralgias, and lymphadenopathy.

History

- Fever
- Rash (urticarial, morbilliform, scarlatiniform)
- Arthralgias
- Myalgias
- Lymphadenopathy
- Facial and neck edema
- Chest pain
- Shortness of breath

Physical-Exam

- Fever
- Rash
- Lymphadenopathy
- Arthritis
- Edema
- Splenomegaly
- Peripheral neuritis
- Myocarditis/pericarditis
- Anaphylaxis

ESSENTIAL WORKUP

- History of a possible offending agent and time course of 6–21 days before onset of symptoms
- Physical exam revealing rash as well as joint, muscular, cardiac, neurologic, or renal insult from vasculitic type process

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Decreased complement levels
- CBC, possible eosinophilia
- Elevated ESR
- Hypergammaglobulinemia
- Urine with proteinuria or hematuria

Imaging

Consider CXR.

Diagnostic Procedures/Surgery

Biopsy is the only means of definitive diagnosis.

DIFFERENTIAL DIAGNOSIS

- Vasculitides (e.g., polyarteritis nodosa, Goodpasture, Wegener)
- Rashes (e.g., erythema multiforme, toxic epidermal necrolysis)
- Immunologic (e.g., systematic lupus erythematosus, polymyositis, anaphylaxis)
- Infectious (e.g., tick-borne disease, Rocky Mountain spotted fever, mononucleosis)



TREATMENT

PRE HOSPITAL

- ABC stabilization
- Anaphylaxis treatment as indicated.

INITIAL STABILIZATION/THERAPY

ABCs if a severe systemic reaction is present

ED TREATMENT/PROCEDURES

- Symptomatic relief until the disease spontaneously resolves in 1–13 wk
- Antihistamines
- Antipyretics
- NSAIDs
- Prednisone is controversial

MEDICATION

- Acetaminophen: 325–650 mg PO/PR (peds: 10–15 mg/kg) q4–6h
- Diphenhydramine: 50–100 mg (peds: 5 mg/kg/d, div., max. dose 50 mg/dose or 300 mg/24h) q6–8h
- Hydroxyzine: 25–50 mg (peds: 0.5 mg/kg/dose) q6–8h
- Ibuprofen: 200–800 mg PO (peds > 6 mo: 5–10 mg/kg) q6–8h
- Prednisone: 5–60 mg/d PO (peds: 0.5–2 mg/kg/d), 2-wk taper



FOLLOW-UP

DISPOSITION

Admission Criteria

- Involvement of the airway
- Relapse of symptoms and signs after initial steroids
- Immunosuppression
- Concomitant serious disease

- Sociologic considerations

Discharge Criteria

Stable; most cases are self-limiting.

Issues for Referral

Skin testing with heterologous antisera is performed routinely to avoid anaphylaxis to future administration of heterologous serum.

FOLLOW-UP RECOMMENDATIONS

Primary care follow-up

PEARLS AND PITFALLS

- Identification and cessation of the offending antigen is crucial in the treatment of serum sickness.
- Significant morbidity comes from a failure to diagnose when the serum sickness is not considered on the differential.

ADDITIONAL READING

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- Pilette C, Coppens N, Houssiau FA, et al. Severe serum sickness-like syndrome after omalizumab therapy for asthma. *J Allergy Clin Immunol*. 2007;120(4):972–973.



See Also (Topic, Algorithm, Electronic Media Element)

- Anaphylaxis
- Vasculitis



CODES

ICD9

- 999.51 Other serum reaction due to administration of blood and blood products
- 999.52 Other serum reaction due to vaccination
- 999.59 Other serum reaction

ICD10

- T80.61XA Oth serum reaction due to admin blood/products, init
- T80.62XA Other serum reaction due to vaccination, initial encounter
- T80.69XA Other serum reaction due to other serum, initial encounter

SEXUAL ASSAULT

Lauren M. Smith



BASICS

DESCRIPTION

Specific legal definition varies from state to state:

- Nonconsensual completed or attempted penetration between the penis and vulva or penis and anus
- Nonconsensual contact between the mouth and the penis, vulva, or anus
- Nonconsensual penetration of the anal or genital opening with a finger, hand, or object
- Nonconsensual intentional touching, directly or through clothing, of the genitalia, vagina, anus, groin, inner thigh, or buttocks

ETIOLOGY

- Lifetime prevalence of sexual assault in US is 18% in women, 5% in men
- 72% of female rape victims are raped by someone they know; however, men are primarily raped and physically assaulted by strangers and acquaintances, not intimate partners.
- Women who are disabled, pregnant, or attempting to leave their abusers are at increased risk of intimate partner rape.
- Prevalence of sexual assault in men is higher in those who are gay, bisexual, veterans, prison inmates or seeking mental health services
- Nearly 25% of women and 7% of men have been raped or sexually assaulted by a current or former partner.



DIAGNOSIS

SIGNS AND SYMPTOMS

- Victims might not disclose assault:
 - Most will reveal the history only in response to direct questions.
- Tachycardia or pounding heart beat
- Headaches
- Nausea
- Back pain
- Skin problems
- Menstrual symptoms
- Sudden weight change
- Sleeping disorders
- Abdominal pain

- Trouble breathing
- Associated injuries:
 - Of those with injuries, 70% report no injury at presentation.
 - Lacerations of perineum
 - Vulvar trauma
 - Laceration of vaginal wall (more common in younger patients, near introitus)
 - Multiple contusions
 - Abrasions
 - Human bite
 - Lacerations or puncture wound to extremity
 - Burns
 - Depressed skull fracture

Pediatric Considerations

- ~ 54% of rapes of women occur before the age of 18.
- Must follow state laws regarding child abuse
- Most of the physical exams in child sexual abuse cases are normal
- In prepubertal children, an exam will most likely not require a speculum exam. If a speculum exam is warranted, it should be done under sedation; consider involving a sexual assault examiner.
- In interviewing the child, ask open-ended questions.
- Use toys and dolls to have the child explain what happened.
- Early psychiatric intervention is necessary.

Pregnancy Considerations

Women who are pregnant have higher rates of abuse/assault

History

- Obtain complete history even if patient does not wish to file charges, including:
 - Date, time, and place of assault
 - Physical description of assailants
 - Number of assailants
 - Types of penetration: Vaginal, oral, rectal
 - Assailant ejaculation: Ask if assailant used condom
 - Any bodily fluid exchange
 - Use of force, weapons, restraints, drugs, or alcohol
 - Ask if victim has memory loss or loss of consciousness
 - Victim's activity since assault:
 - Changed clothes
 - Douched
 - Bathed

- Urinated
- Defecated
- Eaten
- Tampon use
- Full gynecologic history
- Last voluntary intercourse
- Sperm may be mobile up to 5 days in cervix and 12 hr in vagina
- Address all physical complaints.

Physical-Exam

- Use local evidence kit even if victim is unsure of reporting to police.
- Female chaperone required if male physician
- If clothes soiled, photograph prior to undressing, with patient's consent.
- Note emotional state of victim.
- Note general appearance of clothes:
 - Staining
 - Tears
 - Mud
 - Leaves
 - Wood lamp for seminal stains
 - Have patient disrobe while standing on sheet and place all clothes in paper bag.
- Plastic causes mold and increases bacterial counts.
- Only the patient should handle the clothing.
- Arrange for change of clothes.
- Complete physical exam should be done with emphasis on:
 - Abrasions
 - Lacerations
 - Bites
 - Scratches
 - Foreign bodies
 - Ecchymosis
 - Dried semen on skin
- Forensic collection:
 - Fingernail scrapings
 - Scalp or pubic hair samples
 - If oral penetration, swab between teeth for acid phosphatase (assay for semen) and sperm.
 - Throat culture for *Gonococcus* and *Chlamydia* if oral sex
- Gynecologic exam:
 - Explain all steps and allow patient to pace exam.
 - Comb and collect pubic hair per local protocol.
 - Lubricate speculum with water (not lubricant).

- Look for genital trauma even in asymptomatic patients.
- May use toluidine blue to identify small pelvic lacerations from traumatic intercourse:
 - Best applied to vaginal mucosa at introitus
- Special attention to hymen as 1 of the most common places for trauma
- Lacerations to vaginal wall near introitus more common in younger patients
- Aspirate secretions pooled in posterior fornix and place in sterile container to be examined for sperm and acid phosphates:
 - If no secretions in posterior fornix, wipe with cotton tip.
 - Swab and microscopically examine for sperm and acid phosphates.
- Swab for *Gonococcus* and *Chlamydia*:
 - Controversial; evidence can be used by defense to show promiscuity.
- Colposcope allows visualization of small lesions and enables photography of findings (performed by many sexual assault nurse examiner [SANE] programs)
- Rectal exam and cultures for *Gonococcus* and *Chlamydia* if there was penetration or attempted penetration

ESSENTIAL WORKUP

- Obtain written consent prior to any exam, test, or treatment.
- Allow patient to pause and proceed at comfortable pace.
- Allow advocate to stay with patient during exam with patient's consent.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Syphilis serology
- Hepatitis B and C panel
- HIV testing and counseling
- Drug testing (if suspect victim was drugged, can be used against victim if other agents detected)
- Blood type
- Pregnancy test
- *Gonococcus* culture
- *Chlamydia* culture
- Other labs as needed based on injuries

Imaging

As indicated by injuries

Diagnostic Procedures/Surgery

As indicated by injuries



TREATMENT

PRE HOSPITAL

- Treat patient in a kind, nonjudgmental manner.
- C-spine immobilization for patients with head/neck trauma

INITIAL STABILIZATION/THERAPY

Treat life-threatening injuries.

ED TREATMENT/PROCEDURES

- Place patient in quiet, private room.
- Assure patient of confidentiality regarding name and reason for visit.
- Regularly assure patient of safety.
- Enforce nonjudgmental behavior by staff.
- Designate nursing and medical provider for entire stay who is familiar with evidence collection kit.
- Have SANE perform exam if available.
- Contact community or in-hospital advocate to stay with patient while in ED.
- Alert hospital security to possibility of assailant presenting to ED.
- Contact police if patient consents or local law requires.
- Collect evidence as outlined above and according to local law.
- Offer pregnancy prophylaxis if not currently pregnant
- Administer prophylactic therapy for *Gonococcus*, *Chlamydia*, *Trichomonas*
- Consider prophylactic HIV treatment
- Consider prophylactic therapy or vaccine for hepatitis B

MEDICATION

ALERT

Risk of pregnancy after rape is ~ 5%

Pregnancy Prophylaxis

Hormonal therapy if within 72 hr:

- Levonorgestrel 0.75 mg PO 1st dose stat and repeat in 12 hr (preferred) or Levonorgestrel 1.5 mg PO, single dose
- Ethinyl estradiol 100 µg PO and levonorgestrel 0.5 mg PO 1st dose stat, repeat in 12 hr (less side effects but less effective)

ALERT

All patients should be offered prophylaxis for STIs

STI Prophylaxis

- Ceftriaxone 250 mg IM once or Cefixime 400 mg PO single dose (*Gonococcus*)

- Doxycycline 100 mg PO BID for 7 days *or* azithromycin 1 g PO, single dose (*Chlamydia*)
- Metronidazole (Flagyl) 2 g PO, 1 dose (*Trichomonas*)

ALERT

If PCN allergic, treat with Azithromycin 2 gm po single dose for *Gonococcus* and *Chlamydia*

Hepatitis B

If not already immunized, start hepatitis B vaccination in the ED, HBIG is not required unless assailant is known hepatitis B positive

HIV Prophylaxis

IF WITHIN 72 HR

- **High-risk exposures** (source known to be HIV+ or is an intravenous drug user [IVDU], or history of men having sex with men) – Lopinavir/ritonavir (Kaletra) 200 mg/50 mg 2 tablets twice daily plus emtricitabine/tenofovir (Truvada) 200 mg/300 mg once daily for 28 days
- Emtricitabine/tenofovir (Truvada) 200 mg/300 mg once daily for exposures from persons other than those noted above, or lamivudine plus zidovudine (Combivir) 1 tab po twice a day for 28 days
- If HIV prophylaxis medications are started, baseline CBC, BMP, and LFTs should be obtained.



FOLLOW-UP

DISPOSITION

Admission Criteria

Serious traumatic injury

Discharge Criteria

- Medical follow-up for culture and HIV test results and monitoring of HIV prophylactic medication side effects (if applicable)
- Psychological follow-up
- Safe place for patient to go to

Issues for Referral

- Mental health services and counseling
- For all pediatric cases, the Department of Children and Family Services should be contacted.

FOLLOW-UP RECOMMENDATIONS

Follow-up should be provided for repeat HIV testing at 6 wk, 3 mo, and 6 mo

PEARLS AND PITFALLS

- ~70% of rape victims do not tell their doctors or seeking mental health services
- Most victims will not disclose assault, unless in response to direct questions.
- Most of the pediatric exams in alleged sexual assault cases will be normal (80–96%)
- Extragenital trauma may be more common than genital
- Over 600 SANE/SART(specially trained forensic examiners) programs exist in US; use of a SANE, if available, may improve medical, legal, and psychological care of sexual assault victims

ADDITIONAL READING

- Campbell R, Patterson D, Lichy LF, et al. The effectiveness of sexual assault nurse examiner (SANE) programs: A review of psychological, medical, legal, and community outcomes. *Trauma Violence Abuse*. 2005;6:313–329.
- Linden JA. Clinical practice. Care of the adult patient after sexual assault. 2011;365(9):834–841.
- Sommers MS. Defining patterns of genital injury from sexual assault: A review. *Trauma Violence Abuse*. 2007;8:270–280.
- Tjaden P, Thoennes N. Extent, nature, and consequences of rape victimization: Findings from the National Violence Against Women. Washington DC: U. S. Department National Institute of Justice and the Centers for Disease Control and Prevention; 2006.



CODES

ICD9

- 995.53 Child sexual abuse
- 995.83 Adult sexual abuse
- V71.5 Observation following alleged rape or seduction

ICD10

- T74.21XA Adult sexual abuse, confirmed, initial encounter
- T74.22XA Child sexual abuse, confirmed, initial encounter
- Z04.41 Encounter for exam and obs following alleged adult rape

SHOCK

Nathan Shapiro • Christopher M. Fischer



BASICS

DESCRIPTION

- Inadequate supply of blood flow to tissues to meet the demands of the tissues
- Tissue oxygen requirements are not fulfilled.
- Toxic metabolites are not removed.
- If untreated, inevitable progression from inadequate perfusion to organ dysfunction and ultimately to death.
- Major categories of shock:
 - Hypovolemic shock:
 - Decreased blood volume
 - Suspect hemorrhage if acute onset
 - Severe dehydration if progressive onset and elevated hematocrit, BUN, and creatinine
 - Obstructive (cardiogenic) shock:
 - Decreased cardiac output and tissue hypoxia with adequate intravascular volume and myocardial dysfunction
 - Venous congestion with increase in central venous pressure
 - Compensatory increase in SVR
 - May be caused by cardiac dysfunction, obstruction to inflow of blood to the heart, or obstruction to outflow of blood from the heart
 - Septic shock:
 - An initial infectious insult overwhelms the immune system.
 - Biochemical messengers (cytokines, leukotrienes, histamines, prostaglandins) cause vessel dilatation.
 - Capillary endothelium becomes disrupted and the vessels leak.
 - Drop in SVR leads to inadequate tissue perfusion.
 - Secondarily, decreased cardiac output from “cardiac stun” resulting in cold septic shock
 - Neurogenic shock:
 - Spinal cord insults disrupt sympathetic stimulation to vessels.
 - Loss of sympathetic tone causes arteriodilating and vasodilatation.
 - Lesions proximal to T4 disrupt sympathetic, spares vagal innervation causing bradycardia.
 - Anaphylactic shock:
 - An antigen stimulates the allergic reaction.
 - Mast cells degranulate.
 - Histamine releases, along with autocooids, stimulate an anaphylaxis

- cascade.
- Vascular smooth muscle relaxes.
- Capillary endothelium leaks.
- Drop in SVR leads to inadequate tissue perfusion.
- Pharmacologic agents may cause shock through smooth muscle dilation or myocardial depression.

ETIOLOGY

- Hypovolemic shock:
 - Abdominal trauma, blunt or penetrating
 - Abortion—complete, partial, or inevitable
 - Anemia—chronic or acute
 - Aneurysms—abdominal, thoracic, dissecting
 - Aortogastric fistula
 - Arteriovenous malformations
 - Blunt trauma
 - Burns
 - Diabetes
 - Diarrhea
 - Diuretics
 - Ruptured ectopic pregnancy
 - Epistaxis
 - Fractures (especially long bones)
 - Hemoptysis
 - GI bleed
 - Mallory–Weiss tear
 - Penetrating trauma
 - Placenta previa
 - Postpartum hemorrhage
 - Retroperitoneal bleed
 - Severe ascites
 - Splenic rupture
- Toxic epidermal necrolysis:
 - Vascular injuries
 - Vomiting
- Cardiogenic shock:
 - Cardiomyopathy
 - Conduction abnormalities and arrhythmias
 - MI
 - Myocardial contusion
 - Myocarditis
 - Pericardial tamponade
 - Pulmonary embolus

- Tension pneumothorax
- Valvular insufficiency
- Ventricular septal defect
- Vasogenic shock:
 - Acute respiratory distress syndrome
 - Bacterial infection
 - Bowel perforation
 - Cellulitis
 - Cholangitis
 - Cholecystitis
 - Endocarditis
 - Endometritis
 - Fungemia
 - Infected indwelling prosthetic device
 - Intra-abdominal infection or abscess
 - Mediastinitis
 - Meningitis
 - Myometritis
 - Pelvic inflammatory disease
 - Peritonitis
 - Pyelonephritis
 - Pharyngitis
 - Pneumonia
 - Septic arthritis
 - Thrombophlebitis
 - Tubo-ovarian abscess
 - Urosepsis
- Anaphylactic:
 - Drug reaction (most commonly to aspirin, β -lactam antibiotics)
 - Exercise (rare)
 - Food allergy (peanuts, tree nuts, shellfish, fish, milk, eggs, soy, and wheat account for 90% of food-related anaphylaxis)
 - Insect sting
 - Latex
 - Radiographic contrast materials
 - Synthetic products
- Pharmacologic:
 - Antihypertensives
 - Antidepressants
 - Benzodiazepines
 - Cholinergics
 - Digoxin
 - Narcotics

- Nitrates
- Neurogenic:
 - Spinal cord injury



DIAGNOSIS

SIGNS AND SYMPTOMS

Generalized shock:

- Hypotension
- Decreased peripheral pulses
- Tachycardia
- Tachypnea
- Decreased urine output
- Diaphoresis
- Obtundation
- Lethargy

History

Standard medical history with a goal of deducing the etiology of the shock and important precipitating factors

Physical-Exam

- Standard physical exam to assist in determining the etiology (e.g., wounds, cardiac exam signs of cellulitis and urticarial rash, etc.)
- Targeted physical exam to focus on the type of shock state:
 - Hypovolemic (classic symptoms):
 - Neck veins are flat.
 - Mucous membranes are dry.
 - Extremities are cold.
 - Cardiogenic shock (classic symptoms):
 - Jugular venous distension is present.
 - Mucous membranes are moist.
 - Extremities are cold.
 - Early septic shock (classic symptoms):
 - Neck veins are flat.
 - Mucous membranes are dry.
 - Extremities are warm.
 - During late shock, extremities may become cold and mottled.

ESSENTIAL WORKUP

- Identify type or types of shock present.
- Identify underlying cause of shock.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Hemoglobin/hematocrit
- WBC:
 - High: Nonspecific marker of infection
 - Low: Neutropenic infections
- Electrolytes
- Blood glucose:
 - High: Diabetic ketoacidosis or septic shock
 - Low: Pediatric sepsis
- Prothrombin time/partial thromboplastin time
- Cardiac enzymes
- Urinalysis
- β -human chorionic gonadotropin
- Lactic acid level:
 - Good surrogate marker of shock state

Imaging

- CXR
- ECG
- Abdominal US
- CT abdomen:
 - Requires that the patient 1st be stabilized
 - In the setting of abdominal trauma and in search for suspicion of abdominal infection

Diagnostic Procedures/Surgery

EKG:

- Assess for ischemia and other disorders of cardiac muscle:
- Electrical alternans or low voltage with cardiac tamponade
- Right-heart strain with pulmonary embolism



TREATMENT

PRE HOSPITAL

- ABCs per standard protocol
- Fluid resuscitation as warranted

INITIAL STABILIZATION/THERAPY

- Large-bore IV access:
 - When possible, central venous access and monitoring

- Fluid resuscitation in noncardiogenic shock patients
- Control bleeding with direct pressure measures.
- Stabilization of a fractured pelvis with sheet or commercial device or external fixation

ED TREATMENT/PROCEDURES

- Hypovolemic shock:
 - _ Identify source of volume depletion
 - _ Aggressive fluid resuscitation keeping systolic blood pressure (SBP) > 100 mm Hg until definitive treatment
 - _ 2–3 L crystalloid initially
 - _ Packed RBCs if 2–3 L crystalloids do not improve SBP
 - _ Identify source of bleeding and rapidly move toward definitive treatment.
 - _ Thoracotomy and aortic cross-clamping in refractory shock with penetrating torso trauma
- Cardiogenic shock:
 - _ Ease work of breathing with intubation
 - _ Insult-specific therapy (e.g., thrombolytics for MI, pericardiocentesis for pericardial tamponade)
 - _ Treat dysrhythmias.
 - _ Vasopressors (norepinephrine or dopamine) as needed
- Septic shock:
 - _ Aggressive crystalloid fluid resuscitation
 - _ Titrate fluid to urine output > 30 cc/hr
 - _ Blood product transfusion to maintain HCT 30–35%
 - _ Early antimicrobial therapy
 - _ Inotropic support as needed
 - _ Norepinephrine as preferred 1st-line infusion
- Anaphylactic shock:
 - _ Intubation for airway compromise
 - _ Epinephrine
 - _ Subcutaneous in noncritical settings
 - _ IV drip for immediate life threats or refractory hypotension
 - _ H1 blockers (diphenhydramine)
 - _ H2 blockers (cimetidine)
 - _ Corticosteroids (hydrocortisone or methylprednisolone)
 - _ Nebulized β 2-antagonists for bronchospasm
 - _ Patients taking β -blockers may be more likely to experience severe symptoms of anaphylaxis
- Pharmacologic shock:
 - _ Decontamination of overdoses with charcoal
 - _ Inotropic agents as needed
 - _ Drug-specific antidotes

- Neurogenic shock:
 - Supportive therapy
 - Traction and fracture stabilization
 - Corticosteroids

MEDICATION

- Albuterol: 2.5 mg/2.5 cc nebulizer PRN
- Calcium gluconate: 100–1,000 mg IV at 0.5–2 mL/min
- Cimetidine: 300 mg IV
- Diphenhydramine: 50–100 mg IV over 3 min
- Dobutamine: 5–40 µg/kg/min IV:
 - Dopaminergic: 1–3 µg/kg/min IV
 - β-effects: 3–10 µg/kg/min IV
 - α/β-effects: 10–20 µg/kg/min IV
 - α-effects: 20 µg/kg/min IV
- Epinephrine:
 - 1–4 µg/min IV infusion
 - Endotracheal 1 mg (10 mL of 1:10,000) once followed by 5 quick insufflations
 - Place 1 mg in 250 mL D₅W = 4 µg/mL
- Glucagon: 1–5 mg IV bolus initial, then 1–20 mg/h infusion
- Hydrocortisone: 5–10 mg/kg IV
- Methylprednisolone: 1–2 mg/kg IV
- Naloxone: 0.01 mg/kg IV initial, titrate to effect
- Norepinephrine: Start 2–4 µg/min IV, titrate up to 1–2 µg/kg/min IV
- Phenylephrine: 40–180 µg/min IV



FOLLOW-UP

DISPOSITION

Admission Criteria

- All patients in shock need to be admitted.
- ICU criteria:
 - All patients with persistent shock need ICU monitoring.
- Patients with shock definitively reversed may be admitted to non-ICU setting (e.g., tension pneumothorax that has been decompressed and chest tube placed).

Discharge Criteria

Patients who are in shock should not be discharged home from the ED.

Issues for Referral

- Traumatic hypovolemic shock (hemorrhagic shock) patients may require a trauma center.
- Patients with cardiogenic shock due to MI may require cardiac catheterization or additional cardiac surgery support.
- Septic shock due to necrotizing fasciitis may require advanced surgical support.
- Neurogenic shock with spinal cord injury will require neurosurgical care.

PEARLS AND PITFALLS

- Identify the etiology of shock.
- Aggressively resuscitate the patient, 1st with IV fluids and next with vasopressor support to minimize hypoxic exposure.

ADDITIONAL READING

- Havel C, Arrich J, Losert H, et al. Vasopressors for hypotensive shock. *Cochrane Database Syst Rev.* 2011;(5):CD003709.
- Puskarich MA. Emergency management of severe sepsis and septic shock. *Curr Opin Crit Care.* 2012;18(4):295–300.
- Strehlow MC. Early identification of shock in critically ill patients. *Emerg Med Clin North Am.* 2010;28(1):57–66, vii.



CODES

ICD9

- 785.50 Shock, unspecified
- 785.51 Cardiogenic shock
- 785.59 Other shock without mention of trauma

ICD10

- R57.0 Cardiogenic shock
- R57.1 Hypovolemic shock
- R57.9 Shock, unspecified

SHOULDER DISLOCATION

Doodnauth Hiranman • Wallace A. Carter



BASICS

DESCRIPTION

- Shoulder is a very dynamic joint, prone to injury.
- Anterior dislocation (90–96%):
 - Injury is from direct or indirect forces on the abducted and externally rotated arm.
 - Injury may also result from a direct blow to posterolateral aspect of shoulder.
- Posterior dislocation:
 - Often missed
 - Forces on the adducted and internally rotated arm result in posterior dislocation of humeral head in relation to glenoid fossa.
 - Most common mechanism is seizure and sudden contraction of all the posterior muscle groups.
 - Other mechanisms include electrocution and direct blow to anterior shoulder.
- Inferior dislocation (rare):
 - Luxatio erecta
 - Hyperabduction of arm, tear of rotator cuff, and rotation of arm 180° above head
 - Commonly seen after a fall from a height:
 - Arm has struck object on descent and is thrust above the head.
 - Often accompanied by neurovascular injury and fracture

Pediatric Considerations

Dislocation is rare in children: Epiphyseal fractures must be suspected.

Geriatric Considerations

Dislocation is often accompanied by fracture.

ETIOLOGY

- Falls from height
- Impact injuries
- Distraction injuries of upper arm
- Seizures
- Electrocution

SIGNS AND SYMPTOMS

- Severe pain in the affected shoulder
- Anterior dislocation:
 - Shoulder is squared off.
 - Prominent acromion process and palpable anterior fullness
 - Arm is held in slight abduction and external rotation.
- Posterior dislocation:
 - Coracoid process is prominent, with a palpable posterior bulge.
 - Arm is held in slight adduction and internal rotation.
- Inferior dislocation (luxatio erecta):
 - Rare but easy to identify
 - Arm is shortened and fixed above head as if raised to ask a question.
- Head of humerus may be palpable on the lateral chest wall.

ESSENTIAL WORKUP

- Evaluate neurovascular status of distal arm.
- Retest neurovascular status after any manipulation.
- Dislocation requires prompt treatment:
 - Incidence of post-traumatic arthritis increases with time dislocation is untreated.
 - Plain films of the shoulder should be obtained immediately.
 - Even in clinically obvious cases, films should be obtained before manipulation, unless a significant delay will result.
 - An impacted humeral head fracture may be converted to a displaced humeral head fracture if manipulated.

DIAGNOSIS TESTS & INTERPRETATION***Imaging***

- At least 2 views should be obtained:
 - Anteroposterior (AP):
 - To visualize dislocation or fracture
 - Trans-scapular Y or axillary view:
 - To visualize if anterior or posterior
- Anterior dislocation:
 - Posterolateral compression fracture of the humeral head (Hill–Sachs deformity)
 - Corresponding lesion on anterior glenoid rim is the Bankart lesion:
 - These do not require treatment.
 - Fractures of the greater tuberosity of the humeral head are seen in 15–35%:
 - If there is > 1 cm displacement after reduction, surgical intervention

may be necessary.

- Posterior dislocation:
 - Often missed on AP film
- Degree of overlap on radiographic film is smaller and displaced superiorly, producing the meniscus sign.
- Rotated humerus yields “light bulb on a stick” finding on AP view:
 - Reverse Hill-Sachs deformity from compression fracture of the anterior medial humeral head may also be seen.

DIFFERENTIAL DIAGNOSIS

- Fracture of the humeral head
- Fracture of the humeral shaft
- Acromioclavicular injury
- Septic shoulder joint
- Hemarthrosis in shoulder joint
- Scapular fracture
- Cervical spine injury



TREATMENT

PRE HOSPITAL

Neurovascular injury should be identified and the arm splinted in the position of most comfort.

INITIAL STABILIZATION/THERAPY

- Airway management and resuscitate as indicated.
- Exclude more serious injuries, especially in multitrauma patient.
- Ensure no injury to axillary nerve or vessels.

ED TREATMENT/PROCEDURES

- Adequate analgesia and muscle relaxation are essential for successful reduction:
 - Procedural sedation with a short-acting opioid and a benzodiazepine *OR*
 - Methohexital or etomidate alone
 - In the cooperative patient, intra-articular block only (20 cc of lidocaine 1% or bupivacaine 0.5%) into shoulder joint
- Anterior dislocation reduction techniques:
 - Scapular manipulation:
 - Patient seated, traction to arm in horizontal plane, countertraction with other hand on clavicle
 - 2nd person adducts tip of scapula medially, moving glenoid fossa
 - Stimson:
 - Patient in prone position with arm dangling over side, hang 10–15 lb around wrist; muscle fatigued over 20–30 min

- Can concurrently use scapular manipulation
- Only 1 person required
- Traction/countertraction:
 - Patient in supine position with continuous longitudinal traction to affected arm
 - Countertraction from sheet wrapped around chest
 - Arm internally or externally rotated if unsuccessful after several minutes
- External rotation:
 - Patient supine; elbow at 90°; gentle, slow external rotation of arm
 - Should be done slowly and with cooperative patient
- Posterior dislocation reduction techniques:
 - May use Stimson or traction/countertraction techniques with manipulation of humeral head anteriorly
- Inferior dislocation (luxatio erecta) reduction techniques:
 - Patient in supine position; gentle longitudinal traction to distract humeral head
 - Gentle countertraction with sheet draped over trapezius and chest
 - Arm slowly rotated from 180–0°
- Postreduction care:
 - Postreduction films
 - Place in sling and swath or shoulder immobilizer immediately after reduction.
 - Shoulder should remain immobilized for 2–3 wk in young patients.
 - Immobilization time should be less in older patients to avoid frozen shoulder.

MEDICATION

- Bupivacaine 0.5%: 20 cc intra-articular to shoulder
- Diazepam: 5–10 mg IV (peds: 0.2 mg/kg)
- Etomidate: 0.2 mg/kg IV (adult and peds)
- Fentanyl: 50–100 µg IV (peds: 2–4 µg/kg)
- Lidocaine 1%: 20 cc intra-articular to shoulder
- Methohexital: 1–1.5 mg/kg IV (peds: Not routinely used)
- Midazolam: 2–5 mg IV (peds: 0.035–0.1 mg/kg)
- Morphine: 2–8 mg IV (peds: 0.1 mg/kg); use preservative-free formulation.
- Propofol: 1–2 mg/kg IV



FOLLOW-UP

DISPOSITION

Admission Criteria

- Failure to reduce shoulder may require admission for reduction under general anesthesia or open reduction.
- Patients with neurovascular compromise

Discharge Criteria

- Patients with successful reductions, confirmed by plain films, may be discharged with shoulder in appropriate immobilizer and with orthopedic follow-up.
- Recurrent dislocation may require elective surgery.
- Patients with residual neurapraxia from injury or manipulation may be safely discharged with instructions that most symptoms will resolve, but should have neurology follow-up.

Issues for Referral

- Patients with residual neurapraxia should be advised to see a neurologist.
- Routine orthopedic consultation should be advised with all successful reductions.

PEARLS AND PITFALLS

Make sure to document sensory exam of axillary nerve prior to reduction.

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CODES

ICD9

- 831.00 Closed dislocation of shoulder, unspecified

- 831.01 Closed anterior dislocation of humerus
- 831.02 Closed posterior dislocation of humerus

ICD10

- S43.006A Unsp dislocation of unspecified shoulder joint, init encntr
- S43.016A Anterior dislocation of unspecified humerus, init encntr
- S43.026A Posterior dislocation of unspecified humerus, init encntr

SICK SINUS SYNDROME

David F. M. Brown • Nirma D. Bustamante

BASICS

DESCRIPTION

- Collective term used to describe dysfunction in the sinus node's automaticity and impulse generation
- Mechanism:
 - Caused by progressive degeneration of the intrinsic functions of the sinoatrial (SA) node
 - Characterized by periods of unexplained sinus node dysfunction leading to bradyarrhythmias, often without appropriate atrial or junctional escape rhythms
- Syndrome includes:
 - Chronic SA nodal dysfunction
 - Frequently depressed pacemakers
 - Arteriovenous nodal conduction disturbances
 - Sluggish return of SA nodal activity after DC cardioversion
- Presents in all age groups (mean age >65 yr)
- Male/female ratio is 1:1

ETIOLOGY

- Intrinsic causes:
 - Most common cause: Idiopathic degenerative fibrosis of sinus node
 - Coronary artery or SA nodal artery disease
 - Cardiomyopathy
 - Ion channel mutations/familial SSS
 - Leukemia and metastatic disease
 - Infiltrative cardiac or collagen vascular disease, including amyloidosis
 - Surgical trauma
- Inflammatory diseases:
 - Rheumatic heart disease
 - Chagas disease
 - Pericarditis and myocarditis
- Extrinsic causes (not true SSS but similar presentation):
 - Drugs:
 - β -blockers, calcium channel blockers, clonidine
 - Digoxin, amiodarone
 - Lithium, phenytoin
 - Autonomically mediated syndromes (cholinesterase deficiency)

- Hyperkalemia/hypokalemia
- Hypothyroidism
- Hypothermia
- Hypoglycemia
- Sepsis/infection

Pediatric Considerations

Associated with congenital abnormalities and subsequent surgical repair, as well as with congenital SA nodal artery deficiency

DIAGNOSIS

SIGNS AND SYMPTOMS

Symptoms represent CNS hypoperfusion from bradydysrhythmia or traditional cardiovascular presentations

History

- Asymptomatic
- Palpitations/fatigue
- Syncope/presyncope/dizziness
- Anginal equivalents (chest pain/SOB)
- Activity intolerance
- Sudden death

Physical-Exam

- Bradycardia
- Alternating bradycardia and atrial tachycardia
- Altered mental status
- Cyanosis
- Transient ischemic attack/stroke

ESSENTIAL WORKUP

- Ascertaining etiology
- 12-lead EKG
- CXR

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Serum electrolytes (including magnesium and calcium)
- Cardiac markers
- Digoxin level, if appropriate

- Thyroid function testing

Imaging

EKG:

- Most common finding: Chronic, inappropriate sinus bradycardia
- Sinus pauses or SA block
- Atrial fibrillation with slow ventricular response
- Prolonged pauses after cardioversion or carotid massage
- Bradyarrhythmias may alternate with supraventricular tachydysrhythmia.
- Tachy-brady syndrome: Bursts of atrial tachycardia interspersed with bradycardia

Diagnostic Procedures/Surgery

Most electrophysiologic studies are no longer recommended due to poor sensitivity and specificity.

DIFFERENTIAL DIAGNOSIS

- Other bradydysrhythmias
- Other tachyarrhythmias: In particular, be careful to distinguish SSS from atrial fibrillation, because DC cardioversion or the use of nodal agents in presumed Afib can be harmful if SA node dysfunction coexists.
- Electrolyte derangements
- Medication toxicity: β -blockers, calcium channel blockers, clonidine, digoxin
- Excessive vagal tone



TREATMENT

PRE HOSPITAL

- Advanced life support transport
- Oxygen supplementation
- Cardiac monitoring
- Atropine if bradycardic and hemodynamically unstable
- Transcutaneous pacing for unstable patients

INITIAL STABILIZATION/THERAPY

- Atropine if a bradydysrhythmia is causing unstable signs/symptoms: Angina, mental confusion, or hypotension
- Transcutaneous pacing if atropine unsuccessful
- If this fails, emergent transvenous pacing

ED TREATMENT/PROCEDURES

Supraventricular tachydysrhythmia alternating with bradycardia:

- Unstable:

- Cardiovert
- Anticipate subsequent profound bradycardia
- Stable patients:
 - Cardiac monitoring
- Digoxin, diltiazem, verapamil, or magnesium can be used for tachydysrhythmia
- Any medication may cause profound bradycardia
- Bradycardia:
 - Discontinuation of medications that alter sinus node function
 - Correct reversible causes of SA nodal depression: O₂, warming, glucose

ALERT

Rewarming is critical in hypothermia as atropine may cause myocardial instability:

- Anticoagulate patients with atrial fibrillation and tachy-brady syndrome.

MEDICATION

- Atropine: 0.5–1 mg IV/ET:
 - Repeat q5min as necessary, max. dose of 0.04 mg/kg (peds: 0.02 mg/kg, min., 0.1 mg)
- Diltiazem: 0.25 mg/kg IV over 2 min followed in 15 min by 0.35 mg/kg IV over 2 min
- Verapamil: 2.5–5 mg IV bolus over 2 min:
 - May repeat with 5–10 mg q15–30min max. 20 mg
 - Peds < 1 yr: 0.1–0.2 mg/kg over 2 min; repeat q30min 1–15 yr: 0.1–0.3 mg/kg over 2 min, max. dose 5 mg/dose, can repeat once.
- Digoxin: 0.5 mg IV initially then 0.25 mg IV q4h until desired effect (max. 1 mg IV)
- Isoproterenol: 2–3 µg/min IV, titrate to goal heart rate/BP, max. 10 µg/min (peds: 0.1 µg/kg/min)—do *not* coadminister with epinephrine and *only* use in unstable patient
- Epinephrine: 1 mg IV (peds: 0.01 mg/kg IV): For cardiac arrest
- Glucagon: 0.05–0.15 mg/kg IV (peds: 0.05–0.10 mg/kg)
- Heparin: Load 80 U/kg IV; infusion at 18 U/kg/h
- Magnesium: 1–2 g IV

First Line

1st-line definitive therapy is a permanent demand pacemaker to provide a “floor” to bradydysrhythmia:

- Patients with additional tachydysrhythmias will require additional nodal agents.

Second Line

No clear evidence to distinguish between 1st- and 2nd-line treatment.



FOLLOW-UP

DISPOSITION

Admission Criteria

- New onset
- Symptomatic: CHF, syncope, chest pain, dizziness
- Persistent bradyarrhythmia or tachydysrhythmia
- Advanced age; > 60 yr
- Patients should be admitted to a telemetry floor with cardiology consultation.
- Most will require permanent pacing.

Discharge Criteria

- Asymptomatic, otherwise healthy patients can be evaluated as outpatients.
- Holter monitoring

Issues for Referral

- Need for formal cardiac electrophysiology evaluation
- Need for permanent pacemaker placement

FOLLOW-UP RECOMMENDATIONS

Geriatric Considerations

- High incidence of CAD is present in patients with sick sinus syndrome, so a complete cardiovascular risk-factor evaluation and prevention is needed.
- Patient with atrial fibrillation and tachy-brady syndrome need long-term anticoagulation.
- All patients require evaluation by a cardiologist or EP specialist for permanent pacemaker.

PEARLS AND PITFALLS

- Patients who are asymptomatic on ED arrival may have normal EKGs. Consider obtaining a rhythm strip or Holter monitor if clinical suspicion remains high.
- Use of any nodal agents (BB, CCB, or digoxin) in patients with SSS-related tachydysrhythmia risks SA block or SA arrest and should only be administered when prepared for transcutaneous pacing.

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See Also (Topic, Algorithm, Electronic Media Element)

Bradydysrhythmia



CODES

ICD9

427.81 Sinoatrial node dysfunction

ICD10

I49.5 Sick sinus syndrome

SICKLE CELL DISEASE

Steven H. Bowman • Marcus E. Emebo • Mary E. Johnson



BASICS

DESCRIPTION

- Sickle cell disease (SCD) is an autosomal recessive hemoglobinopathy characterized by abnormal hemoglobin (HbS) which polymerizes under stress and deforms RBCs, resulting in hemolysis, vaso-occlusion, and subsequent tissue ischemia/infarction
- HbS production secondary to a single amino acid substitution in hemoglobin gene
- Occurs in people of African, Mediterranean, Middle Eastern, and Asian descent; areas where malaria is endemic
- Severity variable even among the same phenotype
- Genotypes and severity in African Americans:
 - HbSS, severe
 - HbSC, mild to moderate severity
 - HbS β -thalassemia, mild to moderate severity
 - HbAS, sickle cell trait:
 - No manifestation of disease
 - At risk for sudden death with extreme physical exertion, severe hypoxia, severe dehydration, or maternal labor
- Chronic hemolytic anemia associated with progressive vasculopathy manifested by systemic and pulmonary hypertension, cholelithiasis, cutaneous leg ulcers, and priapism
- Acute vaso-occlusive crisis (VOC) can occur in essentially any organ systems:
 - Bone/joint crises:
 - Vaso-occlusion of bone microvasculature causes infarction
 - Long bones, ribs, sternum, spine, and pelvis affected
 - Dactylitis, or “hand–foot syndrome,” occurs at ages 6–24 mo
 - Acute chest syndrome:
 - Vaso-occlusion of pulmonary vasculature
 - Fat embolism from infarcted bone marrow and/or infections (viral or bacterial) may contribute
 - Associated *Chlamydia pneumoniae* and *Mycoplasma pneumoniae* isolated in sputum and *Streptococcus pneumoniae* bacteremia
 - High mortality (2–14%)
 - 50% of sickle cell patients will experience at least 1 episode
 - Radiographic pulmonary infiltrate with fever and respiratory symptoms makes it difficult to distinguish from pneumonia
 - More common in children
 - Splenic sequestration:

- Splenic sinusoids become congested with sickled RBCs, obstructing outflow
- Estimated 6–17% of SCD deaths
- Circulatory collapse may be rapidly fatal
- More common in children <5 yr old
- Aplastic crisis:
 - Bone marrow suppression usually occurs secondary to viral infection, most commonly Parvovirus B19
 - Hallmark acute anemia with low reticulocyte count
 - Acute bone marrow suppression significantly worsens chronic anemia
 - Generally self-limited
 - More common in children
- Cerebrovascular accident/transient ischemic attack (CVA/TIA):
 - Secondary to vaso-occlusion by sickled cells and thromboembolism in children and older patients, respectively
 - Children with SCD have a 300-fold increased risk of CVA/TIA
 - Most events occur before the age of 10 and after the age of 29 yr
- Bacterial infection:
 - Sepsis is the leading cause of death in patients with SCD
 - Increased risk of bacteremia, meningitis, and osteomyelitis
 - Impaired splenic function impairs ability to fight encapsulated organisms
 - *S. pneumoniae*, *Haemophilus influenzae*, *Staphylococcus aureus*, *Escherichia coli*, and *Salmonella* are leading organisms
 - Children <5 yr of age have 400-fold increase in pneumococcal infections.
- Priapism:
 - Painful, sustained, unwanted erection > 3 hr
 - More commonly low-flow (ischemic) priapism than high-flow (nonischemic)

Pediatric Considerations

- Acute sickle cell complications in children carry high morbidity and should be screened for aggressively
- Infections commonly precipitate crisis
- Confirm immunization history (pneumococcal and *H. influenzae* type b)
- Determine if child is receiving prophylactic penicillin, normally indicated in children ≤5 yr old
- Overwhelming infection highest in children <3 yr of age

Pregnancy Considerations

- Variable frequency of crisis episodes, not uncommon for increased frequency

- Anemia is more profound
- Increased rates of hypertensive disorders of pregnancy, asymptomatic bacterial infections, UTI, and pyelonephritis leading to septicemia
- Increased risk of spontaneous abortions, antepartum bleeding, and premature rupture of membranes
- Increased risk of preterm labor, intrauterine growth restriction, and low birth weight

ETIOLOGY

Common crisis precipitants:

- Infection (bacterial and viral)
- Dehydration
- Hypoxemia
- Acidosis
- Emotional stress
- Surgery/trauma
- Weather changes
- Pregnancy
- Toxins

DIAGNOSIS

SIGNS AND SYMPTOMS

- May present with either:
 - Pain crisis
 - Complications of the disease
 - Combination of above
- May not demonstrate usually autonomic signs of acute pain
- Sickle cell pain crisis:
 - Bone/joint crisis:
 - Pain in extremities, back, sternum, or joints
 - Variable extremity and joint swelling/warmth
 - Hand–foot syndrome in infants; swelling in hands and feet and a reluctance to walk or use hands
 - Abdominal crisis:
 - Abdominal pain without peritonitis
 - Variable nausea, vomiting, diarrhea
 - Priapism: Prolonged painful erection
- Complications/progression of disease:
 - Acute chest syndrome:
 - Chest pain
 - Fever
 - New pulmonary infiltrates on chest radiographs

- Respiratory symptoms
- Hypoxemia
- Splenic sequestration crisis:
 - Abdominal pain, splenomegaly
 - Fatigue, lethargy, pallor
 - Hypotension, tachycardia, syncope, shock
- Aplastic crisis:
 - Variable fever, headache, nausea, vomiting
 - Fatigue, pallor, tachycardia
- CVA/TIA:
 - Focal neurologic deficit
 - Mental status changes
 - Seizure

History

- Genotype
- Onset of current symptoms
- Previous crises
- Immunizations
- Surgical history
- Determine typical vs. atypical crisis

Physical-Exam

Conduct a thorough physical exam:

- Vital signs: BP, HR, temperature, O₂ saturation
- General appearance: Jaundice, pallor
- Cardiopulmonary exam:
 - Rales, wheezing, tachypnea
 - Peripheral edema, elevated JVD
 - Gallops, murmurs
- Abdominal exam:
 - Organomegaly, tenderness, peritonitis
- Musculoskeletal exam
 - Erythema on extremities
 - Warm, swollen hands and feet in children
- Neurologic exam:
 - Focal neurologic impairment
 - Cranial nerve palsy

ESSENTIAL WORKUP

Conduct a thorough physical exam, with focus on signs of infection or ischemia.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC:
 - Compare Hb with prior values if available
 - Leukocytosis is common and does not necessarily indicate infection
- Reticulocyte count is generally elevated in SCD individuals, and decreased with aplastic crisis
- Complete metabolic panel (CMP):
 - Be aware that creatinine may appear normal despite baseline chronic renal dysfunction
 - Elevated total bilirubin levels may indicate intravascular hemolysis
- Markers of hemolysis (total bilirubin, haptoglobin, and LDH) may be present at variable degrees
- Serial arterial blood gases and A-a gradients helpful in acute chest syndrome
- Cultures: Blood, urine, throat, and CSF (if indicated)
- Type and screen (or cross)
- Urine pregnancy test in women

Imaging

- Radiographs should be directed to confirm diagnosis:
 - Chest radiograph if pneumonia or acute chest syndrome suspected
 - Extremity radiographs if osteomyelitis suspected
- IV contrast may exacerbate or precipitate a crisis
- Head CT/MRI to evaluate stroke

Diagnostic Procedures/Surgery

- Lumbar puncture if CNS infection or subarachnoid hemorrhage is suspected
- Arthrocentesis for acute arthritis

DIFFERENTIAL DIAGNOSIS

- Sick cell crises may mimic or obscure more serious underlying pathology (e.g., acute abdomen, MI, PE, nephrolithiasis)
- Suspect other diagnoses if pain is more severe or atypical



TREATMENT

INITIAL STABILIZATION/THERAPY

- Identify and treat high morbidity complications
- Establish venous access
- Assess pain and initiate therapy

ED TREATMENT/PROCEDURES

- Choice of analgesics dependent on patient, severity of presentation, and prior agents:
 - Reassess pain frequently (e.g., every 15–30 min) and titrate until improvement
 - IV opiates (e.g., morphine, hydromorphone, fentanyl) 1st line, consider adjunct agents
 - Adjuncts: Acetaminophen, NSAIDs (use with caution given impaired renal function)
 - Caution with meperidine as metabolites may accumulate and pose seizure risk
 - If no venous access, PO and subcutaneous analgesics preferred over IM
- Hydration:
 - Oral hydration if patient tolerating po
 - Parenteral IV solution 0.45% NS for adults and children or 0.2% NS for infants
 - Avoid over-hydration, at risk for:
 - Hyperchloremic metabolic acidosis which promotes RBC sickling
 - Atelectasis which precipitates acute chest syndrome
- Complication-specific therapy:
 - Acute chest syndrome:
 - Oxygen, bronchodilators, incentive spirometer
 - Consider exchange transfusion for worsening respiratory symptoms, hypoxemia, and increasing A–a gradient
 - Splenic sequestration:
 - Simple transfusion, promotes remobilization of RBCs
 - Be aware risk of precipitating VOC after raising Hb levels
 - Ideal treatment is prevention: Chronic transfusions, splenectomy
 - Aplastic crisis:
 - Simple transfusion
 - Isolation from pregnant healthcare workers
 - Priapism:
 - 1st line: Intracavernosal aspiration with α -adrenergic agonist (e.g., epinephrine, terbutaline) irrigation
 - 2nd line: Exchange transfusion if failed aspiration
 - Empiric antibiotics: Sepsis, pneumonia, and osteomyelitis
 - Exchange transfusion may be required for complications such as CVA and priapism
- Consultations:
 - Hematology especially if exchange transfusion required
 - Neurology/neurosurgery for acute CNS events
 - Urology for priapism



FOLLOW-UP

DISPOSITION

Admission Criteria

- Refractory pain
- Complications: Acute chest syndrome, sequestration crisis, aplastic crisis, CVA/TIA, refractory priapism
- Signs of bacterial infection or fever of undetermined etiology
- Symptomatic anemia
- ICU admission for hemodynamic instability, worsening hypoxemia in acute chest syndrome, and severe acute CNS events.

Discharge Criteria

- Resolution of pain crisis
- No indications for admission
- Follow-up arranged with hematologist

Issues for Referral

Meticulous primary care can limit the frequency and severity of pain crises.

FOLLOW-UP RECOMMENDATIONS

If discharged, patient should see PCP or hematologist in 1–2 days.

PEARLS AND PITFALLS

- Distinguish typical sickle cell crisis from acute life-threatening complications
- Treat pain aggressively with appropriately selected and administered analgesic agents
- Patients with acute pain may not demonstrate typical signs, such as tachycardia or diaphoresis

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Anemia



CODES

ICD9

- 282.41 Sickle-cell thalassemia without crisis
- 282.61 Hb-SS disease without crisis
- 282.63 Sickle-cell/Hb-C disease without crisis

ICD10

- D57.1 Sickle-cell disease without crisis
- D57.20 Sickle-cell/Hb-C disease without crisis
- D57.40 Sickle-cell thalassemia without crisis

SINUSITIS (RHINOSINUSITIS)

Cory A. Siebe • Maria E. Moreira



BASICS

DESCRIPTION

- Inflammation of mucous membranes lining the paranasal sinuses and nasal passages with or without fluid collection in the sinus cavities
- Classifications:
 - Acute: Signs and symptoms for < 4 wk
 - Subacute: Signs and symptoms for 4–8 wk
 - Chronic: Signs and symptoms for > 8 wk in spite of antibiotic treatment
 - Recurrent: 3 or more episodes per year

ETIOLOGY

- Acute rhinosinusitis pathophysiology:
 - Viral upper respiratory infection or allergies causes mucous membrane inflammation
 - Inflammation causes obstruction of sinus ostia, decreased mucociliary clearance, and thickening of secretions
 - Viruses are the primary cause, but 0.5–2.2% develop into bacterial infection after bacteria become trapped and multiply, resulting in suppuration
 - Nosocomial rhinosinusitis associated with nasogastric and nasotracheal tubes
 - Immunocompromised patients at higher risk for rhinosinusitis
- Subacute and chronic rhinosinusitis pathophysiology:
 - Multifactorial, role of bacteria remains elusive
 - Allergic inflammation causing narrowed ostia and blocked drainage
 - Immune dysfunction leading to increased infectious risk
 - Impaired ciliary function leading to decreased mucous clearance
 - Odontogenic infection causing maxillary sinusitis
 - Fungus ball
 - Anatomical obstruction or polyps obstructing sinus ostia
- Microbiology:
 - Acute rhinosinusitis:
 - Nontypable *Haemophilus influenzae*
 - *Streptococcus pneumoniae*
 - *Moraxella catarrhalis*
 - *Staphylococcus aureus*
 - Anaerobes
 - Viruses: Parainfluenza, adenovirus, rhinovirus, influenza

- Chronic rhinosinusitis:
 - Same as acute, often polymicrobial, with increasing anaerobes and gram negatives
- Nosocomial rhinosinusitis:
 - *S. aureus*
 - Streptococcal species
 - *Pseudomonas*
 - *Klebsiella*
- Immunocompromised patients with rhinosinusitis:
 - Bacteria as above
 - Fungal pathogens (*Aspergillus*)

Pediatric Considerations

- Nontypable *H. influenzae* more common than *S. pneumoniae* as cause of acute bacterial rhinosinusitis in children
- Ethmoid and maxillary sinuses present at birth
- Frontal and sphenoid sinuses do not emerge until age 6–7 yr
- Rhinosinusitis more common in children
- Periorbital/orbital cellulitis is a common complication of ethmoid rhinosinusitis in children:
 - Periorbital swelling, fever, ptosis, proptosis, and painful or decreased extraocular movements

DIAGNOSIS

SIGNS AND SYMPTOMS

- Facial–dental pain, headache, halitosis, hyposmia, cough
- Purulent nasal discharge and postnasal drainage
- Fever
- Frontal sinusitis:
 - Pain of the lower forehead
 - Pain worsened when lying on the back; improves when upright
- Maxillary sinusitis:
 - Malar facial pain
 - Maxillary dental pain
 - Referred ear pain
 - Pain worsens with head upright or bending forward and improves with reclining
- Ethmoid sinusitis:
 - Retro-orbital pain
 - Periorbital edema
- Sphenoid sinusitis (very uncommon):

- Pain over the occiput or mastoid
- Pain worse when lying on back or bending forward

History

- Acute viral rhinosinusitis:
 - Symptoms typically resolve in 7–10 days
- Acute bacterial rhinosinusitis needing antibiotic treatment can present in 3 different patterns:
 - Pattern 1: Persistent symptoms lasting > 10 days without improvement
 - Pattern 2: Severe symptoms or:
 - Temperature $\geq 39^{\circ}\text{C}$ and purulent nasal discharge for 3–4 days at the beginning of illness
 - Pattern 3: Worsening symptoms:
 - Return of symptoms after a 5–6- day duration of upper respiratory infection that was improving
- Other important history:
 - Symptom history and time course
 - Allergy history
 - Recent NG or NT tube placement
 - Immunocompromised state

Physical-Exam

- Vital signs, toxic/nontoxic appearance
- Edema of the nasal mucous membranes and turbinates
- Purulence in the nares or posterior pharynx
- Warmth, tenderness, or cellulitis over sinus
- Sinus tenderness on palpation
- Periorbital edema
- Failure of transillumination of maxillary sinuses:
 - Observed through the palate
- Dental exam revealing abscess or tenderness of maxillary teeth

ESSENTIAL WORKUP

- Clinical diagnosis based on history and physical exam
- Determine if patient fits pattern of acute bacterial rhinosinusitis that should be treated with antibiotics (see “History”)

DIAGNOSIS TESTS & INTERPRETATION

Lab

Lab studies not helpful for diagnosis or management

Imaging

- Imaging unnecessary in uncomplicated cases
- Plain-film radiography:
 - Normal films do not exclude bacterial cause
 - Waters view can be ordered, but has moderate sensitivity in diagnosing maxillary sinus abnormality and poor sensitivity in diagnosing lesions in other sinuses
 - Odontogenic maxillary sinusitis may be missed by dental exam and panorex films, but is apparent as periapical lucency on cone beam CT or sinus CT
- CT:
 - Preferred if imaging is necessary
 - Warranted in patients with complicated rhinosinusitis, severe headache, seizures, focal neurologic deficits, periorbital edema, or abnormal intraocular muscle function
 - IV contrast if concern for osteomyelitis or abscess

Diagnostic Procedures/Surgery

- Sinus aspirate culture:
 - Gold standard for making a microbial diagnosis but not routinely performed
- Culture of discharge may have benefit but remains unstudied and is not typically performed
- Functional endoscopic sinus surgery (FESS):
 - Restores physiologic sinus drainage

Pediatric Considerations

FESS is a safe and effective treatment in children

DIFFERENTIAL DIAGNOSIS

- Uncomplicated viral or allergic rhinitis
- Otitis media
- Dacryocystitis
- Migraine and cluster headache
- Dental pain
- Trigeminal neuralgia
- Temporomandibular joint disorders
- Giant cell arteritis/temporal arteritis
- Rhinitis medicamentosa (decongestants, β -blockers, antihypertensives, birth control pills)
- Nasal polyp, tumor, or foreign body
- CNS infection
- Granulomatous or ciliary disease
- Aspergillosis
- Rhinocerebral mucormycosis:
 - Rare rapidly progressive fungal infection

- Occurs in diabetics and the immunocompromised
- Orbital/facial pain out of proportion to exam
- Lethargy, headache in a systemically ill-appearing patient
- Black eschar or pale area on the palate or nasal mucosa

Pregnancy Considerations

- Rhinitis of pregnancy:
 - Estrogen has cholinergic effect on mucosa
 - Worse during 3rd trimester
 - Resolves within 2 wk postpartum



TREATMENT

PRE HOSPITAL

No special considerations

INITIAL STABILIZATION/THERAPY

Toxic-appearing patients may require airway management and fluid resuscitation.

ED TREATMENT/PROCEDURES

- Identifying rhinosinusitis needing antibiotics
- Counseling and reassurance to patients requesting antibiotics for mild symptoms <10 days duration

MEDICATION

- Nonantibiotic therapies:
 - Pain control
 - Saline nasal irrigation may be beneficial
 - Oral corticosteroids as adjunctive to oral antibiotics are effective, but data limited
 - Intranasal steroids recommended as adjunct to antibiotics primarily in those with allergies:
 - Beclomethasone dipropionate: 1 spray per nostril QD/TID/BID
 - Dexamethasone sodium phosphate: 2 sprays per nostril BID/TID
 - Antihistamines recommended for patients with underlying allergy
 - Nasal or oral decongestants not recommended (phenylephrine, pseudoephedrine, oxymetazoline)
 - Expectorants may be helpful:
 - Guaifenesin:
 - Adult: 200–400 mg PO; not > 2.4 g/24 h
 - Peds 2–5 yr: 50–100 mg PO; not > 600 mg/24 h;
 - Peds 6–11 yr: 100–200 mg PO; not > 1.2 g/24 h

- **Antibiotics:**
 - Amoxicillin–clavulanate: 250–500 mg PO TID or 875 mg PO BID (peds: 40 mg/kg/d, based on the amoxicillin component)
 - If high risk (systemic toxicity w/fever $\geq 39^{\circ}\text{C}$, attendance at daycare, age < 2 or > 65 yr, recent hospitalization, abx use in last month, or immunocompromised) use amoxicillin–clavulanate: 2 g PO BID (peds: 90 mg/kg/d, based on amoxicillin component)
 - Doxycycline: 100 mg PO BID (alternative for initial empiric therapy in adults)
- 2nd- and 3rd-generation oral cephalosporins no longer recommended for empiric monotherapy due to resistance among *S. pneumoniae*. Can use following combination:
 - Cefpodoxime: 200–400 mg PO BID (peds: 10 mg/kg/d PO BID) or
 - Cefuroxime: 250–500 mg PO BID (peds: 15 mg/kg/d PO BID) +
 - Clindamycin: 150–300 mg PO q6h (peds: 8–16 mg/kg/d PO split q6–8h, MRSA-suspected use 40 mg/kg/d PO split q6–8h)
- Macrolides (clarithromycin and azithromycin) not recommended due to high rates of resistance amongst *S. pneumoniae* (30%)
- Trimethoprim–sulfamethoxazole (TMP/SMX) not recommended due to high rates of resistance among *S. pneumoniae* and *H. influenzae* (30–40%)
- Type 1 penicillin allergy:
 - Levofloxacin: 500 mg PO per day (peds: 8 mg/kg) children under 50 kg max. dose 250 mg/d. Children over 50 kg max. dose 500 mg/d.
 - Moxifloxacin: 400 mg PO per day (adult)
- If symptoms not improved after 3–5 days of 1 antibiotic, switch to another antibiotic
- Recommended duration of therapy:
 - Acute: 10–14 days in children; 5–7 days in adults
 - Chronic: 3–6-wk course of antibiotics (controversial), douche, and nasal steroids

First Line

Supportive care

Second Line

Antibiotics



FOLLOW-UP

DISPOSITION

Admission Criteria

- Evidence of spread of infection beyond the sinus cavity or toxic-appearing patients
- Immunocompromised/diabetic patients with extensive infection
- Multiple sinus or frontal sinus involvement
- Extremes of age
- Severe comorbidity
- ENT evaluation and aspiration if patient is severely ill, immunocompromised, or has pansinusitis and is ill-appearing

Discharge Criteria

Most cases of uncomplicated rhinosinusitis may be managed on an outpatient basis.

Issues for Referral

- Complications of acute infection
- Immunocompromised patients
- Chronic rhinosinusitis or nasal polyps
- Concerns for osteomyelitis, CNS infection, or abscess
- Acute rhinosinusitis–aspergillosis

FOLLOW-UP RECOMMENDATIONS

If patient has no relief with initial treatment and nonantibiotic therapies, follow up with PCP or ENT.

PEARLS AND PITFALLS

- Patients presenting with < 10 days of mild symptoms should be treated with supportive care
- Patients presenting with ≥ 10 days of symptoms, severe symptoms at 4–5 days with fever, or worsening after initial improvement can be diagnosed with acute bacterial rhinosinusitis and should be treated with antibiotics
- Term rhinosinusitis preferred, since inflammation of sinuses rarely occurs without inflammation of the nasal mucosa

ADDITIONAL READING

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- Venekamp RP, Thompson MJ, Hayward G, et al. Systemic corticosteroids for acute sinusitis. *Cochrane Database Syst. Rev.* 2011;(12):CD008115.



CODES

ICD9

- 461.9 Acute sinusitis, unspecified
- 473.0 Chronic maxillary sinusitis
- 473.9 Unspecified sinusitis (chronic)

ICD10

- J01.90 Acute sinusitis, unspecified
- J32.0 Chronic maxillary sinusitis
- J32.9 Chronic sinusitis, unspecified

SKIN CANCER

John J. D'Angelo

BASICS

DESCRIPTION

- Most common cancer in US
- Increasing incidence
- 1 in 6 will have skin cancer during their lifetime
- Actinic keratosis:
 - Premalignant lesion
 - Thickened scaly growth caused by sunlight or other artificial light source
 - Found on areas of body with high sun exposure
 - 0.1–10% may transform into squamous cell carcinoma (SCC)
- Nonmelanoma skin cancer:
 - Less commonly fatal
 - Fast growing
 - May be destructive if left untreated
 - Basal cell carcinoma (BCC):
 - Cells arise from epidermis
 - Most common skin cancer
 - Account for 75% of all nonmelanoma skin cancers
 - Male > female, 3:2.
 - Locally invasive without risk of distant metastasis
 - Most important risk factor is sun exposure
 - More common in fair-skinned patients
 - Most lesions are on the head and neck
 - SCC:
 - 2nd most common skin cancer
 - 20% of cases of skin cancer
 - Most arise from precancerous actinic keratosis lesions
 - Male > female
 - Most important risk factor is sun exposure, especially sunburn
 - 70% occur on head and neck
 - More common in older, fair-skinned patients
 - Risk of regional lymph node and distant metastasis
 - SCC lesions of mucosal surfaces are more aggressive
- Melanoma:
 - 5% of all diagnosed skin cancer in US
 - 62,000 new cases in 2008
 - 15% are fatal

- 75% of skin cancer cause deaths
- Arises from melanin-producing cells
- Most important risk factor is sun exposure, especially sunburn
- Additional risk factors:
 - Fair skin; blond/red hair
 - Multiple common melanocytic nevi
 - Atypical nevi
 - Immunosuppression
 - Positive family history
 - History of nonmelanoma skin cancer (BCC or SCC)
 - ≥ 5 sunburns in early life doubles the risk for malignant melanoma
- Risk of regional lymph node and distant metastasis

ETIOLOGY

- UV irradiation:
 - Both UVA and UVB rays
 - Sun exposure
 - Tanning beds
- SCC often associated with human papilloma virus (HPV)
- Immunosuppression may predispose to SCC
- Vitamin D metabolism may play a role

DIAGNOSIS

SIGNS AND SYMPTOMS

- Actinic keratosis:
 - Rough, pink, circumscribed lesions < 1 cm in diameter
 - A lesion may have both actinic keratosis and SCC
- BCC:
 - May be single or multiple
 - Usually painless
 - Usually appears in sun-exposed areas of skin
 - Erosion or bleeding with mild trauma may be 1st symptoms
 - Nodular BCC:
 - Most common
 - Waxy or pearly papule, possibly with telangiectasia
 - Well-demarcated borders
 - May have central ulceration
 - Pigmented BCC:
 - Similar to nodular BCC with brown, blue, or black coloration
 - Often mistaken for superficial spreading or nodular melanoma
 - Cystic BCC:
 - Bluish/gray cystic nodules

- May be mistaken for benign cysts
- Superficial BCC:
 - Scaly patch-like or papule surrounded by small, clear micropapules
 - Pink, red, or brown
- Micronodular BCC:
 - Well-defined border
 - Aggressive
 - Rarely with ulceration
- Morpheaform BCC:
 - Poorly defined borders
 - May appear “scar like”
 - Aggressive
 - Ulceration and bleeding are rare
- SCC:
 - Characteristic lesion is raised, firm, keratotic papule or plaque
 - Often enlarging
 - Usually asymptomatic but may be ulcerated and painful as invasion occurs
 - Ulcers often crust and ooze
 - Cranial nerve involvement may indicate an aggressive tumor with perineural invasion:
 - Facial numbness, asymmetry, weakness, or pain
- Melanoma:
 - Pigmented skin lesion:
 - 2% will be amelanotic
 - Features suggestive of melanoma (the *ABCDEs* of melanoma):
 - Asymmetry (not regularly round or oval)
 - Border irregularity (notched or poorly defined)
 - Bleeding (spontaneous)
 - Color variegation (shades or combinations of brown, tan, red, white, or blue-black)
 - Diameter > 6 mm
 - Elevation/Enlargement
 - Lesions rarely symptomatic unless ulcerated
 - Superficial spreading melanoma:
 - 70% of all malignant melanomas
 - May have a wide variety of colors
 - Often arise from dysplastic nevus
 - Usually < 3 cm
 - Slight elevation and induration is common
 - Often have satellite lesions
 - Nodular melanoma:
 - 10–15% of melanomas
 - The most symmetric of the different melanomas

- Dark brown or black
- Often exophytic
- Lentigo maligna melanomas (LMM):
 - Always starts as *lentigo maligna*, a macular insitu malignancy which is an evolving lesion of melanoma
 - *LMM* occurs after the lesion begins vertical growth into the dermis – typically indicated by papular or nodular areas
 - Irregular shape and multiple colors
- Acral lentiginous melanoma:
 - Equal among black and white patients
 - Most common form of melanoma found in Asians and African Americans
 - Occur on palms, soles, and subungual region with predilection for soles of feet
 - May be mistaken for subungual hematoma
 - Involvement of the proximal nail fold (Hutchinson sign) is an indicator of melanoma
- Mucosal lentiginous melanoma:
 - Develops from mucosal epithelium in respiratory, GI, and GU tracts
 - Often diagnosed at a later stage of disease
 - Very rare
- Metastatic melanoma:
 - Presentation related to affected organ system
 - Lymphangitic spread with local to regional lymphadenopathy
 - Typical visceral sites of hematogenous spread include liver, lung, bone, brain, and intestines

ESSENTIAL WORKUP

All suspicious lesions require biopsy, a procedure rarely done in ED

DIAGNOSIS TESTS & INTERPRETATION

Lab

No specific testing is required

Imaging

- CXR may show pulmonary involvement by metastatic melanoma
- Head or body CT scan may show visceral involvement by metastatic SCC or melanoma
- Cross-sectional imaging does not rule out metastasis

Diagnostic Procedures/Surgery

Biopsy usually performed by consultant

DIFFERENTIAL DIAGNOSIS

- For BCC:
 - SCC
 - Bowen disease
 - Actinic keratosis
 - Paget disease
 - Benign nevus
 - Melanoma
- For SCC:
 - Actinic keratosis
 - BCC
 - Keratoacanthoma
 - Melanoma
 - Wart
- For melanoma:
 - Atypical nevus
 - Common nevus
 - Actinic keratosis
 - Pigmented BCC
 - SCC



TREATMENT

PRE HOSPITAL

No specific pre-hospital care is required

INITIAL STABILIZATION/THERAPY

- No specific stabilization is usually required beyond basic wound care
- Stabilization may be required for invasion into vascular structures or edema from intracranial metastasis

ED TREATMENT/PROCEDURES

- Skin lesions themselves require no specific ED treatment
- Treat complications of visceral involvement by metastatic melanoma, SCC or locally invasive BCC.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Admission typically only occurs due to complications associated with visceral

involvement or invasive spread

- Admission is rarely required because of the dermatologic lesions themselves

Discharge Criteria

Patients are generally discharged with instructions on obtaining biopsy and/or further evaluation

Issues for Referral

Discharged patients should be advised to consult a dermatologist or primary care physician experienced with skin biopsy.

FOLLOW-UP RECOMMENDATIONS

- Biopsy is required for diagnosis of skin cancer
- Urgent follow-up with dermatologist or primary care physician is advised
- Ensure adequate documentation of conversation with patient regarding urgency of follow-up
- Patients with nonmelanoma skin cancer have a 30–50% chance of developing additional skin cancer within 5 yr

PEARLS AND PITFALLS

- Advise patient to obtain urgent follow-up for any suspicious lesion
- 1 in 6 people will have skin cancer during their lifetime
- Protection from UVA and UVB rays is key to preventing skin cancer

ADDITIONAL READING

- Arora A, Attwod J. Common skin cancers and their precursors. *Surg Clin North Am.* 2009;89:703–712.
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- Ricotti C, Bouzari N, Agadi A, et al. Malignant skin neoplasms. *Med Clin North Am.* 2009;93:1241–1264.



CODES

ICD9

- 173.90 Unspecified malignant neoplasm of skin, site unspecified
- 173.91 Basal cell carcinoma of skin, site unspecified

- 173.92 Squamous cell carcinoma of skin, site unspecified

ICD10

- C44.90 Unspecified malignant neoplasm of skin, unspecified
- C44.91 Basal cell carcinoma of skin, unspecified
- C44.92 Squamous cell carcinoma of skin, unspecified

SLEEP APNEA

Ajay Bhatt

BASICS

DESCRIPTION

- Disorder characterized by cessation of breathing during sleep:
 - Defined as apneic episodes > 10 sec with brief EEG arousals or > 3% oxygenation desaturation
- Risk factors:
 - Obesity
 - Male
 - > 40 yr of age
 - Upper airway anomalies
 - Myxedema (hypothyroidism)
 - Alcohol/sedative abuse
 - Smoking
- Associated illness:
 - Various dysrhythmias, particularly atrial fibrillation and bradyarrhythmia
 - Right and left heart failure
 - MI
 - Stroke
 - Motor vehicle accidents
 - Hypertension poorly controlled by medical therapies

EPIDEMIOLOGY

- Affects about 9% of middle-aged men and 4% of middle-aged women
- 80% of moderate or severe cases undiagnosed in middle-aged adults

ETIOLOGY

3 classifications of sleep apnea:

- Obstructive (84%) is due to upper airway closure despite intact respiratory drive:
 - Also known as Pickwickian syndrome
 - Pharyngeal airway is narrowed
- Central (0.4%) is due to lack of respiratory effort despite patent upper airway.
- Complex (15%) is due to a combination of obstructive and central sleep apnea.

DIAGNOSIS

SIGNS AND SYMPTOMS

- Excessive daytime sleepiness

- Snoring
- Irritability

History

- Significant other apnea report
- Difficulty sleeping
- Decreased attention/concentration
- Depression
- Decreased libido/impotence

Physical-Exam

- Hypertension, hypoxemia
- Obesity
- Craniofacial anomalies
- Macroglossia
- Enlarged tonsils
- Elevated jugular veins (secondary to pulmonary hypertension)
- Large neck circumference

ESSENTIAL WORKUP

- Pulse oximetry
- ECG
- Chest radiograph

DIAGNOSIS TESTS & INTERPRETATION

Lab

ABG is the best test to demonstrate hypercarbia and hypoxemia.

Imaging

- Consider lateral neck soft tissue radiograph to rule out other etiologies of upper airway obstruction.
- Chest radiograph to assess other etiologies of hypoxemia
- Chest CT rarely indicated

Diagnostic Procedures/Surgery

Polysomnogram (PSG) is required for diagnosis:

- > 5 apneic episodes per hour
- Not a consideration for ED management

DIFFERENTIAL DIAGNOSIS

- Asthma
- Cheyne–Stokes breathing

- COPD
- Diaphragmatic paralysis
- High altitude–induced periodic breathing
- Hypothyroidism
- Left heart failure
- Narcolepsy
- Obesity hyperventilation syndrome
- Primary pulmonary hypertension



TREATMENT

PRE HOSPITAL

Caution not to overventilate patient with chronic CO retention

INITIAL STABILIZATION/THERAPY

Chin lift/jaw thrust maneuver, oxygen as needed, oral or nasal airway devices

ED TREATMENT/PROCEDURES

- Proper technique is required for airway management:
 - Supplemental oxygen as needed
 - Bag-valve-mask ventilation may be difficult:
 - Consider the use of nasal and oral airways
 - 2-person technique to ensure a good seal
- Continuous positive airway pressure (CPAP) is the standard of treatment:
 - Acts as a pneumatic splint by maintaining upper airway patency
 - BiPAP is an alternative for patients requiring high pressures or with comorbid breathing disorders.
 - Long-term CPAP therapy decreases BP, insulin resistance, metabolic syndrome, and risk of cardiovascular disease.

ALERT

Endotracheal intubation

- Higher prevalence of difficult intubation:
 - Patients frequently have higher Mallampati scores.
 - Excess pharyngeal tissue in lateral walls often obstructs airway visualization.
 - Patients have overall lower arterial oxygen saturation.
- Plan and consider several methods of definitive airway control:
 - Have alternative devices (laryngeal mask airway, bougie) available.
 - Be prepared to perform cricothyroidotomy if necessary.
- Use neuromuscular blockade only if successful oral intubation is reasonably likely

and bag-mask ventilation is easy.

- Positive end-expiratory pressure for ventilated patients

MEDICATION

- Insufficient evidence to recommend any medication for treatment
- See Airway Management for details on induction agents and neuromuscular blockade.
- Wakefulness-promoting agents (modafinil and armodafinil) are approved as an adjunct to CPAP patients with excessive sleepiness.

ALERT

Avoid sedative use:

- Relaxes the upper airway and worsens airway obstruction and snoring

Long-term Management

- Gold Standard
 - CPAP compliance and weight loss strongly recommended by the American College of Physicians
- Surgical considerations:
 - Most intend to reduce or bypass the excessive pharyngeal/airway resistance that occurs during sleep.
 - Efficacy is unpredictable; no good randomized trials
 - Not a consideration for ED management
- Dental devices:
 - Currently recommended by the American Academy of Sleep Medicine (AASM)
 - Available appliances include tongue repositioning and mandibular devices or soft-palate lifters.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Ventilatory failure, especially if intubation is necessary
- Hemodynamic instability

Discharge Criteria

- Maintenance of O₂ saturation > 85% for several hours using oxygenation or ventilation equipment available to the patient at home
- Very low likelihood of decompensation overnight
- Patients with sleep apnea who present after motor vehicle crashes:

- Manage initially like other blunt trauma patients.
- Later, consider the increased risk with sleep apnea and intervene to prevent future accidents.

FOLLOW-UP RECOMMENDATIONS

- PCP referral for sleep apnea and associated comorbidities
- Encourage compliance, use of CPAP
- Referral of patients with suspected sleep apnea to a pulmonologist
- Encourage weight loss and diet control
- Cardiology referral is appropriate when sleep apnea is complicated by heart failure or dysrhythmias.

PEARLS AND PITFALLS

- Sleep apnea increases risk of cardiovascular disease, stroke, and diabetes mellitus.
- CPAP is the standard of treatment.
- Avoid the use of sedatives.
- Preparation is essential, as sleep apnea increases intubation complications.
- Primary care referral and CPAP compliance education improve therapy.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Airway Management
- Dyspnea

The author gratefully acknowledges Mark Sagarin for his previous edition of this chapter.



CODES

ICD9

- 327.21 Primary central sleep apnea
- 327.23 Obstructive sleep apnea (adult)(pediatric)
- 780.57 Unspecified sleep apnea

ICD10

- G47.30 Sleep apnea, unspecified
- G47.31 Primary central sleep apnea
- G47.33 Obstructive sleep apnea (adult) (pediatric)

SLIPPED CAPITAL FEMORAL EPIPHYSIS

Virag Shah

BASICS

DESCRIPTION

- Femoral epiphysis translates (slips) posteriorly and inferiorly relative to the femoral head/neck
- Classification systems:
 - Degree of femoral head “slip” as a percentage of femoral neck diameter:
 - (Mild, grade 1) < 33.3%
 - (Moderate, grade 2) 33.3–50%
 - (Severe, grade 3) > 50%
 - Temporal:
 - Acute: < 3 wk of symptoms
 - Chronic: > 3 wk of symptoms
 - Acute on chronic: > 3 wk of symptoms, now with acute pain
 - Stability:
 - Stable: Bears weight w/or w/o crutches
 - Unstable: Unable to bear weight
- Epidemiology:
 - Peak age: 12–14 yr (boys), 11–12 yr (girls)
 - Male > female (1.6:1)
 - Bilateral slips: 20% at presentation; additional 20–40% progress to bilateral
 - Atypical SCFE: Endocrinopathy associated:
 - Patient may be < 10 yr age, > 16 yr age, or weight < 50th percentile
 - High risk of bilateral SCFE (up to 100%)

ETIOLOGY

- Proximal physis position changes in adolescence from horizontal to oblique; hence hip forces shift from “compression” to “shear”
- Shear force > strength of femoral physis
- Weakest point of physis = zone of hypertrophy
- Risk factors:
 - Obesity: May contribute to shear forces
 - Down syndrome
 - Endocrinopathy such as hypothyroidism, growth hormone deficiency, renal osteodystrophy (2° hyperparathyroidism): May contribute to growth plate weakening

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Determine chronicity of symptoms and whether or not the patient can bear weight
- Pain in the knee, thigh, groin, or hip (referred pain from the obturator nerve):
 - Vague and dull for weeks in chronic SCFE
 - Severe and sudden onset in acute SCFE, often in the setting of trauma

Physical-Exam

- If stable, presents with limp or exertional limp
- If unstable (patient cannot ambulate), avoid further ambulation attempts
- Commonly presents with leg externally rotated
- Restricted internal rotation, abduction, and flexion (cannot touch thigh to abdomen)
- Anterior hip joint tenderness
- Test: Apply gentle passive hip flexion → if hip externally rotates + abducts → highly suggestive of SCFE
- Gait:
 - Antalgic (patient takes short steps on affected side to minimize weight-bearing during “stance” phase of gait)
 - Trendelenburg (shift of torso over affected hip; sign of moderate/severe slip)
 - Waddling (sign of bilateral SCFE)

ESSENTIAL WORKUP

- Plain radiographs:
 - Further imaging with aid from consultant
- Orthopedic consultation

DIAGNOSIS TESTS & INTERPRETATION

Lab

- If no diagnostic radiographic abnormality, the practitioner may consider the following to help risk stratify possible alternative diagnoses:
 - CBC with differential, sedimentation rate, C-reactive protein
- If endocrinopathy suspected, consider thyroid function testing

Imaging

- Both hips should be imaged for comparison
- Some clinicians prefer cross-table lateral view in acute SCFE instead of frog-leg view (theoretical risk of worsening displacement)
- Anteroposterior radiograph:
 - Widened or irregular physis

- Bird’s beak appearance of the epiphysis “slipping” off of the femoral head
- Klein line: Parallel line drawn from superior border of the femoral neck; line intersects the epiphysis in normal patient
- Lateral radiograph (frog-leg or cross-table)

Diagnostic Procedures/Surgery

If septic hip is suspected, aspiration and fluid analysis may be needed to exclude.

DIFFERENTIAL DIAGNOSIS

- Legg–Calvé–Perthes:
 - Typically seen in 4–9-yr-old age range
- Septic arthritis of hip
- Osteomyelitis
- Toxic synovitis
- Femur or pelvic fractures
- Inguinal or femoral hernia



TREATMENT

PRE HOSPITAL

Patient should be immobilized for transport, as with suspected hip fracture or dislocation.

INITIAL STABILIZATION/THERAPY

- Immobilize hip; keep nonweight bearing
- Do not attempt reduction.

ED TREATMENT/PROCEDURES

- SCFE is an urgent orthopedic condition; delay in diagnosis may lead to chronic irreversible hip joint disability.
- Consult orthopedics immediately for definitive immobilization or operative intervention.

MEDICATION

Pain management as indicated; avoid oral medications if operative intervention is planned



FOLLOW-UP

DISPOSITION

Admission Criteria

- Acute, acute on chronic and bilateral SCFE requires orthopedic admission for urgent operative fixation (usually insitu single cannulated screw fixation)
- Chronic SCFE may be managed with delayed operative fixation

Discharge Criteria

None (no role for observation or attempts at closed reduction due to risk of complications, including osteonecrosis and/or chondrolysis)

FOLLOW-UP RECOMMENDATIONS

Should be arranged by orthopedic specialist

PEARLS AND PITFALLS

- Klein line can be a helpful tool in picking up the abnormality on plain radiograph
- Remember to examine the hip when a child presents with knee or thigh pain

ADDITIONAL READING

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CODES

ICD9

- 732.2 Nontraumatic slipped upper femoral epiphysis
- 732.9 Unspecified osteochondropathy

ICD10

- M93.003 Unspecified slipped upper femoral epiphysis (nontraumatic), unspecified hip
- M93.013 Acute slipped upper femoral epiphysis (nontraumatic), unspecified hip

- M93.023 Chronic slipped upper femoral epiphysis (nontraumatic), unspecified hip

SMALL-BOWEL INJURY

Barry J. Knapp



BASICS

DESCRIPTION

2 general causes:

- Blunt visceral trauma
- Penetrating: Visceral injury (96% of gunshot wounds, 50% of stabbings)—serosal tear, bowel wall hematoma, perforation, bowel transection, mesenteric hematoma/vascular injury

ETIOLOGY

- Blunt:
 - 3rd most commonly injured organ (5–10% of all blunt trauma victims)
 - Motor vehicle accidents
 - Nonvehicular trauma: Abuse/assault, bicycle handlebars, large-animal kick
 - Blast victims
- Mortality rate from small-bowel injury is 33%.
- Mesenteric tears may initially be asymptomatic:
 - Deceleration injury at fixed points (e.g., ligament of Treitz)
 - Shearing mechanisms near fixed points (e.g., ileocecal junction, adhesions)
 - Compressive force against anterior spine
 - Bursting or “blowout” at antimesenteric margin from sudden closed-loop intraluminal pressure rise
- Associated injuries:
 - Liver and splenic lacerations; thoracic and pelvic fractures
 - Seatbelt syndrome: Abdominal wall ecchymosis, small-bowel injury; Chance fracture of L1, L3
- Penetrating:
 - Small bowel is the 2nd most commonly injured organ (32%) in anterior abdominal stabbing.
 - Small-bowel injury is most common in gunshot wounds (49%).

Pediatric Considerations

- Blunt:
 - Less common in children (1–8% of all blunt pediatric trauma)
 - Lower chance of intestinal injury in vehicular accidents when both shoulder and lap belts are worn.
 - Be cautious of nonpenetrating trauma: Airgun accidents at close range (< 10 ft)

- Consider the possibility of nonaccidental trauma.



DIAGNOSIS

SIGNS AND SYMPTOMS

- Physical signs and symptoms are unreliable
- Delays in diagnosis are common
- Presence of a “seatbelt sign” doubles the risk for small-bowel injury.
- Initial presentation may be mild:
 - Uniformly, patients will progress to serious signs/symptoms.
- Delays in diagnosis add to morbidity and mortality:
 - Mortality is 2% when diagnosis is made within 8 hr; 31% when made after 24 hr.

History

- History of blunt or penetrating abdominal trauma
- Must consider in ill children without a definite history of trauma (child abuse)

Physical-Exam

- In awake, alert patients look for:
 - Abdominal tenderness (87–98%)
 - Abdominal pain (85%)
 - Peritoneal signs (67%)
- Many patients will have:
 - Abdominal wall bruising (54%)
 - Hypotension (38%)
 - Guaiac-positive rectal exam (5%)
- Small-bowel injury may initially be obscured by abnormal mental status, severe associated injuries.
- Small-bowel injury not initially apparent may be indicated by:
 - Progressive abdominal pain
 - Intestinal obstruction
 - Decreased urine output
 - Tachycardia

ESSENTIAL WORKUP

- Initial physical exam should note all wounds and areas of tenderness.
- CT for all medically stable patients
- For patients with a negative CT scan in which there is high suspicion of bowel injury, further evaluation or serial exams are indicated.
- For medically unstable patients, diagnostic peritoneal lavage (DPL) is superior to US in determining presence of a hollow viscus injury.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- No diagnostic test has proven highly sensitive in the prediction of small-bowel injury.
- Serum amylase, lipase, and liver function tests have poor sensitivity for acute injury.

Imaging

- Plain radiography of chest/abdomen:
 - _ Not useful for small-bowel injury
 - _ Incidence of pneumoperitoneum visible on plain radiograph is only 8%.
- CT:
 - _ Diagnostic standard for solid-organ injury and head trauma but is less sensitive for hollow viscus injuries
 - _ Newest-generation helical CT scanners have a sensitivity of 88% and a specificity of 99%.
 - _ The benefits of oral contrast are controversial; it is acceptable to use IV contrast only
 - _ Blunt trauma:
 - Used in stable patients
 - Indications for CT in blunt trauma include abdominal tenderness, hypotension, altered mental status (GCS < 14), costal margin tenderness, abnormal CXR, HCT < 30% and hematuria
 - Specific signs for small-bowel injury on CT are pneumoperitoneum (sensitivity 50–75%) and extravasation of contrast (sensitivity 12%).
 - Signs on CT suggestive of small-bowel injury include unexplained free intraperitoneal fluid (most sensitive 93%), thickened bowel wall > 3 mm (61% sensitive), intramural hematomas (75–88% sensitive), interloop fluid, mesenteric streaking.
 - _ Penetrating: CT is not recommended because sensitivity is only 14%; false-negative result rate is 18%.
- US: Not sensitive in hollow viscus injury because air in bowel makes visualization difficult

Diagnostic Procedures/Surgery

- DPL:
 - _ Invasive but may be helpful in unstable patients or in patients with clinically suspicious but nondiagnostic abdominal CT
 - _ Sensitive for hemoperitoneum but not source of bleeding
 - _ Positive if RBC count of > 100,000/mm³
 - _ Lavage amylase > 20 IU/L and leukocyte count > 500/mm³ (late markers of

- small-bowel injury)
- Lavage microscopy for succus/vegetable matter/feces is specific for small-bowel injury but not sensitive.
- Lavage alkaline phosphatase (> 3 IU/L) is reported to be a useful immediate marker of small-bowel injury.
- Laparoscopy: Plays a key role in diagnosing small-bowel injury in stable patients with progressive signs or symptoms

DIFFERENTIAL DIAGNOSIS

- Hemoperitoneum owing to vascular insult
- Solid visceral organ injury or gastric/colon/rectum perforation
- Vertebral injury and associated ileus

Pediatric Considerations

Delay in diagnosis of 1–2 days is common and increases morbidity.



TREATMENT

PRE HOSPITAL

ALERT

- Patients should be transported to the nearest trauma center.
- Do not attempt to replace eviscerated abdominal contents; cover with moist gauze, blanket, and transport.
- Do not remove impaled objects in the abdomen; stabilize the object with gauze and tape and transport.

INITIAL STABILIZATION/THERAPY

- Standard advanced trauma life support protocols, including airway, breathing, and circulation management
- Aggressive fluid resuscitation, central line suggested with pressure infusion of warmed IV fluid (lactated Ringer solution or normal saline)
- Cover eviscerated small bowel with moist gauze; do not remove impaled foreign body in ED.

ED TREATMENT/PROCEDURES

- Immediate transfer to OR is required for patients with an indication for laparotomy:
 - Evisceration
 - Abdominal pain with hypotension
 - Positive DPL or abdominal CT
 - Thoracic abdominal herniation visualized on chest radiograph
 - Impaled foreign body

- Penetrating gunshot wound to the abdomen
- Tetanus and antibiotic prophylaxis should be given for penetrating abdominal wounds and blunt injury requiring surgical exploration.
- Local wound exploration is safe for abdominal stab wounds.
- Serial abdominal exams and observation for otherwise stable patients
- Judicious analgesia as BP permits after diagnosis is established

MEDICATION

- Cefotetan (Cefotan): 1–2 g (peds: 20 mg/kg) IV q12h *or*
- Cefoxitin (Mefoxin): 1–2 g (peds: 40 mg/kg) IV q6h *or*
- Ceftizoxime (Cefizox): 1–2 g (peds: 50 mg/kg) IV q8–12h +
- Metronidazole: 500 mg (peds: 7.5 mg/kg) IV q6h



FOLLOW-UP

DISPOSITION

Admission Criteria

- Indication for laparotomy
- Abnormal mental status/intoxication with abdominal injury
- Presence of abdominal pain, tenderness (even with a negative workup) mandates admission for observation and serial exams.
- Stab and gunshot wounds that violate the abdominal fascia, positive DPL, or worsening findings on clinical exam

Discharge Criteria

- Minimal mechanism blunt trauma in a sober patient with normal exam result who has no abdominal pain and will receive adequate follow-up
- Explicit discharge instructions to return for worsening signs/symptoms are important to identify those with unsuspected injury.
- Penetrating wounds that do not violate abdominal fascia

FOLLOW-UP RECOMMENDATIONS

Discharged patients who develop abdominal complaints should return promptly to the ED.

PEARLS AND PITFALLS

- Small-bowel injury should be considered in any blunt/penetrating abdominal trauma victim.
- Initial presentation of patients with small-bowel injuries may be unimpressive.
- Presence of a “seat belt sign” doubles the risk for small-bowel injury.
- CT scanning may miss a significant percentage of small-bowel injuries.

- Observation and serial exams are an important aspect of detecting occult injuries.

ADDITIONAL READING

- CDC Fact Sheet “Blast Injuries: Abdominal Blast Injuries” 2009. Available at www.emergency.cdc.gov/Blastinjuries.
- Cordle R, Cantor R. Pediatric trauma. In: Rosen P, ed. *Rosen’s Emergency Medicine: Concepts and Clinical Practice*. 7th ed. St. Louis, MO: CV Mosby; 2009.
- Diercks DB, Mehrotra A, Nazarian DJ. Clinical policy: Critical issues in the evaluation of adult patients presenting to the emergency department with acute blunt abdominal trauma. *Ann Emerg Med*. 2011;57:387–404.
- Gross E, Martel M. Multiple trauma. In: Rosen P, ed. *Rosen’s Emergency Medicine: Concepts and Clinical Practice*. 7th ed. St. Louis, MO: CV Mosby; 2009.
- Herr S, Fallat ME. Abusive abdominal and thoracic trauma. *Clin Ped Emerg Med*. 2006;7:149–152.



CODES

ICD9

- 863.20 Injury to small intestine, unspecified site, without open wound into cavity
- 863.29 Other injury to small intestine, without mention of open wound into cavity
- 863.30 Injury to small intestine, unspecified site, with open wound into cavity

ICD10

- S36.409A Unsp injury of unsp part of small intestine, init encntr
- S36.429A Contusion of unsp part of small intestine, init encntr
- S36.439A Laceration of unsp part of small intestine, init encntr

SMOKE INHALATION

Trevonne M. Thompson



BASICS

DESCRIPTION

- Suspect smoke inhalation in anyone involved in a fire within a closed space or with a history of loss of consciousness.
- May cause direct injury to the upper (supraglottic) airway structures
- May cause chemical/irritant effect to lower airway structures
- May cause systemic toxicity from inhaled substances

ETIOLOGY

- Direct heat injury from heated gases/smoke:
 - Limited to supraglottic structures because of the heat-dissipating properties of the upper airway
- Irritant effect from smoke components
- Systemic toxicity from inhaled cellular toxins:
 - Carbon monoxide
 - Hydrogen cyanide

ALERT

Inhalation of steam can be rapidly fatal:

- Steam has ~4,000 times the heat-carrying capacity of hot air.
- Can rapidly cause obstructive glottic edema, thermally induced tracheitis, and hemorrhagic edema of the bronchial mucosa



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Exposure to a fire or heavy smoke
- Typically in a confined space
- Maintain high index of suspicion with history of loss of consciousness

Physical-Exam

- May have a normal physical exam with symptoms developing during the 24-hr interval following exposure
- Upper airway (supraglottic):
 - Nasopharyngeal irritation

- Hoarseness
- Stridor
- Cough
- Lower airway:
 - Chest discomfort
 - Hemoptysis
 - Bronchospasm
 - Bronchorrhea
- May have symptoms and signs of carbon monoxide and/or cyanide toxicity

ALERT

The following signs are suggestive of significant inhalation injury:

- Facial and upper cervical burns
- Carbonaceous sputum
- Singed eyebrows and nasal vibrissae

ESSENTIAL WORKUP

- Pulse oximetry:
 - May be falsely elevated in cases of carbon monoxide exposure
- ABG measurement:
 - Hypoxia
 - Metabolic acidosis in cases of carbon monoxide or hydrogen cyanide
- Chest radiography:
 - Initial radiograph typically normal
 - May show signs of pulmonary injury over the next 24 hr

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Electrolytes, BUN, creatinine, glucose
- CBC
- Coagulation profile
- Creatine phosphokinase when indicated in burn patients
- Carboxyhemoglobin to evaluate for potential carbon monoxide exposure
- Cyanide level:
 - In suspected cases of cyanide exposure, do not wait for the level before initiating therapy.
 - May send lactate level as a marker of cyanide toxicity
- Pregnancy test

Diagnostic Procedures/Surgery

- Peak expiratory flow rate:
 - Low peak flow associated with more severe injury

- PaO₂/FiO₂ ratio:
 - A ratio of < 300 after initial resuscitation is associated with the development of respiratory failure.

DIFFERENTIAL DIAGNOSIS

- Irritant gas exposure
- Asphyxiant gas exposure
- Cardiogenic pulmonary edema
- COPD exacerbation
- Asthma exacerbation
- Pneumonia



TREATMENT

PRE HOSPITAL

- 100% oxygen by face mask
- Intubation for patients with agonal breathing
- Rapid transport to ED for those with stridor:
 - May need advanced airway management
- Albuterol nebulizer therapy for bronchospasm

INITIAL STABILIZATION/THERAPY

- 100% oxygen via face mask
- Intubation:
 - Respiratory distress
- Drooling
- Stridor:
 - Refractory hypoxia
 - CNS depression
 - Significant facial/upper airway burns
- Establish IV access.

ED TREATMENT/PROCEDURES

- Inhaled or nebulized albuterol as needed for bronchospasm
- Corticosteroids as needed for patients with history of asthma or COPD
- Intubated patients:
 - Low endotracheal tube cuff pressure
 - Frequent suctioning
 - Positive end-expiratory pressure
- If indicated, treat for carbon monoxide toxicity:
 - 100% oxygen
 - Hyperbaric oxygen in appropriate cases when available

- If indicated, treat for cyanide toxicity:
 - 100% oxygen
 - Hydroxocobalamin (preferred)
 - If only older nitrite-containing cyanide antidote kit is available
 - Sodium nitrite should be used with caution in cases of significant carbon monoxide exposure
 - Sodium thiosulfate can be used safely with CO exposures

MEDICATION

- Albuterol nebulization: 2.5–5 mg in 2.5 mL of normal saline q20min:
 - Alternatively, 15 mg nebulizer treatment continuous over 1 hr
- Methylprednisolone 40 mg IV (peds: 1–2 mg/kg)
- Prednisone: 40–60 mg PO (peds: 1–2 mg/kg)
- Sodium thiosulfate 12.5 g (50 mL of 25% solution) slow IV infusion (peds: 412.5 mg/kg or 1.65 mL/kg of 25% solution)
- Hydroxocobalamin 5 g IV infused over 15 min (peds: 70 mg/kg)



FOLLOW-UP

DISPOSITION

Admission Criteria

- Intubated
- Significant associated burns
- Persistent dyspnea, hoarseness, odynophagia, carbonaceous sputum
- Persistent cough
- Asthma/COPD with bronchospasm
- Significant carbon monoxide or cyanide exposure
- Comorbid medical illnesses

Discharge Criteria

- Minimal exposure history
- Asymptomatic
- Significant exposure history, asymptomatic after 4–6 hr observation

Issues for Referral

- In cases of significant associated burn injuries, transfer to burn facility as appropriate.
- In cases of significant carbon monoxide toxicity, transfer to hyperbaric oxygen facility as appropriate.

FOLLOW-UP RECOMMENDATIONS

Burn follow-up for patients with associated burns.

PEARLS AND PITFALLS

- In suspected cases of cyanide exposure, do not wait for the level before initiating therapy.
- Order carboxyhemoglobin to evaluate for potential carbon monoxide exposure.

ADDITIONAL READING

- Peck MD. Structure fires, smoke production, and smoke alarms. *J Burn Care Res.* 2011;32(5):511–518.
- Rehberg S, Maybauer MO, Enkhbaatar P, et al. Pathophysiology, management and treatment of smoke inhalation injury. *Expert Rev Respir Med.* 2009;3(3):283–297.
- Toon MH, Maybauer MO, Greenwood JE, et al. Management of acute smoke inhalation injury. *Crit Care Resusc.* 2010;12(1):53–61.



See Also (Topic, Algorithm, Electronic Media Element)

- Carbon Monoxide
- Cyanide
- Hyperbaric Oxygen



CODES

ICD9

- 506.2 Upper respiratory inflammation due to fumes and vapors
- 508.2 Respiratory conditions due to smoke inhalation
- 947.1 Burn of larynx, trachea, and lung

ICD10

- J68.2 Upper resp inflam d/t chemicals, gas, fumes and vapors, NEC
- J70.5 Respiratory conditions due to smoke inhalation
- T27.0XXA Burn of larynx and trachea, initial encounter

SNAKE ENVENOMATION

Patrick M. Lank • Timothy B. Erickson

BASICS

DESCRIPTION

- Pit viper venom:
 - Mixture of proteolytic enzymes and thrombin-like esterases:
 - Enzymes cause local muscle and subcutaneous tissue necrosis.
 - Esterases have defibrinating anticoagulant effect, leading to venom-induced consumption coagulopathy (VICC) in severe envenomations.
- Bite location:
 - Extremity bites most common
 - Head, neck, or trunk bites more severe than bite on extremities
- Severe envenomation:
 - Direct bite into artery or vein
 - Neurotoxic envenomations
- Bite mark significance:
 - Pit viper bite: Classically includes 1 or 2 puncture marks
 - Nonvenomous snakes and elapids: Horseshoe-shaped row of multiple teeth marks
- 25% of all pit viper bites are dry and do not result in envenomation.

ETIOLOGY

Venomous Snakes Indigenous to US

- Pit vipers (Crotalinae):
 - Account for 95% of all envenomations
 - Rattlesnakes, cottonmouths, and copperheads
- Coral snakes (Elapidae):
 - Neurotoxic
 - Western coral snakes, found in Arizona and New Mexico
 - More venomous eastern coral snakes, found in Carolinas and Gulf states

International Exotic Venomous Snakes

Occur in zoos or in owners of exotic snakes

Pediatric Considerations

- 30% of all snakebites involve patients younger than 20 yr. 12% of all snakebites are 9 yr or younger.
- Because of their low body weight, smaller children and infants are more

vulnerable to severe envenomation with systemic symptoms.



DIAGNOSIS

SIGNS AND SYMPTOMS

- Local (Crotaline):
 - Classic skin changes:
 - 1 or 2 puncture wounds
 - Pain and swelling at site
 - Swelling and edema of involved extremity:
 - Within 1 hr in severe envenomations
 - Tender proximal lymph nodes
 - Ecchymosis, petechiae, and hemorrhagic vesicles develop within several hours.
- Systemic (Crotaline):
 - Weakness, dizziness
 - Diaphoresis
 - Nausea
 - Scalp paresthesias
 - Periorbital fasciculations
 - Metallic taste
 - Severe bites can lead to:
 - Coagulopathy (VICC)
 - Hypotension
 - Pulmonary edema
 - Hematuria
 - Rhabdomyolysis
 - Renal failure
 - Cardiac dysfunction
 - Potential elevated compartment pressure in involved extremity
- Symptoms (Crotaline):
 - Primarily neurotoxic, leading to weakness, diplopia, confusion, delayed respiratory depression:
 - Local effects may be deceptively minimal.

History

- Description of snake
- Geographic location of bite

Physical-Exam

Search for manifestations of bites as described above.

ESSENTIAL WORKUP

- Careful exam of wound site and involved extremity:
 - Essential in judging severity of envenomation
 - Mark wound margins to follow progression
- Assess for anaphylactic reactions

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC
- Coags
- Fibrinogen, d-dimer
- Electrolytes, BUN/creatinine, glucose
- Creatine phosphokinase (CPK)
- UA
- Type and cross-match with moderate to severe envenomation.

Imaging

Plain radiographs if foreign body suspected

DIFFERENTIAL DIAGNOSIS

- Nonvenomous snakes (in the US):
 - Narrow head
 - Round pupils
 - No rattles
- Pit vipers:
 - Triangular- or arrow-shaped head
 - Vertical or elliptical pupils
 - Heat-sensing pits just behind the nostrils and in front of eyes
 - ± Rattles
- Coral snakes (applies only in US, not internationally):
 - “Red on yellow—kill a fellow”
 - “Red on black—venom lack”



TREATMENT

PRE HOSPITAL

- Retreat well beyond striking range of snake.
- Immobilize extremity in functional position at the level of heart.
- Keep physical activity minimal.
- Remove rings, watches, and all constrictive clothing.
- It is ill-advised to transport a snake to a health care facility for identification

purposes:

- If you are close enough to get a good picture with a camera/phone, you are too close to a potentially venomous snake.
- Even severed head can envenomate.
- Controversies:
 - Pre-hospital local wound maneuvers are NOT recommended because they cause worse local tissue damage and increase the risk of infection. These include:
 - Incision and drainage
 - Mechanical suction devices
 - Oral suction
 - Tourniquets
 - Cryotherapy
 - Electrocutation
 - Pressure immobilization
 - Incision attempts by inexperienced can lead to severe tendon, nerve, and vascular damage.

Pediatric Considerations

- Envenomation more likely to be severe.
- Severity due to relatively low body weight of small child with same volume of venom.

INITIAL STABILIZATION/THERAPY

- Airway, breathing, and circulation management (ABCs)
- Maintain euvolemia with 0.9% normal saline (NS) to maintain renal blood flow
- Wound monitoring
- Immobilize bitten extremity

ED TREATMENT/PROCEDURES

- Supportive care
- Monitor for compartment syndrome:
 - Repeated measurements of extremity circumference every 15–20 min until local progression/swelling subsides.
 - A true compartment syndrome is unlikely following rattlesnake envenomation.
 - Elevated compartment pressures are treated with more antivenom, as surgical intervention with fasciotomy causes more damage to the area.
 - Surgical therapy considered only in incredibly rare cases and should only be considered in consultation with a regional poison center and medical toxicologist
- Analgesia with IV opioids
- Tetanus prophylaxis if needed

- Broad-spectrum antibiotics not routinely indicated
- Steroids not indicated except for reactions to antivenom (see below)
- Routine use of blood products not indicated
- Wound severity:
 - Minimal:
 - Local swelling and tenderness
 - Moderate:
 - Extremity swelling
 - Evidence of systemic toxicity
 - Severe:
 - Obvious toxicity
 - Unstable vital signs
 - Coagulopathy
 - Elapid envenomation
 - Lab abnormalities

Antivenom

- Indications for Crotalid antivenom therapy:
 - More than minimal extremity swelling
 - Extremity swelling that is progressing
 - Clinical signs of systemic toxicity
 - Unstable vital signs
 - Coagulopathy (low platelets or fibrinogen, elevated PT)
- CroFab:
 - Fundamental treatment for North American pit viper envenomation
 - High-affinity purified ovine Fab antibody fragment antivenom
 - CroFab causes less frequent hypersensitivity reactions than older polyvalent antivenom
 - Pediatric antivenom dose = adult antivenom dose
 - Dosing: 4–6 vials initially
 - Reconstitute each CroFab vial with 25 mL sterile water. Dilute in 250 mL 0.9% NaCl and infuse over 1 hr.
 - If hypotensive or with serious active bleeding, initial dose is 8–12 vials
 - Evaluate for envenomation control 1 hr after antivenom bolus infusion. Control is defined by stable wound appearance, improving coagulation studies, and hemodynamic stability.
 - If envenomation control achieved after 1st bolus of antivenom, may need maintenance antivenom therapy at 2 vials q6h × 3 doses.
 - If envenomation control not achieved after 1st bolus of antivenom, repeat initial bolus and reassess. Discuss with regional poison center or medical toxicologist.
- Victims of envenomation who develop an allergic reaction to antivenom:
 - Stop infusion of antivenom

- Administer antihistamines, corticosteroids, and fluids. Consider epinephrine for severe reactions.
- Discussion of risks/benefits of restarting antivenom should take place with regional poison center or medical toxicologist
- Coral snake antivenom:
 - No longer being manufactured, but stockpile exists in geographically appropriate locales.
 - Effective against more toxic eastern coral snake but not against western coral snakes
 - After proper skin testing, 3–5 vials of antivenin recommended.
 - Treatment complications include anaphylaxis and serum sickness.
 - Coral snake venom is neurotoxic; watch for respiratory depression, control airway
- International exotic venomous snakes:
 - Specific antivenoms may be available at local zoos or through the Antivenom Index.

Pediatric Considerations

- Proportionally more antivenin per body weight
- Standard adult doses required

Pregnancy Considerations

- If mother has systemic signs of envenomation toxicity, fetus is also at risk; timely antivenom therapy is still indicated.
- Consult obstetrician

Treatment Assistance

- Contact local poison center 800-222-1222, medical toxicologist, local zoo, or regional herpetologist.
- Call Antivenom Index at 602-626-6016 in Tucson, Arizona, for assistance in treatment of exotic snakes not indigenous to US



FOLLOW-UP

DISPOSITION

Admission Criteria

- 24-hr observation after control of envenomation progression for patients requiring antivenom administration after pit viper bites.
- 24-hr observation for asymptomatic patients with elapid bites.
- ICU admission for:
 - Patients receiving antivenom

- Evidence of moderate to severe envenomation, especially in children
- All victims of elapid bites and for symptomatic exotic snake envenomations

Discharge Criteria

Suspicious bite that shows no signs or symptoms of envenomation for 6–8 hr and has normal lab panel:

- Dry bites may be observed for 8 hr and discharged if there is no development in local toxicity and if lab studies normal.
- Minor envenomations should be observed for 12–24 hr and have labs repeated 6 hr after presentation, then again before discharge.
- Discharge with follow-up in 24 hr.

FOLLOW-UP RECOMMENDATIONS

PCP or toxicology follow-up 1 wk after antivenom therapy to assess for possible serum sickness or envenomation wound infection.

PEARLS AND PITFALLS

- Avoid overly aggressive pre-hospital care interventions. It is best to rapidly transport to closest medical center.
- Be sure to administer proper dose of antivenom in a timely fashion when clinically indicated.

ADDITIONAL READING

- American College of Medical Toxicology, American Academy of Clinical Toxicology, American Association of Poison Control Centers, et al. Pressure immobilization after North American Crotalinae snake envenomation. *Clin Toxicol (Phila)*. 2011;49:881–882.
- Corneille MG, Larson S, Stewart RM, et al. A large single-center experience with treatment of patients with crotalid envenomations: Outcomes with and evolution of antivenin therapy. *Am J Surg*. 2006;192:848–852.
- Cox MR, Reeves JK, Smith KM. Concepts in Crotaline snake envenomation management. *Orthopedics*. 2006;29(12):1083–1087.
- Cumpston KL. Is there a role for fasciotomy in Crotalinae envenomations in North America? *Clin Toxicol (Phila)*. 2011;49:351–365.
- Lavonas EJ, Ruha AM, Banner W, et al. Unified treatment algorithm for the management of crotaline snakebite in the United States: Results of an evidence-informed consensus workshop. *BMC Emerg Med* 2011;11:2.



CODES

ICD9

989.5 Toxic effect of venom

ICD10

- T63.001A Toxic effect of unsp snake venom, accidental, init
- T63.011A Toxic effect of rattlesnake venom, accidental, init
- T63.021A Toxic effect of coral snake venom, accidental, init

SPIDER BITE, BLACK WIDOW

Tarlan Hedayati • Christopher S. Lim



BASICS

DESCRIPTION

- Syndromes caused by envenomation by black widow spider bite
- Mechanism of toxicity:
 - Females are responsible for human envenomations
 - Venom contains potent neurotoxin, α -latrotoxin:
 - Causes cation-channel opening presynaptically, resulting in increased neurotransmitter release into synapses and neuromuscular junctions
 - Increased neurotransmitter release causes increased neurologic, motor, and autonomic effects
- Morbidity and mortality are dose dependent
- Severity of envenomation depends on:
 - Premorbid health of victim:
 - HTN or cardiovascular disease increase risk
 - Size and age of victim:
 - Children (i.e., smaller size for a given dose of venom) are at greater risk of morbidity and mortality.
 - Number of bites
 - Location of bite wounds
 - Size and condition of spider
- Rarely fatal

ETIOLOGY

Black widow spider features:

- Appearance:
 - Glossy black with red markings shaped like an hourglass or a pair of spots on the ventral aspect of the globular abdomen
 - Females have 25–50 mm leg spans and 15 mm long bodies
- Found throughout North America, except the far north and Alaska
- Prefer dark sheltered hideaways such as garages, barns, outhouses, woodpiles, and low-lying foliage
- Most bites occur during the warmer months when spiders are defending their webs and egg clutches



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- History of spider bite very unreliable and species usually not identified
- Bite:
 - Described as a pinprick or pinch, if felt at all
- Local complaints (within minutes of bite):
 - Pain:
 - Sharp, burning at the bite site
 - Usually resolves spontaneously within minutes or hours
 - May become worse and spread proximally from the bite
- Systemic complaints (within 15–60 min):
 - Cardiac:
 - Palpitations
 - Chest pain or tightness
 - Pulmonary:
 - Shortness of breath
 - Cough
 - Neuromuscular:
 - Headache
 - Dizziness
 - Painful regional muscle cramps and spasms
 - Cramping may progress to larger muscle groups
 - Arm bites may lead to arm and chest muscle tightness and dyspnea
 - Leg bites may lead to abdominal pain and leg spasms
 - Cutaneous dysesthesias and hyperesthesias
 - Localized or diffuse diaphoresis
 - GI:
 - Nausea, vomiting
 - Abdominal pain
 - Genitourinary:
 - Painful persistent erection
 - Gynecologic:
 - Pregnant patients may develop uterine contractions and preterm labor
 - Skin:
 - Pruritus
 - Psychiatric:
 - Anxiety
 - Sense of impending doom

Physical-Exam

- Vital signs may be abnormal:
 - HTN or hypotension
 - Tachycardia or bradycardia

- Fever
- Tachypnea
- Cardiac:
 - Dysrhythmias
 - Myocarditis
- Pulmonary:
 - Bronchorrhea
 - Pulmonary edema
 - Respiratory failure:
 - Usually due to respiratory muscle weakness
- Abdomen:
 - Rigidity
- Genitourinary:
 - Priapism
- Neurologic findings:
 - Tetanic contractions, fasciculations or tremors of extremities
 - Spasm and rigidity in large muscle groups
 - Autonomic instability
 - Seizure
- Skin:
 - Local:
 - 2 pinpricks from the spider’s fangs
 - Tender and blanched skin with surrounding erythema (“target lesion”)
 - Swelling
 - Localized sweating
 - Diffuse:
 - Urticaria
 - Piloerection
 - Generalized diaphoresis
- Psychiatric:
 - Acute toxic psychosis
 - Agitation or restlessness

ESSENTIAL WORKUP

Diagnosis is based on:

- Clinical presentation
- Careful inquiry to elicit spider bite history
- Identification of spider (if possible)

DIAGNOSIS TESTS & NTERPRETATION

Lab

- No specific blood tests for black widow spider venom
- CBC:
 - WBC may be mildly elevated
- Electrolytes, calcium
- BUN, creatinine
- Lipase, LFTs
- Creatine kinase:
 - Elevated in patients with significant muscle spasm
- Cardiac enzymes
- Pregnancy test
- Urinalysis:
 - May demonstrate albuminuria
- ABGs in rare cases with pulmonary edema
- ECG and cardiac monitoring for:
 - Patients with known cardiac disease
 - Patients with chest pain, unstable vital signs or dysrhythmias
 - May show digitalis effect transiently

Imaging

- CXR for respiratory complaints
- Abdominal imaging to rule out other causes of pain

DIFFERENTIAL DIAGNOSIS

- Acute surgical abdomen (e.g., appendicitis, cholecystitis, pancreatitis, AAA)
- Ureterolithiasis/nephrolithiasis
- Sympathomimetics (e.g., cocaine, amphetamines)
- Hypocalcemia
- Tetanus
- Muscular injury or strain
- Hypertensive emergency
- MI/acute coronary syndrome
- Anxiety disorder
- Allergic reaction



TREATMENT

PRE HOSPITAL

- ABCs/ACLS
- Immobilize the wound site and apply cool compresses or ice for comfort during transport to hospital
- Supportive measures (analgesics, anxiolytics) may be required for patients with systemic symptoms

- Negative-pressure venom extraction devices have not been recommended for widow spider bites
- Every effort should be made by caregivers at the scene to find and bring in the responsible spider for identification

INITIAL STABILIZATION/THERAPY

- ABCs
- ACLS as needed
- Fetal monitoring for pregnant patients

ED TREATMENT/PROCEDURES

- Clean the bite site thoroughly
- Tetanus prophylaxis
- Antiemetics for nausea and vomiting
- Analgesics
- Antihistamines
- Benzodiazepines for agitation and restlessness
- Muscle cramps/spasm therapy:
 - Benzodiazepines
 - Narcotics
- Antihypertensive agents for symptomatic HTN
- Antivenin:
 - Elicit history of allergy to horse or horse serum
 - Indications:
 - Moderate to severe symptoms that do not respond to symptomatic measures
 - Significant HTN
 - Respiratory distress
 - Symptomatic and pregnant
 - Priapism
 - Severe rhabdomyolysis
 - Compartment syndrome
 - Seizures
- Perform a skin test for sensitivity to horse serum prior to antivenin administration (test kit included in the antivenin package)
- Watch for type I immediate hypersensitivity reaction in the 1st 20 min:
 - Occurs in up to 25% of recipients
 - Consider pretreatment with antihistamines or SC epinephrine 1:1,000
 - Treat anaphylactic reactions with steroids, antihistamines, epinephrine, and cardiopulmonary support
 - Due to the small quantity of antivenin used, if serum sickness reactions occur, they are usually mild
 - Effectiveness is usually apparent within 2 hr of the 1st treatment and

- repeated doses are rarely necessary
- Antivenin may help prevent persistent neuropathic symptoms

MEDICATION

- Antivenin: 1 ampule (2.5 mL) diluted into 50–100 mL of D₅W or NS (peds: Same dose) IV over 1 hr
- Diphenhydramine: 10–50 mg IV or IM q6–8h (peds: 5 mg/kg/d div. QID)
- Lorazepam: 1–2 mg IV or IM (peds 0.01 mg/kg IV or IM)
- Morphine sulfate: 2–10 mg (peds: 0.1 mg/kg) IV or IM PRN (titrate to patient response)
- Sodium nitroprusside: 0.5–10 mcg/kg/min if diastolic > 120 mm Hg
- Tetanus prophylaxis



FOLLOW-UP

DISPOSITION

Admission Criteria

- Pediatric, elderly, pregnant, or symptomatic patients
- Significant cardiovascular symptoms and signs, or severe HTN, particularly in presence of premorbid cardiac disease or chronic HTN
- Respiratory distress or pulmonary edema
- Persistent symptoms not responding to aggressive management and specific antivenin

Discharge Criteria

- Asymptomatic patients with no positive identification of a black widow spider can be released after observation for 1–2 hr
- Asymptomatic patients with no comorbid illness with a positive identification of the black widow spider should be observed for a minimum of 4–6 hr and discharged if their condition does not change
- All discharged patients must be instructed to watch for the following symptoms and to seek appropriate follow-up:
 - Hematuria
 - Rash
 - Joint pain
 - Lymphadenopathy
 - Shortness of breath
 - Signs of infection
- Discharged patients who received antivenin should be instructed to watch for signs of serum sickness:
 - Type III delayed hypersensitivity

- Uncommon
- Occurs 5 days–3 wk post treatment
- Treat with antihistamines and steroids

Issues for Referral

Toxicology consult for patients requiring admission or antivenin administration

FOLLOW-UP RECOMMENDATIONS

- In most untreated patients, symptoms peak after 2–3 hr and then begin to resolve, occasionally recurring episodically over the following few days
- In otherwise healthy adults, complete resolution of symptoms occurs within 2–3 days
- Neurology follow-up if persistent neurologic symptoms last weeks to months including:
 - Fatigue
 - Generalized weakness or myalgias
 - Paresthesias
 - Headache
 - Insomnia
 - Impotence
 - Polyneuritis

PEARLS AND PITFALLS

- Widow bites in infants may present as intractable crying
- A high fever and WBC count should prompt consideration of alternatives to spider bites (e.g., infection)

ADDITIONAL READING

- Boyer LV, Binford GJ, McNally JT. Spider bites. In: Auerbach, ed. *Wilderness Medicine*. 5th ed. Philadelphia, PA: Mosby; 2007.
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See Also (Topic, Algorithm, Electronic Media Element)

Spider Bite, Brown Recluse



CODES

ICD9

989.5 Toxic effect of venom

ICD10

T63.311A Toxic effect of venom of black widow spider, acc, init

SPIDER BITE, BROWN RECLUSE

Tarlan Hedayati • Christopher S. Lim



BASICS

DESCRIPTION

Local or systemic illness caused by brown recluse spider bite envenomation

ETIOLOGY

- Brown recluse spider (also known as the fiddleback spider) features:
 - Appearance:
 - Delicate body and legs spanning 10–25 mm
 - Tan- to dark-brown with darker violin-shaped marking visible on the upper aspect of the head
 - 3 pairs of eyes
 - Found widely throughout the south-central part of US
 - Habitat: Typically warm and dry locations, indoors or outdoors such as wood piles, bundles of rags, cellars, under rocks, or in attics
 - Bites are typically defensive
- Mechanism of toxicity:
 - Venom is a complex cocktail of enzymes and peptides that:
 - Binds to RBC and causes hemolysis
 - Causes prostaglandin release and activates complement cascade
 - Causes lipolysis and tissue necrosis
 - Triggers platelet aggregation and thrombosis
 - Triggers allergic response to venom antigenic properties
 - May lead to shock and DIC in rare cases
 - Toxicity proportional to:
 - The amount of venom relative to the size of patient
 - Location of envenomation on the body

Pediatric Considerations

- Children are more vulnerable to a given amount of venom than healthy adults
- Fatality more common in children due to severe intravascular hemolysis



DIAGNOSIS

SIGNS AND SYMPTOMS

Diagnosis is based not only on the clinical presentation but also on a reliable history of a spider bite.

History

- An isolated cutaneous lesion is the most common presentation
- Bite sites are usually located in areas under clothing where spider gets trapped between clothing and skin
- Local wound symptom onset:
 - Bite onset is usually asymptomatic, but some may report burning or stinging sensation
 - 1–24 hr later, patients may report aching or pruritis locally
- Systemic features:
 - Rare complication
 - More common in children than adults
 - Develop during the 1st 1–3 days postenvenomation.
 - Patient may report:
 - Fever, chills
 - Weakness, malaise
 - Nausea, vomiting, diarrhea
 - Dyspnea
 - Myalgias, muscle cramps, arthralgias
 - Jaundice
 - Petechial or urticarial rash
 - Generalized pruritic rash
 - Hematuria or dark urine

Physical-Exam

- Bite wound:
 - Usually no visible injury if examined within the 1st 1–3 days
 - There may be a pinprick lesion, local blanching and induration, or erythema.
 - Tissue injury may develop at bite site:
 - Initially, bite mark may be surrounded by edema
 - Next, an erythematous border will develop around a purple center with a thin ring of ischemia between the 2
 - Serous or hemorrhagic bullae may form in the center after 24–72 hr
 - Blister may gradually enlarge and darken with the development of and eschar of skin and subcutaneous fat necrosis over 3–4 days
 - Eschar sloughs off 2–5 wk later leaving an ulcer in its place
 - Necrosis develops most extensively where subcutaneous fat is greatest
 - Lower-extremity blisters may spread distally under the influence of gravity
 - Local response is not dependent on the extent of envenomation and cannot be used to predict the likelihood or severity of subsequent systemic illness
- Skin:

- Jaundice
- Petechia
- Urticaria
- Generalized maculopapular rash

ESSENTIAL WORKUP

- Careful inquiry required to elicit the spider bite history
- Routine lab testing not necessary unless systemic toxicity present.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Spider venom can be detected in skin lesions, but widespread clinical testing is not available yet
- CBC:
 - Hemolytic anemia
 - Thrombocytopenia, particularly with DIC
 - Leukocytosis
- Electrolytes:
 - Hyperkalemia or acidosis in renal failure
- BUN, creatinine
- Creatine kinase may be elevated in rhabdomyolysis
- Prothrombin time/partial thromboplastin test may be prolonged in DIC
- α -dimer and fibrin degradation products may be elevated in DIC
- Fibrinogen may be decreased in DIC
- Urinalysis:
 - Hemoglobinuria
 - Proteinuria

Imaging

- CXR in systemic toxicity
- Soft tissue radiograph of bite site

DIFFERENTIAL DIAGNOSIS

- Angioedema
- Bacterial soft tissue infection; MRSA
- Burn
- Cutaneous anthrax
- Diabetic ulcer
- Decubitus ulcer
- Erythema nodosum
- Fungal infection
- Gonococcal hemorrhagic lesion

- Herpes simplex
- IV drug use or “skin popping”
- Vascular insufficiency with secondary ulcer
- Lyme disease
- Neoplastic lesion
- Other arachnid envenomation
- Poison ivy or oak
- Pyoderma gangrenosum
- Sporotrichosis
- Stevens–Johnson syndrome
- Thrombosis
- Vasculitis
- Warfarin use



TREATMENT

PRE HOSPITAL

- Loosely immobilize wound site
- Elevate the affected extremity
- Cover bite with cool compresses
- Transport to hospital when patient experiences immediate onset of symptoms
- Supportive measures for patients with systemic symptoms

ALERT

Every effort should be made by caregivers at the scene to find and bring in the responsible spider for identification.

INITIAL STABILIZATION/THERAPY

IV fluids, oxygen, cardiac monitoring if the patient is experiencing signs of systemic collapse

ED TREATMENT/PROCEDURES

- Cleanse the bite site thoroughly
- Tetanus prophylaxis
- Analgesics
- Antibiotics:
 - Appropriate if wound appears infected
 - Not indicated prophylactically
 - Antistaphylococcal
- Dapsone:
 - Controversial: Consider for severe toxicity.
 - Screen for G6PD deficiency before initiating
 - Monitor for methemoglobinemia, hemolysis, and leukopenia during therapy

- Excision of necrotic wound:
 - Not indicated in the 1st 8 wk because may cause more severe ulcer formation
- Hemoglobinuria:
 - Treated with IV fluids and alkalinization
 - Monitor renal, fluid, and electrolyte status
- Dialysis for renal failure
- Pressors for shock state
- Blood products in severe hemolysis, DIC
- Specific antivenin:
 - Not commercially available
 - Not FDA approved for use in US
- Therapies requiring further investigation:
 - Topical or systemic steroids
 - Hyperbaric therapy (has been shown to decrease wound size in animal model)
 - Topical nitroglycerin
 - Negative pressure wound therapy, or vacuum-assisted closure

MEDICATION

- Antibiotics:
 - Clindamycin: 150–300 mg PO q6h (peds: 8–16 mg/kg/d PO div. QID)
 - Severe skin infections:
 - Vancomycin: 1 g IVPB q12h (peds: 10 mg/kg q6h)
- Dapsone: Progressive dosage of 50–200 mg/d (peds: 2 mg/kg/24 h PO)
- Methylprednisolone: 125 mg IV bolus followed by prednisone 30–50 mg/d for 5 days (peds: methylprednisolone 1–2 mg/kg IV, prednisone 1–2 mg/kg PO)
- Morphine sulfate: 2–10 mg (peds: 0.1 mg/kg) IV or IM PRN

Pediatric Considerations

- Use dapsone only in severe cases because of increased potential for side effects such as:
 - Hepatitis
 - Methemoglobinemia
 - Hemolytic anemia
 - Leukopenia



FOLLOW-UP

DISPOSITION

Admission Criteria

- Significant local reaction or signs of systemic toxicity
- Lower threshold for children, patients with significant comorbidities

Discharge Criteria

- No evidence of systemic toxicity or severe progression of local wound necrosis after envenomation
- Daily reassessment by primary physician, including blood work, until 3–4 days after envenomation to evaluate for systemic toxicity
- Patients should be advised about prolonged course for skin healing with consideration for surgical excision after 8 wk
- Patients should be advised about potential for extensive scarring, infection, and recurrent ulceration

Pediatric Considerations

Longer observation period or admission because of the higher mortality in this population

Issues for Referral

Consider consultation with:

- General surgery or plastic surgery for wound management
- Hyperbaric specialist for wound management
- Toxicologist
- Nephrologist for cases of renal failure
- Intensivist in cases of shock or DIC

FOLLOW-UP RECOMMENDATIONS

- Primary care physician for continued evaluation of wound
- General surgery or plastic surgery for management of complicated wounds
- Hyperbaric specialist for wound management

PEARLS AND PITFALLS

- Remember the limited range of brown recluse spiders and the rarity of arachnidism as a cause of necrotic skin wounds
- In the absence of a reliable spider bite by history, other diagnoses must be carefully sought and excluded
- Be sure to screen for G6PD deficiency as it causes methemoglobinemia and hemolysis in patients receiving dapsone
- Have a low threshold for admitting pediatric patients, adults with systemic symptoms, or anyone with a large, painful, or infected wound

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Spider Bite, Black Widow



CODES

ICD9

989.5 Toxic effect of venom

ICD10

T63.331A Toxic effect of venom of brown recluse spider, acc, init

SPINAL CORD SYNDROMES

Stephen R. Hayden



BASICS

DESCRIPTION

- Anterior cord syndrome:
 - Results from flexion or axial loading mechanism or direct cord compression from vertebral fractures, dislocations, disc herniation, tumor, or abscess
 - Rarely, can be caused by laceration or thrombosis to the anterior spinal artery
- Brown-Séquard syndrome:
 - Hemisection of the spinal cord, classically as a result of a penetrating wound
 - Rarely unilateral cord compression
- Central cord syndrome:
 - Most commonly occurs in elderly patients who have pre-existing cervical spondylosis and stenosis
 - Forced hyperextension causes buckling of the ligamentum flavum, creating a shearing injury to the central portion of the spinal cord.
- Dorsal cord syndrome:
 - Associated with hyperextension injuries
- Complete cord syndrome:
 - Blunt or penetrating trauma that results in complete disruption of spinal cord
 - Symptoms that remain > 24 hr generally are permanent.

ETIOLOGY

- Spinal cord syndromes result from localized disruption of neurotransmission and exhibit mixed motor and sensory deficits. The most common mechanism is trauma.
- Patients with arthritis, osteoporosis, metastatic disease, or other chronic spinal disorders are at risk of developing spinal injuries as the result of minor trauma.



DIAGNOSIS

SIGNS AND SYMPTOMS

History

Acute loss of motor and/or sensory function usually following a traumatic event

Physical-Exam

- Anterior cord syndrome:
 - Bilateral spastic paralysis and loss of pain and temperature sensation below the level of the lesion
 - Preservation of dorsal column function (proprioception and position sense)
- Brown-Séquard syndrome (lateral cord syndrome):
 - Ipsilateral spastic paresis and loss of dorsal column function (proprioception and position sense)
 - Contralateral loss of pain and temperature sensation
 - Deficits usually begin 2 levels below the injury.
- Central cord syndrome:
 - Loss of motor function affects upper extremities more severely than lower extremities.
 - Most profound deficits occur in the distal upper extremities.
 - Sensory loss is more variable.
- Dorsal cord syndrome:
 - Loss of proprioception, position sensation, and coordination below the level of the lesion
- Complete cord syndrome:
 - Flaccid paresis below the level of the injury
 - Low BP and heart rate, flushed skin, priapism may be present (loss of sympathetic tone).
- Sensory deficit levels:
 - C2: Occiput
 - C4: Clavicular region
 - C6: Thumb
 - C8: Little finger
 - T4: Nipple line
 - T10: Umbilicus
 - L1: Inguinal region
 - L5: Dorsum of the foot
 - S5: Perianal area
- Motor deficit levels:
 - C5: Elbow flexion
 - C7: Elbow extension
 - C8: Finger flexion
 - T1: Finger abduction
 - L2: Hip flexion
 - L3: Knee extension
 - L4: Ankle dorsiflexion
 - S1: Ankle plantar flexion

ESSENTIAL WORKUP

- Detailed neurologic exam, focused on determining if any deficit exists and

- attempting to define the level of injury
- A neurosurgical consultation if deficit exists is recommended in most cases

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Basic preoperative lab studies are indicated.
- Consider sedimentation rate and C-reactive protein to risk-stratify other potential diagnoses.

Imaging

All areas of clinical suspicion should be imaged with plain radiographs.

Geriatric Considerations

Cases in which plain radiographs may be difficult to interpret due to severe DJD, the use of CT may be more appropriate.

- CT of the spine when plain films are normal or ambiguous:
 - _ CT allows assessment of the spinal canal and any impingement by bone fragments.
- MRI is the imaging modality of choice for detection of spinal cord damage; in the acute setting, the indications for MRI are:
 - _ Neurologic deficits not explained by plain films or CT
 - _ Clinical progression of a spinal cord lesion
 - _ Determination of acute surgical candidacy
 - _ Disadvantages of MRI include:
 - The inability to adequately monitor the patient while undergoing the study
 - The incompatibility with certain metal devices
 - The time to complete the exam

Diagnostic Procedures/Surgery

- Myelography is used with CT when MRI is not available or cannot be performed.
- A lumbar puncture may be required if considering Guillain–Barré, multiple sclerosis, or transverse myelitis.

DIFFERENTIAL DIAGNOSIS

- Dorsal root injury
- Peripheral nerve injury
- Guillain–Barré syndrome
- Multiple sclerosis
- Transverse myelitis
- Epidural abscess
- Cerebral vascular accident



TREATMENT

PRE HOSPITAL

- Full spinal immobilization
- IV access should be established for fluid resuscitation in the setting of neurogenic shock.
- Patients should be transported to the nearest trauma center:
 - Prompt evaluation and neurosurgical intervention may lead to a better outcome.

Pediatric Considerations

Cervical collars must be the appropriate size for the child; splinting the head and body with towels and tape is a reasonable alternative.

INITIAL STABILIZATION/THERAPY

- Spinal immobilization must be maintained at all times.
- Intubation must proceed with in-line spinal immobilization.
- IV fluids should be administered at maintenance levels unless shock is present:
 - Spinal trauma may cause hypotension due to loss of sympathetic tone; fluid administration is 1st-line treatment.
 - Other causes of hypotension (e.g., hemorrhage) should be sought before being attributed to spinal cord injury (SCI).
 - Generally, hypovolemic shock causes tachycardia, whereas neurogenic shock results in bradycardia.
 - If BP does not improve after a fluid challenge and no other cause for hypotension can be found, vasopressor use may be necessary; α -agonist is preferred.

ED TREATMENT/PROCEDURES

- Other injuries must be treated as indicated.
- Level of SCI should be determined as a baseline to follow for improvement or deterioration.
- A neurosurgeon must be consulted once an SCI is suspected, even when plain films are normal; early surgical decompression or immobilization may reduce morbidity.
- The patient with an SCI should be managed at an appropriate regional trauma or spinal center:
 - If necessary, transfer should occur as soon as management of other injuries allow.
- IV antibiotics and tetanus prophylaxis are given to patients with a penetrating injury.
- IV vasopressor support may be required to treat neurogenic shock.

MEDICATION

- Phenylephrine: 0.5–2 µg/kg bolus then 50–100 µg/min drip
- Ancef: 1,000 mg q8h

ALERT

In the early 1990s, the use of high-dose methylprednisolone infusion was widely adopted as standard of care following the reports of the 2nd and 3rd National Acute Spinal Cord Injury Study (NASCIS II, NASCIS III); however, extensive systematic review of this therapy and the evidence to support it has demonstrated that this therapy is not recommended for routine use in SCI.



FOLLOW-UP

DISPOSITION

Admission Criteria

All patients with spinal cord syndrome must be admitted to an ICU setting.

Discharge Criteria

No patient with symptoms suggestive of SCI should be discharged.

PEARLS AND PITFALLS

- A detailed neurologic exam and attempt to document the spinal level of neurologic symptoms is critical.
- Involve neurosurgical consultants early, as outcome is time-dependent in many cases.
- EM physicians should not start methylprednisolone treatment for acute SCI.

ADDITIONAL READING

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- Schouten R, Albert T, Kwon BK. The spine-injured patient: Initial assessment and emergency treatment. *J Am Acad Orthop Surg.* 2012;20(6):336–346.
- Theodore N, Aarabi B, Dhall SS, et al. Transportation of patients with acute traumatic cervical spine injuries. *Neurosurgery.* 2013;72(suppl2):35–39.



CODES

ICD9

- 344.89 Other specified paralytic syndrome
- 952.02 C1-C4 level with anterior cord syndrome
- 952.03 C1-C4 level with central cord syndrome

ICD10

- G83.81 Brown-Sequard syndrome
- G83.82 Anterior cord syndrome
- S14.129A Central cord synd at unsp level of cerv spinal cord, init

SPINE INJURY: CERVICAL, ADULT

Gary M. Vilke



BASICS

DESCRIPTION

- Injury to the neck that results in injury to the spinal cord, cervical spine, or ligaments supporting the cervical spine
- May have more than 1 mechanism concurrently
- Flexion injuries:
 - Simple wedge fracture: Usually a stable fracture
 - Anterior subluxation: Disruption of the posterior ligament complex without bony injury; potentially unstable injury
 - Clay shoveler's fracture: Avulsion fracture of the spinous process of C7, C6, or T1; stable fracture
 - Flexion teardrop fracture: Extremely unstable fracture; may be associated with acute anterior cervical cord syndrome
 - Atlanto-occipital dislocation: Unstable injury
 - Bilateral facet dislocation: Can occur from C2–C7; unstable injury
- Flexion/rotation injuries:
 - Unilateral facet dislocation “locked” vertebra: Stable injury
 - Rotary atlantoaxial dislocation: Unstable injury
- Extension injuries:
 - Extension teardrop fracture: An avulsion fracture of the anteroinferior corner of the involved vertebral body; unstable in extension and stable in flexion
 - Posterior arch of C1 fracture: Arch is compressed between the occiput and the spinous process of the axis during hyperextension; unstable fracture
 - Avulsion fracture of the anterior arch of the atlas: Horizontal fracture of C1 and prevertebral soft tissue swelling on the lateral C-spine
 - Hangman fracture: Traumatic spondylolisthesis of the axis involving the pedicles of C2; unstable fracture
 - Hyperextension dislocation: Described as the syndrome of the paralyzed patient with a radiographically normal-appearing C-spine
- Extension–rotation injury:
 - Pillar fracture: Generally stable fracture
- Vertical compression (axial loading) injuries:
 - Jefferson fracture: Burst fracture of both the anterior and posterior arches of C1; extremely unstable fracture
 - Burst fracture: A comminuted fracture of the vertebral body with variable retropulsion of the posterior body fragments into the spinal canal

ETIOLOGY

- Blunt trauma is the major cause of neck injuries:
 - Automobile accidents account for >50%.
 - Falls account for ~20%.
 - Sporting accidents account for 15%.
 - Minor trauma in patients with severe arthritis may result in cervical injuries.
- Penetrating trauma

DIAGNOSIS

SIGNS AND SYMPTOMS

- Neck pain, tenderness on palpation
- Numbness, weakness, paresthesias of upper or lower extremities
- Always assume a C-spine injury in any patient with:
 - Altered mental status (unconscious, intoxicated, on drugs, or hypoxic) following trauma or if events are unknown but trauma is likely
 - Inability to communicate (mentally retarded, language barrier, or intubated) following trauma or if events are unknown but trauma is likely
 - Distracting injury
 - Blunt trauma involving head or neck
- Incomplete cervical cord syndromes (see separate chapter):
 - Brown-Séquard syndrome: Hemisection of cord from penetrating injury (ipsilateral motor paralysis/contralateral sensory hypesthesia)
 - Anterior cord syndrome: Cervical flexion injury causing cord contusion (paralysis/hypesthesia with sparing of position/touch/vibratory sensations)
 - Central cord syndrome: Patients with cervical degenerative arthritis with forced hyperflexion (deficits greater in upper extremities relative to lower extremities)

History

- Obtain history of head or neck trauma.
- Identify history of ankylosing spondylitis or other brittle bone diseases.
- Specific symptoms:
 - Neck pain
 - Weakness
 - Numbness or tingling
 - Stinger

Physical-Exam

- Direct visualization of neck for bruising or deformity
- Palpation over the spinous processes

- Motor, sensory, and reflex exam of upper and lower extremities

ESSENTIAL WORKUP

Complete physical exam and radiographic imaging if clinically indicated

DIAGNOSIS TESTS & INTERPRETATION

Imaging

- Standard radiographs include 3 separate views: Lateral, anteroposterior, and open-mouth views of the odontoid while still immobilized.
- Lateral radiograph must include C1–T1; a swimmer's view may be necessary to view lower levels.
- Supine oblique views may help in identifying subtle rotational injuries.
- CT should be obtained when C-spine fractures, dislocations, or soft tissue swelling is seen on plain films or for unexplained neck pain/neurologic deficit with normal radiograph.
- CT (helical) is considered a good alternative to plain films and is favored in certain patients, including intubated victims of blunt trauma.
- Flexion–extension views may be needed to evaluate for dynamic ligamentous injuries if static radiographs are negative and the alert, cooperative patient still complains of pain.
- MRI has become a valuable tool in evaluating patients with neurologic deficits, including spinal cord injury without radiographic abnormality.

DIFFERENTIAL DIAGNOSIS

- Cervical muscle strain injury (whiplash)
- C-spine dislocation
- Cervical fracture dislocation
- Complex or simple cervical fractures



TREATMENT

PRE HOSPITAL

- If C-spine injury suspected, immobilize with a hard collar, neck pads, and backboard.
- Immobilized patients require constant observation in case of vomiting.
- Immobilize C-spine in patients with penetrating neck wounds only if a neurologic deficit is present.
- If the weapon is still embedded, immobilize the neck to avoid further injury and do not remove the impaling object unless it directly impedes breathing.

INITIAL STABILIZATION/THERAPY

- Immobilize the spine using a rigid collar and backboard plus tape/towels or

lightweight foam pads along the side of the neck.

- Stabilize the airway, establish IV access, and support circulation:
 - Preferred method is careful orotracheal rapid sequence intubation with inline spinal immobilization.
 - Fiberoptic intubation set should be at the bedside and considered if available.

ED TREATMENT/PROCEDURES

- Assess patient for other injuries; remember that the abdominal exam in a C-spine-injured patient is unreliable and further objective testing is indicated.
- Patients with ankylosing spondylitis or other brittle bone diseases are at risk for fracture and cord injury with even trivial mechanisms.
- Patients may be clinically cleared and do not require C-spine radiograph (based on NEXUS) if they:
 - Have no altered level of alertness
 - Are not intoxicated
 - Have no tenderness in the posterior midline cervical spine
 - Have no distracting painful injury
 - Have no focal neurologic deficit
- If a neurologic deficit is present, consult neurosurgery.
- If the radiographs or CT is abnormal, consult neurosurgery or the orthopedic spine service.
- If the radiographs are normal but the alert and cooperative patient is having severe neck pain, consider flexion–extension films, CT, or MRI; if abnormal, consult neurosurgery.

MEDICATION

High-dose steroid protocol for patients with neurologic deficits due to fractures or dislocations.

First Line

Methylprednisolone: 30 mg/kg IV bolus then 5.4 mg/kg/h over the next 23 hr; begin within 8 hr of injury



FOLLOW-UP

DISPOSITION

Admission Criteria

- C-spine fractures or dislocations associated with a neurologic deficit or any unstable fracture or dislocation should be admitted to the ICU or a monitored setting.

- Stable C-spine fractures or dislocations should be admitted.
- Isolated spinous process fractures that are not associated with any neurologic deficit or instability on plain films.
- Simple cervical wedge fractures with no neurologic deficit.

Discharge Criteria

- Patients with acute cervical strain “whiplash”
- Musculoskeletal injuries that are associated with mild to moderate pain, no neurologic deficit, and normal radiographs

Issues for Referral

- The patient with a radiographically normal C-spine but continuous pain may be discharged with a hard collar and appropriate orthopedic follow-up.
- Patients with persistent symptoms from stinger should be followed up in 3–4 wk for EMG.

FOLLOW-UP RECOMMENDATIONS

Return to ED for evaluation if pain increases or numbness, weakness, stingers, or other clinical changes develop.

PEARLS AND PITFALLS

- Trivial neck injuries in patient with ankylosing spondylitis or other brittle bone diseases may result in significant injuries.
- All the NEXUS criteria need to be applied to safely rule out a clinically significant spinal fracture without imaging.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Ankylosing Spondylitis
- Head Trauma, Blunt

- Spinal Cord Syndromes



CODES

ICD9

- 805.00 Closed fracture of cervical vertebra, unspecified level
- 839.00 Closed dislocation, cervical vertebra, unspecified
- 959.09 Injury of face and neck

ICD10

- S12.9XXA Fracture of neck, unspecified, initial encounter
- S13.101A Dislocation of unspecified cervical vertebrae, init encntr
- S19.9XXA Unspecified injury of neck, initial encounter

SPINE INJURY: CERVICAL, PEDIATRIC

Roxanna A. Sadri



BASICS

DESCRIPTION

- Relatively rare, present in 1–2% of patients with severe blunt trauma
- Children <8 yr of age are more likely to have upper cervical spine injuries (C1–C3) and are at risk of growth plate injuries:
 - Spinal fulcrum is higher (C2–C3 at birth)
 - Relatively larger head to body
 - Weaker cervical musculature
 - Ligamentous laxity
 - Immature vertebral joints
- Children >8 yr of age:
 - Increased incidence of pancervical injuries
 - Vertebral body and arch fractures
 - Lower cervical spine injuries more common
- Special considerations:
 - Down syndrome
 - Klippel–Feil syndrome
 - Morquio syndrome
 - Larsen syndrome
- Spinal cord injury without radiographic abnormality (SCIWORA):
 - Based on study population, incidence from 4.5–35% of children with spinal injuries
 - More common in children <8 yr of age
 - May present as definite spinal cord injury:
 - Spinal shock
 - Neurologic deficits
 - Symptoms may be transient and have resolved by time of evaluation:
 - Paresthesias
 - Burning sensation of hands
 - Weakness
 - Symptoms often occur immediately after injury but may have delayed onset (i.e., minutes to days).

ETIOLOGY

- Birth – breech vaginal delivery
- <8 yr – MVC and falls
- >8 yr – MVC and sports injuries

SIGNS AND SYMPTOMS

- Local cervical spine pain
- Limited range of motion
- Neurologic deficit (may be transient)
- May be masked by altered mental status or distracting injury
- Abnormal vital signs:
 - Hypotension
 - Bradycardia
 - Hypoventilation or apnea
- Neck signs:
 - Tender to palpation over cervical spine
 - Limited range of motion
 - Muscle spasm
- Neurologic signs:
 - Paresthesias or sensory deficit
 - Flaccid tone
 - Loss of rectal tone
 - Paralysis
- Paralysis:
 - Anterior cord syndrome:
 - Hyperflexion injury
 - Paralysis
 - Loss of pain sensation, preservation of light touch, and proprioception
 - Central cord syndrome:
 - Hyperextension injury
 - Weakness upper greater than lower extremities
 - Burning sensation in hands and fingers
 - Brown-Séquard syndrome:
 - Cord hemisection
 - Ipsilateral paralysis
 - Contralateral loss of pain
 - Horner's syndrome:
 - Disruption of sympathetic chain
 - Ipsilateral ptosis, miosis, anhidrosis
 - Also consider carotid dissection
 - Quadriplegia
 - Absent reflexes
- Preverbal child may be unable to express symptoms and may not cooperate during exam.

ESSENTIAL WORKUP

- Obtain cervical spine radiographs for:
 - Cervical spine tenderness
 - Altered mental status
 - Neurologic deficit (even if transient)
 - Distracting injury
 - Mechanism of injury
- Additional imaging studies (CT, MRI) may be indicated if plain radiographs are inconclusive OR clinical exam suggests injury
- Nexus criteria can be applied safely to children > 8 yr of age, but not younger

DIAGNOSIS TESTS & INTERPRETATION

Imaging

- Cervical spine radiographs:
 - Standard initial views: Anteroposterior, cross-table lateral, and open-mouth odontoid
 - Cross-table lateral identifies ~80% of fractures, dislocations, and subluxations
 - Addition of AP and odontoid increases sensitivity
 - Need to visualize all 7 cervical vertebrae and C7–T1 junction
 - Space between anterior arch of C1 and anterior aspect of odontoid process:
 - 5 mm or smaller in children and 3 mm in adults
 - Thickening of prevertebral soft tissue:
 - Suggests underlying fracture or ligamentous injury
 - Also occurs with neck flexion, expiration, swallowing
 - Too much variability exists for measurements to be highly sensitive.
 - Soft tissue below the glottis should be approximately twice as thick as above the glottis.
 - Pseudosubluxation of C2:
 - Normal variant
 - A result of ligamentous laxity and often resolves by the age of 8 yr
 - C2 anteriorly displaced on C3
 - Posterior cervical line retains normal relationships.
 - Line drawn between anterior aspect of spinous processes of C1 and C3 should pass within 2 mm of anterior aspect of spinous process of C2.
 - Larger than 2-mm space suggests underlying hangman fracture.
 - Can be applied only at C1–C3
 - Anterior vertebral wedging of C3 and C4:
 - May be mistaken for compression fracture
 - Epiphyseal growth plates may resemble fractures:
 - Posterior arch of C1 fuses by 4 yr of age.

- Anterior arch of C1 fuses by age 6 yr of age.
- Base of odontoid fuses with body of C2 by 7 yr of age.
- Flexion and extension views:
 - Limited use
 - May be useful if suspected occult ligamentous injury
 - Negative cervical spine films
 - No neurologic abnormalities
- CT scan:
 - If fracture suspected despite negative plain radiographs
 - For further definition of fracture identified on plain radiographs
 - Suspicion of a fracture seen on plain radiographs
 - Inadequate radiographs
- MRI:
 - Suspected spinal cord injury with or without abnormalities found on plain radiographs or CT

DIFFERENTIAL DIAGNOSIS

- Cervical muscle strain
- Torticollis
- Cervical adenitis
- Retropharyngeal abscess
- Meningitis



TREATMENT

PRE HOSPITAL

- Immobilize all infants and children with potential cervical spine injuries
- Appropriate size cervical collar
- Tape, towels, padding in combination with car seat or spine board if formal collar not available
- Place padding under neck, shoulders, and back, as relatively larger cranium can cause flexion
- In setting of sports injuries, helmets should be left on

INITIAL STABILIZATION/THERAPY

- Maintain cervical spine immobilization.
- Logroll patient.
- Maintenance of inline cervical spine immobilization if intubation is required.

ED TREATMENT/PROCEDURES

- Any trauma patient with neurologic deficit consistent with spinal cord injury should have methylprednisolone considered.
- Neurosurgical consultation:

- True subluxation
- Fracture
- Transient or persistent neurologic deficit

MEDICATION

CONTROVERSIAL

- Methylprednisolone: Loading dose 30 mg/kg IV over 1 hr; maintenance infusion 5.4 mg/kg/hr over next 23 hr; initiate within 8 hr of injury
- Can cause immunosuppression and increase risk of infection
- Recommend discussion with neurosurgery prior to initiation



FOLLOW-UP

DISPOSITION

Admission Criteria

- Altered mental status
- Signs/symptoms of spinal cord injury
- Fracture
- Obtain appropriate consultation:
 - Neurosurgery
 - Orthopedic spine

Discharge Criteria

- Completely normal mental status
- No radiographic abnormalities
- No transient or persistent neurologic deficit
- Educate parents:
 - SCIWORA can present with delayed onset of symptoms.
 - Patient should return to hospital if paresthesias, weakness, or paralysis is present.

FOLLOW-UP RECOMMENDATIONS

- Follow up with orthopedic surgeon or neurosurgeon as directed
- If concussion suspected, follow-up suggested
- Children with significant trauma should have psychological follow-up.

PEARLS AND PITFALLS

- Maintain appropriate immobilization during evaluation.
- In most cases, plain radiographs can be used as initial screening tool.

- Be aware of unique features of pediatric cervical spine.
- Symptoms of SCIWORA can be transient or delayed.

ADDITIONAL READING

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- Caviness AC. Evaluation of cervical spine injuries in children and adolescents. UpToDate. Available at <http://www.uptodate.com/contents/evaluation-of-cervical-spine-injuries-in-children-and-adolescents>.
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- Swischuk LE. *Imaging of the Cervical Spine in Children.* New York, NY: Springer-Verlag; 2004.



CODES

ICD9

- 805.00 Closed fracture of cervical vertebra, unspecified level
- 847.0 Sprain of neck
- 959.09 Injury of face and neck

ICD10

- S12.9XXA Fracture of neck, unspecified, initial encounter
- S13.4XXA Sprain of ligaments of cervical spine, initial encounter
- S19.9XXA Unspecified injury of neck, initial encounter

SPINE INJURY: COCCYX

Gary Schwartz

BASICS

DESCRIPTION

- Usually results from a fall that ends with the victim in sitting position
- Fall usually occurs from standing height
- Can occur during childbirth
- More common in women

ETIOLOGY

See “Description.”

DIAGNOSIS

SIGNS AND SYMPTOMS

- Tenderness localized over the coccyx
- Ecchymosis over the gluteal fold
- Pain with sitting, especially when leaning forward, and with defecation

History

Patient or witness to provide full history of accident including any earlier events that might influence mechanism of fall or insult

Physical-Exam

A full physical exam:

- Including rectal exam to assess tenderness or mobility of coccyx
- No evidence of neurologic deficit should be found in isolated coccygeal fractures.

ESSENTIAL WORKUP

Most often isolated injury, but if other spinal injury of concern, spinal immobilization should be instituted.

DIAGNOSIS TESTS & NTERPRETATION

Imaging

Routine radiographic imaging unnecessary:

- Concern about unnecessary radiation to gonads when diagnosis can be made clinically
- Imaging is indicated if concern for other spine injuries.

- Radiographs can be hard to interpret because coccyx has normal variant positions that can be confused with fracture.
- Lateral radiograph is a best view for fracture and dislocation.

DIFFERENTIAL DIAGNOSIS

- Coccygodynia
- Levator ani syndrome
- Pilonidal cyst
- Perirectal abscess



TREATMENT

PRE HOSPITAL

- Pain management
- Assess for other injuries

INITIAL STABILIZATION/THERAPY

- Usually none required; if patient unstable, consider other diagnoses.
- Medicate for pain.

ED TREATMENT/PROCEDURES

- Pain medication
- Reduction of displaced coccygeal fracture, but rarely necessary.

TREATMENT GENERAL MEASURES

Recommend donut-shaped seat cushion for comfort.

MEDICATION

- Medication for pain if and as needed
- Stool softener

SURGERY/OTHER PROCEDURES

Reduction may be attempted if displaced coccygeal fracture evident, but rarely needed or successful.



FOLLOW-UP

DISPOSITION

Admission Criteria

Admission is generally not required.

Discharge Criteria

Coccygeal fracture can be managed on an outpatient basis unless other intercurrent injury makes admission necessary.

ADDITIONAL READING

- Cwinn AA. Pelvis. In: Marx J, ed. *Rosen's Emergency Medicine: Concepts and Clinical Practice*. 5th ed. St. Louis, MO: Mosby; 2002:632–633.
- Gutierrez PR, Más Martínez JJ, Arenas J. Salter-Harris type I fracture of the sacro-coccygeal joint. *Pediatr Radiol*. 1998;28:734.
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CODES

ICD9

- 805.6 Closed fracture of sacrum and coccyx without mention of spinal cord injury
- 847.4 Sprain of coccyx
- 959.19 Other injury of other sites of trunk

ICD10

- S32.2XXA Fracture of coccyx, initial encounter for closed fracture
- S33.8XXA Sprain of oth parts of lumbar spine and pelvis, init encntr
- S39.92XA Unspecified injury of lower back, initial encounter

SPINE INJURY: LUMBAR

Stephen R. Hayden

BASICS

DESCRIPTION

- Flexion compression fracture:
 - Wedge compression:
 - If < 50% anterior compression of the vertebral body, injury considered stable
 - No ligamentous injury
 - No neurologic deficit
 - Burst fracture:
 - Vertebral body fracture with retropulsion of bone into the neural canal
 - Kyphosis evident on lateral radiograph
 - Posterior ligamentous injury
 - Anterior compression, lower extremities, calcaneal fractures
 - Possible neurologic deficit
- Flexion distraction (lap belt injury):
 - Abdominal injuries likely
 - Chance fracture:
 - Purely bony injury; fracture line through spinous process, pedicles, and vertebral body
 - No kyphosis evident on lateral radiograph
 - Often no neurologic deficit
 - Facet dislocation:
 - Mostly soft tissue injury; no fracture
 - Complete disruption of posterior ligaments and intervertebral disc
 - Neurologic deficit may be present.
- Flexion rotation:
 - Unstable injury
 - Neurologic deficit often present
- Extension:
 - Unstable, uncommon
 - Disruption of anterior longitudinal ligament and intervertebral disc
 - Neurologic sequelae rare but possible
- Shear injuries (translational injuries):
 - Anterior, posterior, or lateral translation of superior vertebral segment over the inferior segment
 - Complete ligamentous disruption

- Neurologic deficit present
- Simple fractures:
 - Isolated spinous process fracture:
 - Ligamentous disruption
 - No neurologic deficit
 - Isolated transverse process fracture:
 - Ligamentous disruption
 - Neurologic deficit possible; rare isolated root injury

ETIOLOGY

- Blunt trauma with axial distraction, axial compression, or translational forces applied to lumbar region
- Fall from height landing on the feet (associated calcaneal fractures) or on the buttocks
- Motor vehicle accidents (MVA)

Pediatric Considerations

- Rare reports of child abuse presenting as lower extremity flaccid paralysis owing to lumbar spine fracture.
- Spinal cord terminates at L3 in newborn and recedes to T12 by adulthood; direct cord damage possible in children with high lumbar fractures.
- End plate avulsion fractures: Adolescent injury usually at L4–L5 or L5–S1 level; ligament pulls off vertebral body end plate; associated neurologic findings.

DIAGNOSIS

SIGNS AND SYMPTOMS

- Pain or localized tenderness to palpation in lumbar midline
- Ecchymosis or deformity overlying lumbar region; palpable deformity; paraspinal muscle spasm
- Increased interspinous distance by palpation
- Step-off (anterior or posterior displacement of spinous process) by palpation
- Neurologic deficits referable to lumbar spinal nerves:
 - Loss of bladder control
 - Motor: Hip flexion (L1–L4), leg extension (L3, L4), ankle dorsiflexion (L4, L5), toe extension (L5)
 - Sensory: Inguinal crease (L1), medial thigh (L2–L3), knee (L4), lateral calf (L5)
 - Reflexes: Knee jerk (L2–L4)
- Pain may be masked by associated distracting injuries (e.g., pelvis, calcaneal fractures).
- Patients with multiple injuries and altered mental status have an unreliable

clinical exam and require imaging.

History

Mechanism to suggest forces applied to lumbar region:

- MVA
- Fall
- Direct impact to lumbar region

Physical-Exam

- Midline lumbar tenderness or deformity
- Neurologic findings involving lumbar spinal nerves

Geriatric Considerations

- Consider abuse in cases of uncertain mechanism.
- Increase suspicion of bleeding consequences, such as spinal hematomas, in patients taking Coumadin or other anticoagulants.

ESSENTIAL WORKUP

Following criteria associated with higher risk of thoracolumbar (TL) spine injuries and should be imaged:

- TL pain or tenderness to palpation
- Decreased level of consciousness (Glasgow Coma Scale <14)
- Drug intoxication, altered pain perception
- Neurologic deficits (described above)
- Painful, distracting injuries
- Severe injury mechanism (e.g., rollover MVA, auto vs. pedestrian, fall > 10 ft)
- Lumbar radiographs (described under Imaging)
- Careful neurologic exam including:
 - _ Assessment of rectal tone
 - _ Bulbocavernosus and cremasteric reflexes
 - _ Perineal sensation

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Standard multitrauma labs if indicated:
- CBC
- Chemistry panel
- Coagulation studies
- Urinalysis

Imaging

- Lumbar radiography with minimum of anteroposterior and lateral views. Characteristics of *unstable* fractures include:
 - Widening of interspinous, interlaminar, or interpedicular distance
 - Kyphosis $> 20^\circ$
 - Translation > 2 mm
 - Vertebral body height loss $> 50\%$
 - Articular process fracture
- Radiographs may not identify burst fractures in 25% of cases.
- If a fracture is identified, entire spine should be imaged to evaluate for associated injuries.
- Spinous process fracture, transverse process fracture, or simple transverse sacral fracture require lumbar flexion–extension films if patient is neurologically intact and there is no evidence of unstable injury.
- CT or MRI should be performed for further evaluation of suspected fractures or fractures identified on plain films to assess spinal cord integrity.

Diagnostic Procedures/Surgery

Consider postvoid residual urinary catheterization or ultrasound to identify urinary retention.

DIFFERENTIAL DIAGNOSIS

- Contusion
- Pathologic fracture (metastatic cancer)
- Osteoporosis
- Pelvic fracture
- Traumatic herniated disc
- Low posterior rib fracture
- Tuberculous spondylitis (Pott disease)
- Ankylosing spondylitis
- Osteogenesis imperfecta (pediatric)
- Congenital scoliosis with hemivertebra (mistaken for lateral wedge fracture)
- Child abuse
- Spinal hematoma
- Epidural abscess



TREATMENT

PRE HOSPITAL

It is difficult to determine whether an injury is stable in the field; any patients with suspected spinal injuries should be immobilized to prevent further injury.

INITIAL STABILIZATION/THERAPY

- Immobilization while tending to immediate life-threatening conditions
- Airway, breathing, and circulation management

ED TREATMENT/PROCEDURES

- Maintain spinal immobilization.
- High-dose methylprednisolone protocol for any neurologic deficit (best in concert with specialist consultation)
- Consultation with orthopedic spine or neurosurgery service
- Appropriate analgesia
- The following stable injuries may be treated conservatively if CT confirms stability of injury and patient is neurologically intact:
 - _ Isolated spinous process, transverse process fracture
 - _ Chance fracture
 - _ Anterior wedge compression (<50%) fracture
 - _ Stable burst fracture
- Total contact orthotic devices may be useful; limited activities; sleep prone; avoid pillows and soft mattresses, which may worsen deformity.

MEDICATION

- Narcotic pain medication in absence of contraindications
- High-dose steroid protocol: Methylprednisolone: 30 mg/kg IV load over 1 hr, then 5.4 mg/kg/h for the next 23 hr; initiate in ED within 8 hr of injury.

First Line

- Tylenol: 1 g (peds: 15 mg/kg) PO q4h PRN
- Motrin: 400–800 mg (peds: 10 mg/kg) PO q6h PRN
- Dilaudid: 1–2 mg (peds: 0.015 mg/kg) IV/IM q3h PRN
- Morphine: 2–10 mg (peds: 0.1–0.2 mg/kg) IV/IM q3h PRN
- Toradol: 30 mg IV or 60 mg IM (peds: 0.5 mg/kg IV or 1 mg/kg IM) q6h PRN:
 - _ Use half Toradol dose for patients >65 y

Second Line

- Flexeril: 5–10 mg PO q8h PRN
- Soma: 350 mg PO q8h PRN
- Zofran: 4–8 mg IV/PO (peds: >4 y, 4 mg IV/PO) q8h PRN
- Compazine: 5–10 mg IV/IM/PO (peds: 2.5 mg PR/PO) q8h PRN
- Phenergan: 12.5–25 mg IM/PO/IV (peds: 0.5–1 mg/kg IM/PR) q8h PRN:
 - _ IV Phenergan NOT recommended due to reports of tissue necrosis



FOLLOW-UP

DISPOSITION

Admission Criteria

Patients with traumatic lumbar fractures should be admitted for stabilization procedures, parenteral pain control, management of possible ileus, and evaluation for associated injuries.

Discharge Criteria

- Neurologically intact patients with stable injuries evaluated in conjunction with a spine surgeon
- Patients with simple compression (wedge) fractures with no neurologic deficit may be considered for outpatient management if adequate pain control and appropriate follow-up can be arranged.
- Simple transverse sacral fracture, isolated spinous process fracture, and isolated transverse process fracture may also be considered for outpatient management.
- The patient must be neurologically intact with a stable living situation; CT scan and flexion–extension films must confirm fracture stability.

Issues for Referral

Patients discharged with stable injuries should have primary care or orthopedic appointment in 1 wk to monitor for recovery and evaluate for potential complications.

FOLLOW-UP RECOMMENDATIONS

Return to ED for new neurologic symptoms or pain not controlled by discharge medications. Otherwise, follow-up as described above.

PEARLS AND PITFALLS

- Lumbar fractures are rare in pediatrics. Aggressively pursue causative factor if mechanism is not evident.
- Older individuals may have underlying medical cause of lumbar pathology. Pursue alternative causes of pain, such as hematomas and infections. Be vigilant in patients taking anticoagulants.
- CT should follow compression fractures seen on plain films to assess for stability and potential canal involvement.
- Otherwise healthy, ambulatory patients with simple post MVA low back pain may be safely discharged without imaging if the exam is otherwise reassuring.

ADDITIONAL READING

- Boran S, Lenahan B, Street J, et al. A 10-year review of sports-related spinal injuries. *Ir J MedSci*. 2011;180(4):859–863.
- Hanck J, Muñiz A. Cervical spondylodiscitis, osteomyelitis, and epidural abscess mimicking a vertebral fracture. *J Emerg Med*. 2012;42(3):e43–e46.
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See Also (Topic, Algorithm, Electronic Media Element)

- Pediatric Trauma
- Spinal Cord Syndromes
- Trauma, Multiple



CODES

ICD9

- 722.10 Displacement of lumbar intervertebral disc without myelopathy
- 805.4 Closed fracture of lumbar vertebra without mention of spinal cord injury
- 847.2 Sprain of lumbar

ICD10

- M51.26 Other intervertebral disc displacement, lumbar region
- S32.009A Unsp fracture of unsp lumbar vertebra, init for clos fx
- S33.5XXA Sprain of ligaments of lumbar spine, initial encounter

SPINE INJURY: THORACIC

Lauren N. Elliott



BASICS

DESCRIPTION

- The following forces account for most thoracic fractures and dislocations:
 - Axial compression
 - Flexion–rotation
 - Shear
 - Flexion–distraction
 - Extension
- 3 anatomically distinct columns; if 2 of the 3 columns are disrupted, the spinal column is unstable:
 - Posterior column: Posterior bony arch and interconnecting ligamentous structures
 - Middle column: Posterior aspects of the vertebral bodies, posterior annulus fibrosis, and posterior longitudinal ligament
 - Anterior column: Anterior longitudinal ligament, anterior annulus fibrosis, and anterior vertebral body
- Major vs. minor fractures:
 - Minor:
 - Isolated articular fracture
 - Transverse process fracture
 - Spinous process fracture
 - Pars interarticularis fracture
 - Major:
 - Compression fracture
 - Burst fracture
 - Seat belt injury
 - Fracture–dislocation
- Compression fracture (anterior or lateral flexion):
 - Fracture of anterior portion of vertebral body with intact middle column
 - May be posterior column disruption
 - Type A: Fracture through both end plates
 - Type B: Fracture through superior end plate
 - Type C: Fracture through inferior end plate
 - Type D: Both end plates intact
- Burst fracture (axial loading):
 - Fracture through middle column of spine
 - May have spreading of posterior elements and laminar fractures with

possible retropulsion into the spinal canal and potential neurologic compromise

- Type A: Fracture through both end plates
- Type B: Fracture through superior end plate
- Type C: Fracture through inferior end plate
- Type D: Burst in middle column with rotational injury leading to subluxation
- Type E: Burst in middle column with asymmetric compression of anterior column
- Seat belt injury (flexion–distraction):
 - Distraction of posterior and middle columns with anterior column intact
 - Typically caused by lap belts used without shoulder harness
 - Type A: Through bone
 - Type B: Primarily ligamentous
 - Type C: Disruption of bone through middle column
 - Type D: Through ligaments and disc with no middle column fracture
- Fracture dislocations:
 - Failure of all 3 columns following compression, tension, rotation, or shear forces
 - Type A: Flexion–rotation; fall from height
 - Type B: Shear-violent force across long axis of trunk
 - Type C: Flexion–distraction; bilateral facet dislocation

ETIOLOGY

- Thoracic spine is rigid owing to the support of the rib cage and the costovertebral articulations:
 - The spinal canal is narrowest in the thoracic spine
- Traumatic thoracic spine fractures require enormous forces. Motor vehicle and motorcycle collisions, pedestrian's struck, and falls (particularly from height > 10 ft) account for most fractures:
 - A small percentage are caused by penetrating injuries (see “Spinal Cord Syndromes”)
 - 50% of all spinal fractures and 40% of all spinal cord injuries occur at the thoracolumbar junction (T11–L2)

Pediatric Considerations

- Suspect child abuse if thoracic spine injury without clear history of motor vehicle trauma.
- Posterior rib fractures raise index of suspicion for abuse and require closer survey of thoracic spine and entire body for occult injury.

Geriatric Considerations

Increased brittleness of bones in elderly (>65 yr) predispose to fractures with less

severe mechanism, falls from lesser height.

DIAGNOSIS

SIGNS AND SYMPTOMS

- Significant force is required to produce thoracic vertebral fractures.
- Pain at the fracture site or impingement of nearby structures by bone fragments.
- Because of the stabilizing influence of the rib cage, a tremendous amount of force is needed to cause thoracic spine dislocations:
 - Concomitant internal injury should be suspected
 - Thoracic spine fracture–dislocation is less common than thoracolumbar fracture–dislocation but has higher incidence of neurologic impairment
 - Spinal injury at another anatomic level should heighten suspicion for thoracic injury and vice versa
- Common signs and symptoms:
 - Localized soft tissue defect
 - Ecchymosis or hematoma:
 - Scapular contusions
 - Step-offs, deformity, or widening of disc space (more specific)
 - Pain or tenderness (more sensitive):
 - Localized—pain and tenderness over spinous process
 - Referred—paraspinal, anterior chest, or abdomen
 - Paraspinal muscle spasm
 - Paresthesia or dysesthesia
 - Weakness (focal or global)
 - Distal areflexia, flaccid plegia
 - Bowel or bladder incontinence
 - Priapism
 - Loss of temperature control
 - Spinal shock—hypotension with bradycardia

History

- Mechanism of injury
- Comorbidities

ESSENTIAL WORKUP

- Rapid evaluation of ABCs.
- Primary and secondary trauma survey.
- Detailed neurologic exam, including rectal tone and perianal sensation.
- Thorough spine exam for deformity or tenderness.
- Any midline tenderness elicited on exam, distracting injury, altered mental status or intoxication with concerning mechanism mandates plain film spine

radiography.

- If fracture present, determine whether it is stable or unstable.
- Assess for bulbocavernosus reflex in spinal shock.

DIAGNOSIS TESTS & INTERPRETATION

Imaging

- Midline pain or tenderness, significant motor vehicle accident, or falls from height are indications for screening with anteroposterior and lateral plain film views of the spine.
- Thin-cut CT scanning is indicated in any patient with evidence of spinal fracture on plain films and patients with normal plain films and significant pain or tenderness and mechanism for severe injury. Patients with mediastinal widening on plain film should also have evaluation for thoracic spine injury or aortic injury.
- Any finding of a fracture anywhere in the thoracic spine mandate imaging of the entire spine with plain radiographs at a minimum as 10% patients will have an additional fracture.
- Data from the CT of the chest/abdomen/pelvis are increasingly being reformatted to clear the thoracolumbar spine in trauma patients:
 - _ More sensitive than plain radiographs without additional cost or radiation
- MRI for further evaluation of suspected spinal cord injury, compression, or ligamentous tear.

DIFFERENTIAL DIAGNOSIS

- Arthritis (degenerative and rheumatoid)
- Ankylosing spondylitis
- Spina bifida
- Congenital malformation
- Degenerative disc disease
- Neoplasm
- Pathologic fracture:
 - _ Osteoporosis
 - _ Benign or malignant bone tumors



TREATMENT

PRE HOSPITAL

- If the patient's positioning initially prevents placement of a long spinal board, a short board should be placed until the patient is fully extricated.
- Patients with neurologic deficit should be transported directly to a trauma center.

INITIAL STABILIZATION/THERAPY

- Manage airway and resuscitate as indicated:
 - Airway intervention should be done with inline cervical immobilization
 - Identify hypotension that may be secondary to hemorrhage vs. neurogenic
 - Patients with hypotension in setting of trauma should be treated as hypovolemic from hemorrhage until proven otherwise
 - Consider fluid resuscitation with crystalloid followed by blood products if indicated
- Preserve residual spinal cord function and prevent further injury by stabilizing the spine.

ED TREATMENT/PROCEDURES

- Perform all needed resuscitation and diagnostic tests with the patient in full spinal immobilization. This does not require the use of the spinal board which should be removed promptly on arrival during the initial exam.
- If spinal cord injury is suspected, consider the administration of high-dose steroids and consult a spine surgeon.
- If spinal fracture or ligamentous injury is suspected without neurologic impairment, arrange CT or MRI scanning while consulting neurosurgery or orthopedic surgery.
- Pain control should be administered as soon as possible; NSAIDs, opiates, and benzodiazepines are the mainstays of treatment.
- Neurogenic hypotension presents with bradycardia or normal heart rate and patient will be warm from peripheral vasodilation. This is in contrast to the tachycardia and cool extremities seen with hypovolemic shock:
 - Neurogenic hypotension should be treated with crystalloid bolus but may require vasopressors

MEDICATION

- High-dose steroid administration is rapidly falling out of favor due to lack of evidence supporting use and risk of untoward effects of steroids.
- If given, must be within 8 hr of injury as indicated by regional/hospital protocol.
- Methylprednisolone: 30 mg/kg IV bolus over 15 min followed 45 min later by a maintenance infusion of 5.4 mg/kg/h for the next 23 hr if started within 3 hr of injury; consider continuing for 48 hr if started 3–8 hr after injury.
- High-dose steroid treatment not recommended > 8 hr after injury.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients with significant spinal cord or column injury should be treated at a

regional trauma center.

- Unstable spinal column injury
- Spinal cord or root injury
- Ileus
- Pain control
- Concomitant traumatic injury
- ICU-level care based on severity of injuries

Discharge Criteria

Stable minor fractures after orthopedic or neurosurgical evaluation.

FOLLOW-UP RECOMMENDATIONS

Outpatient neurosurgical or orthopedic follow-up as indicated after appropriate ED or inpatient evaluation and treatment.

PEARLS AND PITFALLS

- Suspect and evaluate for thoracic spine injury in any trauma patient.
- CT evaluation is indicated for any patient with significant mechanism, pain, or tenderness; distracting injury or injury at another spinal level; intoxication or altered mental status.
- Maintain spinal immobilization until cleared by radiologic and clinical exam.
- Early consultation with spine surgeon if presence of fracture, neurologic deficit, or instability.
- Treatment with high-dose steroids is currently an area of controversy. Begin treatment within 8 hr of injury if initiating high-dose steroid protocol.

ADDITIONAL READING

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- Inaba K, Munera F, McKenney M, et al. Visceral torso computed tomography for clearance of the thoracolumbar spine in trauma: A review of the literature. *J Trauma.* 2006;60:915–920.



CODES

ICD9

- 805.2 Closed fracture of dorsal [thoracic] vertebra without mention of spinal cord injury
- 839.21 Closed dislocation, thoracic vertebra
- 847.1 Sprain of thoracic

ICD10

- S22.009A Unsp fracture of unsp thoracic vertebra, init for clos fx
- S23.101A Dislocation of unspecified thoracic vertebra, initial encounter
- S23.3XXA Sprain of ligaments of thoracic spine, initial encounter

SPLENIC INJURY

Albert S. Jin



BASICS

DESCRIPTION

- The spleen is formed by reticular and lymphatic tissue and is the largest lymph organ.
- The spleen lies posterolaterally in the left upper quadrant (LUQ) between the fundus of the stomach and the diaphragm.

ETIOLOGY

- The spleen is the most commonly injured intra-abdominal organ:
 - _ In nearly 2/3 of cases, it is the only damaged intraperitoneal structure
 - _ Blunt mechanisms are more common
- Motor vehicle accidents (auto–auto, pedestrian–auto) are the major cause (50–75%), followed by blows to the abdomen (15%) and falls (6–9%)
- Mechanism of injury and kinematics are important factors in evaluating patients for possible splenic injury.
- Splenic injuries are graded by type and severity of injury [American Association for the Surgery of Trauma (AAST) criteria]:
 - _ Grade I:
 - Hematoma: Subcapsular, <10% surface area
 - Laceration: Capsular tear, <1 cm in parenchymal depth
 - _ Grade II:
 - Hematoma: Subcapsular, 10–50% surface area; intraparenchymal, <5 cm in diameter
 - Laceration: Capsular tear, 1–3 cm in parenchymal depth and not involving a trabecular vessel
 - _ Grade III:
 - Hematoma: Subcapsular, >50% surface area or expanding, ruptured subcapsular or parenchymal hematoma; intraparenchymal hematoma, ≥5 cm or expanding
 - Laceration: >3 cm in parenchymal depth or involving the trabecular vessels
 - _ Grade IV:
 - Laceration: Involving the segmental or hilar vessels and producing major devascularization (>25% of spleen)
 - _ Grade V:
 - Laceration: Completely shattered spleen
 - Vascular: Hilar vascular injury that devascularizes the spleen

Pediatric Considerations

- Poorly developed musculature and relatively smaller anteroposterior diameter increase the vulnerability of abdominal contents to compressive forces.
- Rib cage is extremely compliant and less prone to fracture in children but provides only partial protection against splenic injury.
- Splenic capsule in children is relatively thicker than that of an adult; parenchyma of spleen seems to contain more smooth muscle than in adults.
- Significant abdominal injury occurs in only about 5% of child abuse cases but is the 2nd most common cause of death after head injury.

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- In blunt trauma, note the type and direction (horizontal or vertical) of any deceleration or compressive forces:
 - Injuries are caused by compression of the spleen between the anterior abdominal wall and the posterior thoracic cage or vertebra (e.g., lap-belt restraints).
- In penetrating trauma, note the characteristic of the weapon (type and caliber), distance from the weapon, or the type and length of knife or impaling object:
 - Injuries result from a combination of the kinetic energy and shear forces of penetration.

Physical-Exam

- Systemic signs from acute blood loss:
 - Syncope, dizziness, weakness, confusion
 - Hypotension or shock
- Local signs:
 - LUQ abdominal tenderness
 - Palpable tender mass in LUQ (Balance sign)
 - Referred pain to the left shoulder (Kehr sign)
 - Abdominal distention, rigidity, rebound tenderness, involuntary guarding
- Contusions, abrasions, or penetrating wounds to the chest, flank, or abdomen may indicate underlying splenic injury.
- Fractures of lower left ribs are commonly seen in association with splenic injuries.

Pediatric Considerations

Age-related difficulties in communication, fear-induced uncooperative behavior, or a concomitant head injury make clinical exam less reliable.

ESSENTIAL WORKUP

- History and physical exam are neither specific nor sensitive for splenic injury.
- Adjunctive imaging studies are required.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- No hematologic lab studies are specific for diagnosis of injury to the spleen.
- Obtain baseline hemoglobin, type and cross-match, and chemistries.

Imaging

- Plain abdominal radiographs:
 - Too nonspecific to be of value
 - CXR findings suggestive for splenic injury:
 - Left lower rib fracture(s)
 - Elevation of left hemidiaphragm
 - Medial displacement of gastric bubble (Balance sign)
 - Left pleural effusion
- Ultrasound:
 - Routinely performed at bedside in trauma patients as part of focused assessment with sonography (FAST)
 - Primary role is detecting free intraperitoneal blood, which may suggest splenic injury
 - Does not image solid parenchymal damage well
 - Technically compromised by uncooperative patient, obesity, substantial bowel gas, and subcutaneous air
- CT scan:
 - Noncontrast CT is procedure of choice in stable patient due to speed and accessibility
 - Depicts the presence and extent of splenic injury and adjacent organs, including the retroperitoneum
 - Provides the most specific information in patients stable enough to go to the CT scanner
- MRI:
 - May be applicable to subset of hemodynamically stable patients who cannot undergo CT scan (e.g., allergic to IV contrast)
- Angiography:
 - Has been added to the diagnostic and treatment options for selected cases

Diagnostic Procedures/Surgery

- Diagnostic peritoneal lavage (DPL):
 - Extremely sensitive for the presence of hemoperitoneum although

- nonspecific for source of bleeding and does not evaluate retroperitoneum
- Largely replaced by the FAST exam in most major trauma centers.

DIFFERENTIAL DIAGNOSIS

- Intraabdominal organ injury, especially liver
- Injury to retroperitoneal structures
- Thoracic injury



TREATMENT

PRE HOSPITAL

- Obtain details of injury from pre-hospital providers.
- IV access
- Penetrating wounds or evisceration should be covered with sterile dressings.

INITIAL STABILIZATION/THERAPY

- Airway management (including C-spine immobilization)
- Standard Advanced Trauma Life Support (ATLS) resuscitation measures:
 - Adequate IV access, including central lines and cutdowns, as dictated by the patient's hemodynamic status
 - Fluid resuscitation, initially with 2 L of crystalloid (NS or lactated Ringer solution), followed by blood products as needed

ED TREATMENT/PROCEDURES

- Immediate laparotomy may be appropriate in the acutely injured and hemodynamically unstable patient with presumed hemoperitoneum and splenic injury.
- Most patients with acute splenic injury either are hemodynamically stable or stabilize rapidly with relatively small amounts of fluid resuscitation.
- Adjunctive diagnostic procedures supplementing the physical exam should be performed early in the evaluation, followed by laparotomy when indicated by positive diagnostic findings.
- Gunshot wounds to the anterior abdomen are routinely explored in the OR.
- Stab wounds can be managed by local wound exploration, followed by US or DPL when intraabdominal penetration is suspected.
- Operative vs. nonoperative management:
 - Patients with signs and symptoms of intraabdominal hemorrhage, those with operative indications based on imaging//diagnostic procedures, and those who fail nonoperative management should undergo laparotomy.
 - Angiographic embolization is an option in hemodynamically stable patient
 - Splenectomy vs. splenic salvage depends on the grade of splenic injury.
 - >70% of all stable patients are currently being treated via nonoperative management:

- Hemodynamic stability
- Negative abdominal exam
- Absence of contrast extravasation on CT
- Absence of other clear indications for exploratory laparotomy
- Absence of associated health conditions that carry an increased risk for bleeding (e.g., coagulopathy, hepatic failure, anticoagulant use, coagulation factor deficiency)
- Injury grades I–III

Geriatric Considerations

- Patients > 55 yr should be considered for operative management due to decreased physical tolerance to traumatic insult (splenic capsule thins with age) and reduced physiologic reserve.
- Embolization is relatively contraindicated in patients > 55 yr due to higher failure rates in these patients.

Pediatric Considerations

- Nonoperative management of splenic injuries is considered safe:
 - _ Concerns for overwhelming postsplenectomy infection/sepsis



FOLLOW-UP

DISPOSITION

Admission Criteria

All patients with splenic injury require hospitalization for definitive laparotomy or observation with serial abdominal exams, serial hematocrit determinations, and bed rest.

Discharge Criteria

Only asymptomatic patients objectively demonstrated not to have splenic or other traumatic injury may be discharged.

ADDITIONAL READING

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- Gomez D, Haas B, Al-Ali K, et al. Controversies in the management of splenic trauma. *Injury.* 2012;43:55–61.
- Izu BS, Ryan M, Markert RJ, et al. Impact of splenic injury guidelines on hospital stay and charges in patients with isolated splenic injury. *Surgery.*

2009;146(4):787–791.

- St Peter SD, Keckler SJ, Spilde TL, et al. Justification for an abbreviated protocol in the management of blunt spleen and liver injury in children. *J Pediatr Surg*. 2008;43:191–194.
- Tinkoff G, Esposito TJ, Reed J, et al. American Association for the Surgery of Trauma Organ Injury Scale I: Spleen, liver, and kidney, validation based on the National Trauma Data Bank. *J Am Coll Surg*. 2008;207(5):646–655.



CODES

ICD9

- 865.00 Injury to spleen without mention of open wound into cavity, unspecified injury
- 865.01 Injury to spleen without mention of open wound into cavity, hematoma without rupture of capsule
- 865.02 Injury to spleen without mention of open wound into cavity, capsular tears, without major disruption of parenchyma

ICD10

- S36.00XA Unspecified injury of spleen, initial encounter
- S36.029A Unspecified contusion of spleen, initial encounter
- S36.039A Unspecified laceration of spleen, initial encounter

SPONDYLOLYSIS/SPONDYLOLISTHESIS

Lisa G. Lowe Hiller



BASICS

DESCRIPTION

- Spondylolysis:
 - Bony defect at the pars interarticularis (the isthmus of bone between the superior and inferior facets)
 - Can be unilateral or bilateral
 - Bilateral form has a much higher likelihood of slippage or spondylolisthesis than the unilateral form.
- Spondylolisthesis:
 - The slipping forward of 1 vertebra upon another
 - Spondylolysis can contribute to spondylolisthesis, which is noted in ~5% of the population. It is 2–4 times more common in males.
 - Of those with spondylolysis, 50% will have some degree of spondylolisthesis develop during their lifetime, and 50% of those will be symptomatic:
 - Literature does not associate athletic activity with increased slippage.
 - Spondylolisthesis predisposes to nerve root impingement and frequently sciatica.
- Classification:
 - Type 1—dysplastic: Congenital defect of the neural arch or intra-articular facets is often associated with spina bifida occulta
 - Type 2—isthmic: Stress fracture from repetitive microtrauma through the neural arch
 - Type 3—degenerative: Long-standing segmental instability
 - Type 4—traumatic
 - Type 5—pathologic: Generalized or focal bone disease
 - Spondylolisthesis is divided into 4 grades based on degree of slippage (Meyerding grading system):
 - Grade I: Up to 25% of the vertebral body width
 - Grade II: 26–50% of vertebral body width
 - Grade III: 51–75% of vertebral body width
 - Grade IV: 76–100% of vertebral body width
 - The most common location for spondylolisthesis is L5 displaced on the sacrum (85–95%), followed by L4 on L5.

Pediatric Considerations

- Spondylolysis is one of the most common causes of serious low back pain in children, although it is most often asymptomatic.

- Symptoms most often present during adolescent growth spurt from age 10–15 yr.
- Seen commonly in athletic teens; particularly in sports involving back hyperextension (e.g., gymnastics, diving, football).
- Acute symptoms are related to trauma.

ETIOLOGY

Unknown; theories include congenital pars anomalies, alterations in bone density, and recurrent subclinical stress injury.

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Onset often gradual, unless traumatic
- Often associated with feeling of stiffness or spasm in paravertebral muscles
- Pain in the back and proximal legs aggravated by standing and walking
- Sitting or forward bending relieves pain.
- Pain occurs after varying amounts of exercise, with standing, or with coughing:
 - Aggravating factors can include repetitive hyperextending movements.
 - Alleviating factors can include rest, although the course is variable and slow and usually requires sitting or stooping positions.
- Systemic/neurologic symptoms: Minimal, unless there is significant trauma or “slip.”

Physical-Exam

- Hyperlordotic posture:
 - Trunk may appear shortened.
 - Rib cage approaches iliac crests.
- Hamstring tightness:
 - Knees flexed to allow patient to stand upright
- Only “typical” finding is 1-legged hyperextension:
 - Standing on 1 leg and leaning backward reproduces pain on ipsilateral side.
- Palpation may reveal step-off with a prominent spinous process of L5 in significant spondylolisthesis.
- Neurologic exam is usually normal:
 - If abnormal, pain and sensorimotor loss is in a dermatomal distribution.
 - Consider herniation or spondylolisthesis.

Pediatric Considerations

- Spondylolysis in a child <10 yr is rare; these patients should be watched for the following:

- Constant pain lasting several weeks
- Pain occurring spontaneously at night
- Pain that interferes repeatedly with school, play, or sports
- Pain associated with marked stiffness, limitation of motion, fever, or neurologic signs
- Pain at the lumbosacral junction

DIAGNOSIS TESTS & INTERPRETATION

Lab

There are no required lab studies.

Imaging

- Lumbosacral spine radiographs:
 - Lateral and oblique radiographs of spine most helpful.
 - Spondylolysis will manifest as a radiolucent defect in the pars interarticularis, visible as a “collar” or “broken neck” on the oblique view “Scottie dog”
 - Secondary radiographic signs may include sclerosis of the contralateral pedicle and spina bifida occulta at the level of the spondylolysis.
 - Majority (80–95%) found at L5–S1 level, 15% at L4–L5.
 - Spondylolisthesis will manifest as forward slipping of one vertebral body on another (seen on lateral view).
- Single photon emission computed tomography (SPECT)—better specificity for linking back pain to spondylolysis.
- CT scan:
 - Pathology more clearly demonstrated than on plain films
 - Can identify other spinal pathology
 - Plays an important role for orthopedics in management decisions through identification of new stress fractures and healing of old stress fractures.
 - If a CT scan is obtained in the ED, sagittal reconstructions should be performed and the CT scanner should be at minimum a 16-slice scanner.
 - Outpatient evaluation unless history of recent trauma.
- MRI—exact role not yet clarified in literature:
 - Useful for defining nerve root impingement and central canal and neuroforaminal narrowing.
 - May be useful in the assessment of acuity of abnormality.
 - Can identify alternate pathologic diagnoses.

Pediatric Considerations

- Lower threshold for ordering imaging studies.
- Progressive slipping more likely to occur than in adults.

DIFFERENTIAL DIAGNOSIS

- Tuberculosis (Pott disease)
- Discitis
- Bone or spinal cord tumor
- Pyelonephritis
- Retroperitoneal infection
- Injury to muscles or joints of back
- Congenital hip dislocation
- Rickets
- Ruptured intervertebral disc
- Vascular claudication
- Osteomyelitis
- Osteoid osteoma
- Aortic aneurysm



TREATMENT

PRE HOSPITAL

Spinal precautions are not needed unless there is a history of recent trauma.

INITIAL STABILIZATION/THERAPY

Vigorous attempts at traction should not be pursued.

ED TREATMENT/PROCEDURES

- Pain control and muscle relaxants as clinically needed
- Supportive therapy if symptoms are mild
- Restrict activities if repetitive trauma is likely aggravating cause (e.g., sports) for 3–6 wk, followed by reintroduction of activity when asymptomatic.
- Consider antilordotic braces (controversial) or physical therapy.
- Orthopedic consult or referral if symptoms are moderate to severe or unresponsive to supportive care
- Surgical intervention typically consists of spinal fusion in the flexed position:
 - 50% of symptomatic patients with spondylolisthesis may require surgery.
- All symptomatic patients with grade III or IV spondylolisthesis should probably undergo surgery.
- Exercises are not of proven benefit.

Pediatric Considerations

- Activity restriction is not necessary if minimal or no symptoms.
- Literature suggests good outcome for young athletes with conservative treatment.

MEDICATION

- Muscle relaxants:
 - E.g.—methocarbamol: 1,000–1,500 mg PO QID (peds: Safety and effectiveness for children < 12 yr of age not established)
 - Diazepam: 2–10 mg PO TID–QID
 - Cyclobenzaprine: 5–10 mg PO TID (peds: Safe for ages > 15 yr old)
- NSAIDs:
 - E.g.—ibuprofen: 200–800 mg PO TID–QID (peds: 5–10 mg/kg PO q6h)
- Opioids (doses can vary on oral medications):
 - Example—morphine sulfate: 0.1 mg/kg up to 2–4 mg increments IV.
 - Acetaminophen/hydrocodone: 5/500 mg 1–2 tabs PO QID; do not exceed acetaminophen 4 g/24 h (peds: Do not exceed 5 doses of 10–15 mg/kg acetaminophen in 24 hr)
 - Acetaminophen/oxycodone: 5/325 mg 1–2 tabs PO QID; do not exceed acetaminophen 4 g/24 h (peds: Do not exceed 5 doses of 10–15 mg/kg acetaminophen in 24 hr)
 - Acetaminophen/codeine: 300/30 mg 1–2 tabs PO QID (peds: 0.5–1 mg/kg codeine PO q4–6h; max. 60 mg/dose codeine; 1 g/dose, 75 mg/kg/d up to 4 g/d > 3 yr old); do not exceed acetaminophen 4 g/24 h (peds: Do not exceed 5 doses of 10–15 mg/kg acetaminophen in 24 hr)



FOLLOW-UP

DISPOSITION

Admission Criteria

- Inability to walk
- Inability to cope at home due to pain or social situation
- New or progressive neurologic deficit

Discharge Criteria

- Orthopedic follow-up arranged
- Social support system in place
- Pain control
- Patient education

Pediatric Considerations

Close follow-up is mandatory.

ADDITIONAL READING

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- Tsirikos AI, Garrido EG. Spondylolysis and spondylolisthesis in children and adolescents. *J Bone Joint Surg Br.* 2010;92(6):751–759. doi:10.1302/0301-620X.92B6.23014.



CODES

ICD9

- 738.4 Acquired spondylolisthesis
- 756.11 Spondylolysis, lumbosacral region
- 756.12 Spondylolisthesis

ICD10

- M43.00 Spondylolysis, site unspecified
- M43.10 Spondylolisthesis, site unspecified
- M43.16 Spondylolisthesis, lumbar region

SPONTANEOUS BACTERIAL PERITONITIS

Alison Foster-Goldman • Christopher T. Richards

BASICS

DESCRIPTION

- Infection of ascites fluid without an evident intra-abdominal surgically treatable source:
 - Ascites fluid polymorphonuclear leukocyte count (PMN) $> 250/\text{mL}$ with a positive bacterial peritoneal fluid culture
- Must be distinguished from secondary bacterial peritonitis:
 - Nonsurgical management of secondary bacterial peritonitis carries 100% mortality.
 - Surgical management of spontaneous bacterial peritonitis (SBP) carries 80% mortality
- Up to 30% yearly incidence of SBP in patients with ascites

ETIOLOGY

- Mechanism:
 - Portal hypertension causes translocation of intestinal bacteria through edematous gut mucosa to the peritoneal cavity
 - Variceal bleeding increases the risk of SBP due to a compromised barrier between the GI tract and blood stream
 - Transient bacteremia with low serum complement
 - Decreased host defense mechanisms
 - Impaired activity of reticuloendothelial system phagocytosis and opsonization
 - Can also seed ascitic fluid via bacteremia from infections outside of the gut
- Usually seen in the setting of cirrhosis:
 - Rare in other conditions causing ascites (nephrotic syndrome or CHF)
- Predominant organisms:
 - 63% aerobic gram-negative (*Escherichia coli*, *Klebsiella*, others)
 - 15% gram-positive (Streptococci)
 - 6–10% enterococci
 - $< 1\%$ anaerobic
- Gram-positives account for 50% of cases in patients who are on prophylactic therapy with fluoroquinolones.

DIAGNOSIS

SIGNS AND SYMPTOMS

Up to 30% of patients with SBP have no signs or symptoms of infection.

History

- Abdominal pain: Diffuse, constant, often very mild
- Fever, chills
- Diarrhea from bacterial overgrowth
- Worsening ascites
- Altered mental status
- Fatigue, myalgias

Physical-Exam

- Fever is the most common sign:
 - _ A lower threshold for fever ($> 37.8^{\circ}\text{C}$ or $> 100^{\circ}\text{F}$) is maintained for cirrhotic patients owing to baseline hypothermia
 - _ 80% of patients with SBP have fevers and chills
- Altered mental status
- Ascites
- Abdominal tenderness:
 - _ Development of a rigid abdomen may not occur because of the separation of visceral and parietal pleura due to ascites

ESSENTIAL WORKUP

- Paracentesis is the mainstay of diagnosis unless patient has peritoneal dialysis
- Coagulopathy does not have to be corrected before the procedure (except for platelets $< 20,000$)
- Procedure:
 - _ Use ultrasound guidance when available
 - _ Location (with patient supine):
 - 3–5 cm cephalad and medial to anterosuperior iliac spine, lateral to the rectus sheath OR
 - 2 cm caudad to the umbilicus (ensure bladder emptying beforehand)
 - _ 40–50 mL should be aspirated, then change needles to avoid contamination:
 - 10 mL for each culture bottle
 - 10 mL for cell count, chemistries, Gram stain (lithium–heparin tube, EDTA tube, and sterile container)
 - _ Inoculate culture bottles with peritoneal fluid immediately at the bedside

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Routine ascitic fluid assays:
 - _ Cell count and differential:
 - Count bands as PMNs

- Total protein
- Albumin
- Culture
- Gram stain
- Optional fluid assays:
 - o Glucose
 - o LDH (from lysed PMNs)
 - o Amylase
- Characteristics of ascitic fluid consistent with SBP:
 - PMNs $> 250/\text{mm}^3$
 - Diagnosis suggested when:
 - o WBC $> 1,000/\text{mm}^3$
 - o WBC $> 250/\text{mm}^3$ with $> 50\%$ PMNs
 - Total protein $< 1 \text{ g/dL}$
 - pH < 7.34
 - Normal amylase
 - Positive culture:
 - o Only 30–50% of cultures become positive; this rate increases with high volume bedside inoculation of culture bottles
 - Positive Gram stain
 - Glucose $< 50 \text{ mg/dL}$
 - Ascites LDH $>$ serum LDH
 - Lactoferrin > 242 shows promise as marker for SBP
 - Serum–ascites albumin gradient $> 1.1 \text{ g/dL}$ consistent with portal hypertension
 - If hemorrhagic ascites ($> 10,000 \text{ RBC}/\text{mm}^3$), subtract $1 \text{ PMN}/\text{mm}^3$ for every $250 \text{ RBC}/\text{mm}^3$ in ascites fluid interpretation
- Blood tests (usually reflect underlying disease):
 - CBC with differential
 - Basic metabolic panel
 - PT/PTT
 - LFTs (including albumin)
 - Blood cultures
 - UA and culture

Imaging

- Abdominal ultrasound:
 - Confirms presence of ascites
 - Helps guide paracentesis
- Chest radiograph
- Abdominal radiographs: Flat-plate and upright to evaluate for perforation or

obstruction

- Water-soluble contrast CT if suspect secondary bacterial peritonitis

Diagnostic Procedures/Surgery

Surgery consultation to consider exploratory laparotomy if free air on x-ray or extravasation of contrast on CT

DIFFERENTIAL DIAGNOSIS

- Secondary bacterial peritonitis:
 - _ Due to perforation or abscess
 - _ Polymicrobial Gram stain or 2 of the following:
 - Ascites total protein > 1 g/dL
 - Ascites glucose < 50 mg/dL
 - Ascites LDH > 1/2 upper limit of normal serum LDH or LDH > 225
 - _ Orange ascites with bilirubin > 6 mg/dL suggests ruptured gallbladder
- Acute hepatitis:
 - _ Fever, leukocytosis, abdominal pain \pm ascites
 - _ Ascites PMNs < 250/mm³
- Culture-negative neutrocytic ascites:
 - _ Ascites PMNs > 250/mL, culture negative
- Monomicrobial non-neutrocytic bacterascites:
 - _ Due to colonization phase of SBP
 - _ Ascites PMNs < 250/mm³, monomicrobial culture
 - _ Treated like SBP if symptomatic
- Polymicrobial bacterascites:
 - _ Due to accidental gut perforation (1 in 1,000 paracenteses)
 - _ Ascites PMNs < 250/mm³, polymicrobial culture
- Pancreatitis:
 - _ Elevated ascites amylase
- Peritoneal carcinomatosis or tuberculous peritonitis:
 - _ Secondary bacterial peritonitis criteria with non-PMN predominance and lack of fever



TREATMENT

PRE HOSPITAL

- IV fluids for hypotension
- Blood glucose for altered mental status
- Supplemental oxygen for respiratory complaints

INITIAL STABILIZATION/THERAPY

- ABCs

- Prompt antibiotic treatment and IV fluids for septic shock

ED TREATMENT/PROCEDURES

- Administer platelets before paracentesis only if platelet count is $< 20,000/\text{mm}^3$
- Give empiric antibiotics immediately after paracentesis for:
 - Ascites PMNs $> 250/\text{mm}^3$ or
 - Temperature $> 37.8^\circ\text{C}$ or
 - Altered mental status or
 - Abdominal pain/tenderness or
 - Clinical features most consistent with SBP
- Antibiotic options:
 - Ceftriaxone or cefotaxime
 - Ampicillin–sulbactam, piperacillin–tazobactam or aztreonam
 - Avoid aminoglycosides, fluoroquinolones
 - Add metronidazole for secondary bacterial peritonitis
- IV albumin is helpful in preventing renal impairment and reducing mortality in diagnosed SBP

Prognosis

- In-hospital noninfection–related mortality is 20%
- Can be precursor to hepatorenal syndrome
- 1- and 6-mo mortality rates after an episode of SBP are 32% and 69%, respectively

MEDICATION

First Line

- Cefotaxime: 2 g IV q8h
- Albumin for high-risk patients: 1.5 g/kg IV on day 1 and 1 g/kg IV on day 3

Second Line

- Ceftriaxone: 2 g IV q8h
- Piperacillin–tazobactam: 3.375 g IV q6h
- Ampicillin–sulbactam: 1.5–3 g IM/IV q6h
- Aztreonam: 0.5–2 g IM/IV q6–12h



FOLLOW-UP

DISPOSITION

Admission Criteria

- Admit all patients for IV antibiotics and gastroenterology consultation
- ICU admission for septic shock or severe hepatic encephalopathy

Discharge Criteria

- All patients with suspected or known SBP should be admitted.
- If patient refuses admission and has no signs of shock, encephalopathy, azotemia, or GI bleeding, a dose of IV ceftriaxone and a course of oral fluoroquinolones followed by close follow-up may be considered

Issues for Referral

- Hepatology and gastroenterology referral may be indicated
- Prophylaxis with norfloxacin or trimethoprim/sulfamethoxazole

ALERT

Infections related to continuous abdominal peritoneal dialysis:

- Symptoms: Cloudy peritoneal fluid (90%), abdominal pain (80%), and fever (50%)
- Signs: Abdominal tenderness 70%
- Diagnosis: Peritoneal WBCs > 100/mL with > 50% PMNs and positive Gram stain or culture:
 - _ Fluid should be accessed by trained personnel
- Microbiology:
 - _ > 50% of cases are due to gram-positives, most commonly staphylococci
 - _ *E. coli* is an *uncommon* cause of peritonitis in patients with chronic ambulant peritoneal dialysis
- Treatment:
 - _ Antibiotics are given through the intraperitoneal (IP) route
 - _ 1st choice: Cefazolin (1 g IP per day) + ceftazidime (1 g IP per day)
 - _ Vancomycin (2 g IP every week) is an alternative to cefazolin
 - _ Amikacin 2 mg/kg/day IP

FOLLOW-UP RECOMMENDATIONS

Gastroenterology or PCP follow-up for patients with SBP

PEARLS AND PITFALLS

- Rule out secondary bacterial peritonitis first
- Bedside inoculation of blood culture bottles with ascitic fluid increases culture yield
- Maintain high suspicion for SBP, since many patients are asymptomatic

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Ascites
- GI Bleeding
- Hepatitis
- Hepatorenal Syndrome
- Abdominal Pain

We wish to acknowledge the previous authors of this chapter for their contributions on this topic: Michael Schmidt, Amer Aldeen, and Lucas Roseire.



CODES

ICD9

567.23 Spontaneous bacterial peritonitis

ICD10

K65.2 Spontaneous bacterial peritonitis

SPOROTRICHOSIS

Matthew Hinderaker • Adam Kellogg



BASICS

DESCRIPTION

- Lymphocutaneous:
 - Most common form
 - Inoculation of fungus (*Sporothrix schenckii*) into skin/soft tissue
 - Disease with or without hematogenous spread after traumatic inoculation with soil or plant material
 - Secondary to animal bites/scratches, especially from cats, trauma
 - Increased risk: Farmers, gardeners, landscapers, forestry workers
- Pulmonary:
 - Inhalation of conidia aerosolized from soil/plant decay
 - Increased risk: Alcoholics, diabetics, COPD, steroid users
- Multifocal extracutaneous:
 - Cutaneous inoculation and hematologic spread
 - Increased risk: HIV/immunosuppressed patients

ETIOLOGY

- Fungal infection caused by *S. schenckii*:
- Dimorphic fungus
- Occurs as mold on decaying vegetation, moss, and soil in temperate and tropical environments
- Animal vectors, notably cats and armadillos



DIAGNOSIS

SIGNS AND SYMPTOMS

- Several clinical manifestations/syndromes
- Determined by mode of inoculation and host factors
- Lymphocutaneous:
 - Initial lesions appear days to weeks after inoculation
 - Begin as papules, become nodular, often ulcerate:
 - Distal extremities more commonly involved
 - Size: Millimeters to 4 cm
 - Pain absent or mild
 - Drainage is nonpurulent
 - Systemic symptoms usually absent
 - Secondary nodular lesions develop along lymphatics draining the original

- site.
- May wax and wane over years if untreated
- Fixed cutaneous:
 - Plaque-like or verrucous lesion at the site of inoculation (typically face and extremities)
 - Ulceration uncommon
 - Do not manifest lymphangitic progression
 - Common in endemic regions of South America
- Extracutaneous:
 - Osteoarticular:
 - Secondary to local or hematologic inoculation
 - Septic arthritis more common than osteomyelitis
 - Joint inflammation, effusion, and pain
 - Single or multiple joint involvement of extremities: Knee, elbow, wrist, ankle
 - Indolent onset, few systemic symptoms
 - Tenosynovitis, septic arthritis, bursitis, nerve entrapment syndrome
 - Usually poor outcome due to delayed diagnosis
- Pulmonary:
 - Syndrome resembles mycobacterial infection (TB)
 - Fever, weight loss, fatigue, night sweats
 - Productive cough, hemoptysis, dyspnea
 - Uniformly fatal if untreated
- Multifocal extracutaneous (disseminated):
 - Low-grade fever, weight loss
 - Diffuse cutaneous lesions
 - Arthritis/osteolytic lesions/parenchymal involvement
 - Chronic lymphocytic meningitis
 - Ocular adnexa, endophthalmitis
 - Genitourinary, sinuses
 - Can be fatal if untreated
 - Often occurs in immunocompromised host

History

- Activity with exposure to soil, moss, organic material, or to cats in endemic areas
- Fixed cutaneous or lymphocutaneous: Healthy host
- Disseminated/extracutaneous: Diabetics, COPD, HIV/AIDS

Physical-Exam

- Fixed cutaneous/lymphocutaneous: Lesions found on exam
- Disseminated: Nonspecific findings

ESSENTIAL WORKUP

Diagnosis dependent on isolation *S. schenckii* from site of infection:

- Culture from aspirated material, tissue biopsy, or sputum

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Blood tests not indicated with cutaneous disease
- Cultures of sputum, synovial fluid, CSF, blood as indicated by extracutaneous manifestations
- No reliable serologic assays available

Imaging

- Pulmonary:
 - Chest radiograph reveals cavitary lesions
- Extracutaneous/disseminated:
 - Consider bone scan in immunocompromised host.

Diagnostic Procedures/Surgery

- Lymphocutaneous/fixed cutaneous:
 - Biopsy reveals pyogranulomatous inflammation, 3–5 mm cigar-shaped yeast
- Pulmonary:
 - Gram stain of sputum may yield yeast; sputum cultures often positive
- Extracutaneous/disseminated:
 - CSF reveals lymphocytic meningitis, increased protein/decreased glucose

DIFFERENTIAL DIAGNOSIS

- Lymphocutaneous:
 - Leishmaniasis
 - Nocardiosis
 - *Mycobacterium marinum*
 - Tularemia
- Fixed cutaneous:
 - Bacterial pyoderma
 - Foreign-body granuloma
 - Inflammatory dermatophyte infections
 - Blastomycosis
 - Mycobacteria
- Osteoarticular:
 - Rheumatoid arthritis
 - Gout
 - Tuberculosis
 - Bacterial arthritis
 - Pigmented villonodular synovitis

- Pulmonary and meningitis:
 - Mycobacterial infections
 - Histoplasmosis
 - Coccidioidomycosis
 - Cryptococcal disease



TREATMENT

INITIAL STABILIZATION/THERAPY

Airway/hemodynamic stabilization for severely ill patients with extracutaneous manifestations

ED TREATMENT/PROCEDURES

- Lymphocutaneous/fixed cutaneous:
 - Itraconazole (drug of choice): Efficacious, but expensive and potential for hepatotoxicity, has numerous drug–drug interactions, black box in heart failure
 - Terbinafine: Less expensive alternative if failure of itraconazole, only in cutaneous disease
 - Saturated solution of potassium iodide (SSKI): Inexpensive but bitter taste and side effects (anorexia, nausea, diarrhea, fever, salivary gland swelling) lead to limited acceptability
 - Local heat therapy for cutaneous disease ($> 35^{\circ}\text{C}$) inhibits fungal growth, use in pregnant patients or others who cannot tolerate medication, therapy may take 3–6 mo
- Pulmonary:
 - Itraconazole or amphotericin B in early disease, effective in $\sim 30\%$ of cases
 - More advanced disease often requires resection plus amphotericin B
- Osteoarticular:
 - Itraconazole: 1st-line therapy for more than 1 yr, amphotericin B if refractory
- Disseminated:
 - Amphotericin initially
 - Itraconazole in stable, immunocompetent patients
 - HIV and sporotrichosis: Suppressive therapy with itraconazole is recommended after initial infection

MEDICATION

- Amphotericin B: Lipid form 3–5 mg/kg daily (preferred, especially in pregnancy and peds); if using deoxycholate form (pt with no risk of renal dysfunction) 0.7–1 mg/kg daily and infuse over 2 hr
- Itraconazole: Lymphocutaneous: 100–200 mg (peds: 6–10 mg/kg/d, max. 400 mg)

- PO TID for 3 days, then 100–200 mg per day for 2–4 wk after lesions resolve, pulmonary/osteoarticular: 200 mg PO TID for 3 days, then BID for 12 mo
- SSKI: 5 drop (peds: 1 drop) in water or juice TID; increase by 5 drops per dose each week up to a max. 40–50 drops TID (peds: max. of 1 drop/kg) as tolerated, for 6–12 wk or until lesions resolve
 - Terbinafine: Lymphocutaneous only: 250–500 mg PO per day for 2–4 wk after lesions healed



FOLLOW-UP

DISPOSITION

Admission Criteria

- Systemic signs/symptoms
- Pulmonary, CNS, multifocal disease
- Immunosuppressed host with disseminated disease

Discharge Criteria

Lymphocutaneous/fixed cutaneous form, nontoxic

Issues for Referral

Infectious disease consultant as appropriate

FOLLOW-UP RECOMMENDATIONS

Infectious disease specialist, dermatology, appropriate specialist given disease involvement (orthopedics, neurology)

PEARLS AND PITFALLS

Fixed cutaneous, lymphocutaneous, pulmonary, extracutaneous/disseminated disease secondary to *S. schenckii*:

- Inoculation with soil, moss, or organic material (skin break or inhalation)
- Contact with cats
- Healthy hosts develop fixed cutaneous/lymphocutaneous disease, immunocompromised hosts develop extracutaneous/disseminated disease
- Disseminated disease presents with nonspecific symptoms that often result in delayed diagnosis and poor outcome.
- Oral itraconazole is 1st-line therapy except for disseminated disease, where amphotericin is used initially

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CODES

ICD9

117.1 Sporotrichosis

ICD10

- B42.0 Pulmonary sporotrichosis
- B42.1 Lymphocutaneous sporotrichosis
- B42.9 Sporotrichosis, unspecified

STAPHYLOCOCCAL SCALDED SKIN SYNDROME

Roger M. Barkin



BASICS

DESCRIPTION

- Results from the actions of a soluble epidermolytic exotoxin produced by *Staphylococcus aureus*:
 - _ Produced at a distant site of infection or colonization
 - _ Disseminates hematogenously
 - _ Lyses desmosomes of granular cells in the superficial epidermis
 - _ Results in generalized intradermal exfoliation
- Typically affects infants and children <6 yr of age:
 - _ Adults have specific staph antibodies allowing them to localize, metabolize, and excrete the staph toxins.
 - _ Infants and children are unable to metabolize and excrete toxin efficiently.
 - _ Immunocompromised adults and those with severe renal dysfunction are also susceptible
- Presentation determined by age and extent of rash:
 - _ Classic staphylococcal scalded skin syndrome
 - _ Pemphigus neonatorum
 - _ Bullous impetigo
 - _ Generalized in the newborn: Ritter disease
- Typically, coagulase-positive phage group II *Staphylococcus*:
 - _ Phage groups I and III also implicated

ETIOLOGY

- Colonization often without overt infection
- Concurrent infection or break of skin barrier:
 - _ Nasopharynx
 - _ Urinary tract
 - _ Minor skin abrasions
 - _ Circumcision site
 - _ Conjunctivitis
 - _ Umbilicus/omphalitis
 - _ Impetigo
 - _ Endocarditis and septicemia
- Often no focus identified



DIAGNOSIS

SIGNS AND SYMPTOMS

- Constitutional symptoms:
 - Malaise
 - Fever
 - Irritability
 - Child may appear well, ill, or overtly toxic
 - Abrupt onset
- Scarlatiniform erythematous rash (sandpaper like) resembling a “sunburn”—erythroderma
- Exquisitely tender skin
- Areas of prominence:
 - Around the flexor areas of the neck
 - Intertriginous areas, especially axilla and groin
 - Near the eyes and mouth
 - Increased erythema in skin creases
- Facial edema with radial crusting fissures around the eyes, nose, and mouth
- Flaccid bullae:
 - Within 1–3 days after onset of rash
 - Initially over flexures (axillae, groin, body orifices)
 - Bullae migrate through epidermis with light lateral pressure; epidermis separates with minor pressure (Nikolsky sign).
 - Rupture within hours
 - Epidermis separates with minor trauma.
 - Epidermis is shed in sheets.
 - Denuded areas are moist, sensitive, and painful.
 - Complete healing within 2 wk, no scarring
- Purulent conjunctivitis
- Mucous membranes not affected
- Complications rare:
 - Hypothermia
 - Fluid and electrolyte imbalance
 - Secondary infection
 - Pneumonia
 - Septicemia
 - Cellulitis
 - Osteomyelitis

ESSENTIAL WORKUP

- Clinical presentation is diagnostic.
- Determine location/source of toxin producing *Staphylococcus*.
- Assess systemic nature of infection.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC and urinalysis:
 - _ Assess for sepsis if source not obvious.
- Electrolytes:
 - _ Indicated if signs of dehydration or extensive rash
- Blood cultures (rarely positive)

Imaging

Indicated as need to determine location/source of infection

Diagnostic Procedures/Surgery

- Fluid aspirated from bullae:
 - _ Sterile in staphylococcal scalded skin syndrome
 - _ Consistent with hematogenous dissemination of the toxin
- Isolation of staphylococci from a site other than the blisters:
 - _ Commonly conjunctivae, nasopharynx, or blood
- Skin biopsy or frozen histologic section:
 - _ Determine level of epidermal/dermal separation (cleavage is in granular layer of dermis).
 - _ Indicated for children on medications, those > 6 yr, and in cases of mixed presentation

DIFFERENTIAL DIAGNOSIS

- Infection:
 - _ Scarlet fever:
 - Involves the mucous membranes
 - Strawberry tongue
 - Painful desquamation does not occur
- Bullous impetigo:
 - _ Turbid or cloudy bullous fluid
- Bullous varicella:
 - _ Tzanck prep or viral base reveals giant cells.
 - _ 5 days after the onset of varicella
- Toxic shock syndrome:
 - _ Rapid development of clinical signs and symptoms
 - _ Mucous membrane and multiorgan involvement
- Toxic epidermal necrolysis or drug eruption:
 - _ Much more common in adults
 - _ Severely afflicted mucous membranes
 - _ Full-thickness epidermal necrosis
- Dermatologic:
 - _ Erythema multiforme
 - _ Epidermolysis hyperkeratosis

- Epidermolysis bullosa
- Pemphigus vulgaris
- Scald injury
- Secondary rash of an underlying disorder:
 - Lymphoma
 - Aspergillosis
 - Irradiation
 - Graft-versus-host reaction
 - Kawasaki disease



TREATMENT

PRE HOSPITAL

- 9% NS fluid bolus if dehydration present
- Initial burn treatment

INITIAL STABILIZATION/THERAPY

- Management is similar to an extensive 2nd-degree burn:
 - Involvement of large body surface area will require IV fluids.
- Provide adequate analgesia.
- Undress and place child on sterile linen.
- Limit handling of child.
- Apply moist sterile dressings.
- Avoid excess heat loss.

ED TREATMENT/PROCEDURES

- Topical burn creams are of no proven benefit.
- Steroids are contraindicated.
- IV antibiotics effective against penicillinase-resistant *S. aureus*:
 - Cefazolin
 - Nafcillin
 - Vancomycin if methicillin-resistant *S. aureus* (MRSA) suspected
- Oral antibiotics for mild involvement:
 - Dicloxacillin
 - Erythromycin
 - Cephalexin

MEDICATION

- Cefazolin: 50–100 mg/kg/24 h IV div. QID
- Cephalexin: 25–100 mg/kg/24 h PO div. QID
- Dicloxacillin: 12–25 mg/kg/24 h PO div. QID
- Erythromycin: 30–50 mg/kg/24 h PO div. QID
- Nafcillin: 1–2 g IV q6h (peds: Newborns, 50–100 mg/kg/24 h IV div. q6h; children,

100–200 mg/kg/24 h IV div. q6h)

- Vancomycin: 40 mg/kg/24h IV q 6 hrs



FOLLOW-UP

DISPOSITION

Admission Criteria

- Children < 1 yr
- All toxic-appearing children
- Widespread skin involvement
- Dehydration and/or electrolyte derangement

Discharge Criteria

- Older, well-appearing children with mild involvement
- Oral antibiotics for 7 days
- Follow-up within 48 hr

Issues for Referral

- Infectious disease consultant
- Surgeon if source needs excision/drainage

ADDITIONAL READING

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CODES

ICD9

695.81 Ritter's disease

ICD10

L00 Staphylococcal scalded skin syndrome

STERNOCLAVICULAR JOINT INJURY

Christopher M. Tedeschi • Wallace A. Carter



BASICS

DESCRIPTION

- Sternoclavicular joint (SCJ) is the only joint that connects the upper limb to the trunk.
- Among the least frequently injured joints in the body
- Most commonly due to athletic or vehicular injuries
- Congenital or spontaneous dislocation and subluxation are rarely seen
- SCJ stability depends on ligamentous attachments, primarily anterior and posterior sternoclavicular ligaments, interclavicular ligament, and costoclavicular ligament

ETIOLOGY

- Injury to the SCJ can be from sprains, subluxations, or dislocations of the ligamentous structure
- In sprains, ligamentous capsule remains intact
- Subluxation occurs when sternoclavicular ligament ruptures while costoclavicular ligament remains intact
- Complete ligamentous disruption leads to dislocation
- The SCJ can dislocate anteriorly or posteriorly. A large force is required. A greater force is required to displace the clavicle posteriorly.
- Direction of dislocation depends on the shoulder position:
 - Anterior dislocation more likely when the acromion is posterior to the manubrium.
 - Posterior dislocation more likely when the acromion is anterior to the manubrium.
- Anterior dislocation is more common (more than 90% of dislocations):
 - Caused by a posteriorly directed force to the anterolateral aspect of the shoulder
 - Reciprocal anterior displacement of the medial clavicle
 - May be associated with pneumothorax, hemothorax, pulmonary contusion, and rib fractures
 - Subluxation and dislocation may occur spontaneously.
- Posterior SCJ dislocation results from:
 - Anterior-to-posterior blow to the medial clavicle
 - Anteriorly directed force to the lateral aspect of the ipsilateral shoulder
 - A blow to the contralateral shoulder when the injured side is braced against an immobile object

- Posterior dislocation is a surgical emergency:
 - Indications for immediate reduction:
 - Compression or tear of trachea, esophagus, or great vessels
 - Recurrent laryngeal nerve injury

Pediatric Considerations

- The medial epiphyseal growth plates of the clavicles are last to ossify, and fuse between ages 22 and 25:
 - Until fusion, growth plate is the weakest part of the joint
- Fractures through the medial epiphysis mimic SCJ dislocations:
 - Most commonly Salter–Harris type I or II fractures
 - True dislocations of the SCJ are extremely rare in children because of strong ligamentous attachments.



DIAGNOSIS

SIGNS AND SYMPTOMS

- Pain and swelling localized to the medial clavicle and SCJ with appropriate mechanism
- Affected arm supported across the chest by the contralateral arm
- Inability to abduct or externally rotate arm
- If subluxed or sprained, the SCJ is tender on direct palpation and with shoulder movement:
 - No deformity or significant AP mobility
- If the SCJ is dislocated, shoulder appears shortened:
 - Head tilts toward injured side due to sternocleidomastoid muscle spasm
- In anterior dislocation, medial end of the clavicle is visibly prominent and palpable.
- In posterior dislocation, there may be a sulcus of the SCJ area through which the lateral border of the manubrium may be palpated:
 - Dislocation may be masked by significant swelling over the SCJ region, and may mimic anterior dislocation.
- Posterior dislocation may be accompanied by signs of vascular compromise or damage to mediastinal structures:
 - Signs of shock
 - Difficulty breathing or speaking
 - Upper extremity pain or neurologic symptoms

History

- High-energy direct blow, most often from athletic injuries or motor vehicle collisions
- Sprains and subluxations may be associated with other injuries of the shoulder

girdle.

Physical-Exam

- Tenderness and swelling in sprains and subluxations
- In anterior dislocation, prominence of medial clavicle
- For any concern of posterior dislocation, assess for signs of airway or neurovascular compromise:
 - Dysphagia or respiratory distress may signify compression or disruption of trachea or esophagus.
 - Assess pulses in upper extremities
 - Hoarseness may signify injury to the recurrent laryngeal nerve.
 - Motor or sensory deficits suggest brachial plexus injury
 - Assess venous return in upper extremities:
 - Venous compression may lead to engorged upper extremity veins or venous thrombosis

ESSENTIAL WORKUP

- Comprehensive trauma evaluation and resuscitation for other life-threatening injuries
- Special attention to respiratory, neurologic, and vascular status
- A posterior dislocation implies substantial mechanism of injury; other life-threatening injuries must be ruled out.
- Appropriate analgesia for patient comfort

DIAGNOSIS TESTS & INTERPRETATION

Imaging

- Difficult to assess SCJ injury with routine radiographs:
 - May demonstrate asymmetry of the SCJ compared with contralateral side
 - More useful to assess coexisting bony, pulmonary, and mediastinal injury
 - Chest x-rays may be read as normal and further imaging is warranted if index of suspicion is high
- US can reliably demonstrate SCJ dislocations:
 - May be useful in the initial ED evaluation of unstable patients with chest trauma
 - Use high-frequency linear probe
 - In anterior dislocation, medial clavicle seen anterior relative to manubrium compared to contralateral side
- CT scan is best to evaluate the SCJ:
 - Useful when plain films are inconclusive
 - Accurately differentiates fractures from dislocations
 - Demonstrates the position of the medial clavicle
 - Shows detailed anatomy of the thoracic outlet and mediastinum

- Contrast CT can show related vascular injuries and is the imaging modality of choice.
- MRI can be useful in demonstrating ligamentous and soft tissue SCJ injuries:
 - The articular disc is the most vulnerable soft tissue structure in SCJ injury.
 - Can demonstrate specific ligamentous injuries in the setting of joint subluxation
 - Better suited after the initial period of diagnosis and treatment
 - Can help distinguish true dislocation from physeal injury in pediatric patients

DIFFERENTIAL DIAGNOSIS

- Sternoclavicular sprain, subluxation, or dislocation
- Medial clavicle fracture
- Septic arthritis
- Osteomyelitis of medial clavicle



TREATMENT

PRE HOSPITAL

- Attention to airway and vital signs, and neurovascular status of affected extremity
- Affected arm should be splinted in the position of comfort before transport to the ED.

INITIAL STABILIZATION/THERAPY

- Endotracheal intubation for signs of airway compromise or as needed in the trauma patient
- Emergent SCJ reduction for:
 - Unstable or compromised airway
 - Signs of shock
 - Diminished pulses
 - Hoarseness
 - Dysphagia
 - Neurovascular compromise:
 - Upper extremity weakness
 - Paresthesia

ED TREATMENT/PROCEDURES

- *Sprains and subluxations* may be treated symptomatically with ice, NSAIDs, sling immobilization, and orthopedic follow-up.
- *Anterior dislocations* may be reduced in the ED:
 - Procedural sedation for adequate pain control and muscle relaxation
 - Rolled towel placed between the shoulder blades in the supine position:
 - Longitudinal traction applied to the extended arm with shoulder

- abducted 90°
- Assistant applies gentle pressure over the displaced end of the clavicle.
- After reduction, immobilize with a well-padded figure-of-8 dressing.
- Many anterior dislocations remain unstable after reduction.
- Surgery rarely indicated, as deformity is mainly cosmetic
- *Posterior dislocations* require urgent reduction best achieved in the OR under general anesthesia:
 - Orthopedic and thoracic surgery consults
 - Closed reduction is preferred (and often successful) but may not be possible in injuries >48 hr.
 - If surgeon not immediately available, emergent reduction in the ED may be necessary:
 - Relieve serious airway, neurologic, or vascular compromise
 - Adequate sedation and analgesia are essential
 - Patient placed supine with a roll between shoulder blades
 - Affected arm is abducted and extended
 - Increased traction as arm is brought into extension
 - If unsuccessful, a sterile towel clamp is used to grasp medial clavicular head and apply gentle anterior traction

MEDICATION

Procedural sedation:

- Etomidate: 0.1 mg/kg IV
- Fentanyl: 1–2 µg/kg IV
- Ketamine: Peds: 1 mg/kg IV – up to 2 additional doses of 0.5 mg/kg IV PRN
- Midazolam: 0.01 mg/kg (peds: 0.05–0.1 mg/kg) IV q2–3min
- Propofol: Initial bolus 1 mg/kg IV, then 0.5 mg/kg q3min as needed (adults and peds)



FOLLOW-UP

DISPOSITION

Admission Criteria

- Posterior dislocations of the SCJ require admission for possible reduction in the OR and evaluation for potential intrathoracic complications.
- Coexisting injury significant enough to warrant hospitalization

Discharge Criteria

- SCJ sprains
- Anterior dislocations of the SCJ without neurovascular compromise or other

significant injury

- Appropriate outpatient orthopedic follow-up arranged

Issues for Referral

Outpatient referral to an orthopedist should be recommended for patients with any significant SCJ injuries.

FOLLOW-UP RECOMMENDATIONS

- It is difficult to achieve long-term stability after closed reduction of dislocations, so close orthopedic follow-up is advisable.
- Simple sling sufficient for sprains
- Figure-of-8 dressing for more severe injuries
- Repeat MRI or CT imaging may be beneficial.
- Even for mild sprains and subluxations, high-risk activity should be avoided for up to 3 mo.

PEARLS AND PITFALLS

- Since SCJ injuries are rare, this potentially life-threatening injury may be missed during ED evaluation and resuscitation.
- Posterior dislocations mandate early thoracic and cardiothoracic surgery consultation.
- Posterior dislocation may be mistaken for anterior due to marked swelling over the joint.
- In the pediatric population, a Salter–Harris fracture may mimic a dislocation.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Acromioclavicular Joint Injury
- Arthritis, Septic

- Clavicle Fracture
- Trauma, Multiple



CODES

ICD9

- 839.61 Closed dislocation, sternum
- 848.41 Sprain of sternoclavicular (joint) (ligament)

ICD10

- S43.60XA Sprain of unspecified sternoclavicular joint, initial encounter
- S43.203A Unspecified subluxation of unspecified sternoclavicular joint, initial encounter
- S43.206A Unspecified dislocation of unspecified sternoclavicular joint, initial encounter

STEVENS–JOHNSON SYNDROME

Herbert G. Bivins • James Comes

BASICS

DESCRIPTION

- Stevens–Johnson syndrome (SJS) is an idiosyncratic, severe mucocutaneous disease:
 - Blistering of < 10% of the body surface area (BSA)
 - 95% of patients have mucous membrane lesions:
 - Usually at 2 or more sites
 - 85% have conjunctival lesions
 - Lesions often involving face, neck, and central trunk regions become confluent over hours to days
- On a continuum with toxic epidermal necrolysis (TEN); but thought to be a distinct disease entity from erythema multiforme (EM):
 - SJS: < 10% of BSA
 - SJS–TEN overlap syndrome: 10–30% of BSA
 - TEN: > 30% of BSA, can affect up to 100% BSA

ETIOLOGY

- The most common causes include medications and infections:
 - Damage to the skin is thought to be mediated by cytotoxic T lymphocytes and mononuclear cells aimed at keratinocytes expressing (drug-related) antigens
 - Cytokines from activated mononuclear cells probably contribute to cell destruction and systemic manifestations
- Causative medications:
 - Antibiotics (e.g., penicillin, sulfonamide)
 - Anticonvulsants
 - Oxicams
 - NSAIDs
 - Allopurinol
- Infections:
 - *Mycoplasma pneumoniae*
 - Herpes simplex

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- *Prodrome:*
 - Usually 1–3 days prior to development of skin lesions
 - Fever
 - Headache
 - General malaise
 - Upper respiratory infection (URI) symptoms
 - Arthritis, arthralgias, and myalgias prior to mucocutaneous lesions
- **Skin:** Mild to moderate skin tenderness followed by skin pain, burning sensation, and paresthesias
- **Eye:** Conjunctival burning or itching
- **Mucous membranes:** Painful micturition, painful swallowing
- **Drug exposure precedes symptoms usually by 2 wk:**
 - Re-exposure may result in onset of symptoms within 48 hr
- **Risk factors include HIV, genetic factors, viral infections, and underlying immunologic diseases**

Physical-Exam

- **Rash:** Target lesions, erythematous or purpuric macules with or without confluence, and raised flaccid blisters or bullae with skin detachment that spread with lateral pressure (Nikolsky sign) on erythematous areas
- **Mucous membrane:** Erythematous tender erosions of the mouth, pharynx, trachea, genitalia, or anus; possibly pseudomembrane formation
- **Eye:** Mild to severe conjunctivitis with possible formation of pseudomembranes and corneal ulcers

ESSENTIAL WORKUP

A complete history and physical exam with careful attention to mucous membranes, percentage of blistering, and identification of likely etiology

DIAGNOSIS TESTS & NTERPRETATION

Lab

- **Electrolytes**
- **Liver enzymes may be mildly elevated**
- **CBC:**
 - **Anemia and lymphopenia are common**
- **UA**

Imaging

Chest radiography if pneumonia is a consideration

Diagnostic Procedures/Surgery

Skin biopsy of lesions and mucous membranes demonstrates necrosis of the entire epidermal layer with formation of subepidermal split above basement membrane

DIFFERENTIAL DIAGNOSIS

- SJS if < 10% of BSA
- Overlapping SJS and TEN (skin detachment between 10% and 30% of BSA plus widespread macules or flat atypical target lesions)
- TEN (skin detachment > 30% of the BSA plus widespread macules or flat atypical targets)
- EM
- Thermal burns
- Phototoxic reactions
- Exfoliative dermatitis
- Pustular drug eruptions
- Bullous fixed drug eruptions
- Paraneoplastic pemphigus
- Graft-versus-host disease in bone marrow transplant patients
- Toxic shock syndrome
- Staphylococcal scalded skin syndrome

Pediatric Considerations

Staphylococcal scalded skin syndrome is in the pediatric differential diagnosis of severe blistering mucocutaneous diseases



TREATMENT

PRE HOSPITAL

- ABCs
- Observe universal precautions
- IV access if indicated
- Transport to burn center if > 30% of body surface involved

INITIAL STABILIZATION/THERAPY

- Endotracheal intubation and ventilatory support may be required for impending respiratory failure (more commonly associated with TEN)
- IV fluids

ED TREATMENT/PROCEDURES

- Fluid replacement:
 - Fluid losses may be significant
- Recognize and treat underlying infections:
 - Sepsis is the primary cause of death, frequently from gram-negative

pneumonia

- Secondarily infected cutaneous lesions can be treated with débridement of blisters, compresses, and systemic antibiotics

- Corticosteroids are controversial
- Prophylactic antibiotics may be indicated if systemic steroids are given
- Intravenous immunoglobulin (IVIG) may be beneficial
- Mild systemic symptoms may be treated with acetaminophen or NSAIDs provided they are not the cause of the mucocutaneous reaction
- Mucous membrane lesions are extremely painful and may require parenteral analgesics
- Large extensive bullae should be débrided, ideally in a burn unit

MEDICATION

- Acetaminophen: 500 mg PO/PR q4–6h (peds: 10–15 mg/kg/dose; do not exceed 5 doses/24 h); do not exceed 4 g/24 h
- Acyclovir: 5–10 mg/kg IV q8h (for herpes simplex virus infections)
- Ibuprofen: 300–800 mg PO (peds: 5–10 mg/kg/dose)
- Morphine sulfate: 0.1 mg/kg/dose IV

First Line

- Fluid replacement
- Treat underlying etiology
- Treat secondary infections
- Analgesia

Second Line

- IVIG
- Corticosteroids



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients with SJS should be admitted to the hospital
- Patients with extensive epidermal detachment should be admitted to a burn center or a specialized intensive care unit

Discharge Criteria

Patients with EM minor may be discharged with appropriate and timely follow-up

Issues for Referral

Patients must be made aware of the likely offending drug (and its class) and that it must never be administered to them again

FOLLOW-UP RECOMMENDATIONS

Follow-up with PCP and/or dermatologist

PEARLS AND PITFALLS

- SJS may begin like an influenza illness. Lesions appear 1–3 days after the prodrome
- The diagnosis is clinical and biopsy is supportive
- *M. pneumoniae* and herpes simplex are more common triggers in children than in adults

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Erythema Multiforme
- Toxic Epidermal Necrolysis



CODES

ICD9

- 695.13 Stevens-Johnson syndrome

- 695.14 Stevens-Johnson syndrome-toxic epidermal necrolysis overlap syndrome

ICD10

- L51.1 Stevens-Johnson syndrome
- L51.3 Stevens-Johnson synd-tox epdrml necrolysis overlap syndrome

STING, BEE

Daniel T. Wu

BASICS

DESCRIPTION

- Injection of hymenoptera venom causes:
 - Release of biologic amines
 - Local or systemic allergic reactions
- Reactions are:
 - Usually IgE-mediated type I hypersensitivity reactions
 - Rarely type III (Arthus) hypersensitivity reactions

ETIOLOGY

- Hymenoptera—order of the phylum Arthropoda
- Includes bees (Apidae family), wasps and hornets (Vespidae family), fire ants (Formicidae family)

DIAGNOSIS

SIGNS AND SYMPTOMS

History

History and physical exam—keys to diagnosis

Physical-Exam

5 types of reactions to stings:

- Local reaction:
 - Most common type of reaction
 - Local pain, erythema, and edema at sting site
 - Symptoms occur immediately and resolve within 1–2 hr
- Large local reaction:
 - Similar to local reaction but affects larger area or entire limbs
 - Peaks at 48 hr and can last several days
 - Mild to moderate fever
- Systemic reaction:
 - Includes anaphylaxis
 - Can be fatal (usually owing to respiratory failure)
 - Respiratory:
 - Wheezing
 - Coughing

- Stridor
- Shortness of breath
- Hoarseness
- Angioedema
- GI:
 - Nausea
 - Vomiting
 - Diarrhea
 - Abdominal pain
- Cardiovascular:
 - Hypotension
 - Chest pain
 - Tachycardia
 - Shock
- Other:
 - Urticaria
 - Pruritus
 - Flushing
- Symptoms occur within 15–20 min and last ≤ 72 hr
- Toxic reaction:
 - Result of multiple stings and large doses of venom
 - Symptoms similar to anaphylaxis
- Unusual reactions:
 - Owing to unusual immune response
 - Vasculitis
 - Nephrosis
 - Serum sickness
 - Neuritis
 - Encephalitis
 - Reaction delayed (days to weeks after sting)

ESSENTIAL WORKUP

- History and physical exam key to diagnosis
- No radiologic or lab test will confirm hymenoptera envenomation or anaphylaxis

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC, electrolytes, BUN, creatinine, glucose, arterial blood gases (ABGs):
 - Not routine
 - Consider when significant systemic effects present

Diagnostic Procedures/Surgery

ECG:

- When significant systemic effects present in patients at risk for cardiovascular disease

DIFFERENTIAL DIAGNOSIS

- Insect bites sometimes cause pain; stings always cause pain.
- Cellulitis:
 - _ Difficult to distinguish between large local reactions and cellulitis
 - _ Infections of hymenoptera envenomations are rare and usually caused by wasp envenomations.
 - _ Local reaction can resemble periorbital cellulitis.
- Gout
- Soft tissue trauma
- Systemic/toxic reactions:
 - _ Pulmonary embolus
 - _ Anaphylaxis from different agent
 - _ Hyperventilatory syndrome/anxiety
 - _ Acute coronary syndrome



TREATMENT

PRE HOSPITAL

Most deaths occur within 1st hour owing to either respiratory obstruction or anaphylaxis causing cardiovascular and respiratory collapse.

INITIAL STABILIZATION/THERAPY

Acute Severe Systemic Reaction/ Anaphylaxis

- ABCs:
 - _ Intubation/ventilation with rapidly increasing signs of laryngeal compromise
 - _ Oxygen
 - _ 0.9% normal saline (NS) IV access
- Epinephrine SC/IV
- Antihistamines IV
- Corticosteroids
- When signs of systemic reactions:
 - _ Assess for patent airway
 - _ Establish IV access

ED TREATMENT/PROCEDURES

- Systemic reactions:

- Epinephrine for respiratory symptoms/hypotension
- Antihistamines—H₁ (diphenhydramine) and H₂ (cimetidine, ranitidine, or famotidine) blockers
- Steroids (prednisone, methylprednisolone, or dexamethasone)
- Inhaled β -agonist for wheezing/shortness of breath
- For persistent hypotension:
 - o 0.9% NS IV fluid resuscitation
 - o Vasopressor (epinephrine/ α -adrenergic) for hypotension resistant to IV fluids
- Removal of remnants of stinger at site of envenomation (bees may leave stingers with venom sacs) by scraping, not squeezing
- Local reactions:
 - Cool compress
 - Elevation
 - Remove constrictive clothing or jewelry
 - Topical antihistamine/topical steroidal cream as needed
 - Oral antihistamine or steroids as needed

MEDICATION

- Albuterol, β -agonist (inhaled): 3 mg in 5 mL NS (peds: 0.1 mg/kg of 5 mg/mL concentration) via nebulization
- Cimetidine: 300 mg (peds: 5 mg/kg) IV/IM/PO
- Diphenhydramine:
 - 50–100 mg (peds: 1 mg/kg) IV for severe reactions
 - 25–50 mg (peds: 1 mg/kg) PO QID for severe local reactions
- Epinephrine:
 - 0.1 mg: 1 mL of 1:10,000 dilution (peds: 0.01 mg/kg 0.1 mL/kg of 1:10,000 dilution up to 1 mL) IV over 5 min for shock
 - 0.3 mg (0.3 mL of 1:1,000 dilution); (peds: 0.01 mg/kg up to 0.5 mg) SC for severe reactions but not in shock
- Famotidine: 40 mg IV (peds: 1 mg/kg/d div. BID IV)
- Methylprednisolone: 125 mg (peds: 1–2 mg/kg) IV
- Norepinephrine: 2–4 μ g/kg/min (peds: 0.1 μ g/kg/min) titrated continuous infusion
- Prednisone: 60 mg (peds: 1–2 mg/kg) PO
- Ranitidine: 50 mg IV/IM (peds: 2–4 mg/kg/d div. q6–8h IV/IM)



FOLLOW-UP

DISPOSITION

Admission Criteria

- Worsening symptoms, airway compromise
- Persistent unstable vital signs require ICU admission.
- Life-threatening reaction requires 24-hr observation.
- Systemic reaction requires minimum of 6 hr of observation.

Discharge Criteria

- Minimal isolated local reaction
- Systemic reactions that resolve and do not recur during 6-hr observation period

Issues for Referral

Follow-up:

- Provide patients with life-threatening reactions, emergency anaphylaxis kits (EpiPen; peds: EpiPen Jr if < 15 kg), and medical identification bracelets (Medi-Alert).
- Systemic reaction requires follow-up for possible immunotherapy.

FOLLOW-UP RECOMMENDATIONS

Allergist follow-up for patients with systemic reactions.

PEARLS AND PITFALLS

- Treat patients who present with systemic reactions to bee stings aggressively.
- Provide prescriptions for EpiPen to patients discharged after presenting with life-threatening reactions to bee stings.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Anaphylaxis



CODES

ICD9

989.5 Toxic effect of venom

ICD10

T63.441A Toxic effect of venom of bees, accidental, init

STING, SCORPION

Frank LoVecchio



BASICS

DESCRIPTION

- Scorpion venom is neurotoxic:
 - Sodium channels opening
 - Prolonged firing of neurons
- Autonomic, somatic, and cranial nerve excitation occurs.
- Symptoms begin within minutes of bite.
- Symptoms persist 1–72 hr.

ETIOLOGY

- *Centruroides* species found in Southern US, Mexico, Central America, and Caribbean
- Many other species in Asia, Africa, Israel, South America, and Middle East

Pediatric Considerations

- Can be misdiagnosed as seizures, amphetamine poisoning, or meningitis
- Higher mortality and severity of illness



DIAGNOSIS

SIGNS AND SYMPTOMS

- Onset within minutes, progressing to maximum severity in ~1–2 hr but may persist \leq 48–72 hr.
- Scorpion species determines symptomatology (*Centruroides sculpturatus*, aka *Centruroides exilicauda* or bark scorpion, is the only species in US causing symptoms).
- Local tissue effects:
 - No erythema
 - Pain
 - Hyperesthesia
- Autonomic effects:
 - Sympathetic symptoms:
 - Tachycardia
 - Hypertension
 - Hyperthermia
 - Pulmonary edema
 - Agitation

- Perspiration
- Parasympathetic effects:
 - Hypotension
 - Bradycardia
 - Hypersalivation
- Somatic effects:
 - Involuntary muscle contractions
 - Restlessness
- Cranial nerve effects:
 - Roving eye movements
 - Blurred vision
 - Nystagmus
 - Tongue fasciculations
 - Loss of pharyngeal muscle control

ESSENTIAL WORKUP

- Identification of scorpion species not needed if scorpion is native to US (see above).
- Maintain high clinical suspicion in endemic areas
- Assess envenomation grade severity:
 - Grade I: Local pain and/or paresthesias at site
 - Grade II: Local pain and/or paresthesias at a remote site
 - Grade III: Either cranial/autonomic *or* somatic skeletal neuromuscular dysfunction
 - Grade IV: Both cranial/autonomic *and* somatic skeletal muscle dysfunction

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Grades I and II envenomations:
 - None
- Grades III and IV envenomations:
 - BUN, creatinine
 - Electrolytes
 - UA
 - CBC
- Severely agitated patients:
 - Creatine kinase
 - Urine myoglobin
- Severe respiratory distress:
- ABGs

Imaging

- Chest radiograph for respiratory symptoms
- ECG for tachycardia

DIFFERENTIAL DIAGNOSIS

- Snake, spider, insect envenomation
- Tetanus
- Diphtheria
- Botulism
- Overdose/dystonic reaction
- Seizures
- Infections



TREATMENT

PRE HOSPITAL

- Evaluate ABCs
- IV access

INITIAL STABILIZATION/THERAPY

- ABCs
- Endotracheal intubation if necessary
- IV
- O₂
- Monitor

ED TREATMENT/PROCEDURES

- Mild envenomations—grades I and II:
 - Oral analgesics
 - Tetanus prophylaxis
- Severe envenomations—grades III and IV:
 - Antivenom (Anascorp), expensive therapy
 - Tetanus prophylaxis
 - Hypertensive urgencies/emergencies (rare):
 - Standard therapy such as labetalol
 - Hypotension:
 - IV fluid resuscitation and pressor therapy with dopamine
 - Severe agitation:
 - Midazolam
 - Treatment for rhabdomyolysis if present

MEDICATION

- Antivenom: Centruroides (scorpion) (Rx: Anascorp infuse 3 vials IV over 10 min);

monitor for up to 60 min after completing infusion to determine if symptoms are resolved. Additional doses may be used if needed; infuse 1 vial at a time at 30–60 min intervals.

- Dopamine: 2–5 µg/kg/min IV; increase in 5–10 µg/kg/min as needed
- Midazolam: 1–2 mg (peds: 0.01–0.05 mg/kg) IV
- Labetalol: 20 mg (peds: 0.3–1 mg/kg/dose) q10min
- Fentanyl: 50–150 µg (peds: 1–3 µg/kg) IV
- Tetanus toxoid: 0.5 mL IM (peds: Same dose)

Pediatric Considerations

Antivenom doses are the same in children because dosage is based on venom burden.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Grades III and IV envenomations require admission to ICU.
- If antivenom is given with resolution of symptoms, observe for 1–2 hr if asymptomatic.

Discharge Criteria

- Grades I and II envenomations after a short observation period (3–4 hr after sting occurred) for progression of symptoms
- Grades III and IV envenomations given antivenom with resolution of symptoms can be discharged.
- If patient received antivenom, discuss signs and symptoms of delayed serum sickness.
- Discuss possibility of persistence of pain and paresthesias at site.
- Encourage patient to return for progression of symptoms.

Pediatric Considerations

Toddlers are more likely to have early airway involvement.

FOLLOW-UP RECOMMENDATIONS

Primary care follow-up if antivenin given.

PEARLS AND PITFALLS

- Maintain high index of suspicion for scorpion stings in endemic areas when patients present with typical symptoms.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Botulism
- Rhabdomyolysis
- Seizures
- Spider Bite, Black Widow
- Tetanus



CODES

ICD9

989.5 Toxic effect of venom

ICD10

T63.2X1A Toxic effect of venom of scorpion, accidental, init

STREPTOCOCCAL DISEASE

Scott C. Sherman



BASICS

DESCRIPTION

- Increase in frequency of aggressive streptococcal necrotizing skin infection noted in 1980s and dubbed “flesh-eating bacteria.”
- Affects otherwise healthy patients aged 20–50 yr who did not have underlying predisposing diseases.
- Rapid progression of shock and multiorgan dysfunction, with death occurring within 1–2 days.
- Incidence is 3–4 per 100,000 in industrialized countries
- Invasive infections caused by group A *Streptococcus* (GAS) include:
 - Necrotizing fasciitis (NF):
 - Progressive, rapidly spreading soft tissue infection located within the deep fascia and subcutaneous fat
 - Streptococcal toxic shock syndrome (STSS):
 - May occur in patients with GAS associated NF.
 - Portals of entry for streptococci include vagina, pharynx, mucosa, and skin.
 - Unknown cause in 50% of cases.
 - “Other” invasive disease defined as isolation of GAS from a normally sterile body site (i.e., sepsis, bacteremic pneumonia, septic arthritis, etc.)
- Occurs sporadically, with occasional outbreaks in long-term care facilities and hospitals.
- Rate of invasive GAS disease 6 times the annual incidence of meningococcal disease.

STSS Case Definition

- Isolation of GAS from sterile or nonsterile body site
- Hypotension
- 2 or more of the following:
 - Renal impairment
 - Coagulopathy
 - Liver abnormalities
 - Acute respiratory distress
 - Extensive tissue necrosis (NF)
 - Erythematous rash

ETIOLOGY

- NF:
 - GAS is causative in 10% of cases. Blunt trauma is risk factor.
 - Mixed anaerobic and aerobic organisms are found in 70% of cases.
 - *Staphylococcus aureus*, *Clostridium* species, and other enteric organisms
- Streptococcal toxic shock syndrome:
 - Occurs when susceptible host is infected with virulent strain
 - M protein types 1, 3, and 28 are most common.
 - Pyrogenic exotoxins (e.g., A, B, and C) produce fever and shock via activation of tumor necrosis factor and interleukins.
 - Nonsteroidal anti-inflammatory drugs appear to mask or predispose patients.
 - Risk factors:
 - Age < 10 or > 60 yr
 - Cancer
 - Renal failure
 - Leukemia
 - Severe burns
 - Corticosteroids

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- *Pain*:
 - Most common initial symptom of NF:
 - Occurs in 85% of cases
 - Out of proportion to physical findings
 - Often abrupt in onset and severe
 - Often requires palliative IV narcotics
 - Usually involves an extremity
 - May mimic peritonitis, pelvic inflammatory disease, pneumonia, acute MI, or pericarditis

Physical-Exam

- *Fever* most common sign:
 - Can present with hypothermia, especially if patient is in shock
- *Altered mental status* present in 55% of cases
- *Soft tissue infection* (erythema and swelling) present in 80%:
 - Indistinct borders, blisters, bullae
 - No lymphangitis or lymphadenopathy
- *Influenza-like syndrome* in 20%:

- Fever
- Chills
- Myalgias
- Nausea, vomiting
- Diarrhea
- Shock:
 - Present at admission or within 4–8 hr in *all* patients
 - Frequently persists despite fluids, antibiotics, and vasopressors
- Renal failure:
 - Precedes onset of shock in many cases
 - Dialysis often necessary
 - Kidney function returns to normal within 4–6 wk in survivors.
- ARDS:
 - Occurs in 55% of patients

ESSENTIAL WORKUP

- Suspect NF when pain is out of proportion to exam.
- Obtain plain films to search for presence of air in soft tissues.
- Blood cultures should be obtained.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC with differential:
 - Mild leukocytosis with left shift initially
- Electrolytes, BUN, and creatinine
- Calcium level:
 - Hypocalcemia in association with fat necrosis from NF
- Urinalysis:
 - Hemoglobinuria if renal involvement
- Serum creatine phosphokinase:
 - An elevated or rising level correlates with NF or myositis.
- Aerobic and anaerobic blood cultures
- Wound cultures
- PT/PTT/INR/DIC panel

Imaging

- Plain films:
 - Gas in soft tissues in 25–75% of cases of NF, but not as frequently associated with group A β -hemolytic streptococcal infection
 - More common in mixed anaerobic infections
- CT scan:
 - Asymmetric thickening of deep fascia

- Gas
- MRI:
 - High signal intensity of the fascia in T2-weighted images associated with NF

Diagnostic Procedures/Surgery

Aspiration of involved areas with Gram stain and culture may be useful

DIFFERENTIAL DIAGNOSIS

- Sepsis
- Cellulitis
- Erysipelas
- NF/myositis secondary to infection by another pathogen



TREATMENT

PRE HOSPITAL

Stabilize as appropriate

INITIAL STABILIZATION/THERAPY

- Maintain ABCs.
- Treat shock with fluids and vasopressors as needed:
 - Hypotension is often intractable, and up to 10–20 L/day may be required.
- Intubation and mechanical ventilation for:
 - ARDS
 - Severe shock
 - Ventilatory failure

ED TREATMENT/PROCEDURES

- Broad-spectrum antibiotics immediately after cultures until the presence of GAS has been confirmed:
 - Clindamycin is a potent suppressor of GAS bacterial toxin synthesis and inhibits M protein synthesis
- Early surgical consultation. Most patients will require an operative procedure (e.g., fasciotomy, surgical débridement, exploratory laparotomy, intraocular aspiration, amputation, or hysterectomy):
 - Immediate surgery is indicated if there is:
 - Extensive necrosis or gas
 - Compartment syndrome
 - Profound systemic toxicity
- Droplet precautions for the 1st 24 hr of antibiotic therapy
- Reports of successful use of IV immunoglobulin
- Hyperbaric oxygen therapy still controversial

MEDICATION

- NF due to invasive streptococcal disease (NOTE: In the ED, empiric treatment should be initiated until monomicrobial NF caused by GAS has been confirmed):
 - Clindamycin: 900 mg IV (peds: 40 mg/kg/d), *and*
 - Penicillin G: 4 million U IV (peds: 250,000 U/d), *or*
 - Vancomycin: 15 mg/kg IV (peds: 10 mg/kg q6h) if patient has penicillin allergy
- Empiric treatment of NF from all causes (*Clostridium perfringens*, GAS, methicillin-resistant *S. aureus* [MRSA], mixed anaerobes/aerobes):
 - Piperacillin/tazobactam 3.5 g IV *and*
 - Clindamycin 900 mg IV *and*
 - Vancomycin 1 g IV
 - For patients with a penicillin allergy treat with aztreonam 2 g IV, clindamycin 900 mg IV, vancomycin 1 g IV, and metronidazole 500 mg IV



FOLLOW-UP

DISPOSITION

Admission Criteria

ICU admission required for all patients with suspected invasive streptococcal infection. Mortality from GAS NF ~ 20%, but with both NF and STSS, mortality rate increases to 70%.

Discharge Criteria

None

PEARLS AND PITFALLS

- Hypotension and shock may require large volumes of IV fluids and vasopressors.
- Broad-spectrum antibiotics should be administered until the presence of GAS can be confirmed.
- Surgical consultation should be obtained for débridement.

ADDITIONAL READING

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- Nuwayhid ZB, Aronoff DM, Mulla ZD. Blunt trauma as a risk factor for group A streptococcal necrotizing fasciitis. *Ann Epidemiol.* 2007;17:878–881.
- Steer AC, Lamagni T, Curtis N, et al. Invasive group A streptococcal disease: Epidemiology, pathogenesis, and management. *Drugs.* 2012;72(9):1213–1227.



See Also (Topic, Algorithm, Electronic Media Element)

- [Pharyngitis](#)
- [Toxic Shock Syndrome](#)



CODES

ICD9

- [040.82 Toxic shock syndrome](#)
- [041.01 Streptococcus infection in conditions classified elsewhere and of unspecified site, streptococcus, group A](#)
- [728.86 Necrotizing fasciitis](#)

ICD10

- [A48.3 Toxic shock syndrome](#)
- [B95.0 Streptococcus, group A, causing diseases classd elswhr](#)
- [M72.6 Necrotizing fasciitis](#)

BASICS

DESCRIPTION

- High-pitched audible wheezing and vibratory harsh sounds mainly on inspiration.
- Impedance of air movement through the upper airway.
- It implies a laryngotracheal airway obstruction.

ETIOLOGY

- Congenital:
 - Laryngomalacia
 - Laryngeal webs/rings
- Vocal cord dysfunction:
 - Congenital
 - Surgical injury
 - Postintubation trauma
 - Thyroid malignancy
 - Mediastinal mass
 - Neural abnormalities (e.g., meningomyelocele, Arnold–Chiari malformation)
- Subglottic stenosis:
 - Postoperative scarring
 - After radiation therapy
 - After prolonged endotracheal intubation
- Subglottic hemangioma
- Infection:
 - Bacterial tracheitis
 - Epiglottitis
 - Viral croup
 - Peritonsillar abscess
 - Retropharyngeal abscess
 - Supraglottitis
 - Uvulitis (e.g., Quincke disease)
 - Ludwig angina
 - Diphtheria
 - Tetanus
- Extrinsic compression:
 - Trauma
 - Hematoma

- Vascular anomalies (e.g., rings)
- Intraluminal obstruction of the trachea:
 - Foreign body
 - Tracheomalacia
 - Cyst
 - Invasive tumors
 - Squamous cell
 - Lymphomas
 - Thyroid masses/carcinomas
 - Laryngeal or tracheal papilloma
- Angioedema

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Breathing difficulties
- Audible stridor (worsens with feeds, crying, and on lying supine)
- Muffled hoarseness “Hot potato” voice in adults
- Feeding difficulties in infants (amount of feeds and regurgitation with GERD)
- Apneas and cyanotic attacks
- Antenatal, perinatal, and birth events (e.g., resuscitation at birth with intubation)
- Anxiety
- Cough
- Drooling
- Sore throat

Physical-Exam

- Tachypnea
- Dyspnea
- Dysphagia
- Fever
- Respiratory distress, worse with agitation
- Nasal flaring, intercostal retractions, subcostal indrawing
- Paradoxical diaphragmatic movement (late finding)
- Audible stridor (inspiratory/biphasic stridor)
- Cyanosis
- Trismus:
 - Peritonsillar abscess, retropharyngeal abscess, Ludwig angina

ESSENTIAL CONSIDERATIONS

- Visualization of the upper airway:
 - Radiographic if symptoms very mild; be careful!
- Direct visualization in OR with a surgeon prepared to perform a cricothyrotomy or tracheostomy is the safest approach.

DIAGNOSIS TESTS & INTERPRETATION

Lab

These tests are not helpful and thus avoidable; may upset a child even more.

Imaging

Radiograph of lateral and posteroanterior neck and chest:

- Not essential
- Only done in extremely mild cases or when there is suspicion of foreign body aspiration

Diagnostic Procedures/Surgery

- Fiberoptic laryngoscopy:
 - Should be performed with an intubating fiberoptic laryngoscope in a setting where a rapid surgical airway can be obtained
- Direct laryngoscopy:
 - Diagnostic study of choice
 - Should be performed in a setting where a rapid surgical airway can be obtained

DIFFERENTIAL DIAGNOSIS

- Stertor:
 - Pharyngeal obstruction while wheezing
- Bronchospasm
- Malingering (patient breathing against a closed glottis)



TREATMENT

PRE HOSPITAL

- Keep child calm, with mother if possible.
- Supply blow-by oxygen.
- Maintain adequate airway.
- Use bag-valve-mask (BVM) if respiratory status deteriorates.
- Intubate if BVM ineffective.
- Provide rapid transport with ED notification.

INITIAL STABILIZATION/THERAPY

- In children: Avoid agitation. Supply blow-by oxygen.
- Use 100% nonrebreathing-type face mask
- Pulse oximetry to check oxygen saturation and monitoring of vitals.

Pediatric Considerations

- Avoid agitating child.
- Watch for rapid deterioration of respiratory status.

ED TREATMENT/PROCEDURES

- Airway management:
 - Stridor comprises a difficult airway passage:
 - Be prepared to create an airway surgically before intubation.
 - If time permits, perform intubation in OR with surgeon and pediatric anesthesiologist present.
 - Intubate with tube 1 or 2 sizes smaller than would be normally used.
- Oral awake intubation:
 - Ketamine induction
 - Patient is sedated but continues to ventilate during procedure.
- Avoid blind nasotracheal intubation.
- Oral intubation is preferred initially. After oral intubation the oral tube is replaced by a nasal tube of the same size.
- Provide surgical airway if intubation fails or sudden deterioration in respiratory status occurs.
- Postintubation ceftriaxone in cases of infectious cause
- Sedation/paralysis for duration of intubated status after airway is secured.
- Extubation could be attempted when an air leak develops around the tracheal tube, which can take around 2–10 days.
- Controversies:
 - Heliox therapy
 - Racemic epinephrine therapy
 - Early intubation

MEDICATION

- Atropine: 0.02 mg/kg IV
- Ceftriaxone: 1–2 g IV
- Diazepam: 2–10 mg IV (peds: 0.2–0.3 mg/kg)
- Etomidate: 0.3 mg/kg IV
- Fentanyl: 3 µg/kg IV
- Ketamine: 1–2 mg/kg IV or 4–7 mg/kg IM
- Lidocaine: 1.5 mg/kg IV
- Midazolam: 1–5 mg IV (0.07–0.3 mg/kg for induction)
- Vecuronium: 0.1 mg/kg IV
- Nebulized epinephrine: 1 mL of 1:1,000 diluted to 5 mL with normal saline

- Dexamethasone: 0.15 mg/kg oral/IV



FOLLOW-UP

DISPOSITION

Admission Criteria

All cases of stridor that are not completely resolved during the ED course mandate admission of patient to hospital.

Discharge Criteria

Stridor fully resolved or identified as a nonstridorous abnormal breathing sound.

Issues for Referral

Consultation with an otolaryngologist or a pediatric surgeon prior to airway visualization

PEARLS AND PITFALLS

- Attempting visualization of the airway without the backup needed for an emergency tracheostomy is a pitfall.
- Laryngoscopy findings determine the indications for other complementary exams such as barium swallow, polysomnography, echocardiography, CT, or magnetic resonance scans of neck and thorax.
- Patients, especially children with stridor, often have associated abnormalities involving respiratory tract which mandates not only endoscopic exam of the larynx, but also the tracheobronchial system.

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- Halpin LJ, Anderson CL, Corriette N. Stridor in children. *BMJ*. 2010;340:c2193.
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CODES

ICD9

- 748.2 Web of larynx
- 748.3 Other anomalies of larynx, trachea, and bronchus
- 786.1 Stridor

ICD10

- Q31.0 Web of larynx
- Q31.5 Congenital laryngomalacia
- R06.1 Stridor

SUBARACHNOID HEMORRHAGE

Alfred A. Joshua



BASICS

DESCRIPTION

- Bleeding into the subarachnoid space and CSF:
 - Spontaneous:
 - Most often results from cerebral aneurysm rupture
 - Aneurysms that occur are more likely to rupture (> 25 mm).
 - Traumatic:
 - Represents severe head injury

EPIDEMIOLOGY

- Incidence is 6–16 per 100,000 individuals.
- Affects 21,000 in US annually
- Associated mortality in 30–50% of patients
- Uncommon prior to 3rd decade; incidence peaks in 6th decade

RISK FACTORS

- Previous ruptured aneurysm who have other aneurysms
- Family history
- Hypertension
- Smoking
- Alcohol abuse
- Sympathomimetic drugs:
 - Cocaine, methamphetamine, and ecstasy (MDMA) use
- Gender (female:male 1.6:1)

Genetics

- 3–7-fold increased risk with 1st-degree relatives with subarachnoid hemorrhage (SAH)
- Strongest genetic association represents only 2% of SAH patients:
 - Autosomal dominant polycystic kidney disease, Ehlers–Danlos type IV, familial intracranial aneurysms

Pediatric Considerations

- Most often due to arteriovenous malformation in children
- Although rare in children, SAH is a leading cause of pediatric stroke.

ETIOLOGY

- “Congenital,” saccular, or berry aneurysm rupture (80–90%):

- Occur at bifurcations of major arteries
- Incidence increases with age.
- Aneurysms may be multiple in 20–30%.
- Nonaneurysmal perimesencephalic hemorrhage (10%)
- Remaining 5% of causes include:
 - Mycotic (septic) aneurysm due to syphilis or endocarditis
 - Arteriovenous malformations
 - Vertebral or carotid artery dissection
 - Intracranial neoplasm
 - Pituitary apoplexy
- Severe closed head injury

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Classically a severe, sudden headache:
 - Often described as “thunderclap” or “worst headache of life”
 - Headache is often occipital or nuchal, but may be unilateral.
 - Usually develops within seconds and peaks within minutes
 - Distinct from prior headaches
 - Headache often maximal at onset
- Sentinel headaches and minor bleeding occur in 20–50%:
 - May occur days to weeks prior to presentation and diagnosis
- Seizures, transient loss of consciousness, or altered level of consciousness occur in more than 50% of patients.
- Vomiting occurs in 70%.
- Syncope, diplopia, and seizure are particularly high-risk features for SAH.

Physical-Exam

- Focal neurologic deficits occur at the same time as the headache in 33% of patients:
 - 3rd cranial nerve (CN III) palsy (the “down and out” eye) occurs in 10–15%.
 - Isolated CN VI palsy or papillary dilation may also occur.
- Nuchal rigidity develops in 25–70%.
- Retinal hemorrhage may be the only clue in comatose patient.

ESSENTIAL WORKUP

- Complete neurologic exam and fundoscopic exam
- Emergent noncontrast head CT scan:
 - Diagnoses 93–98% of SAH if performed within 12 hr

- Thin cuts (3 mm) through base of brain improve diagnostic yield.
- CT is less sensitive after 24 hr or if hemoglobin < 10 g/L.
- Lumbar puncture (LP) and CSF analysis must be performed if CT negative and history suggests possibility of SAH.

Pregnancy Considerations

- Incidence slightly increased in pregnancy
- Workup should include CT and LP

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Baseline CBC and differential
- Electrolytes, renal function tests
- Coagulation studies
- Cardiac markers:
 - Troponin I elevated in 10–40%
- CSF analysis (see below)

Imaging

- Chest radiograph for pulmonary edema:
 - Occurs in up to 40% with severe neurologic deficit
- Traditional gold standard: 4-vessel digital subtraction cerebral angiography
- Spiral CT angiography:
 - Useful for operative planning
 - Quite sensitive for detection of aneurysms > 4 mm, less with smaller aneurysms
- MR angiography:
 - MRI is less sensitive for hemorrhage
 - Quite sensitive for detection of aneurysms > 4 mm, less with smaller aneurysms
- Transcranial Doppler ultrasound:
 - May be useful in detecting vasospasm.

Diagnostic Procedures/Surgery

- LP:
 - Presence of erythrocytes in CSF indicates SAH or traumatic tap:
 - If traumatic tap suspected, LP should be performed 1 interspace higher.
 - Diminishing erythrocyte count in successive tubes suggests but does not firmly establish a traumatic tap.
 - Xanthochromia is diagnostic of SAH if performed 12 hr after onset.

- An elevated opening pressure may indicate SAH, cerebral venous sinus thrombosis, or pseudotumor cerebri.
- ECG:
 - ST-segment elevation or depression
 - QT prolongation
 - T-wave abnormalities
 - Often mimics ischemia or infarction
 - Symptomatic bradycardia, ventricular tachycardia, and ventricular fibrillation

DIFFERENTIAL DIAGNOSIS

- Neoplasm
- Arterial dissection
- Aneurysm (unruptured)
- Arteriovenous malformation
- Migraine
- Pseudotumor cerebri
- Meningitis
- Encephalitis
- Hypertensive encephalopathy
- Hyperglycemia or hypoglycemia
- Temporal arteritis
- Acute glaucoma
- Subdural hematoma
- Epidural hematoma
- Intracerebral hemorrhage
- Thromboembolic stroke
- Sinusitis
- Seizure disorder
- Cerebral venous sinus thrombosis
- Cavernous sinus thrombosis



TREATMENT

PRE HOSPITAL

- Initial assessment and history:
 - Level of consciousness
 - Glasgow Coma Scale score
 - Gross motor deficits
 - Other focal deficits
- Patients with SAH may need emergent intubation for rapidly deteriorating level of consciousness.

- IV access should be established.
- Provide supplemental oxygen.
- Monitor cardiac rhythm.
- Patients should be transported to a hospital with emergent CT and ICU capability.

INITIAL STABILIZATION/THERAPY

- Manage airway, resuscitate as indicated:
 - _ Rapid-sequence intubation
 - _ Pretreat with lidocaine and defasciculating dose of nondepolarizing paralytic to blunt increase in intracranial pressure (ICP) during intubation.
 - _ Cardiac monitoring and pulse oximetry
 - _ Establish adequate IV access
- Obtain urgent neurosurgical consultation

ED TREATMENT/PROCEDURES

- Prevent rebleeding:
 - _ Risk of rebleeding highest in the 1st few hours after aneurysmal rupture
- Manage ICP:
 - _ Elevate head of bed to 30°.
 - _ Prevent increases in ICP from vomiting and defecation with antiemetics and stool softeners.
 - _ Treat increased ICP with controlled ventilation and mannitol.
 - _ Maintain central venous pressure > 8 mm Hg and urine output > 50 mL/hr
- BP control:
 - _ Balance HTN-induced rebleeding vs. cerebral hypoperfusion
 - _ Goal mean arterial pressure 100–120 mm Hg, systolic BP < 160:
 - Labetalol, hydralazine, nitroprusside, or nicardipine for hypertension
 - _ Correct hypovolemia:
 - Should start within 96 hr of SAH
 - Treat hypotension with volume expansion.
- Cerebral vasospasm:
 - _ May cause secondary ischemia and infarction after SAH:
 - _ Oral nimodipine improves functional outcome:
 - Discuss with neurosurgeon prior to administration
 - _ Monitor with transcranial Doppler.
- Adequately treat pain.
- Seizures:
 - _ Manage with IV benzodiazepine
 - _ Consider prophylactic anticonvulsants in immediate posthemorrhagic period
- Correct temperature, electrolyte, glucose, or pH abnormalities.
- Treat coagulopathy, thrombocytopenia, and severe anemia.
- Monitor for and correct pulmonary edema and cardiac arrhythmias.
- Antifibrinolytic therapies:

- Discuss with neurosurgeon prior to initiation
- Consider administration immediately after aneurysmal rupture in patients at high risk of rebleeding when this is combined with treatment of aneurysm and monitoring for hypotension.
- When patient is stable, expedited transfer to hospital with neurosurgical capabilities is mandatory.

MEDICATION

- Diazepam: 5–10 mg (peds: 0.2–0.3 mg/kg) IV/IM q10–1min PRN; max. 30 mg (peds: 10 mg)
- Fentanyl: 1–3 µg/kg (adults and peds) IV q1–4h PRN
- Fosphenytoin: 15–20 phenytoin equivalents (PE) per kg (adults and peds) IV × 1; maintenance 4–6 mg/kg/d IV
- Hydralazine: 10–20 mg (peds: 0.1–0.5 mg/kg IV) q30min–4h PRN
- Labetalol: 20 mg IV bolus, then 40–80 mg q10min; max. 300 mg; follow with IV continuous infusion 0.5–2 mg/min (peds: 0.4–1 mg/kg/h IV continuous infusion; max. 3 mg/kg/h)
- Lidocaine: 1–1.5 mg/kg IV × 1 (adults and peds)
- Lorazepam: 2–4 mg (peds: 0.03–0.05 mg/kg/dose; max. 4 mg/dose) IV q15min PRN
- Midazolam: 1–2 mg (peds: 0.15 mg/kg IV × 1) IV q10min PRN
- Morphine: 2–10 mg (peds: 0.05–0.2 mg/kg IV) q2–4h PRN
- Nicardipine: 5–15 mg/h IV continuous infusion (peds: Safety not established)
- Nimodipine: 60 mg PO/NGT q4h; (peds: Safety not established)
- Nitroprusside: 0.25–10 µg/kg/min IV continuous infusion (adults and peds)
- Ondansetron: 4–8 mg (peds: 0.1–0.15 mg/kg max. 4 mg) PO/IM/IV TID PRN
- Phenytoin: 15–20 mg/kg IV load at max. 50 mg/min; max. 1.5 g; maintenance 4–6 mg/kg/d IV; (adult and pediatric)
- Promethazine: 12.5–25 mg (peds > 2 yr old: 0.25–1 mg/kg; max. 25 mg/dose) PO/IM/IV q4–6h PRN

SURGERY/OTHER PROCEDURES

- Per neurosurgical consultant
- Early operative or endovascular intervention may prevent vasospasm and improve outcome.



FOLLOW-UP

DISPOSITION

Admission Criteria

- All patients with SAH should be admitted to an ICU.

- Patients with negative CT findings and equivocal LP findings should be admitted for observation.

Discharge Criteria

- Patients with negative CT and LP findings and onset of symptoms < 2 wk
- Outpatient follow-up for headache treatment and further evaluation

Issues for Referral

Early referral to center with access to neurosurgeons and endovascular specialists (if none at practicing institution)

PROGNOSIS

- Mortality is 12% before arrival to hospital.
- Ultimately fatal in more than 50%.
- In cases of “sentinel bleed” or early detection of aneurysmal rupture, outcomes are improved with early surgical or interventional approaches.

PEARLS AND PITFALLS

- Failure to consider SAH in differential diagnosis for new, acute headache
- Failure to assess previous headache workup as complete (CT and LP)

ADDITIONAL READING

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- Rabinstein AA. The AHA Guidelines for the Management of SAH: What we know and so much we need to learn. *Neurocrit Care*. 2009;10(3):414–417.
- Uysal E, Yanbuluğlu B, Ertürk M, et al. Spiral CT angiography in diagnosis of cerebral aneurysms of cases with acute subarachnoid hemorrhage. *Diagn Interv Radiol*. 2005;11(2):77–82.
- Wolfson A. Blunt neck trauma. In: Wolfson AB, Hendey GW, Hendry PL, et al., eds. *Harwood-Nuss’ Clinical Practice of Emergency Medicine*. Philadelphia, PA: Lippincott Williams & Wilkins; 2005.



CODES

ICD9

- 430 Subarachnoid hemorrhage

- 852.00 Subarachnoid hemorrhage following injury without mention of open intracranial wound, unspecified state of consciousness

ICD10

- I60.9 Nontraumatic subarachnoid hemorrhage, unspecified
- S06.6X0A Traum subrac hem w/o loss of consciousness, init

SUBDURAL HEMATOMA

Stephen R. Hayden



BASICS

DESCRIPTION

- Classification of subdural hematoma (SDH):
 - Acute: Diagnosis within the 1st 3 days
 - Subacute: Diagnosis 3 days–3 wk
 - Chronic: Diagnosis after 3 wk
- CT description:
 - Rarely crosses midline
 - Does cross suture lines
 - Inner margins are often seen to be irregular.
- Acute:
 - Most commonly due to acceleration–deceleration forces and less commonly from direct trauma
 - Sagittal movement of the head causes stretch of parasagittal bridging veins.
 - Other bleeding sites include:
 - Laceration of dura
 - Venous sinus injury
 - Cortical arteries
 - Nontraumatic injuries: Intracerebral aneurysm rupture, arteriovenous malformation, coagulation disorder, arterial HTN, drug or alcohol abuse
- Chronic:
 - Encapsulated hematoma most likely caused by repeated small hemorrhages of bridging veins.

ETIOLOGY

- Acute:
 - Most common type of intracranial hematoma (66–70%)
 - Occurs most commonly at cerebral complexities > falx cerebri > tentorium cerebelli
 - Peak incidence 15–24 yr, 2nd peak >75 yr
 - Represents 26–63% of blunt head injury
 - Motor vehicle crash (MVC) is most common cause overall.
 - Falls and assault more commonly result in isolated SDH (72%) than do MVCs (24%).
 - Elderly patients and those with seizure disorders are at increased risk.
 - Mortality is related to presenting signs and symptoms as well as

comorbidities:

- Mortality is 50% for age >70
- Less than 1/2 present as *simple extra-axial collection* (22% mortality rate)
- ~40% of patients will have *complicated SDH*: Parenchymal laceration or intracerebral hematoma (mortality rate >50%)
- 3rd group *associated with contusion* (30% mortality rate with functional recovery of 20%)
- Coagulopathy: INR >2 increases risk of bleed ×2, INR >3 is associated with larger initial volume and increased expansion
- Chronic:
 - _ Most common in babies or elderly with atrophy:
 - Associated with infarction in underlying brain
- 75% of patients are >50
- <50% have history of trauma
- 50% are alcoholic
- Epilepsy and shunting procedures

Pediatric Considerations

- May occur secondary to trauma at birth
- Nonaccidental trauma more common



DIAGNOSIS

SIGNS AND SYMPTOMS

- Acute:
 - _ 1/5 have diagnosis discovered at autopsy.
 - _ Most commonly misdiagnosed as intoxication or cerebrovascular accident (CVA)
 - _ Headache and altered mental status:
 - 50% unconscious at discovery
- Subacute/chronic:
 - _ Headaches, nausea, vomiting, and seizures are frequent symptoms.
 - _ Presentation varied:
 - Fluctuating mental status
 - Unsteady gait
 - Slow progression of deficits

Pediatric Considerations

Imaging is necessary in infants with persistent vomiting, new seizures, lethargy, irritability, bulging, or tense fontanelles.

Physical-Exam

- Acute:
 - Headache and altered mental status
 - Most common clinical signs are hemiparesis or hemiplegia:
 - Seen in 40–65%
 - SDH opposite motor deficit in 60–85%
 - Pupillary abnormality seen in 28–79%:
 - SDH will be on same side of pupillary abnormality in 70–90%.
 - Seizures may be seen in ~10% initially.
 - Papilledema in <1/3
- Chronic:
 - Presentation is varied and mimics other diseases.

ESSENTIAL WORKUP

Obtain directed history:

- Mechanism of injury kinetics
- Neurologic status: Baseline and at-scene
- Complicating factors:
 - Past medical history, medications
 - Allergies, drug use
 - Rapid neurologic assessment:
- Glasgow Coma scale ([GCS] after fluid resuscitation most important)
- Brainstem reflexes:
 - Anisocoria
 - Pupillary light reflex
 - Corneal, gag, oculocephalic/oculovestibular
 - Head imaging

DIAGNOSIS TESTS & INTERPRETATION

Lab

- ABG, CBC, electrolytes with glucose, prothrombin time (PT), partial thromboplastin time (PTT)
- Blood ethyl alcohol, drug screen

Imaging

- Head CT in coordination with other necessary trauma workup
- Acute:
 - Characteristic CT finding is crescent-shaped clot overlying hemispheric convexity.
 - May have irregular medial border of hematoma
 - Mixed density of clot may represent active bleeding
 - Most (60%) associated with other intracranial lesions

- Intracranial volume of hematoma >2% predicts poor prognosis
- Chronic:
 - MRI is a better choice, as lesion may be isodense on CT from 2–3 wk.
 - MRI volume in diffusion-weighted images correlates with Rankin disability score.
 - CT may show hypodense lesion after 3 wk.
 - Spinal radiographs

Pediatric Considerations

US can be used to visualize cerebral structures if fontanelles are patent.

DIFFERENTIAL DIAGNOSIS

- Acute:
 - Diffuse axonal injury
 - Cerebral contusion
 - Intracerebral bleed
 - Subdural hygroma
 - Epidural hematoma
 - Shaken baby/battered child syndrome
- Chronic:
 - Pseudotumor cerebri
 - Brain tumor
 - Dementia
 - Meningitis
 - CVA/transient ischemic attack
 - Cerebral atherosclerosis
 - Toxic, metabolic, respiratory, or circulatory causes



TREATMENT

INITIAL STABILIZATION/THERAPY

- Manage airway and resuscitate as indicated:
 - Hypoxia is a strong predictor of outcome.
 - Maintain SaO₂ >95%.
 - Rapid-sequence intubation (RSI) is indicated for GCS <9 or for evidence of increased intracranial pressure (ICP).
 - RSI for PaCO₂ >45, anisocoria, drop of GCS by 3, loss of gag reflex, C-spine injury
- Routine hyperventilation is no longer recommended due to resultant diminished cerebral perfusion pressure.
- Controlled ventilation to maintain PCO₂ 35–40 mm Hg:

- NS to maintain mean arterial pressure (MAP) 100–110 is necessary:
 - A single episode of systolic BP < 90 is associated with poor outcome.
- Spine precautions
- Elevate head of bed 20–30° (only after adequate fluid resuscitation to avoid resultant decrease in cerebral blood flow [CBF]).
- Not considered helpful:
 - Steroids
 - Antibiotic prophylaxis
 - Hyperventilation (unless herniation is imminent)
 - Fluid restriction
 - Calcium channel blockers
 - Hypothermia not proven
 - NaCl 3% not yet proven helpful

ED TREATMENT/PROCEDURES

- Acute
- Early neurosurgical intervention (< 4 hr) in comatose patients shows reduced mortality:
 - Burr holes may be used as temporizing measure in deteriorating patients.
 - ICP monitoring is indicated for patients with abnormal CT who are intubated.
 - Subdural evacuating port system has been shown to be equivalent to Burr hole for acute treatment of SDH
- Nonoperative treatment may be indicated for small SDH:
 - < 20 mL of blood, < 1 cm, midline shift < 5 mm, no mass effect, no neurologic deficit
 - This requires frequent neurologic reassessment.
 - 10% go on to require operative intervention.
- Maintain euvolemic state with isotonic fluids:
 - Arterial line placement to monitor MAP, PO₂, and PCO₂
 - Foley catheter to monitor I/O status
- Control ICP:
 - Prevent pain, posturing, and increased respiratory effort:
 - Sedation with benzodiazepines
 - Neuromuscular blockade with vecuronium or rocuronium in intubated patients
 - Etomidate is a good induction agent.
 - Mannitol may be used once euvolemic:
 - Shown to increase MAP > cerebral perfusion pressure and CBF as well as decrease ICP
 - Keep osmolality between 295 and 310.
 - Use furosemide (Lasix) as an adjunct only if normovolemic.
 - Treat HTN:

- Labetalol, nicardipine, or hydralazine
- Treat coagulopathy
- Use fresh frozen plasma 4+ units
- Use prothrombin complex concentrate
- Treat hyperglycemia if present:
 - Associated with increased mortality in traumatic brain injury
- Treat and prevent seizures:
 - Diazepam and phenytoin (Dilantin), levetiracetam: Prophylactic anticonvulsants not indicated

MEDICATION

- Diazepam: 5–10 mg (peds: 0.2–0.3 mg/kg) IV/IM q10–15min PRN; max. 30 mg (peds: 10 mg)
- Dilantin: Adults and peds: Load 18 mg/kg at 25–50 mg/min
- Etomidate: 0.3 mg/kg IV for induction of RSI
- Fentanyl: 2–4 µg/kg
- Hydralazine: 10–20 mg (peds: 0.1–0.5 mg/kg IV) q2–4h PRN
- Labetalol: 20 mg IV bolus, then 40–80 mg q10min; max. 300 mg; follow with IV continuous infusion 0.5–2 mg/min; (peds: 0.4–1 mg/kg/h IV continuous infusion; max. 3 mg/kg/h)
- Lasix: Adults and peds: 0.5 mg/kg IV
- Levetiracetam: 1,500 mg PO/IV q12h
- Lidocaine: As preinduction agent, 1.5 mg/kg IV
- Mannitol: Adults and peds: 0.25–0.5 g/kg IV q4h
- Midazolam: 1–2 mg (peds: 0.15 mg/kg IV × 1) IV q10min PRN
- Nicardipine: 5–15 mg/h IV continuous infusion (peds: Safety not established)
- Pentobarbital: 1–5 mg IV q6h
- Prothrombin complex concentrate: 50 U/kg IV
- Rocuronium: 1 mg/kg for induction
- Thiopental: As induction agent, 20 mg/kg IV



FOLLOW-UP

DISPOSITION

Admission Criteria

- Acute SDH patients should be admitted to the operating room or ICU by the neurosurgical service.
- Subacute subdurals should be admitted to a monitored setting.

Discharge Criteria

Patients with chronic SDH often can be managed as outpatients in conjunction with

neurosurgery, adequate home resources, and appropriate follow-up.

Issues for Referral

All patients need neurosurgical evaluation immediately.

PEARLS AND PITFALLS

The following factors predict prognosis:

- GCS on admission
- Time to treatment
- Pupil abnormalities
- CT volume of hematoma and presence of midline shift
- Midline shift > hematoma volume

ADDITIONAL READING

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CODES

ICD9

- 432.1 Subdural hemorrhage
- 767.0 Subdural and cerebral hemorrhage
- 852.20 Subdural hemorrhage following injury without mention of open intracranial wound, unspecified state of consciousness

ICD10

- I62.00 Nontraumatic subdural hemorrhage, unspecified
- P10.0 Subdural hemorrhage due to birth injury

- S06.5X0A Traum subdr hem w/o loss of consciousness, init

SUDDEN INFANT DEATH SYNDROME (SIDS)

Genie E. Roosevelt



BASICS

DESCRIPTION

- Sudden, unexpected death of an infant <1 yr old who was typically well before being placed down to sleep
- Death remains unexplained after being thoroughly investigated by autopsy, exam of the death scene, investigation of the circumstances, and review of the family and infant medical histories.
- Leading cause of death in infants 1 mo–1 yr of age; the incidence has declined markedly since the initiation of the “Back to Sleep” program in 1994:
 - 1992: 120 deaths/100,000 live births (US)
 - 2001: 56 death/100,000 live births (US)
 - No change from 2001–2006
- Peak occurrence of SIDS at 1–4 mo of age:
 - 90% occur <6 mo of age
 - 2% occur >10 mo of age
- Ethnic differences: 2006 rates per 100,000 live births: All populations, 54.5; non-Hispanic white, 55.6; non-Hispanic black, 103.8; American Indian/Alaska Natives, 119.4; Asian American or Pacific Islander, 22.8; Hispanic, 27.
- Sleeping on back (supine) reduces incidence significantly (“Back to Sleep”). Practice of infants sleeping on their backs began initially in Europe and then in US

ETIOLOGY

- Most likely multifactorial
- SIDS infants likely have predisposing conditions that make them more vulnerable to both internal and external stressors.
- Potential stressors include anemia, congenital diseases, dysrhythmias, electrolyte abnormalities, genetic defects, infection, metabolic disorders, neurologic events, suffocation, trauma, upper airway obstruction.
- Maternal and antenatal risk factors:
 - Alcohol and illicit drug use
 - Intrauterine growth restriction
 - Lower socioeconomic status
 - Poor prenatal care
 - Prior sibling death secondary to SIDS
 - Shorter interval between pregnancies
 - Smoking
 - Younger age

- Infant risk factors:
 - Bed sharing
 - Exposure to environmental smoking
 - Gastroesophageal reflux (GER)
 - Hyperthermia
 - Low birth weight, prematurity
 - Male gender
 - Soft bedding, soft sleeping surface
 - Recent febrile illness
- Supine sleeping position, breast-feeding, and pacifier use are protective.
- Home monitoring has not been shown to prevent SIDS.



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- No significant pre-existing signs or symptoms to alert caretakers
- Unpredictable
- Most infants appear normal when put to bed.
- Death occurs while the infant is sleeping.
- Typically the event is silent with no signs of struggling.
- No clinical or pathologic explanation for death.
- Apparent life-threatening event (ALTE) is an acute event that is frightening to the caretaker:
 - Characterized by apnea (either central or obstructive) causing changes in skin color—cyanosis, pallor, or erythema with limpness, choking, and/or gagging.
 - Infant should be transported to hospital for evaluation and monitoring.
 - Appears well when evaluated by clinicians after recovery from ALTE.
 - Associated with an increased risk of SIDS.

Physical-Exam

- Prior to the event, the infant is seemingly healthy and well appearing, well developed, and well nourished.
- If event was brief and self-limited, may appear well when evaluated after the episode.
- Potential complications for surviving infants include pulmonary edema, aspiration pneumonia, and neurologic sequelae secondary to hypoxia including seizures.

ESSENTIAL WORKUP

- SIDS is a diagnosis of exclusion, so requires an evaluation to identify primary

and/or contributing conditions.

- Thorough investigation of the death scene:
 - Conditions surrounding sleeping space (temperature, surface, bedding, bed sharing)
 - Position in which infant was sleeping
 - Interview of parents, family members, and caregivers
 - Exam of potentially relevant items from the death scene
 - Maintain sensitivity toward family as investigation may be difficult for them.
- Review infant and family histories:
 - Prenatal, perinatal, and postnatal infant medical history
 - Family medical and social histories, particularly mother
- Impact of investigation on family:
 - Family is very vulnerable during the investigation
 - May help them through the grieving process.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Selective studies reflecting nature of episode and patient condition
- Arterial/venous blood gas
- Blood culture and other sepsis workup as indicated
- CBC
- ECG
- Include family member to evaluate for familial dysrhythmias such as prolonged QT syndrome
- EEG
- Electrolytes including calcium, magnesium and phosphorous. Liver function tests
- Toxicology screen
- UA and culture

Imaging

- Chest radiograph to assess cardiopulmonary status
- Skeletal survey to evaluate for child abuse (may be performed by pathologist)
- Head CT if child survives to assess intracranial pathology
- Consider upper GI to evaluate for GER

Diagnostic Procedures/Surgery

- Autopsy:
 - Most states require an autopsy for potential SIDS cases
 - Important that postmortem exam be performed as SIDS is a diagnosis of exclusion

- Involves microscopic exam of vital organs through tissue samples as well as gross exam
- Some postmortem findings in SIDS cases that might establish alternative cause of death:
 - o Congenital cardiomyopathies
 - o Cardiac rhabdomyomas
 - o Tuberous sclerosis
 - o Rare genetic/metabolic diseases
 - o Viral myocarditis
 - o Intracranial arteriovenous malformations

DIFFERENTIAL DIAGNOSIS

- Cardiovascular:
 - Anomalous coronary artery
 - Aortic stenosis
 - Cardiomyopathy
 - Dysrhythmia
 - Myocarditis
- Respiratory:
 - Aspiration
 - Suffocation
- Infection:
 - Botulism
 - Bronchiolitis/respiratory syncytial virus
 - Encephalitis
 - Meningitis
 - Pertussis
 - Sepsis
- CNS:
 - Arteriovenous malformation
 - Central hypoventilation
 - Neuromuscular disorders
 - Seizures
 - Tuberous sclerosis
- GI:
 - GER
 - Diarrhea
 - Pancreatitis
 - Volvulus
- Endocrine/metabolic:
 - Carnitine deficiency
 - Congenital adrenal hyperplasia
 - Glycogen storage disease

- Long- or medium-chain acyl-coenzyme A deficiency
- Urea cycle defect
- Systemic:
 - Child abuse
 - Dehydration
 - Intentional poisoning
 - Hyperthermia



TREATMENT

- Initiate resuscitation at the scene; transport infant to ED and continue protocols en route.
- On very rare occasion and under medical direction, resuscitations have been aborted and the infant is pronounced at the scene; consideration must be given to the emotional, social, and clinical circumstances.

PRE HOSPITAL

- Resuscitation procedures supplemented by support for the family
- Evaluate setting; determine if suspicion of abuse

INITIAL STABILIZATION/THERAPY

- Assess and support ABCs (bedside).
- Administer appropriate medications per protocols by endotracheal tube if IV access unobtainable (atropine, epinephrine, lidocaine, and naloxone).
- Monitor vital signs: BP, heart rate, respirations, and oxygen saturation continuously.
- Conduct a thorough physical exam; look for unintentional as well as intentional traumas.
- Assess the scene, family members, and other caretakers.

ED TREATMENT/PROCEDURES

- Resuscitate patient per established protocols continuing efforts initiated by pre-hospital personnel:
 - Health care providers are encouraged to offer family members the opportunity to be present during resuscitation.
- If resuscitation unsuccessful and no obvious diagnosis found, parents should not be told that SIDS is the cause of death:
 - In speaking with the parents, SIDS may be included among the possible causes of death.
 - A diagnosis cannot be made until completion of an autopsy, investigation of circumstances and death scene, and exploration of the medical histories of the infant and family.
- Family support:

- If resuscitation unsuccessful, attention should then focus on the family; if resuscitation ongoing, communication and support of family is essential.
- All family members and caregivers are affected; they experience grief, guilt, failure, and inadequacy.
- Some parents want to spend quiet time holding their infants after an unsuccessful resuscitation.
- Family is defined variably among different cultures; ED personnel should attempt to be sensitive to cultural needs and expectations of the family.
- Family should be offered support in the ED and supplied with resources of support for beyond the day of the infant's death; local, state, and national SIDS Foundation resources should be made available.
- Support may be obtained from Sudden Infant Death Syndrome Alliance/First Candle, 1314 Bedford Avenue, Suite 210, Baltimore, MD 21208 (800-221-7437) or local SIDS support organization.
- The child's PCP should be involved in the follow-up and support of the family.
- Emergency personnel support:
 - ED debriefing should be conducted for all staff who were involved in the infant's care, including EMS personnel; it is important to allow people to express their feelings and freely process the event in a supportive environment.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Observe all infants who have ALTE for evaluation and monitoring after initial resuscitation and stabilization.
- Most high-risk infants have 1 of the following variables: Obvious need for admission, significant medical history, and > 1 ALTE in 24 hr.

Discharge Criteria

Patients are generally admitted for observation and monitoring for documented episodes and support of family.

Issues for Referral

- All surviving infants should have a pediatric consultation.
- Families will need support.

PEARLS AND PITFALLS

Infants with SIDS or ALTE should be resuscitated appropriately. Autopsies are essential for diagnosis and should be considered mandatory. Use available resources including social workers and chaplains as support for the family is crucial.

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See Also (Topic, Algorithm, Electronic Media Element)

- Abuse, Child
- Apnea, Pediatric
- Resuscitation, Neonatal; Resuscitation, Pediatric



CODES

ICD9

798.0 Sudden infant death syndrome

ICD10

R99 Ill-defined and unknown cause of mortality

SUICIDE, RISK EVALUATION

Helen M. Farrell • Vithya Balasubramaniam



BASICS

DESCRIPTION

- The intentional taking of one's own life
- Suicidal ideation:
 - Passive: A conscious desire not to live
 - Active: Intention to die with or without a plan
- Parasuicidal behavior: Self-injury not intended to cause death (e.g., superficial cutting, cigarette burns, head banging)
- Reckless behavior: Not taking prescribed medications, taking too much of prescribed medications, running into traffic
- Risk-to-rescue ratio—lethality of plan compared with likelihood of rescue:
 - High risk-to-rescue ratio indicates increased severity of attempt.
- Occult presentation:
 - Many individuals at risk for suicidal behavior seek care in the ED for nonbehavioral complaints
 - Improved suicide screening practices may be needed to capture this population.

ETIOLOGY

- 36,891 suicides in US (CDC 2009)
- 12–25 attempts per every completed suicide
- 25.4 per 100,000 males (CDC 2009)
- 7.4 per 100,000 females
- 11.1 per 100,000 general population
- 2 peaks in age group most at risk for suicide:
 - Age 15–24 yr (3rd leading cause of death in this age group)
 - Age > 60 yr (highest rates of any age group, increasing incidence with age)

Risk Factors for Suicidal Behavior

- Depression (bipolar or unipolar)
- Alcohol or drug abuse
- History of physical or sexual abuse
- Unemployment
- Incarceration
- History of head injury or neurologic disorder
- Firearms in the home
- Cigarette smoking

- Positive family history of suicide attempt
- Psychiatric or medical comorbidities
- Gender:
 - Women 3 times more likely to attempt suicide.
 - Men 3 times more likely to complete suicide.
- Psychological:
 - Impulsivity/aggression
 - Depression
 - Anxiety
 - Hopelessness
 - Self-consciousness/social disengagement
 - Poor problem-solving abilities
 - Lack of social supports
 - Widowed
 - Divorced
 - Separated
 - Lack of social supports
 - Recent loss of relationship
 - Anniversary of loss
- Environmental
- Rural areas:
 - Access to firearms
 - Poverty
 - Unemployment

Risk Factors for Completed Suicide

- Male
- Age > 60 yr
- White or Native American
- Widowed/divorced
- Living alone
- Unemployment/poverty
- Past suicide attempt

Methods of Suicide (CDC 2009)

- Firearms (most common among men and 2nd most common in women)
- Overdose (Most common among women); most common means of suicide attempt (70% of failed attempts are by overdose)
- Hanging
- Suffocation

Populations at Highest Risk for Completing Suicide

- > 90% of patients who commit suicide have a psychiatric diagnosis.
- Depression—especially psychotic depression
- Anxiety and panic disorder
- Alcohol or drug intoxication
- Schizophrenia
- Adolescents

Others at Risk for Completing Suicide

- Recent discharge from psychiatric facility
- History of suicidal ideation or suicide attempt
- Serious physical illness present in up to 70% of all suicides, particularly in elderly patients.
- History of incarceration
- Physicians
- Victims of violence/abuse

Interventions that Lower Risk

- Patients with mood disorders (major depression and bipolar disorder) treated with lithium
- Patient with major depression treated with electroconvulsive therapy
- Patients with schizophrenia treated with clozapine
- **NOT** shown to decrease suicide rates: Treatment with selective serotonin reuptake inhibitors (SSRIs) for major depression

Protective factors

- Strong social supports
- Family cohesion
- Peer group affiliation
- Good coping and problem-solving skills
- Positive values and beliefs
- Ability to seek and access help

DIAGNOSIS

SIGNS AND SYMPTOMS

- Depressed mood
- Verbalization of suicidal ideation with or without plan
- Hopelessness
- Helplessness
- Anger/aggression
- Impulsivity
- Psychotic symptoms (i.e., paranoia, command auditory hallucinations)

History

- Obtain history to assess risk:
 - Asking about suicide does not increase risk for attempt
- Degree of suicidal ideation
- Plan immediate risk of self-injury?
 - Means available to complete plan
 - Activity toward initiating plan
 - Patient's expectations of lethality of plan
- Intent: Reasons, goal
- Risk-to-rescue ratio
- Plan or intent to harm others?
- Presence of acute precipitants:
 - Recent losses, lack of social supports
- Risk factors:
 - History of past suicide attempts
 - Psychiatric review of symptoms: Depression, psychosis, panic/anxiety
 - Chronic medical illness
 - Alcohol or drug abuse
- Serial assessment of mental status, consistency of responses
- Factors preventing suicide

Physical-Exam

- As needed to address acute medical issues
- Look for evidence of injuries and signs of self-neglect.

Scoring Systems

- Modified SAD PERSONS Score:
 - Sex: Male 1 point
 - Age <19 or >45 yr 1 point
 - Depression or hopelessness 2 points
 - Previous attempts or psychiatric care 1 point
 - Excessive alcohol or drug use 1 point
 - Rational thinking loss 2 points
 - Separated/divorced/widowed 1 point
 - Organized or serious attempt 2 points
 - No social supports 1 point
 - Stated future intent 2 points
 - Data suggests that patients with a score of <5 can safely be managed as an outpatient

ESSENTIAL WORKUP

- Collateral information from outpatient care givers, family, friends
- Safety plan:

- Would the patient immediately seek help if suicidal ideation recurred?
- Elimination of means of suicide
- Access to other means of suicide
- Support and supervision in the outpatient setting
- Prompt outpatient follow-up with psychiatric therapy
- Patient investment in not attempting suicide
- Identifying reasons for living
- Safety contracts are no guarantee that individuals will not attempt suicide.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Blood–alcohol level
- Serum toxicology screen: Aspirin, acetaminophen, and other medications
- Urine drug screen:
 - Many psychiatric facilities require toxicology screen before placement.
- Carbon monoxide (as indicated)

Imaging

Not routinely indicated

Diagnostic Procedures/Surgery

ECG – as indicated

DIFFERENTIAL DIAGNOSIS

- Normal despondency
- Bereavement
- Adjustment disorder with depressed mood
- Major depressive disorder
- Bipolar disorder
- Organic mental disorder (head injury, dementia, delirium)
- Schizophrenia
- Panic and anxiety disorders
- Alcohol or drug abuse
- Borderline personality disorder
- Antisocial personality disorder
- Accidental death
- Attempted homicide

Pediatric Considerations

- Suicide is a leading cause of death among young people 15–24 yr of age.
- More than 4,000 adolescents commit suicide every year (CDC 2009)
- Rapidly increasing in young black males ages 10–14 yr

- Less evidence available to link suicide in youth to overt psychiatric illness
- Stresses:
 - Prior attempts
 - Family disruption
 - History of psychiatric disorder
 - Depression
 - Disciplinary crisis
 - Broken romance
 - School difficulties
 - Bereavement
 - Rejection
 - History of physical or sexual abuse
- Early warning signs:
 - Progressive declining schoolwork
 - Multiple physical complaints
 - Substance abuse
 - Disrupted family relations
 - Social withdrawal
 - Anhedonia

Geriatric Considerations

- Suicide rates highest in age > 65 yr
- Completed suicide: 83% men
- Risk factors: Divorced, widowed, male, social isolation
- Tend to use more lethal methods
- Lower ratio of attempts to completions



TREATMENT

PRE HOSPITAL

- For potentially dangerous patient who refuses transport to treatment facility; involve police and impose restraint.
- Risk to medics on the scene in cases of firearms or other weapons
- Know state and local laws, availability of mobile crisis units, and when to involve the police.

INITIAL STABILIZATION/THERAPY

- Prevent ability to elope
- Ensure patient safety:
 - Remove sharp objects, belts, shoelaces, and other articles that could be used for self-injury
- Provide safe environment

- Appropriate supervision

ED TREATMENT/PROCEDURES

- Confer with patient's outpatient therapist or physician if possible
- Voluntary admission to psychiatric facility
- Involuntary admission if patient refuses voluntary
- For involuntary psychiatric admission, patient must have psychiatric disorder and 1 of the following:
 - _ Risk for danger to self
 - _ Risk for danger to others
 - _ Inability to care for self

MEDICATION

Treat underlying psychiatric disorder.



FOLLOW-UP

DISPOSITION

Admission Criteria

- If patient endorses suicidal ideation with plan and intent, admission may be needed for safety.
- If impulsivity, anger, or aggression hinder ability to control behavior

Discharge Criteria

- Patient has no suicidal ideation.
- Patient agrees to return to ED immediately or seek psychiatric help if suicidal ideation recurs.
- Patient has passive suicidal ideation without plan or intent.
- Patient has good support network or placement in appropriate crisis housing
- Appropriate outpatient psychiatric follow-up is ensured.
- In some cases, patients who express suicidal ideation while intoxicated may be discharged if no longer suicidal once they are sober.
- Some patients with borderline personality disorder and chronic suicidal ideation are discharged after careful psychiatric evaluation in consultation with long-term outpatient caregivers.

FOLLOW-UP RECOMMENDATIONS

Close psychiatric follow-up for those with acute illness who do not require admission

PEARLS AND PITFALLS

- A careful history will identify risk factors for suicide.

- Access collateral sources of information about patient's recent thoughts and behavior.
- Maintain patient safety during evaluation
- Hospital admission may be required if patient endorses suicidal ideation and plan.

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See Also (Topic, Algorithm, Electronic Media Element)

Depression



CODES

ICD9

- 311 Depressive disorder, not elsewhere classified
- V17.0 Family history of psychiatric condition
- V62.84 Suicidal ideation

ICD10

- R45.851 Suicidal ideations
- Z81.8 Family history of other mental and behavioral disorders
- Z91.5 Personal history of self-harm

SUPRAVENTRICULAR TACHYCARDIA

James G. Adams • Matthew S. Patton

BASICS

DESCRIPTION

- Rhythm that originates ectopically above the His bundle
- Heart rate of 100 bpm or greater
- Irregular narrow complex supraventricular tachycardia (SVT):
 - Atrial fibrillation (AF):
 - Most common form of pathologic SVT seen in the ED
 - 10% of people >75 yr of age have AF.
 - Atrial flutter with variable block
 - Multifocal atrial tachycardia
- Regular narrow complex SVT:
 - Atrial flutter
 - Atrioventricular nodal re-entry tachycardia (AVNRT):
 - 60% of SVT in adults, 70% are female
 - Typically present age 30–40 yr
 - Atrioventricular reciprocating tachycardia (AVRT) involving an accessory pathway
- Wide complex SVT:
 - Conduction is outside of the normal His-Purkinje system.
 - Accessory pathway or a bundle branch block is present.
 - More common in younger patients without structural disease
 - Always suspect a ventricular rhythm with a wide complex rhythm
 - Treat as ventricular tachycardia (VT) unless absolutely certain of SVT

ETIOLOGY

- Atrial tachycardia:
 - Precipitated by a premature atrial or ventricular contraction
 - Electrolyte disturbances
 - Drug toxicity (i.e., theophylline)
 - Hypoxia
 - Increased atrial pressure
- Junctional tachycardia:
 - AV nodal re-entry
 - Myocardial ischemia
 - Structural heart disease
 - Pre-excitation syndromes
- Wolff–Parkinson–White (WPW) syndrome:

- Intrinsic accessory pathway
- Drug and alcohol toxicity
- AF:
 - HTN
 - Coronary artery disease
 - Hypo-/hyperthyroidism
 - Heavy alcohol intake
 - Mitral valve disease
 - Chronic pulmonary disease
 - Pulmonary embolus
 - WPW syndrome
 - Hypoxia
 - Digoxin toxicity
 - Chronic pericarditis
 - Sepsis
- Atrial flutter:
 - Ischemic heart disease
 - Valvular heart diseases
 - CHF
 - Myocarditis
 - Cardiomyopathies
 - Pulmonary embolus
 - Other pulmonary disease

DIAGNOSIS

SIGNS AND SYMPTOMS

- Palpitations (most common)
- Lightheadedness, pressure in the head
- Dyspnea
- Diaphoresis
- Dizziness
- Weakness
- Chest discomfort
- Syncope
- Prominent neck veins “frog sign”
- Signs of instability:
 - Mental status changes
 - Chest pain/ischemia
 - Acute pulmonary edema
 - Hypotension

History

- Abrupt onset of palpitations, lightheadedness, weakness, chest pain:
 - Current symptoms
 - Previous episodes
- Insidious onset of generalized weakness, exercise intolerance, and malaise
- Prior cardiac history
- Medications:
 - Over-the-counter, decongestants
- Illicit drug use

Physical-Exam

- Vital signs:
 - Tachycardia
 - BP normal or hypotensive
 - Respiratory rate normal or tachypneic
- Cardiac:
 - Regular or irregularly irregular rhythm
 - JVD may be present in setting of heart failure
- Pulmonary:
 - Rales may be present in setting of heart failure.

ESSENTIAL WORKUP

- ABCs, assess stable vs. unstable
- History
- EKG

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC
- Electrolytes
- Cardiac enzymes, BNP
- Thyroid function (usually low yield)

Imaging

- CXR:
 - Assess cardiac size
 - Evaluate for pulmonary process
 - More useful in AF/flutter

Diagnostic Procedures/Surgery

- EKG:
 - AF:

- Atrial fibrillatory waves without discernable P-waves
- Irregularly irregular ventricular rate of 100–220
- Atrial flutter:
 - Regular atrial rate usually > 300
 - Beat-to-beat uniformity of cycle length, polarity, and amplitude
 - Sawtooth flutter waves directed superiorly and most visible in leads II, III, aVF
 - AV block, usually 2:1, but occasionally greater or irregular
- Multifocal atrial tachycardia:
 - 3 distinctly different P-waves with varying pulse rate intervals
- Atrial tachycardia:
 - Rate of 100–200 bpm
 - P-wave precedes QRS and is morphologically different from the sinus P-wave.
- Junctional tachycardia:
 - Usually 1:1 conduction, with ventricular rates equaling the atrial rate.
 - May be either paroxysmal or sustained
 - Ventricular rates > 200 bpm in an adult suggest an accessory pathway syndrome such as WPW syndrome.
 - Absence of preceding P-waves
 - Often retrograde P-waves buried in the QRS

DIFFERENTIAL DIAGNOSIS

- Sinus tachycardia:
 - Sepsis
 - Hypovolemia
 - Pericardial tamponade
 - Acute MI
 - Drug intoxication
- Wide complex tachycardias:
 - Distinguish between supraventricular with aberrancy or ventricular origins.



TREATMENT

PRE HOSPITAL

- Supplemental oxygen
- IV access
- Monitor

INITIAL STABILIZATION/THERAPY

- IV access
- Oxygen

- Monitor

ED TREATMENT/PROCEDURES

- AF/atrial flutter:
 - AF is most likely diagnosed when the rhythm is irregular
 - When unstable, then immediate synchronized cardioversion
 - When stable, rate control is a priority:
 - β -blockers or calcium channel blockers, amiodarone, and digoxin
 - Cardioversion in stable patients should not be attempted unless the dysrhythmia is known to be acute (< 48 hr in duration) due to risk of embolism
- WPW syndrome:
 - Consider direct current cardioversion or amiodarone, flecainide, or procainamide.
 - Avoid AV node blocking agents such as adenosine, β -blockers, calcium channel blockers, and digoxin.
- In regular narrow complex SVTs:
 - Vagal maneuvers will occasionally terminate the dysrhythmia:
 - Carotid massage (although beware of carotid disease, especially in elderly)
 - Ice to face in children (mammalian diving reflex)
 - Valsalva maneuver
 - If this is unsuccessful, adenosine is the drug of choice.
 - Adenosine 6 mg will convert 60–80% of SVT
- Wide complex SVT:
 - Try to determine whether VT or SVT with aberrancy
 - If in doubt must be treated as VT
 - Brugada criteria may help identify VT (See “Ventricular Tachycardia”).
 - Verapamil is absolutely contraindicated.
 - Adenosine should be reserved for SVT with aberrancy and is rarely indicated.
 - Electrical cardioversion:
 - Fewer potential complications than antiarrhythmic drugs when mechanism unknown
 - Antidysrhythmic drugs:
 - IV procainamide and IV amiodarone
 - Lidocaine is less effective, although sometimes more readily available.
 - Bretylium lacks any evidence of efficacy.

Pediatric Considerations

- Synchronized cardioversion for unstable patient 0.5–1 J/kg
- SVT is the most common dysrhythmia seen in young adults and children without underlying heart disease:

- Initial vagal maneuvers:
 - Infants: Ice/water bag to forehead × 15 sec
 - Children: Valsalva: “Blow into straw”
- Aberrant conduction:
 - WPW syndrome and AVNRT are the 2 most common forms of SVT seen in children.
- Use verapamil only > 1 yr of age.

Pregnancy Considerations

- Adenosine considered safe
- 2nd-line agents IV propranolol or metoprolol
- Avoid verapamil (maternal hypotension)
- Cardioversion is safe.

MEDICATION

- Adenosine: 6 mg (peds: 0.1 mg/kg up to 6 mg) rapid IVP; if no response after 1–2 min, then 12 mg (peds: 0.2 mg/kg up to 12 mg), may repeat 12 mg (0.2 mg/kg)
- Amiodarone: Load with 15 mg/min IV over 10 min (peds: 5 mg/kg over 20–60 min), then 1 mg/min IV for 6 hr, then 0.5 mg/min IV for 18 hr
- Digoxin: 0.5 mg IV initially, then 0.25 mg IV q4h
- Diltiazem: 0.25 mg/kg IV (usually 10–20 mg) over 2 min, followed in 15 min by 0.35 mg/kg IV over 2 min
- Esmolol: 0.5 mg/kg IV over 1 min; maintenance infusion, 0.05 mg/kg/min IV over 4 min, then 0.1–0.2 mg/kg/min IV continuously
- Lidocaine: 100 mg IV
- Metoprolol: 5–15 mg slow IV push at 5-min intervals to total of 15 mg
- Procainamide: 20–30 mg/min IV up to 17 mg/kg, may increase to 50 mg/min for more urgent situations
- Propranolol: 0.1 mg/kg div. into equal doses at 2–3-min intervals
- Sotalol: Load 10 mg/min IV up to 1–1.5 mg/kg body weight
- Verapamil: 2.5–5 mg IV bolus over 2 min; may repeat with 5–10 mg q15–30min to max. of 20 mg



FOLLOW-UP

DISPOSITION

Admission Criteria

- Possible cardiac ischemic event
- Persistent SVT
- Possible pre-excitation syndrome

- Other underlying metabolic abnormalities

Discharge Criteria

Terminated rhythm without organ hypoperfusion

Issues for Referral

If there are no concerns for underlying cardiac disease or metabolic derangement, a patient with uncomplicated SVT that is successfully treated may be discharged to follow-up with a primary doctor or cardiologist.

FOLLOW-UP RECOMMENDATIONS

The patient should return to the ED if feeling faint, dizzy, numbness or weakness of the face or limbs, or trouble seeing or speaking. Avoid high-risk activities (swimming, piloting, diving, etc.) until further evaluation.

PEARLS AND PITFALLS

- Valsalva maneuvers should, ideally, be attempted with the patient lying flat. Despite the modest likelihood of success, the maneuver is simple and efficient.
- AF is the most worrisome rhythm in patients with an accessory pathway such as WPW. At high ventricular rates, AF can appear deceptively regular, but should not be mistaken for benign SVT.
- When adenosine has no apparent effect, an escalating dose beyond 12–18 mg, is sometimes used. However, if any lower adenosine dose transiently slows the heart rhythm, but the fast rate quickly resumes, then an increased dose is not warranted and an alternative medication should be used.
- A wide complex tachycardia of uncertain etiology should be treated as VT, typically with amiodarone and potentially with procainamide if an accessory pathway is possible.
- Since procainamide can be administered at a maximum rate of 50 mg/min, it takes a minimum of 20 min to administer 1 g, or 30 min to administer 1.5 g. Therefore, request the medicine promptly to optimize timing of administration. If QRS widening or hypotension occur, slow the rate of administration or discontinue the medication.

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- Manole MD, Saladino RA. Emergency department management of the pediatric patient with supraventricular tachycardia. *Pediatr Emerg Care*. 2007;23(3):176–189.
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See Also (Topic, Algorithm, Electronic Media Element)

Ventricular Tachycardia



CODES

ICD9

- 427.0 Paroxysmal supraventricular tachycardia
- 427.89 Other specified cardiac dysrhythmias

ICD10

I47.1 Supraventricular tachycardia

SYMPATHOMIMETIC POISONING

Sean Patrick Nordt



BASICS

DESCRIPTION

- Direct or indirect stimulation of adrenergic receptors in sympathetic and central nervous systems
- Often no correlation between dosage and degree of toxicity
- Cocaine may also block sodium channels of cardiac myocytes, leading to “tricyclic” or class 1a–type dysrhythmias.

Pediatric Considerations

- Sympathomimetic poisoning in children may present similarly to meningitis or other systemic illness.
- Urinary toxicology screening may be only way to discover sympathomimetic poisoning in children presenting with altered mental status.
- Methylphenidate (Ritalin, Concerta) and other sympathomimetics used for ADHD may cross-react with altered mental status.

ETIOLOGY

- Sympathomimetic toxicity can result from use of any sympathetically active drug, including:
 - All amphetamines, methamphetamines, and derivatives (ecstasy, MDMA)
 - Cocaine
 - Synthetic cathinones “Bath Salts”
 - Phencyclidine (PCP)
 - Lysergic acid diethylamide (LSD)
 - Decongestants (rare)
- Drug delivery routes: Inhalation, injection, snorting, or ingestion



DIAGNOSIS

SIGNS AND SYMPTOMS

- Vital signs:
 - Tachycardia:
 - Bradycardia possible for cocaine and some other decongestants
 - Increased BP:
 - Severely intoxicated patients may be hypotensive.
 - Tachypnea
 - Hyperthermia:

○ Often present, may be severe, and is often overlooked

- CNS:
 - Anxiety
 - Headache
 - Agitation
 - Altered mentation
 - Diaphoresis
 - Seizures
 - Stroke
 - Dystonia (rare)
- Cardiovascular:
 - Palpitations
 - Chest pain
 - Myocardial ischemia or infarction
 - Tachydysrhythmias
 - Cardiovascular collapse
 - Murmur (e.g., endocarditis)
- Other:
 - Dilated pupils
 - Dry mucous membranes
 - Urinary retention may cause enlarged bladder.
 - Needle track marks or abscesses on extremities should be sought.
 - Increased or decreased bowel sounds
 - The presence of diaphoresis and bowel sounds may help to differentiate sympathomimetic toxicity from anticholinergic poisoning.

History

- Assess history for possible sympathomimetic agents:
 - Cold preparations
 - Prescription amphetamines
 - Recreational drug use
- Assess for possible coingestions
- Evaluate for symptoms of end organ injury:
 - Chest pain
 - Shortness of breath
 - Headache, confusion, and vomiting

Physical-Exam

- Common findings include:
 - Agitation
 - Tachycardia
 - Diaphoresis

- Mydriasis
- Severe intoxication characterized by:
 - Tachycardia
 - Hypertension
 - Hyperthermia
 - Agitated delirium
 - Seizures
 - Diaphoresis
- Hypotension and respiratory distress may precede cardiovascular collapse
- Evaluate for associated conditions:
 - Cellulitis and soft tissue infections
 - Diastolic cardiac murmurs or unequal pulses
 - Examine carefully for trauma
 - Pneumothorax from inhalation injury
 - Focal neurologic deficits

ESSENTIAL WORKUP

- Monitor vital signs:
 - Increased temperature (>40°C possible):
 - Core temperature recording essential
 - Peripheral temperature may be cool
 - Indication for urgent cooling
 - Ominous prognostic sign
 - BP:
 - Severe hypertension can lead to cardiac and neurologic abnormalities.
 - Late in course, hypotension may supervene.
- ECG:
 - Signs of cardiac ischemia
 - Ventricular tachydysrhythmias
 - Reflex bradycardia

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Urinalysis for:
 - Blood
 - Myoglobin
- Electrolytes, BUN/creatinine, glucose:
 - Hypoglycemia may contribute to altered mental status.
 - Acidosis may accompany severe toxicity.
 - Rhabdomyolysis may cause renal failure.
 - Hyperkalemia—life-threatening consequence of acute renal failure
- Coagulation profile to monitor for potential disseminated intravascular

- coagulation (DIC):
 - _ INR, PT, PTT, platelets
- Creatine phosphokinase (CPK):
 - _ Markedly elevated in rhabdomyolysis
- Urine toxicology screen:
 - _ For other toxins with similar effects (e.g., cocaine)
 - _ Some amphetamine-like substances (e.g., synthetic cathinones, MDMA) may not be detected.
- Salicylate and acetaminophen levels if suicide attempt a possibility or if OTC medications ingested (e.g., cough, cold)
- Venous blood gas, ABG

Imaging

- CXR:
 - _ Adult respiratory distress syndrome
 - _ Noncardiogenic pulmonary edema
- Head CT for:
 - _ Significant headache
 - _ Altered mental status
 - _ Focal neurologic signs
 - _ Subarachnoid hemorrhage, intracerebral bleed

Diagnostic Procedures/Surgery

Lumbar puncture for:

- Suspected meningitis (headache, altered mental status, hyperpyrexia)
- Suspected subarachnoid hemorrhage and CT normal

DIFFERENTIAL DIAGNOSIS

- Sepsis
- Thyroid storm
- Serotonin syndrome
- Neuroleptic malignant syndrome
- Pheochromocytoma
- Subarachnoid hemorrhage
- Drugs that cause delirium:
 - _ Anticholinergics
 - _ Tricyclic antidepressants
 - _ Sympathomimetics
 - _ Ethanol withdrawal
 - _ Sedative/hypnotic withdrawal
 - _ Hallucinogens
 - _ PCP
- Drugs that cause hypertension and tachycardia:

- Sympathomimetics
- Anticholinergics
- Ethanol withdrawal
- PCP
- Caffeine
- Monoamine oxidase inhibitors
- Theophylline
- Nicotine
- Drugs that cause seizures:
 - Camphor
 - Carbamazepine
 - Carbon monoxide
 - Chlorinated hydrocarbons
 - Cholinergics
 - Cyanide
 - Ethanol withdrawal
 - Hypoglycemics
 - Isoniazid
 - Lead
 - Lithium
 - Local anesthetics
 - Phenothiazines
 - Propoxyphene
 - Salicylates
 - Sedative/hypnotic withdrawal
 - Strychnine
 - Sympathomimetics
 - Theophylline
 - Tricyclic antidepressants



TREATMENT

PRE HOSPITAL

- Patient may be uncooperative or violent.
- Secure IV access.
- Protect from self-induced trauma.

INITIAL STABILIZATION/THERAPY

- ABCs
- Establish IV 0.9% NS access
- Cardiac monitor
- Naloxone, dextrose (or Accu-Chek), and thiamine if altered mental status

ED TREATMENT/PROCEDURES

- Decontamination:
 - Gastric lavage not routinely recommended:
 - May consider if recent (within 1 hr) of life-threatening ingestion.
 - Activated charcoal not routinely recommended.
 - Consider activated charcoal with sorbitol in 1st dose if administered.
 - Consider activated charcoal with body stuffer or body packer ingestions.
 - Whole-bowel irrigation with polyethylene glycol solution – electrolyte solution for body packers
- Hypertensive crisis:
 - Initially administer benzodiazepines if agitated.
 - α -blocker (phentolamine) as 2nd-line agent
 - Nicardipine or nitroglycerin IV for severe HTN unresponsive to benzodiazepines
 - Nitroprusside can also be used for severe, unresponsive HTN
 - Avoid β -blockers, which may exacerbate HTN due to unopposed α activity
- Agitation, acute psychosis:
 - Administer benzodiazepines.
 - Use butyrophenones (e.g., haloperidol) *with caution* to manage agitation:
 - May lower seizure thresholds and may prolong QT duration
- Dysrhythmias:
 - Sodium bicarbonate IV push is treatment of choice for ventricular dysrhythmias indicative of sodium channel blocking (i.e., widened QRS complex).
 - Lidocaine for ventricular dysrhythmias refractory to alkalinization, benzodiazepines, and supportive care
- Hyperthermia:
 - Benzodiazepines if agitated
 - Active cooling if temperature $> 40^{\circ}\text{C}$:
 - Tepid water mist
 - Evaporate with fan
- Paralysis:
 - Indicated if muscle rigidity and hyperactivity contributing to persistent hyperthermia
 - Nondepolarizing paralytic preferred
- Rhabdomyolysis:
 - Administer benzodiazepines.
 - Hydrate with 0.9% NS.
 - Maintain urine output at 1–2 mL/min
 - Hemodialysis (if acute renal failure and hyperkalemia occur)
- Seizures:
 - Maintain airway

- Administer benzodiazepines
- Phenobarbital if unresponsive to benzodiazepines

MEDICATION

- Activated charcoal: 1–2 g/kg up to 100 g PO
- Dextrose: D₅₀W 1 amp: 50 mL or 25 g (peds: 1 to 2 mL/kg of D₂₅W; infants: 2.5 to 5.0 mL/kg of D10%) IV
- Diazepam (benzodiazepine): 5–10 mg (peds: 0.2–0.5 mg/kg) IV. Not recommended <6 months of age
- Lorazepam (benzodiazepine): 2–6 mg (peds: 0.03–0.05 mg/kg) IV
- Nicardipine IV infusion at 5 mg/h titrate by 2.5 mg/h q5min to max. 15 mg/h
- Nitroprusside: 0.5–10 µg/kg/min IV (titrated to BP)
- Phenobarbital: 15–20 mg/kg at 25–50 mg/min until cessation of seizure activity; monitor for respiratory depression. Safety not established <6 years of age
- Phentolamine: 1–5 mg IV over 5 min (titrated to BP)
- Sodium bicarbonate: 1 or 2 amps (50 mEq/amp) (peds: 1–2 mEq/kg) IV push



FOLLOW-UP

DISPOSITION

Admission Criteria

- Admit all body packers or stuffers to hospital.
- Severe manifestations of toxicity to monitored bed:
 - Seizures
 - Dysrhythmias
 - Hyperthermia
 - Rhabdomyolysis
 - Severe hypertension
 - Altered mental status
- Ischemic chest pain

Discharge Criteria

Mildly intoxicated patients can be observed and treated in ED until resolution of clinical manifestations.

FOLLOW-UP RECOMMENDATIONS

Patients may need referral for chemical dependency rehab and detoxification

PEARLS AND PITFALLS

- Admit patients with severe or persistent symptoms

- Hyperthermia above 40°C may be life threatening:
 - Treat with aggressive sedation and active cooling
- Recognize rhabdomyolysis and hyperkalemia
- Avoid physical restraints in agitated patients if possible
- Consider associated emergency conditions:
 - Chest pain – acute coronary syndrome
 - Infection in altered patients with fevers and history of IV drug use
 - Traumatic injury with methamphetamine abuse
- Benzodiazepines are 1st-line therapy in symptomatic sympathomimetic intoxication

ADDITIONAL READING

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CODES

ICD9

971.2 Poisoning by sympathomimetics [adrenergics]

ICD10

T44.901A Poiso by unsp drugs aff the autonm nervous sys, acc, init

SYNCOPE

Jarrold Mosier • Samuel M. Keim



BASICS

DESCRIPTION

- Transient loss of consciousness associated with loss of postural tone
- Ultimately, it is the lack of oxygen to the brainstem reticular-activating system, which results in a loss of consciousness and postural tone.
- Most commonly, an inciting event causes a drop in cardiac output.
- Cerebral perfusion is re-established by autonomic regulation as well as the reclined posture, which results from the event.
- Accounts for 3% of ED visits

Pregnancy Considerations

- Pregnant patients frequently experience presyncope or syncope from various causes. 5% of patients experience syncope, 28% experience presyncope throughout their pregnancy.
- Placenta acts as an AV malformation, causing decreased SVR that potentiates orthostatic symptoms.
- Fetus lying on IVC can lead to neurogenic and hypovolemic syncope.
- Pregnant patients at higher risk of DVT/pulmonary embolism (PE), UTI, seizures (preeclampsia), valvular incompetencies. Must exclude these diagnoses in ED evaluation.

Geriatric Considerations

- Elderly with highest incidence as well as increased morbidity
- >1/3 will have numerous potential causes.

ETIOLOGY

- Neurally mediated syncope:
 - Reflex response causing vasodilatation and bradycardia with resulting cerebral hypoperfusion
 - Vasovagal (common faint):
 - Often incited by pain or fear
 - Prodromal findings are usually present.
 - Typically lasts <20 sec
 - Tilt-table testing is the gold standard to diagnose.
 - Carotid sinus syncope:
 - Cough, sneeze
 - GI stimulation (e.g., defecation)

- Micturition
- Orthostatic:
 - Positional changes cause abrupt drop in venous return to heart.
 - Volume depletion:
 - Severe dehydration (e.g., vomiting, diarrhea, diuretics)
 - Hemorrhage (see “Hemorrhagic Shock”)
- Autonomic failure:
 - Diabetic or amyloid neuropathy
 - Parkinson disease
 - Drugs (e.g., β -blockers) and alcohol
- Cardiac arrhythmias:
 - Typically sudden and without prodromal symptoms
 - Tachydysrhythmia or bradydysrhythmia
 - Inherited syndromes (e.g., long QT syndrome, Brugada syndrome)
 - Pacemaker/implantable cardioverter defibrillator malfunction
- Structural cardiac or cardiopulmonary disease:
 - Valvular disease (especially aortic stenosis)
 - Hypertrophic cardiomyopathy
 - Acute myocardial infarction
 - Aortic dissection
 - Pericardial tamponade
- Pulmonary embolus
- Neurologic:
 - Transient spike in intracranial pressure that exceeds cerebral perfusion pressure
 - Postsyncopal headache is almost universal
 - May be presentation of a subarachnoid hemorrhage
- Cerebrovascular steal syndromes



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Prodromal symptoms:
 - Lightheadedness
 - Diaphoresis
 - Dimming vision
 - Nausea
 - Weakness
- The following findings suggest an underlying life threat:
 - Sudden event without warning

- Chest pain or palpitations
- 6 Ps of a syncope history:
 - 1. Prodrome activities
 - 2. Prodrome symptoms—visual symptoms, nausea
 - 3. Predisposing factors—age, chronic disease, family history of sudden death
 - 4. Precipitating factors—stress, postural symptoms
 - 5. Passerby witness—what did they see?
 - 6. Postictal phase, if any—suggests seizure

Physical-Exam

- Evaluate for trauma
- Orthostatic vital signs
- Check for difference in BP in both arms suggesting aortic dissection or subclavian steal syndrome.
- Careful cardiovascular exam, including murmurs, bruits, and dysrhythmias
- Rectal exam to check for GI bleeding
- Urine pregnancy test in reproductive-age female
- Careful neurologic exam

Pediatric Considerations

- Warning signs of a potential serious underlying disease:
 - Syncope during exertion
 - Syncope to loud noise, fright, extreme stress
 - Syncope while supine
 - Family history of sudden death at young age (<30 yr)

ESSENTIAL WORKUP

- ECG immediately upon arrival to check for:
 - Ischemia
 - Dysrhythmias
 - Block
 - Long QT interval
 - Brugada syndrome
 - Wolff–Parkinson–White syndrome
- Detailed history and physical exam will determine diagnosis in 85% of those who eventually obtain a diagnosis.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Driven by history and physical exam
- CBC in suspected occult hemorrhage
- Serum bicarbonate:

- Normal with most syncopal events
- Marked decreased bicarbonate obtained <1 hr after the event:
 - Suggestive of a grand mal seizure rather than syncope
 - If due to seizure, should normalize 1 hr after the event
- Cardiac enzymes in suspected ischemia
- Pregnancy test in reproductive-age female
- Electrolytes in patients with profound dehydration or diuretic use

Imaging

- ECG and monitoring until cardiac etiology ruled out
- Chest radiograph ± CT angiography if congestive heart failure (CHF), dissection, or massive PE suspected
- Head CT if abnormal neurologic exam or transient ischemic attack suspected
- Echocardiogram if concern for structural defects

DIFFERENTIAL DIAGNOSIS

- Seizure is most commonly mistaken for syncope:
 - Key differentiating factor is postictal confusion.
 - Brief tonic movements and urinary incontinence may be seen with syncope.
- Metabolic disorders (e.g., hypoxemia, hyperventilation, hypoglycemia)
- Toxicologic
- Stroke
- Psychogenic syncope
- Malingering
- Breath-holding spells in children



TREATMENT

PRE HOSPITAL

- Oxygen
- Cardiac monitoring
- IV access

INITIAL STABILIZATION/THERAPY

- Advanced cardiac life support (ACLS) interventions for unstable patients
- Oxygen
- Cardiac monitoring
- IV access with normal saline fluid bolus in suspected hypovolemia
- Consider coma cocktail—dextrose, thiamine, and naloxone for persistent altered mental status

ED TREATMENT/PROCEDURES

- ACLS interventions for dysrhythmias
- Standard regimens for acute myocardial infarction
- Control BP for subarachnoid hemorrhage and aortic dissection
- Consider thrombolytics for submassive PE.

MEDICATION

- Dextrose: D₅₀W 1 amp (50 mL or 25 g) IV (peds: D₂₅W 2–4 mL/kg IV)
- Naloxone: 2 mg IV or IM (peds: 0.1 mg/kg)
- Thiamine: 100 mg IV or IM (peds: 50 mg)



FOLLOW-UP

DISPOSITION

Admission Criteria

- San Francisco Syncope Rule identifies patients at high risk for serious short-term outcomes (“CHESS”):
 - History of CHF
 - Hematocrit < 30%
 - Abnormal ECG
 - Patient complaint of shortness of breath
 - Systolic BP < 90
- Other recommendations:
 - Suspected cardiac syncope must be admitted to monitored bed
 - GI bleeds consider intensive care unit bed
 - Admit elderly patients with syncope.

Discharge Criteria

- Neurally mediated syncope or orthostatic syncope from volume depletion may be evaluated on outpatient basis with close follow-up, if patient is reliable and has a good social structure.
- Driving restrictions until cleared

PEARLS AND PITFALLS

- Use of criteria such as the San Francisco Syncope Rule prevents unnecessary admissions.
- Do not assume vasovagal cause in syncope associated with headache or chest pain.

ADDITIONAL READING

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and treatment) of syncope—update 2004. Executive summary. *Eur Heart J*. 2004;25(22):2054–2072.

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CODES

ICD9

- 337.01 Carotid sinus syndrome
- 427.89 Other specified cardiac dysrhythmias
- 780.2 Syncope and collapse

ICD10

- G90.01 Carotid sinus syncope
- R00.1 Bradycardia, unspecified
- R55 Syncope and collapse

SYNDROME OF INAPPROPRIATE ANTIDIURETIC HORMONE SECRETION (SIADH)

Matthew D. Bitner



BASICS

DESCRIPTION

- Most common cause of hyponatremia in hospitalized patients (and doubles inpatient mortality in some studies)
- A *water balance problem* more than 1 of sodium (Na) balance
- Normal regulation of water balance:
 - _ Antidiuretic hormone (ADH):
 - Integral controller of water balance
 - Increases water permeability of the collecting tubules, resulting in free water reabsorption
 - Synthesized by hypothalamus but secreted by posterior pituitary
 - _ Water deprivation (increased plasma osmolality) stimulates secretion as sensed by:
 - Osmoreceptors/atrial stretch receptors
 - Carotid baroreceptors
 - Aortic arch/pulmonary veins
- Hyponatremia:
 - _ *Mild*: Serum sodium < 135 mEq/L
 - _ *Moderate*: Serum sodium < 130 mEq/L
 - _ *Severe*: Serum sodium < 125 mEq/L
 - _ Excess extracellular water *relative* to Na
 - _ Depletional hyponatremia:
 - Sodium depletion can be caused by diet, GI losses, diuretic use, and renal or adrenal disease.
 - Often accompanied by extracellular fluid volume depletion
 - Hyponatremia associated with clinical signs of hypovolemia
 - Increased Hct, BUN, Cr
 - Urinary sodium excretion < 20 mEq/L
 - _ Dilutional hyponatremia:
 - Increased extracellular water in presence of normal or increased total body sodium
 - Can be caused by increased fluid intake (oral, IV), drugs, or medical conditions
 - Euvolemia with edema
 - Normal or decreased Hct, BUN, Cr
 - Urinary sodium excretion > 20 mEq/L

- Inappropriate ADH secretion is a form of dilutional hyponatremia.
- Definition of SIADH:
 - ADH secretion in absence of hyperosmolality or hypovolemia
- Criteria for definition:
 - Essential features:
 - *Hyponatremia*—despite correction for hyperglycemia, hyperproteinemia, or hyperlipidemia
 - *Euvolemia*—no clinical signs of volume depletion (orthostasis, tachycardia) or volume overload (edema, ascites)
 - *Hyposmolality* of the plasma— < 275 mOsm/kg of water
 - Normal renal, adrenal, and thyroid function
 - No recent diuretic use
 - Urine Osm > 100 mOsm/kg of water
 - Supplemental features:
 - Plasma uric acid < 4 mg/dL
 - BUN < 10 mg/dL
 - FENa $> 1\%$
 - Failure to correct hyponatremia after an infusion of normal saline (NS) 0.9%
 - Abnormal water load test (inability to excrete $\geq 90\%$ of a 20 mL/kg water load in 4 hr)

ETIOLOGY

- **Malignant disorders:**
 - ADH-producing tumors
 - Cancer (Small-cell lung, pancreatic, prostate)
 - Pituitary tumors
 - Thymoma
 - Lymphoma
- **Pulmonary disorders:**
 - Pneumonia
 - TB
 - Lung abscess
 - COPD
- **CNS disorders:**
 - Meningitis/encephalitis
 - CVA
 - Head injury
- **Medications:**
 - Thiazides
 - Chlorpropamide
 - Vincristine
 - Anticonvulsants (carbamazepine)

- Antidepressants (tricyclics, SSRIs)
- Antipsychotics
- NSAIDs
- Ecstasy (MDMA)
- Vasopressin analogs (DDAVP, oxytocin, vasopressin)
- **Transient:**
 - Endurance exercise
 - General anesthesia
 - Pain
 - Stress
- **Other:**
 - Hereditary
 - Positive-pressure ventilation
 - HIV/AIDS
 - Idiopathic

ALERT

Cerebral salt-wasting syndrome (CSWS) can mimic SIADH.

- Seen in patients with cerebral tumors or subarachnoid hemorrhage and in neurosurgical patients
- Etiology unclear
- Represents appropriate water resorption in the face of salt wasting (urine Na > 30–40 mmol/L)
- Fluid restriction can help differentiate the 2:
 - In SIADH: Hypouricemia will correct
 - In CSWS: Hypouricemia will persist
- Treatment of CSWS may differ from that of SIADH:
 - Infusion of NS
 - May benefit from fludrocortisone therapy



DIAGNOSIS

SIGNS AND SYMPTOMS

- Serum sodium < 135 mEq/L:
 - May be asymptomatic
- Serum sodium < 130 mEq/L:
 - Weakness/lethargy
 - Weight gain
 - Headache
 - Anorexia
- Sodium serum < 120 mEq/L:
 - Altered mental status
 - Seizure/coma

- Chronic hyponatremia: 50% asymptomatic
- High mortality in acute hyponatremia

History

- Thorough medication history
- Course of illness (acute, subacute, or chronic)

Physical-Exam

- Volume status
- Mental status
- Stigmata of malignancy

ESSENTIAL WORKUP

- Diagnosis is 1 of exclusion, need to evaluate for other causes of:
 - Depletional or dilutional hyponatremia
- Electrolytes, BUN, Cr, glucose, protein, lipids:
 - Hyponatremia (serum Na < 135 mmol/L)
 - Serum hyposmolality (serum Osm < 275 mOsm/kg)
- Urine osmolality:
 - Inability to excrete dilute urine
 - Urine osmolality > 100 mOsm/kg
- Urine sodium:
 - Continued urinary excretion of sodium
 - Urinary sodium > 20 mEq/L

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Serum protein levels
- Lipid levels
- Glucose levels
- Serum osmolality
- LFT and thyroid function test
- Morning cortisol level

Imaging

Consider imaging (CXR, CT head) to screen for pathology causing SIADH (tumors/masses)

DIFFERENTIAL DIAGNOSIS

Causes of Hyponatremia

- See etiologies above

- Increased extracellular fluid (dilutional hyponatremia):
 - _ Renal failure/insufficiency
 - _ CHF
 - _ End-stage liver disease
- Normal extracellular fluid (dilutional hyponatremia):
 - _ SIADH
 - _ Myxedema
 - _ Sheehan syndrome (postpartum hypopituitarism)
 - _ Reset osmostat syndromes (dilute urine at lower than normal sodium levels)
- Decreased extracellular fluid (depletional hyponatremia):
 - _ Increased losses:
 - Excessive sweating (endurance sports)
 - GI losses (vomiting, diarrhea)
 - _ 3rd-space sequestration
 - _ Diuretic use
 - _ Aldosterone deficiency:
 - Addison disease
 - _ Salt-losing nephropathies:
 - Renal tubular acidosis
- Pseudohyponatremia (seen in hyperglycemia, hyperproteinemia, hyperlipidemia)



TREATMENT

PRE HOSPITAL

- In patients with altered mental status, maintenance and protection of the airway are paramount.
- When hypovolemia is suspected, appropriate fluid resuscitation should be initiated.
- Rapid patient evaluation and transport are essential.

INITIAL STABILIZATION/THERAPY

- Severe symptomatic hyponatremia with CNS manifestations
- Endotracheal intubation for patients in need of airway protection
- Identify/treat other causes of altered mental status
- Treat seizures with benzodiazepines
- Proceed to hyponatremia treatment

ED TREATMENT/PROCEDURES

- Most effective treatment of SIADH is successful *eradication of the underlying cause*.
- Initial treatment of hyponatremia caused by SIADH is the same for all causes of euvolemic/hypervolemic hyponatremia.

Mildly Symptomatic Hyponatremia, Chronic Hyponatremia with Minimal Symptoms,

Asymptomatic Hyponatremia

- Serum sodium usually > 125 mEq/L
- Fluid restriction 800–1,000 mL/day alone or in conjunction with:
 - 0.9% NS infusion and/or IV furosemide
- Correct serum sodium by no more than 0.5 mEq/L/hr (5–6 mEq/day):
 - Too rapid correction of serum sodium levels can induce *central pontine myelinolysis*, associated with development of bulbar palsy, quadriplegia, seizures, coma, and death.

Severe Hyponatremia

- Symptomatic patient, serum sodium < 125 mEq/L
- Increase serum sodium by no more than 12 mEq/L in 1st 24 hr at a rate of 1 mEq/L/hr (8–12 mEq/day when serum sodium below 125 mEq/L and slow to 5–6 mEq/day when serum sodium rises to 125 mEq/L).
- Target level: 125 mEq/L
- Treat patients with significant neurologic symptoms with 3% saline solution.
- Serum sodium lab testing every 1–2 hr

Acute Life-threatening Hyponatremia

- Serum sodium usually < 120 mEq/L
- Associated with seizures or coma
- Clinical goal: Stop seizure and improve neurologic status
- Therapeutic goal: Same as for severe hyponatremia
- Administer hypertonic saline solution (3%)
- Stop hypertonic saline when symptoms (i.e., seizures) resolve and transition to NS.
- IV furosemide to promote diuresis and induce a negative fluid balance.
- Once serum sodium = 125 mEq/L, further IV fluid should be in the form of 0.9% saline solution.
- Restoration of serum sodium to normal levels should take place over ≥ 48 hr.
- Drugs that inhibit the secretion/effects of ADH:
 - Indicated when SIADH not self-limited and cause cannot be removed
 - Demeclocycline (blocks effect of ADH)

MEDICATION

- Conivaptan 20 mg IV over 30 min (for severe hyponatremia in concert with admitting physician)
- Demeclocycline: 300 mg PO BID–QID
- Hypertonic saline solution (3% NaCl): 250–500 mL (max. initial dose 5 mL/kg):
 - 25–100 mL/hr
 - Limit rate in rise of serum sodium to 0.5–1 mEq/L/h.
 - Discontinue when seizure resolves or serum sodium of 125 mEq/L is reached.
 - Rise in serum sodium by 4–6 mEq/L is usually sufficient to stop seizures.

- 0.9% NS: Maintenance rates
- Lasix: 1 mg/kg up to 20–40 mg IV



FOLLOW-UP

DISPOSITION

Admission Criteria

- Severe life-threatening hyponatremia
- Symptomatic hyponatremia
- Serum sodium < 125 mEq/L regardless of symptoms
- New-onset SIADH in which underlying cause or complications must be diagnosed and treated
- Patient's compliance an issue

Discharge Criteria

- Asymptomatic chronic hyponatremia
- Serum sodium > 125 mEq/L
- No unstable comorbid factors
- Known diagnosis of SIADH

FOLLOW-UP RECOMMENDATIONS

All patients with hyponatremia that meet discharge criteria still require follow-up to check for resolution, monitoring, and/or diagnosis of the underlying cause of the SIADH/hyponatremia.

PEARLS AND PITFALLS

- SIADH is a diagnosis of exclusion.
- Must evaluate for other causes as well as renal, thyroid, adrenal, cardiac, and hepatic dysfunction.
- Take a thorough medication history.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Hyponatremia

The author gratefully acknowledges the contribution of Arunachalam Einstein on previous editions of this chapter.



CODES

ICD9

- 253.6 Other disorders of neurohypophysis
- 276.1 Hyposmolality and/or hyponatremia

ICD10

- E22.2 Syndrome of inappropriate secretion of antidiuretic hormone
- E87.1 Hypo-osmolality and hyponatremia

SYNOVITIS, TOXIC

Daniel A. Popa • Ian R. Grover

BASICS

DESCRIPTION

- Nonspecific inflammation and hypertrophy of the synovium with an effusion of the hip joint in children
- It can affect any joint but most commonly affects the hip.
- Disease process is self-limiting.
- Most common cause of acute hip pain and a limp in children aged 3–10.
- Also referred to as acute transient synovitis and irritable hip syndrome.
- Age group most affected is 3–6 yr.
- Male > female (2:1)
- Right hip > left

ETIOLOGY

- Cause of toxic synovitis is unknown.
- Infectious etiology is suspected, because an upper respiratory infection precedes the symptoms of transient synovitis in ~50% of cases.

DIAGNOSIS

SIGNS AND SYMPTOMS

- Unilateral hip pain
- Pain in the anteromedial thigh and knee
- Pain with weight bearing
- Limp
- Low-grade fever
- Decreased range of motion (ROM) of the affected hip
- Pain with ROM of the affected hip

History

- Acute onset of unilateral hip pain
- No history of trauma
- Pain with ambulation
- Recent upper respiratory infection

Physical-Exam

- Low-grade fever, usually <38.5°C (101.3°F)
- High-grade fevers are more concerning for septic arthritis

- Nontoxic appearing
- Limited hip ROM due to pain
- Hip is usually held in the flexed and externally rotated position for maximal comfort.

ESSENTIAL WORKUP

- Hip x-rays
- AP pelvis
- CBC, C-reactive protein (CRP), ESR if concerned for septic arthritis

DIAGNOSIS TESTS & INTERPRETATION

Lab

CBC, CRP, ESR:

- May be normal or elevated
- An elevated white blood cell (WBC) count, CRP, or ESR alone does not differentiate toxic synovitis from septic arthritis or osteomyelitis.
- If WBC count, CRP, and ESR are normal, more serious causes of hip pain are less likely.
- If CRP < 2 mg/dL and able to bear weight, more likely to be toxic synovitis

Imaging

- Plain hip films (anteroposterior and frog-leg view):
 - Usually normal
 - May detect an effusion or other causes of hip pain
- US to rule out joint effusion and to guide hip joint aspiration if required
- MRI (rarely indicated):
 - Very useful in diagnosing Legg–Calvé–Perthes (LCP) disease
- Bone scan:
 - Used to differentiate LCP disease from toxic synovitis
 - Can detect osteomyelitis
 - The increased radiation is usually reserved for recurrent cases or cases in which the diagnosis is still in question.

Diagnostic Procedures/Surgery

Joint aspiration:

- Not necessary if the patient is afebrile with a normal WBC count, CRP, and ESR
- Abnormal joint fluid analysis indicates SA (see “Arthritis, Septic”)

DIFFERENTIAL DIAGNOSIS

- SA
- Osteomyelitis
- Soft tissue infection

- LCP disease
- Slipped capital femoral epiphysis
- Juvenile rheumatoid arthritis
- Rheumatic fever
- Chondrolysis
- Gaucher disease
- Osteosarcoma
- Ewing sarcoma
- Osteoid osteoma
- Leukemia
- Tuberculosis of the hip
- Fracture
- Lyme disease
- Psoas abscess
- Sickle cell crisis

Pediatric Considerations

- 4–17% of children have a recurrent episode.
- 10% of recurrent cases may be the presenting feature of a chronic inflammatory condition.
- 2–10% of patients with toxic synovitis later develop LCP disease:
 - Suggested that toxic synovitis may represent an early stage of LCP disease.



TREATMENT

PRE HOSPITAL

- Keep leg in position of comfort.
- Treat with NSAIDs.

ED TREATMENT/PROCEDURES

- Conservative treatment
- Bed rest in position of comfort: Flexion and external rotation
- Initiate NSAIDs
- Apply heat to the area
- Antibiotics and steroids are not indicated
- Some authors recommend no weight bearing for 7–10 days following improvement and return of normal hip function, citing increased risk for recurrence.
- Close follow-up is essential, with repeat radiographs due to association with LCP.

MEDICATION

First Line

- Ibuprofen: 200–600 mg (peds > 6 mo old: 5–10 mg/kg/dose) PO q6h PRN
- Naproxen: 250–500 mg (peds > 6 mo old: 5–10 mg/kg/dose) PO BID PRN

Second Line

Acetaminophen: 500 mg (peds: 10–15 mg/kg, do not exceed 5 doses/24 h) PO/PR q4–6h, do not exceed 4 g/24 h



FOLLOW-UP

DISPOSITION

Admission Criteria

Patients with severe joint pain or a large effusion may require hospitalization for bed rest and analgesics.

Discharge Criteria

All patients who have had more serious causes of hip pain excluded and have been diagnosed with toxic synovitis can be discharged from the hospital with good follow-up.

Issues for Referral

Follow-up with an orthopedic surgeon in 1–2 wk for repeat evaluation.

FOLLOW-UP RECOMMENDATIONS

- Return to the ED immediately for worsening pain in the hip or increasing fever.
- Follow-up with pediatric orthopedic surgeon in 1–2 wk for repeat evaluation.
- Patients should have repeat x-rays done in 6 mo to exclude LCP disease.

PEARLS AND PITFALLS

- Most cases are diagnosed by history and physical exam alone with fever and weight bearing as key elements.
- ~ 50% of children have a history of a preceding viral illness.
- NSAIDs help treat the pain and shorten the course of the illness.
- Nearly all children recover from toxic synovitis within 2 wk and without sequelae.
- ~ 2–10% of children with toxic synovitis develop LCP disease.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Arthritis, Septic
- Hip Injury
- Legg–Calvé–Perthes Disease



CODES

ICD9

727.09 Other synovitis and tenosynovitis

ICD10

- M67.351 Transient synovitis, right hip
- M67.352 Transient synovitis, left hip
- M67.359 Transient synovitis, unspecified hip

SYPHILIS

Jessica Freedman

BASICS

DESCRIPTION

- Sexually transmitted disease
- 12 million new cases diagnosed annually worldwide
- Acquired via mucous membranes/disrupted skin
- Divided into 3 stages:
 - Primary syphilis:
 - Painless chancre or ulcer
 - Secondary syphilis:
 - Replication and hematogenous spread
 - Begins 3–6 wk after primary lesion
 - Late latent secondary phase
 - Tertiary or late syphilis:
 - Very uncommon
 - Cardiovascular and neurologic symptoms

ETIOLOGY

Treponema pallidum:

- Spirochete bacteria

DIAGNOSIS

SIGNS AND SYMPTOMS

Primary (Early) Syphilis

- 21-day incubation period
- No constitutional symptoms
- Chancre:
 - Painless papule at site of inoculation
 - Clean-based, circular, sharply defined borders:
 - Solitary lesions
 - Commonly on penis, vulva, and rectum
 - Bilateral regional lymphadenopathy
 - Heals spontaneously in 3–6 wk
- Rectal chancre:
 - Painful or painless
 - Rectal irritation/discharge

- Painless enlargement of lymph nodes

Secondary (Early) Syphilis

- Occurs 3–6 wk after primary lesion
- Disseminated stage
- Rash (most common):
 - Symmetric, diffuse, polymorphous, papular, or maculopapular rash
 - Rash may be diverse and not fit a pattern
 - Starts on trunk and flexor extremities
 - Spreads to involve palms and soles:
 - Discrete, red/reddish-brown
 - 0.5–2 cm in diameter
- Condyloma lata:
 - Large raised gray/white lesions, painless, moist
 - Mucous membranes:
 - Oral cavity and perineum
 - Very contagious
 - Intertriginous areas
 - Flat rectal warts
- Systemic symptoms:
 - Fever, headache, malaise, anorexia, sore throat, myalgias, and weight loss
- Diffuse lymphadenopathy:
 - Palpable nodes at inguinal, axillary, posterior cervical, femoral, and/or epitrochlear regions
 - Painless, firm, and rubbery
- Less common:
 - “Moth-eaten” alopecia
 - Syphilitic meningitis
 - Scleritis
- Loss of lateral 3rd of eyebrows
- Painless mucosal lesions (mucous patches)
- Secondary stage resolves spontaneously in 1–2 mo

Latent Secondary Syphilis

- Begins after primary and secondary symptoms resolve.
- Period of no symptoms but positive serology:
 - CSF normal
- Late latent stage not infectious except for fetal transmission in pregnant women
- Persists for lifetime or develops into tertiary syphilis

Tertiary (Late) Syphilis

- Occurs in about 15% of patients with untreated latent secondary syphilis

- Can appear 10–20 yr after initial infection
- Neurologic and cardiovascular involvement:
 - Destructive stages of disease
- Neurosyphilis (most common):
 - Asymptomatic:
 - Positive CSF – Venereal Disease Research Laboratories (VDRL)
 - CSF pleocytosis (10–100 lymphocytes)
 - Elevated CSF protein at 50–100 mg/dL
 - Meningitis:
 - Aseptic; CSF with positive VDRL, higher protein, and lower glucose (compared with above)
 - Cranial nerve palsy, including isolated 8th nerve palsy
 - General paresis:
 - Loss of cortical function
 - Argyll Robertson pupils (small fixed pupils that do not react to strong light, but do react to accommodative convergence)
 - Tabes dorsalis (peripheral neuropathy)
- Degeneration of posterior columns/posterior or dorsal roots of spinal cord
- Dementia
- Paresthesias, abnormal gait, and lightning (sudden, severe) pain of extremities/trunk
- Progressive loss of reflexes, vibratory/position sensation
- Positive Romberg sign
- Vision: Optic atrophy
- Pupils: Argyll Robertson pupils
- Urinary incontinence
- Gummas:
 - Late benign syphilis of cutaneous skin/viscera:
 - Bone, brain, abdominal viscera, etc.
- Granulomatous, cellular hypersensitivity reaction:
 - Round, irregular, or serpiginous shape
 - “Great pox”
- Cardiovascular:
 - Thoracic aortic aneurysm (ascending most common):
 - Dilated aorta and aortic valve regurgitation
 - Aortic valve insufficiency
 - Coronary thrombosis
 - Destructive lesions of skeletal structures or skin
- HIV-infected:
 - Strong association with syphilis
- Increased incidence of neurosyphilis

Congenital Syphilis

- In utero infection:
 - Age <2 yr:
 - Hepatosplenomegaly, rash, condyloma lata, rhinitis (snuffles), jaundice (nonviral hepatitis), osteochondritis
- Older children (syphilis stigmata):
 - Interstitial keratitis, nerve deafness, anterior bowing of shins, frontal bossing, mulberry molars, Hutchinson teeth, saddle nose, etc.

ESSENTIAL WORKUP

Rapid plasma reagin (RPR)

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Serology:
 - Nontreponemal test:
 - RPR
 - VDRL
 - Positive 14 days after chancre appears
 - Early false negatives, especially ≤ 7 days after primary chancre
 - Repeat negative test in 2 wk and correlate with disease activity
 - False positives in 1–2% of general population
 - 4-fold change in titer clinically significant
 - 100% sensitivity in secondary syphilis
 - Nonreactive after successful treatment
 - Treponemal antibody test:
 - Fluorescent treponemal antibody absorption (FTA–ABS)
 - Hemagglutination assay for antibody to *T. pallidum* (MHA–TP)
 - More sensitive and specific
 - 1% false-positive rate
 - Confirmatory test
 - Reactive for patient's lifetime
 - More costly and harder to perform
 - Dark-field microscopy:
 - Identifies treponemes from primary and secondary lesions
 - Suspicious early lesions with negative serology (early primary syphilis)
 - False negatives with ointments, creams
 - Oral specimen unsuitable
 - CSF analysis for tertiary neurosyphilis:
 - Tertiary syphilis
 - Positive VDRL/RPR
 - Lymphocytes > 5/mL

- Protein > 45 mg/dL
- Decreased glucose

DIFFERENTIAL DIAGNOSIS

- Genital ulcer:
 - Chancroid (painful)
 - Genital herpes:
 - Vesicular, multiple lesions
 - Lymphogranuloma venereum
 - Granuloma inguinale
 - Superficial fungal infection
 - Carcinoma
- Secondary and tertiary syphilis:
 - Pityriasis rosea
 - Drug-induced rash
 - Acute febrile exanthems
 - Psoriasis
 - Lichen planus
 - Scabies
 - Infectious mononucleosis
 - Viral illness
 - Bacteremia
 - Tertiary syphilis:
 - Psychosis
 - Dementia
 - Multiple sclerosis
 - Meningitis
 - Encephalitis
 - Delirium
 - Unknown overdose



TREATMENT

INITIAL STABILIZATION/THERAPY

Lower BP and establish IV access for aortic dissection.

ED TREATMENT/PROCEDURES

- Treatment other than penicillin with increased relapse rate:
 - Desensitize those allergic to penicillin.
- Pregnancy:
 - Treat with penicillin even in latent syphilis.
 - If patient allergic to penicillin, admit for desensitization.

- Jarisch–Herxheimer reaction:
 - Transient febrile reaction to therapy
 - May be owing to antigen liberation from spirochetes or activation of complement cascade
 - Peaks at 8 hr, resolves in 24 hr
 - Symptoms:
 - Fever, headache, malaise, worsening rash
 - Treat with antipyretics
 - No serious sequelae
- Recommended testing:
 - Sexual partners
 - Concomitant sexually transmitted diseases including HIV
 - Repeat serology test in 6 and 12 mo.

MEDICATION

- Early primary, secondary, early latent (<1 yr):
 - Benzathine penicillin G: 2.4 million U IM
 - Doxycycline: 100 mg PO BID for 14 days
 - Tetracycline: 500 mg PO QID for 14 days
- Late latent (>1 yr) except neurosyphilis:
 - Benzathine penicillin G: 2.4 million U IM 3 times over 2 wk on days 0, 7, and 14
 - Doxycycline: 100 mg PO BID for 4 wk
 - Tetracycline: 500 mg PO QID for 4 wk
- Neurosyphilis:
 - Penicillin G: 3–4 million U IV q4h for 10–14 days
 - Procaine penicillin: 2.4 million U IM daily +
 - Probenecid: 500 mg PO QID for 10–14 days
- Congenital syphilis:
 - Penicillin G: 50,000 U/kg IM q8–12h for 10–14 days; or
 - Procaine penicillin: 50,000 U/kg IM daily for 10–14 days



FOLLOW-UP

DISPOSITION

Admission Criteria

- Neurosyphilis requires IV antibiotics
- Pregnant women allergic to penicillin requiring desensitization

Discharge Criteria

Follow-up care:

- Measure for falling titers in 6 mo and 1 yr after treatment.
- Tertiary/latent (>1 yr):
 - Measure for falling titers in 3, 6, 12, and 24 mo after treatment.

Issues for Referral

Infectious disease consultation for secondary and tertiary syphilis as well as congenital and neurosyphilis

FOLLOW-UP RECOMMENDATIONS

Titers must be monitored.

PEARLS AND PITFALLS

- Syphilis is known as the “great imitator.”
- In patients presenting with unknown rash, think of syphilis and ask about history of genital lesions.
- Be sure to examine mucous membranes of all patients presenting with rash.
- Think of tertiary syphilis with neurologic symptoms of unknown etiology.

ADDITIONAL READING

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CODES

ICD9

- 091.2 Other primary syphilis

- 091.9 Unspecified secondary syphilis
- 097.9 Syphilis, unspecified

ICD10

- A51.0 Primary genital syphilis
- A51.49 Other secondary syphilitic conditions
- A53.9 Syphilis, unspecified

SYSTEMIC LUPUS ERYTHEMATOSUS

Susanne M. Hardy • John P. Lemos

BASICS

DESCRIPTION

- Chronic autoimmune disease; peak onset between ages 15 and 40 yr; characterized by flares and remissions
- Multisystem disease with diverse clinical manifestations:
 - _ Mucocutaneous:
 - Most commonly involved system
 - 4 specific skin rashes
 - _ Arthritis
 - _ Cardiac:
 - Endocarditis
 - Myocarditis
 - CHF
 - Conduction abnormalities
 - Atherosclerosis
 - MI
 - _ Renal:
 - Glomerulonephritis
 - Renal failure
 - _ Pulmonary:
 - Pleural effusion (usually exudative)
 - Pneumonitis/pleuritis
 - Pulmonary hemorrhage
 - Pulmonary embolism
 - Pneumonia
 - Pulmonary edema
 - Pulmonary hypertension
 - _ Neurologic:
 - Lupus cerebritis
 - _ Vascular:
 - Vasculitis
 - Thrombosis
 - Atherosclerosis
 - _ GI:
 - Peritonitis
 - Mesenteric vasculitis and ischemia
 - Pancreatitis

Pediatric Considerations

- Neonatal lupus may occur when maternal autoantibodies cross the placenta:
 - Associated with transient anemia and thrombocytopenia
- Congenital heart block is the most serious complication

Geriatric Considerations

- 10 times greater risk of MI due to atherosclerosis
- High incidence of osteoporosis related to chronic steroid use

RISK FACTORS

Genetics

- More common in females than males (9:1 ratio)
- More common in women of childbearing age
- More common in African Americans
- Higher frequency of systemic lupus erythematosus (SLE) and other autoimmune diseases among 1st-degree relatives

ETIOLOGY

- Autoantibody production against cell nucleus and cytoplasmic structures, leading to inflammatory changes, vasculitis, and immune complex deposition in multiple organ systems
- A significant percentage of patients have an associated antiphospholipid syndrome:
 - Characterized by antibodies against cellular phospholipid components
 - Tendency toward recurrent vascular thrombosis
- Lupus is a chronic disease with several exacerbating factors:
 - Infection
 - Sun exposure
 - Fatigue
 - Trauma
 - Medications (sulfonamides)
 - Stress
 - Diet
- Drug-induced lupus is a milder disease that eventually resolves once the drug is discontinued. Usually presents with skin and joint manifestations while renal and neurologic involvement is rare.
- Common medications include:
 - Chlorpromazine, methyldopa, procainamide, hydralazine, isoniazid, quinidine, minocycline

- 4 of the 11 criteria in the following list are needed to make the diagnosis:
 - Malar rash
 - Discoid rash
 - Photosensitivity rash
 - Oral ulcers
 - Arthritis
 - Serositis
 - Neurologic disorders
 - Hematologic disorders
 - Immunologic disorders
 - Renal disorders
 - Antinuclear antibodies

SIGNS AND SYMPTOMS

- Systemic:
 - Fatigue
 - Fever
 - Weight loss
 - Dyspnea
- Skin:
 - Malar rash (butterfly maculopapular facial)
 - Discoid rash (raised red patches)
 - Photosensitivity rash (subacute cutaneous lupus)
 - Bullous rash (large blisters)
- Musculoskeletal:
 - Myalgias
 - Joint pain
 - Arthritis:
 - Defined as 2 or more peripheral joints
 - Polyarthritis, symmetric, or migratory
- Heart:
 - Chest pain
 - Pericardial rub
 - Murmur
- Vascular:
 - Vasculitis
 - Thrombosis
 - Atherosclerosis
 - Peripheral vascular disease
- Lungs:
 - Dyspnea
 - Tachypnea
 - Pleural rub

- Rales
- Nervous system:
 - Psychosis/depression
 - Headache
 - Seizures
 - Peripheral neuropathies
 - Stroke/cranial nerve deficits
 - Cerebritis
- GI:
 - Painless oral ulcers
 - Abdominal pain
 - Positive stool guaiac suggests mesenteric ischemia

History

- Symptoms commonly accumulate and exacerbate over years, with flares and remissions. A history of fatigue, rashes, and joint pain may point to the diagnosis.
- Patients describe arthralgias out of proportion to physical findings

Physical-Exam

- Check for fever
- Carefully evaluate skin for rashes and vasculitis

ESSENTIAL WORKUP

- Thorough history and physical exam needed to distinguish between major and minor flare-ups
- Major flare-ups:
 - CBC
 - Electrolytes, BUN, creatinine, glucose
 - UA
 - ESR
 - Chest radiograph, ECG, and pulse oximetry for cardiorespiratory symptoms

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC:
 - Leukopenia, thrombocytopenia, normochromic normocytic anemia
 - Degree of hematologic disorders suggests degree of disease activity
- ESR:
 - May be elevated during acute exacerbations
 - Not a good indicator of active disease
- CRP may also be elevated; marked elevation may be a sign of infection
- PTT:

- May be elevated in patients with lupus anticoagulant
- UA:
 - Protein (persistent proteinuria > 0.5 g/day or 3+ persistently)
 - Casts (red blood cell)
 - Hematuria
 - WBCs
- Amylase is elevated in mesenteric ischemia and pancreatitis
- Send antinuclear antibody, rheumatoid factor (RF), antistreptolysin O (ASO) titer if diagnosis unclear
- Anti-Sm and anti-dsDNA are diagnostic
- A false-positive Venereal Disease Research Laboratory (VDRL) test is supportive of the diagnosis
- Joint aspirate typically shows fluid with fewer than 3,000 WBCs
- LP if suspicion for meningitis or encephalitis

Imaging

- CXR:
 - Pneumonitis
 - Pneumonias
 - Pleural effusion
 - Cardiomegaly
- ECG/echocardiogram
- CT chest:
 - Pulmonary embolus
 - Pulmonary hemorrhage
 - Diffuse alveolar hemorrhage
- CT head for change in mental status or neurologic findings (lupus cerebritis is a diagnosis of exclusion)

Pregnancy Considerations

- Pregnancy is not recommended during active disease owing to the high risk of spontaneous abortion
- The effect of pregnancy on disease activity is variable

DIFFERENTIAL DIAGNOSIS

- Hypotension in the known lupus patient may be due to shock from a major flare-up, secondary to acute steroid withdrawal, or the result of sepsis
- Other autoimmune diseases:
 - Rheumatic fever
 - Rheumatoid arthritis
 - Dermatomyositis
 - Overlap syndromes
- Skin changes:

- Urticaria
- Erythema multiforme
- Idiopathic thrombocytopenic purpura
- Multiple sclerosis
- Epilepsy



TREATMENT

INITIAL STABILIZATION/THERAPY

ABCs

ED TREATMENT/PROCEDURES

- Mainstays include NSAIDs, corticosteroids, antimalarials, and immunosuppressive drugs
- Special attention must be given to CNS and renal involvement as well as infections; these are the main determinants of morbidity
- Mild flare-ups—arthralgias, myalgias, fatigue, and rash:
 - NSAIDs (careful with lupus nephritis), acetyl salicylic acid (ASA), topical steroids for rash, sunscreen
 - Topical steroids for most cutaneous manifestations
 - If not sufficient, begin low-dose prednisone
- Major flare-ups—life- or organ-threatening:
 - Methylprednisolone
 - Anticoagulation for thrombosis; give blood products early if needed
 - Psychotropics for neuropsychiatric symptoms
 - Anticonvulsants for seizures
 - If poor response, consult rheumatology before starting cytotoxic medications
- Chronically:
 - Prednisone taper
 - NSAIDs
 - Rheumatologist initiated:
 - Antimalarials: Quinacrine, chloroquine:
 - Side effect is irreversible retinopathy
 - Cyclophosphamide
 - Azathioprine
 - Methotrexate
 - Belimumab (FDA approved for active, autoantibody positive disease in patients under active treatment)
 - Hormonal therapy, mycophenolate mofetil, rituximab, and autologous marrow stem cell transplant are under investigation

MEDICATION

- Methylprednisolone: 15 mg/kg/d IV up to 1 g; consult rheumatologist for peds dosing
- Prednisone: 5–30 mg (peds: < 0.5 mg/kg) PO daily for minor flare
- Prednisone: 1–2 mg/kg/d PO for major flares in adults
- Ibuprofen: 800 mg (peds: 5–10 mg/kg) PO TID



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients who have end-organ disease such as renal, cardiac, or CNS involvement
- Thrombocytopenia with hemorrhage, arterial or venous thrombosis
- Consider admission with pericarditis, myocarditis, pleural effusion or infiltrates, and evidence of vasculitis
- Those with severe end-organ or life-threatening manifestations should be admitted to the ICU
- Patients with lupus should be treated as immunocompromised and suspected or diagnosed infections should be treated aggressively

Discharge Criteria

- Patients may be discharged home with mild flare-ups if afebrile, well hydrated, and not ill appearing
- ESR should not be used as disposition criterion as it may be elevated long after a flare-up has subsided

Issues for Referral

Because lupus is a chronic disease, a rheumatologist or knowledgeable primary care physician (PCP) must follow the patient adequately

FOLLOW-UP RECOMMENDATIONS

PCPs must educate patients regarding sun protection, immunizations, and lowering risks of atherosclerosis

PEARLS AND PITFALLS

- The diagnosis of SLE is complicated and requires a thorough history and physical exam supported by appropriate lab testing
- Chronic steroid therapy leads to immunosuppression
- Renal involvement confers a poor prognosis
- Serum creatinine may be elevated, but is a poor indicator of the disease (urinalysis

- is more sensitive with proteinuria and/or red blood cell casts)
- All patients with SLE should be offered annual, seasonal influenza vaccinations and be sure that pneumococcal vaccination is up to date
- VDRL may be falsely positive

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- Lehrmann J, Sercombe CT. Systemic lupus erythematosus and the vasculitides. *Rosen's Emergency Medicine*. 7th ed. Philadelphia, PA: Mosby Elsevier; 2010.
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CODES

ICD9

- 420.0 Acute pericarditis in diseases classified elsewhere
- 583.81 Nephritis and nephropathy, not specified as acute or chronic, in diseases classified elsewhere
- 710.0 Systemic lupus erythematosus

ICD10

- M32.9 Systemic lupus erythematosus, unspecified
- M32.12 Pericarditis in systemic lupus erythematosus
- M32.14 Glomerular disease in systemic lupus erythematosus

TACHYDYSRHYTHMIAS

James G. Adams • Matthew S. Patton



BASICS

DESCRIPTION

- Any disturbance of the heart's rhythm resulting in a rate > 100 bpm
- Sinus tachycardia:
 - _ Narrow complex regular rhythm at a rate of 100–150 bpm
 - _ Max. rate typically 220 minus age
 - _ Functional response to physiologic stress caused by increased catecholamine tone or decreased vagal stimulation
- Supraventricular tachycardia (SVT):
 - _ A narrow complex tachycardia that originates above the His bundle
- Regular SVT:
 - Atrial tachycardia
 - Junctional tachycardia:
 - Regular tachycardia without preceding depolarization waves
- Irregular SVT:
 - Atrial fibrillation (AF)
 - Atrial flutter
 - Multifocal atrial tachycardia
- Ventricular tachycardia (VT):
 - _ ≥ 3 consecutive ventricular ectopic beats at a rate of 100 bpm
 - _ Most common initiating rhythm in sudden death in patients with previous MI
- Torsades de pointes:
 - _ Paroxysmal form of VT with undulating axis and prolonged baseline QT interval
 - _ Secondary to either congenital or acquired abnormalities of ventricular repolarization
 - _ Often the result of drug therapy or electrolyte disturbances
- VF:
 - _ Oscillations without evidence of discrete QRST morphology
 - _ Accounts for 80–85% of sudden cardiac deaths
 - _ Frequently results from degeneration of sustained VT

ETIOLOGY

- Sinus tachycardia:
 - _ Acute MI
 - _ Anemia

- Anxiety
- CHF
- Drug intoxication
- Hyperthyroidism
- Hypovolemia
- Hypoxia
- Infection
- Pain
- Pericardial tamponade
- Pulmonary embolus
- Atrial tachycardia:
 - Precipitated by a premature atrial or ventricular contraction
 - Electrolyte disturbances
 - Drug toxicity
 - Hypoxia
- Junctional tachycardia:
 - AV nodal re-entry
 - Myocardial ischemia
 - Structural heart disease
 - Pre-excitation syndromes
 - Drug and alcohol toxicity
- AF:
 - HTN
 - Coronary artery disease
 - Hyper-/Hypothyroidism
 - Alcohol intake
 - Mitral valve disease
 - Chronic obstructive pulmonary disease
 - Pulmonary embolus
 - Wolf–Parkinson–White (WPW) syndrome
 - Hypoxia
 - Digoxin toxicity
 - Chronic pericarditis
 - Idiopathic AF
- Atrial flutter:
 - Ischemic heart disease
 - Valvular heart disease
 - CHF
 - Myocarditis
 - Cardiomyopathies
 - Pulmonary embolus
 - Electrolyte abnormalities
 - Recent cardiac surgery

- Multifocal atrial tachycardia:
 - Hypoxic effects of chronic lung disease
 - Theophylline toxicity
- VT:
 - Dilated cardiomyopathy
 - Cardiac ischemia
 - Hypoxia
 - Cardiac scarring/fibrosis
 - After cardiac surgery or congenital anomaly repair
 - Digoxin toxicity
 - Long QT syndrome
 - Electrolyte abnormalities
- Torsades de pointes:
 - Drug toxicity (antiarrhythmic class IA and III agents, antipsychotics, antibiotics, etc.)
 - Hypokalemia
 - Hypomagnesemia
 - Congenital QT prolongation
- VF:
 - Acute MI (most common)
 - Chronic ischemic heart disease
 - Hypoxia
 - Acidosis
 - Anaphylaxis
 - Electrocutation
 - Shock
 - Hypokalemia
 - Initiation of quinidine therapy
 - Massive hemorrhage

DIAGNOSIS

SIGNS AND SYMPTOMS

- Asymptomatic
- Palpitations
- Lightheadedness
- Dyspnea
- Diaphoresis
- Dizziness
- Weakness
- Chest discomfort
- Angina

- Syncope
- Prominent neck veins
- Signs of instability:
 - Hypotension
 - Pulmonary edema
 - Chest pain
 - Mental status changes

History

- Acute onset of palpitations, lightheadedness, generalized weakness, or shortness of breath
- Sudden collapse, often preceded for minutes–hours by chest pain
- Prior history of cardiac disease common (ischemia, CHF)

Physical-Exam

Determine if the patient is hemodynamically stable:

- Assess mental status.
- Assess heart rate.
- Assess BP: Normal or hypotensive
- Cardiac exam

ESSENTIAL WORKUP

- ABCs
- Determination of unstable vs. stable patient
- Detailed history
- 12-lead EKG and rhythm strip to categorize the tachycardia

DIAGNOSIS TESTS & INTERPRETATION

Lab

Studies should be ordered based on the presentation to evaluate underlying metabolic abnormalities or ischemia.

Diagnostic Procedures/Surgery

EKG:

- SVT:
 - Narrow complex, rate usually 130–160
 - Uniformity of polarity and amplitude
 - No P-waves visible
- AF:
 - Irregular, narrow QRS complex, rate < 150--170 bpm
- Atrial flutter:
 - Regular atrial rate, usually > 300

- Beat-to-beat uniformity of cycle length, polarity, and amplitude
- Sawtooth flutter waves directed superiorly and most visible in leads II, III, aVF
- AV block usually 2:1, but occasionally greater or irregular
- Multifocal atrial tachycardia:
 - 3 distinctly different conducted P waves with varying pulse rate intervals
- VT:
 - QRS > 0.12 sec and often > 0.14 sec.
- Torsades de pointes:
 - Wide complex, ventricular rate > 200 bpm
 - QRS structure displays an undulating axis, with the polarity of the complexes appearing to shift around the baseline.
 - Occurrence is often in short episodes of < 90 sec.
- VF:
 - EKG shows oscillations without evidence of discrete QRST morphology.
 - Oscillations are usually irregular and occur at a rate of 150–300 bpm.
 - When the amplitude of most oscillations is 1 mm, the term “coarse” is used.
 - “Fine” VF is used for oscillations < 1 mm.



TREATMENT

PRE HOSPITAL

Cardiopulmonary resuscitation if pulseless

INITIAL STABILIZATION/THERAPY

- IV access
- Oxygen
- Cardiac monitor
- Determine rhythm

ED TREATMENT/PROCEDURES

- Irregular narrow complex (A fib):
 - Rate control
 - β -Blockers or calcium channel blockers
 - Anticoagulation if onset is > 24 hr
 - Cardioversion for *severe* hemodynamic compromise
- Regular narrow-complex tachydysrhythmia:
 - Vagal maneuvers occasionally terminate the dysrhythmia:
 - Beware of carotid disease in elderly.
 - Adenosine:
 - May be diagnostic, revealing underlying AF/atrial flutter
- Stable wide-complex tachycardia:

- Determine whether VT or SVT with aberrancy
- Administration of AV nodal-blocking agents (verapamil, adenosine) may result in VF:
 - With WPW, use amiodarone, flecainide, procainamide, or DC cardioversion.
- Electrical cardioversion should be utilized when mechanism unknown.
- Antidysrhythmic drugs include procainamide and amiodarone.
- Torsades de pointes:
 - Magnesium, overdrive pacing, amiodarone
 - Correct underlying abnormal electrolytes.
 - Consider repletion of serum K to 4.5.
- Polymorphic VT (variable QRS morphology):
 - Ejection fraction (EF) normal:
 - β -Blockers, lidocaine, amiodarone, or procainamide
 - EF abnormal:
 - Amiodarone or lidocaine; then synchronized cardioversion
 - Treat ischemia and correct electrolytes.
- Monomorphic VT:
 - EF normal:
 - Procainamide preferred to amiodarone, sotalol, lidocaine; synchronized cardioversion
 - EF abnormal:
 - Amiodarone or lidocaine
 - Procainamide with caution as may cause hypotension; synchronized cardioversion
- VF or pulseless VT:
 - Treatment per ACLS protocol

MEDICATION

- Adenosine: 6 mg (peds: 0.1 mg/kg up to 6 mg) rapid IV push; if no response after 1–2 min, then 12 mg (peds: 0.2 mg/kg up to 12 mg), may repeat 12 mg (0.2 mg/kg)
- Amiodarone:
 - *VT/SVT with pulse*: 150 mg IV over 10 min (peds: 5 mg/kg IV over 20--60 min, redose up to 15 mg/kg, 300 mg max), then 1 mg/min for 6 hr and 0.5 mg/min for next 18 hr.
 - *VF/pulseless VT*: 300 mg IV push (peds: 5 mg/kg IV), may give 150 mg IV push 3--5 min after if no response (peds: redose up to 15 mg/kg or 300 mg max), followed by infusion as above.
- Diltiazem: 0.25 mg/kg IV (usually 10–20 mg) over 2 min, followed in 15 min by 0.35 mg/kg IV over 2 min
- Epinephrine: 1 mg (peds: 0.01 mg/kg) IV push q3–5min; 2.5 mg (peds: 0.1 mg/kg) endotracheally q3–5min

- Lidocaine: 1–1.5 mg/kg (100 mg) (peds: 1 mg/kg) IV push, may repeat q5–10min, max. dose 3 mg/kg
- Magnesium sulfate: 2 g diluted in 100 mL D₅W IV over 2 min (peds: 25–50 mg/kg, max. 2 g, IV over 10–20 min)
- Metoprolol: 5–15 mg slow IV push at 5-min intervals to total of 15 mg
- Procainamide:
 - *VF/pulseless VT*: 30 mg/min (peds: Not recommended) IV load until rhythm resolves, hypotension, QRS widens > 50% or max. 17 mg/kg, then 1–4 mg/min IV
 - *Perfusing VT*: 20 mg/min (peds: 15 mg/kg IV over 30–60 min) IV load until rhythm resolves, hypotension, QRS widens > 50% or max. 17 mg/kg, then 1–4 mg/min IV
 - *SVT*: 15–17 mg/kg IV at 20–30 mg/min *or* 100 mg IV q5min slow IV push until rhythm resolves or max. dose 1,000 mg (peds: 3–6 mg/kg IV over 5 min, max. 100 mg/dose, may repeat q5–10min as needed to total dose 15 mg/kg)
- Vasopressin: 40 U (peds: Not recommended) IV push once



FOLLOW-UP

DISPOSITION

Admission Criteria

- VT or VF
- Possible cardiac ischemic event
- Persistent SVT
- Underlying metabolic abnormalities

Discharge Criteria

Terminated supraventricular rhythm without organ hypoperfusion

Issues for Referral

Electrophysiologic testing:

- Diagnostic but not required emergently
- Determines therapy for accessory pathways

PEARLS AND PITFALLS

- Always suspect a ventricular rhythm with a wide complex rhythm, especially in the older patient.
- Antidysrhythmic administration may increase success rate of cardioversion.
- Rapid, uninterrupted chest compressions may increase the success rate of

defibrillation for a patient with a pulseless rhythm.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Atrial Fibrillation
- Supraventricular Tachycardia
- Ventricular Tachycardia



CODES

ICD9

- 427.0 Paroxysmal supraventricular tachycardia
- 427.1 Paroxysmal ventricular tachycardia
- 785.0 Tachycardia, unspecified

ICD10

- I47.1 Supraventricular tachycardia
- I47.2 Ventricular tachycardia
- R00.0 Tachycardia, unspecified

TASER INJURIES

Christian M. Sloane

BASICS

DESCRIPTION

- Tasers are part of a class of less lethal weapons referred to as conducted energy weapons (CEWs).
- Most common in US are those made by Taser International; these include the M-26, X-26, and X2 although others exist.
- These devices use a high-voltage low-amperage current to override the subject's ability to control the peripheral nervous system; they cause pain so as to induce subject compliance.
- Handheld devices such as stun guns require the application of 2 exposed probes to the skin (or close to the skin) to cause a localized response (usually pain).
- Other devices, such as the Taser International devices, have barbed probes attached to thin wires that can be shot up to 35 ft to deliver current from a distance.
- Needle lengths of CEW barbs are of varied lengths but generally less than or around 0.5 in.
- The effects of CEWs vary depending on the type of device being used, location, placement, and distance between the probes on the subject's body. If probe spread on the body is <5 cm, effectiveness is less.
- Skin effects
 - May leave marks at site of probe contact, called “signature marks”
 - Small puncture wound from barbs
- Skeletal effects
 - Fractures may result from falls.
 - Vertebral compression fractures have been reported as a result of a Taser discharge.
 - Barbs may penetrate bone.
- Muscle effects
 - Strains possible
 - Rhabdomyolysis possible with repeated prolonged use, though more likely could result from the underlying cause leading to use of the Taser (e.g., excited delirium syndrome [ExDS])
- Cardiovascular effects
 - Theoretically could cause ventricular fibrillation if a charge was delivered over the heart during a vulnerable part of the cardiac cycle. This risk is not easily quantifiable but estimated to be very low.
 - A case of atrial fibrillation has been reported following Taser use.

- No significant effects in otherwise healthy subjects. Does not cause changes in ECG or troponin I.
- Unclear how device would affect pacemakers/automatic internal cardiac defibrillators (AICDs). Energy is low; theoretically should not cause damage. Could cause an AICD to deliver a shock if electrical activity of the CEW is misinterpreted as a dysrhythmia.
- Nervous system effects
 - There have been case reports of skull penetration and seizure.
- Respiratory effects
 - Initial concerns that the CEWs would disrupt ventilation proved unfounded. Research has shown that subjects actually increase ventilation during an application.

ETIOLOGY

The devices are commonly used in law enforcement but may also be used in the military, self-defense, by those wishing to commit a crime, or an accidental discharge of a weapon on to law enforcement personnel.

DIAGNOSIS

SIGNS AND SYMPTOMS

ALERT

Subjects on whom a device has been used may be in a state of ExDS.

History

A history of the use of a device is usually obtained. Important factors are:

- The type of device
- The mode used (probe or drive stun)
- The number of cycles discharged
- The duration of cycles applied
- Location of contact on the body

Physical-Exam

- Pay particular attention to the location of barb strike. Barbs in the skin, though unlikely, may cause injury to underlying structures:
 - Eye
 - Face
 - Neck
 - Groin
 - Genitals
 - Secondary injuries do occur
 - From fall

- From aspiration if device is deployed in the water
- From tetanic muscle contraction
- From barb penetration

ESSENTIAL WORKUP

- All persons who have been exposed to CEW activation should receive a medical evaluation. The scope of that evaluation should depend on the type of use and state of the subject.
- For a subject who subsequently becomes compliant, is alert, and is acting appropriately and/or had CEW darts hit areas that are not medically sensitive, the darts may be removed and an evaluation done at intake to a detention facility.
- Given the risk to a subject who is in a state of ExDS regardless of CEW use, such a person requires an ED evaluation.

Geriatric Considerations

The above groups warrant a medical evaluation, given that there are so few data to guide any definitive statements about their use.

DIAGNOSIS TESTS & INTERPRETATION

Labs should be directed at the underlying reason the person was “tasered.” No labs are required simply because the person was tasered.

Lab

If ExDS is present:

- CBC
- Chemistry panel
- Creatine kinase
- UTOX
- VBG to check for acidosis
- Lactate

Imaging

- Not routine
- If altered level of consciousness with no clear cause, then head CT
- X-ray if Taser barb penetrated bone; this is most likely if it hit a digit or where bone is close to skin (e.g., tibia, nose).
- US if individual is pregnant
- Other imaging guided by suspicion of traumatic/secondary injury

Diagnostic Procedures/Surgery

- Pacemaker interrogation if patient has pacemaker or AICD, since the device may have been damaged or have delivered a shock.
- ECG if there is underlying significant heart disease

- Women who are pregnant > 20 wk should have fetal monitoring.

DIFFERENTIAL DIAGNOSIS

Usually not unclear if device used.



TREATMENT

PRE HOSPITAL

- If patient is acting normally, has normal vital signs, and AO \times 4, appropriate, no special intervention is needed. Depending on jurisdiction, barb may be removed if not in sensitive area (face, eye, groin, neck, genitals). Otherwise, stabilize barb and transport patient to hospital for removal.
- If patient is agitated, treatment as per agitation/ExDS.
- If cardiac dysrhythmia is present, initiate cardiac monitoring, IV access, oxygen, treat if in protocol and scope or practice.
- Treat any secondary traumatic injuries.

INITIAL STABILIZATION/THERAPY

If ExDS, then treat per guidelines, including medications.

ED TREATMENT/PROCEDURES

Initial treatment is steered toward underlying injuries.

- If patient acting normally, normal vital signs, AO \times 4 appropriate, and no complaints of secondary injury, no special intervention needed.
- Update tetanus status as needed.
- Taser barb removal: Using 2 fingers of nondominant hand, stabilize skin around the barb by holding it down. Use dominant hand to grasp barb shaft and pull barb out.
- Treat the puncture wound like any other.
- Approach secondary traumatic injuries per trauma protocols.
- Treat dysrhythmias per protocol.
- Consider and treat ExDS if patient unstable.

MEDICATION

- Tetanus vaccination, dT or dTaP: 0.5 mL IM
- Midazolam (Versed) 5 mg IM/IV for agitation
- Haloperidol (Haldol) 5 mg IM/IV for agitation



FOLLOW-UP

DISPOSITION

Admission Criteria

- Admit for signs of:
 - Cardiac instability
 - Excited delirium syndrome
 - Serious secondary injury

Pregnancy Considerations

Any female who is pregnant should undergo a medical evaluation of the pregnancy; if viable, she should undergo fetal monitoring at an appropriate facility.

Discharge Criteria

Patient acting normally, normal vital signs, AO × 4 appropriate, and no complaints of secondary injury, or secondary injuries treated and stable for discharge

Issues for Referral

Wound care, injury follow-up

PEARLS AND PITFALLS

- These patients may be suffering from ExDS, hence law enforcement became involved or Taser was used. Failure to aggressively treat this life-threatening condition will result in untoward outcome.
- Always screen for possible secondary injury.
- Stable, alert, appropriate subjects do not need much more than simple barb removal (if necessary), a tetanus vaccination update, and wound care.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Excited Delirium Syndrome, Puncture Wounds



CODES

ICD9

- 919.8 Other and unspecified superficial injury of other, multiple, and unspecified sites, without mention of infection
- 994.8 Electrocution and nonfatal effects of electric current

ICD10

- T14.8 Other injury of unspecified body region
- T75.4XXA Electrocution, initial encounter

TEMPORAL–MANDIBULAR JOINT INJURY/SYNDROME

Ben Osborne • Jennifer Dohrman

BASICS

DESCRIPTION

- Myofascial pain causing temporomandibular joint (TMJ) dysfunction
- Prevalence of 40–75% of 1 sign of TMJ disorder
- Most common in 20–50-yr-olds
- Females seek treatment more frequently
- 40% have symptoms that resolve spontaneously
- TMJ is a synovial joint:
 - Allows for hinge and sliding movements
- Articular disorders:
 - Congenital or developmental
 - Degenerative joint disorders:
 - Inflammatory (rheumatoid arthritis)
 - Noninflammatory (osteoarthritis)
 - Trauma
 - TMJ hypermobility:
 - Laxity
 - Dislocation
 - Subluxation
 - TMJ hypomobility:
 - Trismus
 - Fibrosis
 - Infection
 - Neoplasm
- Masticatory muscle disorders:
 - Local myalgias
 - Myositis
 - Muscle spasm
 - Contracture
 - Myofascial pain disorder
- TMJ clicking:
 - May be normal finding; present as a transient finding in 40–60% of the population
- TMJ motion:
 - Typical range is 35–55 mm (maxillary to mandible incisors)
 - Limited by adhesions within the joint or disk displacement or trismus from muscle spasm

- Intra-articular disk disorder:
 - Anterior displacement with reduction:
 - Displacement in closed mouth position
 - Often with a click and variable pain with opening mouth
 - May worsen over time
 - Anterior disk displacement without reduction:
 - Disk is a mechanical obstruction to opening mouth
 - Maximal opening may be 20–25 mm
 - Often difficult to correct

ETIOLOGY

TMJ dysfunction is poorly understood:

- Multifactorial:
 - Bruxism (teeth grinding)
 - Trauma
 - Malocclusion
- Onset may be related to stress

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Preauricular pain:
 - Constant but with fluctuating intensity
 - Dull and aching
 - May be referred to the ipsilateral ear, head, neck, or periorbital region
 - Exacerbated by mandibular movement (pathognomonic)
 - More conspicuous at night and may cause insomnia
 - Often worsens through the day
- Tongue, lip, or cheek biting
- Ear pain
- Ear fullness
- Tinnitus
- Dizziness
- Neck pain
- Headache
- Eye pain

Physical-Exam

- Joint sounds:
 - Popping or clicking sensation with TMJ articulation

- A palpable or audible click with opening and closing
- Not sufficient for diagnosis if not accompanied by pain or other dysfunction
- Misalignment and limited range of motion:
 - Dentoskeletal malocclusion or lateral deviation
 - Open or closed locking of the jaw
- Tenderness over the muscles of mastication and TMJ:
 - Masseter muscle most commonly painful
- Pain with dynamic loading (bite on gauze)

ESSENTIAL WORKUP

- Diagnosis based on history and physical exam
- Exclude other causes of headache and facial pain

DIAGNOSIS TESTS & INTERPRETATION

Lab

No specific lab tests are indicated unless there is concern for other disease process, i.e., ESR may help distinguish temporal arteritis from TMJ dysfunction.

Imaging

- Panorex is the screening radiograph of choice:
 - May demonstrate fracture or intra-articular pathology (i.e., tumor or degenerative joint disease) but usually unremarkable
- CT: Best for evaluating bony structures for fractures, dislocations, etc.
- MRI: Best imaging for nonreducing displaced disks:
 - Allows for better visualization of joints simultaneously

DIFFERENTIAL DIAGNOSIS

- Acute coronary syndrome
- Carotid artery dissection
- Intracranial hemorrhage (subarachnoid hemorrhage)
- Inflammatory diseases:
 - Giant cell (temporal) arteritis
 - Rheumatoid arthritis
- Trigeminal or glossopharyngeal neuralgia
- Vascular headache
- Intraoral and dental pathology
- Herpes zoster
- Salivary gland disorder
- Otitis media, otitis externa
- Sinusitis
- Elongated styloid process pain
- Jaw trauma (fracture or dislocation)



TREATMENT

PRE HOSPITAL

Provide comfort and reassurance

INITIAL STABILIZATION/THERAPY

Make sure airway is patent

ED TREATMENT/PROCEDURES

- Acute therapeutic options:
 - _ Patient reassurance and education—”usually mild and self-limited”
 - _ Rest
 - _ Heat
 - _ Analgesics and anxiolytics
 - _ Urgent reduction of open or closed locking TMJ
 - _ Reduction of TMJ dislocation:
 - Dislocation usually bilateral
 - IV muscle relaxant may be helpful
 - Often requires procedural sedation
 - Monitor airway
 - May face the patient or perform from behind the patient
 - Protect thumbs with gauze and/or tongue depressors
 - Thumbs rest on intraoral surface of mandible
 - Fingers wrap around jaw
 - Firm, progressive downward pressure as jaw is guided 1st in a caudal direction and then posteriorly
 - _ Physical therapy—moist heat or ice packs
 - _ Pain site injections with mixture of steroids/lidocaine
- Outpatient management:
 - _ Combination pharmacotherapy:
 - NSAIDs
 - Muscle relaxants
 - Antidepressants
 - Sedative hypnotics
 - _ Home physical therapy—moist heat or ice packs and mechanically soft diet
 - _ Caution not to open mouth > 2 cm for 2 wk
 - _ Avoid triggers such as gum chewing
 - _ Occlusal appliance worn during sleep
 - _ Referral to dentist or oral–maxillofacial surgeon

MEDICATION

First Line

- Naproxen: 250–500 mg PO BID (peds: 10 mg/kg/d PO div. q12h)
- Cyclobenzaprine: 5–10 mg PO TID (peds: 5–10 mg PO TID if > 15 yr old); caution with hepatic impairment
- Diazepam: 2–10 mg PO BID–TID (peds: < 12 yr old 0.12–0.8 mg/kg/d PO div. q6–8h); poor efficacy when used alone
- Ibuprofen: 600 mg (peds: 10 mg/kg) PO q8h; less effective than naproxen

Second Line

- Nortriptyline: 10–50 mg PO qhs
- Narcotic analgesic
- Sedative hypnotics



FOLLOW-UP

DISPOSITION

Admission Criteria

TMJ syndrome can be managed on an outpatient basis unless a locked or dislocated joint cannot be reduced

Discharge Criteria

Treat as outpatient with pain medication, muscle relaxants, and warm compresses

FOLLOW-UP RECOMMENDATIONS

Patients with TMJ syndrome may need referral to ENT, oral surgeon, or dentist for further care

PEARLS AND PITFALLS

- TMJ locking must be addressed urgently
- If ear pain with no ear findings, evaluate for TMJ
- NSAIDs, rest, and heat are 1st-line therapy

ADDITIONAL READING

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York, NY: McGraw-Hill; 2011.

- Lewis EL, Dolwick MF, Abramowicz S, et al. Contemporary imaging of the temporomandibular joint. *Dent Clin North Am.* 2008;52:875–890.
- Marx JA, Hockberger RS, Walls RM, et al. *Rosen's Emergency Medicine: Concepts and Clinical Practice.* 7th ed. St. Louis, MO: Mosby; 2009.
- Scrivani SJ, Keith DA, Kaban LB. Temporomandibular disorders. *N Engl J Med.* 2008;359:2693–2705.



CODES

ICD9

- 524.60 Temporomandibular joint disorders, unspecified
- 524.62 Temporomandibular joint disorders, arthralgia of temporomandibular joint
- 524.64 Temporomandibular joint sounds on opening and/or closing the jaw

ICD10

- M26.60 Temporomandibular joint disorder, unspecified
- M26.62 Arthralgia of temporomandibular joint
- M26.69 Other specified disorders of temporomandibular joint

TENDON LACERATION

Nicholle D. Bromley

BASICS

ALERT

Tendons near lacerations must be explored through *complete range of motion* to rule out injury.

DESCRIPTION

- Based on mechanism
- External trauma:
 - Penetrating trauma:
 - Gunshot wounds
 - Glass
 - Knives
 - Foreign bodies
 - Blunt trauma:
 - Crushing force or avulsion from hyperextension of a joint
- Internal trauma:
 - Entrapment/laceration from bony fracture (rare)

ETIOLOGY

Tendon injuries grossly categorized into those affecting upper vs. lower extremities:

- Upper-extremity injuries frequently related to the workplace, home, an assault, or attempted suicide
- Lower-extremity injuries most often associated with work or motor vehicle accident

DIAGNOSIS

SIGNS AND SYMPTOMS

- Pain is the cardinal symptom.
- Functional deficit
- Soft tissue damage:
 - Swelling
 - Ecchymosis
 - Lacerations
 - Hemorrhage
- Abnormal resting position of the extremity or large joint instability increases suspicion for tendon injury.

ESSENTIAL WORKUP

- A careful history:
 - Mechanism, time of injury
 - Hand position during injury
 - Hand dominance
 - Drug allergies
 - Medications
 - Past medical history
 - Tetanus vaccination status
- Physical exam:
 - Examine resting position of hand. (At rest there is natural flexion of fingers increasing from radial to ulnar side.)
 - Examine the wound in position of initial injury.
 - Perform neurovascular exam before local anesthesia is instilled.
 - Examine each digit separately.
 - Test strength against resistance.
 - Examine tendon with direct visualization through full range of motion.
- Flexor digitorum profundus injuries:
 - Present with inability to flex the distal interphalangeal (IP) joint
 - Exam involves stabilizing the proximal IP joint in full extension while the patient attempts to flex distal IP joint.
- Flexor digitorum superficialis injuries:
 - Present with inability to flex the proximal IP joint of a digit
 - Usually established by means of standard superficialis tendon test:
 - While holding the uninjured digits in full extension, the patient attempts to flex the affected finger at the proximal IP joint.
 - False negative if profundus is functional.
 - The distal IP joint extension test:
 - May make this diagnosis more apparent
 - Patient is asked to make a precision pinch between thumb and the injured finger.
 - Then asked to flex the proximal IP joint so that the distal IP joint is hyperextended
 - Confirms the integrity of the flexor digitorum superficialis
- Forearm and wrist flexor injuries:
 - Present with inability to flex ulnar or radial side of wrist or to flex the wrist while opposing the thumb to the little finger
- Extensor tendon injuries:
 - Found by weakness or lack of extension of the distal phalanx against resistant
 - Indicates partial or complete disruption
 - Best determined with patient placing palm on flat surface and asking the patient to attempt to extend the fingers individually

- Palpate each tendon.
- Loss of normal tension indicates injury.
- Further explore tendons and wounds after local anesthesia (1% lidocaine or 0.5% bupivacaine) in a bloodless, well-lit surgical field:
 - Tendons near lacerations must be explored through complete range of motion.
 - Best elucidates tendon injuries distal or proximal to a skin wound

Pediatric Considerations

- More difficult to get an adequate exam
- The healing process is usually quicker and more often associated with complete return to preinjury function.

DIAGNOSIS TESTS & INTERPRETATION

Lab

Wounds 1st examined >12 hr after injury or wounds with evident infection should be cultured.

Imaging

- Radiographs are frequently needed to identify radiopaque foreign bodies or fractures.
- High-frequency US can be used to identify complete tendon lacerations:
 - Partial tendon lacerations difficult to image
 - A water bath may help when attempting to image a painful extremity.
- US guidance may help to guide removal of foreign bodies.
- MRI

DIFFERENTIAL DIAGNOSIS

- Always rule out an associated foreign body or fracture.
- Lacerations over the proximal IP joint may involve the lateral bands or the central slip of the extensor mechanism:
 - Boutonnière deformity from improper repair
- Disruption of the extensor tendon distal to the central slip results in a mallet finger deformity.
- “Jersey finger” is a closed traumatic injury with avulsion of the flexor digitorum profundus, seen when a football player grabs the jersey of another player and his finger gets stuck.
- Avulsion of the flexor digitorum superficialis distally may be present with or without an associated avulsion fracture:
 - Suspect when a grasping finger is hit by a fast-moving object (jammed finger).



TREATMENT

PRE HOSPITAL

- Do not remove foreign matter from the patient in the field.
- Immobilize and transport patient.
- Apply direct pressure to control hemorrhage.
- Assess distal neurovascular status for signs of compromise.
- Contact medical control before any attempted reduction.

INITIAL STABILIZATION/THERAPY

- Evaluate extremity and control hemorrhage with direct pressure.
- Remove all jewelry or constricting bands.

ED TREATMENT/PROCEDURES

- Pain control as required
- Administer tetanus toxoid as needed.
- Copious irrigation with 1 L NS
- Broad-spectrum antibiotic, such as a 1st-generation cephalosporin (Cefazolin)
- Tendon lacerations associated with human bites:
 - _ Must be copiously irrigated
 - _ Place on IV antibiotics with coverage of oral anaerobes (ampicillin/sulbactam).
 - _ Immobilize and elevate the hand.
- Remove all foreign bodies and provide débridement of avascular tissue.
- Partial tendon lacerations that involve >20% of the cross-sectional area of the tendon must be repaired.
- Simple extensor tendon lacerations may be repaired in the ED:
 - _ Use a 4-0 or 5-0 nonabsorbable suture in a figure-of-8 or a modified Kessler stitch.
- All suspected flexor tendon, wrist, and distal forearm tendon lacerations require consultation by a hand surgeon, ideally within 12 hr.
- Tendon lacerations over the proximal IP joint may result in a boutonnière deformity:
 - _ Refer to a hand surgeon.
- The superficial nature of multiple tendons, nerves, and vessels on the volar aspect of the wrist renders them easily vulnerable to penetrating trauma:
- “Spaghetti wrist” or “full house”:
 - _ Volar wrist laceration with at least 10 structures involved
 - _ Requires prompt consultation with a hand surgeon
- Tendon lacerations associated with fractures require referral for operative repair.
- If a surgeon is not promptly available:
 - _ Irrigate copiously.

- Close skin without repair of tendon.
- Immobilize injured hand with a bulky volar dressing and splint.
- Wrist in 20–30° of flexion
- Metacarpal joint in 60–70° of flexion
- IP joints in 10–15° of flexion

MEDICATION

- Ampicillin/sulbactam: 3 g IV q6 (peds: 200 mg/kg/d IM or IV div. q6h)
- Cefazolin: 1 g IV piggyback (peds: 100 mg/kg/d IM or IV div. q6h, followed by 40 mg/kg/d PO QID for 5–7 days)
- Tetanus toxoid: 0.5 mL IM (peds: <7 yr–diphtheria–pertussis–tetanus vaccine preferred; in those >7 yr, adult dose tetanus toxoid if immunization series not completed), tetanus immune globulin, as required, 250 IU administered IM



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients with infected tendon lacerations must be admitted for operative débridement.
- Any patients with tendon injury secondary to human bite must be admitted for operative débridement and IV antibiotics.
- Any patients with significant flexor tendon laceration may be admitted for timely operative repair or transferred to the nearest hand surgeon.

Discharge Criteria

- Patients with an extensor tendon laceration that is not infected, nor associated with other significant injury or underlying fracture, which was repaired by the ED physician and is now properly splinted, may be discharged with timely surgical follow-up.
- Patients with an extensor tendon laceration requiring surgeon referral for repair (wrist, forearm, proximal IP joint), which has been properly treated and splinted, with the patient placed on antibiotics, may be discharged for timely surgical follow-up.

PEARLS AND PITFALLS

- Partial lacerations are common but more difficult to diagnosis than complete disruptions because they may demonstrate intact function:
 - Alterations of the normal resting hand position may indicate partial laceration.

- Lacerations over the metacarpophalangeal joint should be considered the result of a human bite until proven otherwise:
 - Look for associated extensor tendon injury while metacarpophalangeal joint flexed.
- It is very important to test strength because tendon injuries with up to a 90% full-thickness laceration can have normal range of motion. Therefore, test strength against resistance.
- Tendon laceration with > 20% cross-sectional area of involvement need repair

ADDITIONAL READING

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- Sokolove PE. Extensor and flexor tendon injuries in the hand, wrist, and foot. In: Roberts JR, Hedges JR, eds. *Clinical Procedures in Emergency Medicine*. 5th ed. Philadelphia, PA: Saunders Elsevier; 2009.
- Wu TS, Roque PJ, Green J, et al. Bedside ultrasound evaluation of tendon injuries. *Am J Emerg Med*. 2012;30(8):1617–1621.



CODES

ICD9

- 848.9 Unspecified site of sprain and strain
- 884.2 Multiple and unspecified open wound of upper limb, with tendon involvement
- 891.2 Open wound of knee, leg [except thigh], and ankle, with tendon involvement

ICD10

- S46.929A Laceration of unspecified muscle, fascia and tendon at shoulder and upper arm level, unspecified arm, initial encounter
- S56.429A Laceration of extensor muscle, fascia and tendon of unspecified finger at forearm level, initial encounter
- S86.909A Unspecified injury of unspecified muscle(s) and tendon(s) at lower leg level, unspecified leg, initial encounter

TENDONITIS

James Killeen



BASICS

DESCRIPTION

- The term “tendinitis” has been used to describe chronic painful tendon injuries before the underlying pathology was understood. This term has led to confusion about the cause, chronicity, and treatment of the underlying disorder. The terms “tendinosis” or “tendinopathy” should be used to describe chronic tendon disorders.
- Overuse syndrome:
 - Clinical syndrome of chronic pain and tendon thickening
 - Synovial cells increase in thickness
 - Excess synovial fluid collection
 - Constant irritation
- If no further injury occurs, the acute process may last from 48 hr–2 wk.
- Tendinopathy is described as fibrosis being present without inflammatory cells and symptoms persist longer than 3 mo.

ETIOLOGY

- Mechanical overload or repetitive microtrauma to the musculotendinous unit:
 - Intrinsic factors:
 - Inflexibility
 - Muscle weakness or imbalance
 - Extrinsic factors:
 - Excessive deviation, frequency, or activity
 - In tendinopathies, the collagen is in a state of disrepair, with proliferation and chronic irritation of neurovascular repair tissue in the tendon and its linings.
- Chemotactic and vasoactive chemical mediators are released:
 - Vasodilatation and cellular edema increasing the number and activity of PMNs



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- The patient’s history should explain what movement led to the injury.
- Repetitive stress and mechanical overload
- The classic inflammatory signs include pain, warmth, erythema, and swelling.

- Pain will resolve quickly after initial movement, only to become a throbbing pain after exercise.

Physical-Exam

- Defined as inflammation of the tendon only
- There is a poor distinction between tendonitis and tenosynovitis (degree of inflammation). These are now termed as tendinopathies.
- Clinical findings:
 - Warmth
 - Presence of an effusion
 - Decreased range of motion
 - Instability
 - Pain on motion
 - Tenderness over tendon site

Specific Conditions Supraspinatus Tendinopathy

Supraspinatus and other rotator cuff tendons:

- Compressed between humerus and acromion
- Overuse of the extremity may lead to microtrauma of the tendons and fibers.
- Neer classification:
 - Stage 1:
 - Age < 25
 - Involved in sports requiring repetitive overhead motion (e.g., swimmers or pitchers)
 - Edema and hemorrhage of the tendon
 - Flexion–abduction motion will elicit pain.
 - “Dull aches”
 - Stage 2:
 - Age 25–40
 - Pain is constant and worsens at night.
 - Active motion is limited by pain.
 - Passive range of motion is preserved.
 - Diffuse, intense pain
 - Fibrosis and thickening of the tendon
 - Stage 3:
 - Partial or complete tendon tears
 - Raising the humerus in a forced forward flexion while preserving scapular rotation causes impingement.

Calcific Tendonitis

- Age older than 40 yr with unknown etiology.
- Any tendon of the rotator cuff can be affected, but there is a predisposition for the supraspinatus.
- Most cases are asymptomatic and are found on routine radiographs.
- Calcium is deposited within the tendon over time, undergoes spontaneous

resorption, causing pain.

- Acute attacks may develop from crystal release.

Bicipital Tendinopathy

- Pain to the anterior shoulder, which radiates down the radius
- Discomfort when rolling on the shoulder or trying to reach a hip pocket or back zipper
- Focal tenderness is between the greater and lesser tuberosities of the humerus.
- Yergason test:
 - _ Elbow at 90° and arm against the body
 - _ Pain increases with resisted supination of the wrist.
- Speed test:
 - _ Pain along the bicipital groove with resisted forward flexion and forearm supination

Lateral Epicondylitis (Tennis Elbow)

- Rotational repetitive motion causes pain.
- Dull ache on the outside of the elbow that increases with grasping and twisting
- Inflammation at the insertion of the common extensor tendon at lateral epicondyle of humerus
- Resisted active dorsiflexion of the wrist on extension of the middle finger against resistance can reproduce pain with the elbow extended.
- Inflammation at site of insertion of the flexor carpi radialis on the medial epicondyle:
 - _ Bowlers, golfers, pitchers
 - _ Active flexing of the wrist against resistance causes pain.

Wrist/Hand

- Inflammatory changes of the synovial lining between tendons and the retinaculum
- De Quervain tenosynovitis:
 - _ Inflammation of the abductor pollicis longus and extensor pollicis brevis
 - _ Finkelstein test:
 - Patient makes fist with thumb curled in palm.
 - Wrist is deviated in the direction of the ulna.
 - Pain occurs in 1st extensor compartment.
 - _ Osteoarthritis of the carpal metacarpal joints or GC tenosynovitis causes the same pain.

Trigger Finger

- Proximal portion of the palmar flexor tendon sheath becomes stenosed and catches as the finger is moved.
- Symptoms vary from pain to locking in flexion.

Ankle

- Achilles tendinopathy:
 - _ Overuse injury commonly seen in males
 - _ Trauma or systemic disease causing inflammation
 - _ With repeated stress, scar tissue formation and degeneration of the tendon

will occur.

- Patient will have pain, reduced range of motion, or morning stiffness
- Achilles tendon rupture
 - Seen more commonly in 30–40-yr-old recreational athletes
 - “Popping sensation”
 - Acute weakness, inability to continue activity
 - Feels like being kicked or hit in back of leg
 - May initially have a gap by palpation, followed by ecchymosis and a boggy sensation
 - Inability to plantar flex the foot with complete rupture
 - Thompson test:
 - Patient lies prone with the feet hanging over the edge of the bed.
 - Physician squeezes the calf muscles and looks for plantar flexion
 - 20–30% of Achilles tendon ruptures are missed at the initial visit because the clinician was falsely reassured by the patient’s ability to plantar flex or walk.
 - The Matles Test: the patient lies prone with knees flexed to 90°. Observe whether the affected foot is dorsiflexed or neutral (both are abnormal) compared to the uninjured side, where the foot should appear plantarflexed.

Pediatric Considerations

- Apophysitis occurs in children at an ossification center subject to traction:
 - Little League elbow at the medial epicondyle
 - Osgood–Schlatter syndrome at tibial tubercle
- Avascular necrosis (AVN):
 - Presents with pain and swelling around a joint
 - Can occur at various locations
 - Well-recognized sites:
 - Capitellum of the humerus
 - Head of the femur
 - Tarsal navicular
 - Metatarsal head
 - Diagnosis is made by plain radiographs.
 - Radiographs are often required to rule out fracture, AVN, osteochondritis dissecans, or bony tumor.

ESSENTIAL WORKUP

Physical exam

DIAGNOSIS TESTS & INTERPRETATION

Lab

CBC, C-reactive protein (CRP), ESR only if more serious infection suspected

Imaging

- Radiographs:
 - Extra-articular from articular etiologies
 - “SECONDS”:
 - Soft tissue swelling
 - Erosions
 - Calcifications
 - Osteoporosis
 - Narrowing
 - Deformity
 - Separation
- Ultrasound
 - Evaluate joint effusions
 - More sensitive than MRI
 - Used more frequently in the emergency setting
 - Focal tendon thickening
 - Focal hypoechoic areas
 - Irregular and ill-defined borders
 - Peritendinous edema
- MRI:
 - Internal morphology of the tendon and the surrounding structures
 - Helps diagnose retrocalcaneal bursitis and insertional tendonitis
 - Reveals tendon thickening and increased signal with chronic tendon abnormalities
- Scintigraphy:
 - ⁹⁹Techetium pertechnetate phosphate (binds with plasma protein) and concentrates in joint space (bursitis)

DIFFERENTIAL DIAGNOSIS

- Septic arthritis
- Fracture
- Osteoarthritis



TREATMENT

PRE HOSPITAL

Immobilize injured extremity as indicated.

INITIAL STABILIZATION/THERAPY

Ice, immobilization pending work-up

ED TREATMENT/PROCEDURES

- General:
 - Rest
 - NSAIDs
 - Ice (10–20 min intervals)
 - Range of motion exercises
 - Eccentric exercise is the application of a load (i.e., muscular exertion) to a lengthening muscle.
 - Local injection for pain control
 - Outpatient management
 - Admit only for surgery or severe disability
 - Allow 6–12 wk to heal
 - Recent studies have described successful investigational therapies
 - Prolotherapy, an ultrasound-guided injection of dextrose and lidocaine to stimulate repair.
 - Sclerotherapy injections of Polidocanol, a sclerosing substance to reduce neovascularity
 - **Aprotinin** is a broad-spectrum protease and matrix metalloproteinase (MMP) inhibitor, injected peritendinously
- Calcific tendonitis
 - Low-energy radio shock-wave therapy has recently shown significant pain relief:
 - Thought to increase the resorption of calcium
 - Cimetidine has been used to decrease pain and calcium deposits.
- Trigger finger:
 - Conservative treatments such as rest, splinting (thumb spica) and NSAIDs for most
 - Some physicians suggest cortisone injections, (84–91% cure rate).
 - Surgical release of A-1 Pulley may be required.
- De Quervain tenosynovitis
 - Rest, ice, NSAIDs
 - Thumb spica splint for 3–5 days often helps
- Achilles tendonitis:
 - Rest, ice, NSAIDs
 - Orthotics or heel wedges
 - Cryotherapy has shown to be useful in controlling inflammation.
 - Achilles rupture should be splinted posteriorly in slight plantar flexion:
 - Refer to orthopedics, as patients often need surgery

MEDICATION

- Ibuprofen: 400–800 mg PO q6–8h (max. 2,400 mg per day); peds: 5–10 mg/kg/dose PO q4–6h (max. 50 mg/kg/d)
- Acetaminophen: 10–15 mg/kg/dose every 4–6 hr as needed; do not exceed

acetaminophen 4 g/24 h (peds: Do not exceed 5 doses of 10–15 mg/kg acetaminophen in 24 hr)



FOLLOW-UP

DISPOSITION

Admission Criteria

Admit patients if require surgery or other more serious illness/injury

Discharge Criteria

Most patients may be managed as outpatients with appropriate referral.

Issues for Referral

- All complete tendon ruptures merit referral for surgical consultation.
- Partial tendon tears and chronic tendinopathy that fail to improve with 3–6 mo of conservative treatment may benefit from consultation with a specialized runners' clinic, physical medicine and rehabilitation specialist, physical therapist, or orthopedic surgeon

FOLLOW-UP RECOMMENDATIONS

Prevention of reinjury is central to follow-up care.

PEARLS AND PITFALLS

- Fluoroquinolones
- Tendinopathy and tendon rupture have been reported uncommonly in adults given fluoroquinolones but have been reported with most fluoroquinolones.

ADDITIONAL READING

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- Woodley BL, Newsham-West RJ, Baxter GD. Chronic tendinopathy: Effectiveness of eccentric exercise. *Br J Sports Med.* 2007;41:188–198.



See Also (Topic, Algorithm, Electronic Media Element)

Tenosynovitis



CODES

ICD9

- 726.0 Adhesive capsulitis of shoulder
- 726.10 Disorders of bursae and tendons in shoulder region, unspecified
- 726.90 Enthesopathy of unspecified site

ICD10

- M65.819 Other synovitis and tenosynovitis, unspecified shoulder
- M75.30 Calcific tendinitis of unspecified shoulder
- M77.9 Enthesopathy, unspecified

TENOSYNOVITIS

James Killeen



BASICS

DESCRIPTION

- Definition
 - Inflammation of the tendon and tendon sheath
- Caused by inflammation, overuse, or infection
- Synovial sheaths cover tendons as they pass through osseofibrous tunnels:
 - Visceral and parietal layers of the synovium lubricate and nourish the tendons.
 - Infection can be introduced into tendon sheath.
- Skin wound
- Hematogenous spread
- Flexor tenosynovitis (FTS) of hand:
 - Typically infectious etiology
 - Penetrating injury especially at flexion creases of the finger is the most common mechanism.
 - High-pressure “injection” injury to fingers
- Air tools
- Paint sprayers
- Hydraulic equipment
- May appear minor on the surface but are associated with high incidence of FTS

ETIOLOGY

- De Quervain tenosynovitis:
 - Caused by overuse
 - Inflammatory in nature
 - 2 thumb tendons: The abductor pollicis longus (APL) and extensor pollicis brevis (EPB).
 - On their way to the thumb, the APL and EPB traverse side-by-side through a thick fibrous sheath that forms a tunnel at the radial styloid process
- GC tenosynovitis:
 - *Neisseria gonorrhoea*
- Nongonococcal infectious tenosynovitis:
 - *Staphylococcus aureus* and Streptococci are most common in penetrating injuries.
 - *Pasteurella multocida* is common with cat bites.
 - *Eikenella corrodens* is common in human bites.
 - *Pseudomonas* is seen in patients with diabetes or marine-associated injuries.

- *Mycobacterium* species may occur in immunocompromised patients.
- Fungal tenosynovitis may occur from puncture wounds due to thorns or woody plants

DIAGNOSIS

SIGNS AND SYMPTOMS

- Cardinal signs of Kanavel for FTS include:
 - Tenderness and symmetric swelling along flexor tendon sheath (sausage digit)
- Flexed position of the digit
- Pain with passive extension of the finger
- Pain with palpation along the tendon sheath

Hand

- De Quervain tenosynovitis:
 - Repetitive pinching motion of thumb and fingers
- Assembly-line workers
- Carpenters
- Landscaping or weeding
 - Pain in the radial aspect of the wrist becomes worse with activity and better with rest.
 - Pain occurs on palpation along the radial aspect of the wrist.
 - Pain occurs with passive range of motion of the thumb.
 - Finkelstein test:
 - Pain occurs with ulnar deviation of the wrist with the thumb cupped in a closed fist.
- GC tenosynovitis:
 - Most commonly affects teenagers, young adults
 - Seen in the ankle, hand, or wrist
 - More commonly seen in women
 - Vaginal or penile discharge usually absent
 - Fever, chills, polyarthralgia are common.
 - Erythema, tenderness to palpation, and painful range of motion of the involved tendon
 - Dermatitis may be present.
 - Hemorrhagic macules or papules on the distal extremities or trunk

Forearm

Traumatic tenosynovitis is seen after a direct blow to the lower portion of the forearm.

Ankle

- Stenosing tenosynovitis:
 - Commonly seen at the inferior retinaculum of the peroneus tendon
 - Patients are usually >40 yr old and have some predisposing trauma.
 - Motion increases the pain.
- Rheumatoid tenosynovitis:
 - Medially, the posterior tibial and flexor hallucis longus tendons are commonly involved.
 - Laterally, the peronei are involved.
 - Anteriorly, the anterior tibial tendon is involved.
 - Motion increases the pain.
 - Spontaneous rupture may occur.

History

- Assess for infectious etiology:
 - History of sexually transmitted disease exposure, penile or vaginal discharge
- Obtain history of mechanism:
 - High-pressure injections
 - Puncture wounds, bites
 - Environmental exposures
- Assess tetanus status and comorbid factors (e.g., diabetes and immunocompromised).

Physical-Exam

- Assess Kanavel signs.
- Document neurovascular status.
- Tubular swelling of the tendon sheath if acute tenosynovitis is present.
- Identify signs and symptoms of systemic illness as well as other potential sites of infection.

ESSENTIAL WORKUP

Thorough history and physical exam will often lead to appropriate diagnosis.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC, ESR:
 - May be of assistance in infectious etiology
- GC cultures (urethra, cervix, rectum, or pharynx) may be useful.
- Liver function tests may be elevated with disseminated *N. gonorrhoea* infection.

Imaging

- Radiographs are low yield, unless a retained radiopaque soft tissue foreign body is suspected.

- MRI has proven accurate in assisting the diagnosis of tenosynovitis:
 - Generally unnecessary in ED

Diagnosis Procedure/Surgery

NA

DIFFERENTIAL DIAGNOSIS

- Ankle, soft tissue injuries
- Bursitis
- Carpal tunnel syndrome
- Cellulitis
- Compartment syndrome
- Endocarditis
- Felon
- *Gonorrhea*
- Gout and pseudogout
- Hand infections
- High-pressure hand injuries
- Soft tissue hand injuries
- Soft tissue knee injuries
- Reiter syndrome
- Rheumatic fever
- Rheumatoid arthritis



TREATMENT

PRE HOSPITAL

- Delay to definitive treatment leads to significant increased morbidity and loss of function.
- Elevation and immobilization of the affected extremity should be performed.

INITIAL STABILIZATION/THERAPY

- Manage airway and resuscitate as indicated:
 - Septic shock
- Elevation, immobilization of affected extremity
- IV access
- Tetanus status
- Procedure
 - Diagnostic arthrocentesis is indicated if joint effusion is present with tenosynovitis:
 - Most patients with disseminated GC infection have coexisting septic arthritis.

- Cultures are negative in 50% of patients.
- 25% GC arthritis is polyarticular.
- Joint fluid glucose is normal.
- WBCs usually are <50,000 and a Gram stain is positive in 25% of the patients.

ED TREATMENT/PROCEDURES

Hand

- High-pressure injection injuries to hand:
 - Surgical emergency
 - Immediate hand surgery consultation
 - Pain management
- Infectious FTS of hand:
 - Immediate hand surgery consultation
 - Broad-spectrum antibiotic coverage
- De Quervain tenosynovitis:
 - Rest, NSAID agents, and thumb spica splint
 - Consider lidocaine/corticosteroid injection if condition is unresponsive.
 - Phonophoresis (application of hydrocortisone gel to the radial styloid area daily) helps relieve pain in minor cases.
- GC tenosynovitis:
 - Admit for IV antibiotic therapy.
 - Penicillin or 1st-generation cephalosporins for initial therapy
 - 2nd-generation cephalosporins as an alternative
 - Surgical drainage may be indicated if antibiotics do not improve the condition.
 - Pain management
- Nongonococcal infectious tenosynovitis:
 - If diagnosis is equivocal, the patient should receive IV antibiotic therapy and consultation with a hand surgeon.
 - Cover for *Staphylococcus*, *Streptococcus*, as well as anaerobic bacterial infection.
 - Consider coverage for *Pseudomonas* for the diabetic or immunocompromised patient.
 - Aminoglycosides may be added for double coverage.
 - Pain management

Forearm

- Traumatic tenosynovitis:
 - Rest, ice, elevation, immobilization
 - NSAIDs

Ankle

- Stenosing tenosynovitis:
 - Rest, ice, elevation, immobilization
 - NSAIDs
- Rheumatoid tenosynovitis:
 - Rest, ice, elevation, immobilization
 - NSAIDs

MEDICATION

- Cefazolin: 1–2 g IV q8h (peds: 50–100 mg/kg/d IV div. q8h)
- Cefotetan: 1–2 g IV q12h (peds: 50–100 mg/kg/d IV div. q12h)
- Cefoxitin: 1–2 g IV q8h (peds: 80–160 mg/kg/d IV div. q6–8h)
- Ceftriaxone: 1–2 g IV q12h (peds: 50–100 mg/kg/d IV div. q12h)
- Clindamycin: 600–900 mg IV q8h (peds: 20–40 mg/kg/d div. q8h)
- Penicillin G: 12–24 mIU IV div. q4–6h (peds: 100,000–400,000 IU/kg/d IV div. q4–6h)
- Timentin: 3.1 g IV q6h (peds: 200–300 mg/kg/d IV div. q4–6h)
- Tobramycin: 1 mg/kg IV q8h or 5 mg/kg IV q24h (peds: 2–2.5 mg/kg IV q8h)
- Zosyn: 3.375 g IV q6h (peds: 200–400 mg/kg/d IV div. q6–8h)



FOLLOW-UP

DISPOSITION

- Patients with FTS require immediate consultation with a hand specialist and admission.
- Patients presenting 24–48 hr may have more conservative therapy to include immobilization, elevation IV antibiotics, and close observation.
- Surgical débridement indicated if patient is not improved within the 1st 24 hr, or physical findings are not resolved within 48 hr.
- Patients presenting longer than 48 hr require surgical débridement in the operating room.
- The hand surgeon may attempt continuous catheter irrigation of the tendon sheath.

Admission Criteria

Infectious or high-pressure etiologies for tenosynovitis should be admitted.

Discharge Criteria

Inflammatory etiologies can be managed as outpatients with appropriate referral.

Issues for Referral

Stenosing and rheumatoid tenosynovitis

FOLLOW-UP RECOMMENDATIONS

Patients whose etiology is inflammatory should be referred for follow-up within 1–2 wk

PEARLS AND PITFALLS

- High-pressure injection injuries may have subtle clinical findings, small puncture wounds
- Early hand surgeon consultation for suspected infectious etiology or high-pressure injection injuries is paramount
- De Quervain tenosynovitis may require thumb spica immobilization in order to improve

ADDITIONAL READING

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- Torralba KD, Quismorio FP Jr. Soft tissue infections. *Rheum Dis Clin North Am*. 2009;35(1):45–62.



CODES

ICD9

- 727.00 Synovitis and tenosynovitis, unspecified
- 727.04 Radial styloid tenosynovitis
- 727.05 Other tenosynovitis of hand and wrist

ICD10

- M65.4 Radial styloid tenosynovitis [de Quervain]
- M65.849 Other synovitis and tenosynovitis, unspecified hand
- M65.9 Synovitis and tenosynovitis, unspecified

TESTICULAR TORSION

Edward Newton

BASICS

DESCRIPTION

- Rotation of the testicle around the spermatic cord and vascular pedicle
- Rotation often occurs medially (two-thirds of cases):
 - _ Ranges from incomplete (90–180°) to complete (360–1,080°) torsion
 - _ Depending on the degree of torsion:
 - Vascular occlusion occurs
 - Infarction of the testicle after more than 6 hr of warm ischemia
- Testicular salvage:
 - _ 73–100% with <6 hr of ischemia
 - _ 50–70% at 6–12 hr
 - _ <20% after 12 hr
 - _ It is still worthwhile to attempt to salvage the testicle up to 24 hr after the onset.
- Testicular infarction leads to atrophy and may ultimately decrease fertility.

EPIDEMIOLOGY

Bimodal distribution of torsion:

- Peak incidences in infancy and adolescence
- 85% of cases occur between ages 12 and 18 yr, with a mean of 13 yr.
- Torsion is rare after age 30 but still possible.

ETIOLOGY

- Congenital abnormality of the genitalia:
 - _ High insertion of the tunica vaginalis on the spermatic cord
 - _ Redundant mesorchium
 - _ Permits increased mobility and twisting of the testicle on its vascular pedicle
- The anatomic abnormality is bilateral in 12%, so both testicles are susceptible to torsion.

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Sudden onset of unilateral testicular pain
- Scrotal swelling and erythema

- Less commonly, torsion may present with pain in the inguinal or lower abdominal area.
- Up to 40% of patients may describe previous similar episodes that remitted spontaneously:
 - Represents spontaneous torsion and detorsion
- Nausea and vomiting occur in 50% of cases.
- Low-grade fever occurs in 25%.
- There is often a history of minor trauma to the testicle preceding the onset of pain.
- Symptoms of urinary infection (dysuria, frequency, and urgency) are absent.

Physical-Exam

- In distinguishing torsion from epididymitis, localized tenderness is helpful early; however, once significant scrotal swelling occurs, the anatomy becomes indistinct.
- Torsion of the appendix testis is less painful and does not threaten the viability of the testicle
- Characterized by the “blue dot” sign
- The affected torsed testicle may lie transversely as opposed to the normal vertical lie.
- Cremasteric reflex is frequently absent on the affected side with testicular torsion.
- Sensitivity 96%; specificity 66%
- Prehn sign:
 - Relief of pain on elevation of the testicle in epididymitis
 - Worsening or no change in the pain with torsion
 - Considered unreliable

ESSENTIAL WORKUP

- The presentation of an “acute scrotum” in a child or adolescent requires rapid assessment and immediate consultation with a urologist.
- These patients require noninvasive flow studies or surgical exploration to confirm torsion.
- 3.3 (ED)–30% (Urology service) of these patients ultimately prove to have testicular torsion.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Elevated WBC count with a left shift is present in 50% of cases.
- Urinalysis is usually normal, but up to 20% of cases of torsion include pyuria.
- There are no lab tests specific for testicular torsion.

Imaging

ALERT

- There are limitations of all flow studies:
 - _ Reflect only the current state of perfusion
 - _ Spontaneously detorsed testicle may show normal or even increased flow.
 - _ Still at high risk for recurrent torsion
- Traditional criterion standard has been technetium-99m radionuclide scans:
 - _ Decreased flow in the torsed testicle compared with the unaffected side
 - _ Frequent time delays in obtaining scans
- Doppler ultrasound:
 - _ Assess testicular blood flow and visualize the torsed spermatic cord directly.
 - _ Has replaced nuclear scanning:
 - Less invasive
 - More readily available test
 - Comparable results
 - _ Overall sensitivity and specificity of 98% and 100%, respectively for torsion but lower in distinguishing between testicular torsion and torsion of the appendix testis.
 - _ Epididymitis will reveal increased flow due to inflammation.
 - _ Torsion will reveal decreased or no blood flow.
 - _ Color-flow Doppler is most commonly available.
 - _ Use of Doppler contrast material may enhance the accuracy.
 - _ High definition ultrasound (HDUS) is emerging as an accurate means of directly imaging the torsed spermatic cord

Pediatric Considerations

- All imaging techniques have technical limitations in infants:
 - _ Testicular vessels are very small.
 - _ Amount of blood flow to the testicle under normal conditions is minimal.
- Scrotal exploration may be required.

Diagnostic Procedures/Surgery

- Scrotal exploration can be done rapidly under local anesthesia to diagnose and treat torsion.
- The “bell-clapper” deformity of both testicles should be corrected by orchiopexy.

DIFFERENTIAL DIAGNOSIS

- Acute hydrocele
- Epididymitis/orchitis
- Henoch–Schönlein purpura
- Incarcerated inguinal hernia
- Testicular neoplasm
- Testicular trauma or rupture of the testicle
- Torsion of the appendix testis (31–70% of acute scrotum cases)
- Other intra-abdominal conditions:

- Appendicitis
- Pancreatitis
- Renal colic



TREATMENT

PRE HOSPITAL

- There is no definitive treatment that can be rendered in the field.
- Pre-hospital personnel must recognize the urgency of acute testicular pain in young patients.
- These patients should be transported to the ED immediately.

INITIAL STABILIZATION/THERAPY

IV fluid, analgesics as appropriate

ED TREATMENT/PROCEDURES

- Rapid triage and assessment
- Exam of testicle to exclude primary neoplasm
- Establish the diagnosis and mobilize appropriate urologic care.
- Applying an ice pack to the scrotum relieves pain:
 - May prolong the viability of the ischemic testicle
- If definitive care is likely to be delayed beyond 4–5 hr from the onset of torsion, manual detorsion may be attempted (26.5–80% successful).
 - Externally rotate the affected testicle opposite the usual medial direction of torsion.
 - Continue until pain is relieved, normal anatomy is restored, or Doppler US shows return of flow.
 - All patients who undergo manual detorsion must be surgically explored.

MEDICATION

Analgesia



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients with confirmed torsion must be admitted for scrotal exploration and bilateral orchiopexy.
- Flow studies that are inconclusive and technical failures mandate further investigation by surgical exploration of the scrotum.
- Admission for urgent surgical exploration of an acute scrotum is mandatory if

there is any potential delay in obtaining a flow study:

- Patients in whom apparent spontaneous detorsion has occurred should undergo elective exploration for bilateral orchiopexy.

Discharge Criteria

- Patients with negative scrotal exploration and those with normal flow studies can be discharged with appropriate urologic follow-up.
- Parameters for return to ED must be discussed because of the possibility of recurrent torsion.
- Patients with an obvious diagnosis other than testicular torsion can be referred for care.

PEARLS AND PITFALLS

- Testicular torsion can mimic acute appendicitis in children.
- Remember that “time is testicle”; emergent workup and consultation are required.
- Maintain a high index of suspicion for testicular torsion in all age groups even though peak incidence is in adolescents and neonates.
- If testicular torsion is diagnosed early, a near 100% salvage rate for the testicle is possible. Orchiopexy is not a guarantee against future torsion, although it does reduce the odds.

ADDITIONAL READING

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- Schmitz D, Safranek S. Clinical inquiries. How useful is a physical exam in diagnosing testicular torsion? *J Fam Pract*. 2009;58:433–434.



See Also (Topic, Algorithm, Electronic Media Element)

- Epididymitis/Orchitis
- Hydrocele



CODES

ICD9

- 608.20 Torsion of testis, unspecified
- 608.21 Extravaginal torsion of spermatic cord
- 608.22 Intravaginal torsion of spermatic cord

ICD10

- N44.00 Torsion of testis, unspecified
- N44.01 Extravaginal torsion of spermatic cord
- N44.02 Intravaginal torsion of spermatic cord

TETANUS

Daniel T. Wu

BASICS

DESCRIPTION

- Rare disease in US but still prevalent in 3rd-world countries
- About 30 cases per year in US
- One-half of the cases involve people >50 yr of age
- Majority of cases in US occur in the unvaccinated, >10 yr since last booster or IVDUs
- 500,000–1,000,000 cases worldwide
- High mortality rates even with treatment
- Incubation period:
 - Inoculation to the appearance of the 1st symptoms:
 - 48 hr to 3 wk or more
 - Period of onset:
 - <7 days—poor prognosis
 - Very poor prognosis if <48 hr from 1st symptom to initial reflex spasm
- Neonatal tetanus:
 - Due to infected umbilical stump
 - Symptom onset in 2nd week of life when maternal antibodies decrease
 - Rare in US but common in 3rd-world countries
 - Worldwide, accounts for over one-half of all tetanus infections

ETIOLOGY

- Clostridium tetani:
 - Slender, motile, heat-sensitive, anaerobic gram-positive rod with a terminal spherical spore
 - Spore characteristics
 - Resistant to oxygen, moisture, temperature extremes
 - Can survive indefinitely until it germinates
 - Ubiquitous in soil and feces
- When inoculated into a wound or devitalized tissue or injected IV as a contaminant of street drugs, the spores germinate under anaerobic conditions and produce 2 toxins.
- Toxins:
 - Tetanolysin:
 - Damages tissue
 - Does not cause clinical manifestations of tetanus infection

- Tetanospasmin:
 - Powerful neurotoxin
 - Disrupts the release of neurotransmitters such as γ -aminobutyric acid (GABA)
 - Responsible for the clinical manifestations
- Muscle spasms
- Autonomic instability
- Uncontrolled motor activity

DIAGNOSIS

SIGNS AND SYMPTOMS

Generalized

- Most common type accounting for about 80% of all cases
- Initial presentation:
 - Muscle stiffness and pain
 - Trismus (initial)
 - Risus sardonicus (characteristic facial appearance)
- Systemic symptoms:
 - Irritability
 - Restlessness
 - Diaphoresis
- Later manifestations:
 - Muscle group rigidity
 - Sudden burst of tonic contractions of muscle groups causing:
 - Opisthotonos
 - Flexion and adduction of the arms
 - Clenching of fists
 - Extension of the lower extremities
 - Diaphragmatic spasm or paralysis:
 - May compromise respiration
- Hypersympathetic state (most common cause of death):
 - Begins in the 2nd week
 - Dysrhythmias
 - BP changes
 - Diaphoresis
 - Hyperthermia

Local

- Less common form of disease, accounting for about 17% of all cases
- Typical localized spasms around area of initial infection may:

- Be mild
- Persist for months before resolving
- Evolve to generalized form (13%)

Cephalic

- Rare variant of disease
- Follows head injury or otitis media
- Spasm of lower cranial and facial muscles:
 - Cranial nerve (CN) palsies, CN VII most common
- May progress to generalized tetanus

Neonatal

- Generalized form of tetanus occurring during the 1st weeks of life
- Often caused by infection of umbilical stump
- Clinical manifestations:
 - Irritability
 - Poor suck
 - Facial grimacing
 - Muscle spasms with touch
- Very high mortality rate (50–100%)
- Incubation period 1–2 wk

History

- Investigate source of infection.
- Acute skin wound not necessary to contract infection
- > 25% of infections occurred in the absence of known acute trauma.
- Infections can occur from abscesses, ulcers, and gangrene.
- Elicit tetanus immunization status.

ESSENTIAL WORKUP

- Perform complete physical exam focusing on cardiovascular and respiratory status, neurologic and CN exam.
- Diagnosis of tetanus is clinical:
 - Suspect in all cases of trismus
 - No wound recalled in one-fifth of cases
 - Full tetanus immunization almost eliminates diagnosis.

DIAGNOSIS TESTS & INTERPRETATION

Often of limited or no benefit for diagnosis but useful for ruling out other etiologies or assessing complications of disease

Lab

- CBC

- Electrolytes, BUN, creatinine, glucose, calcium:
 - For hypocalcemia
- Strychnine level
- ABG, pulse oximetry:
 - For oxygenation status
- Wound culture for *C. tetani*:
 - Positive only about 30% of time
- *C. tetani* titers:
 - Will be useful only after the fact
- CSF analysis:
 - Normal in tetanus
 - Exclude meningitis/encephalitis

Imaging

CT brain for altered mental status:

- Normal in tetanus

DIFFERENTIAL DIAGNOSIS

- Strychnine poisoning
- Jaw muscles usually spared or not involved early in strychnine poisonings
- Dystonic reaction to dopamine blockade
- Infection:
 - Meningitis
 - Rabies
 - Encephalitis
 - Peritonitis
 - Alveolar abscess
- Black widow spider envenomation
- Botulism
- Serotonin syndrome
- Hypocalcemic tetany
- Bell palsy (cephalic form, before trismus)



TREATMENT

PRE HOSPITAL

- Evaluate airway carefully:
 - Endotracheal intubation complicated by trismus, vocal cord paralysis, and facial/neck rigidity
- Avoid excessive stimulation because it may provoke tetany of musculature.

INITIAL STABILIZATION/THERAPY

- ABCs:
 - Prophylactic intubation
 - Require neuromuscular blockade due to trismus
 - Establish IV 0.9% NS
 - Monitor BP and cardiac rhythm (autonomic instability).
- Administer benztropine or diphenhydramine to exclude dystonic reaction.

ED TREATMENT/PROCEDURES

- Focuses on 3 goals:
 - Stabilizing the patient and supportive care
 - Neutralizing the toxin
 - Removing any remaining organism
- Stabilization and supportive care:
 - Secure airway:
 - Prophylactic intubation may be necessary.
 - Paralytic agent may be needed in the setting of trismus:
 - Succinylcholine should be used with caution due to the risk of hyperkalemia from upregulation of acetylcholine receptors.
 - Treat muscle spasms with benzodiazepines; if large doses fail, can administer dantrolene.
- Autonomic instability therapy:
 - Occurs days to weeks after the onset of symptoms
 - Tachydysrhythmia and hypertension:
 - No treatment universally effective
 - α - and β -blockers can be tried but may cause worsening of symptoms (labetalol has been used for its α - and β -blocking effects).
 - Clonidine, magnesium, morphine, fentanyl, and epidural anesthesia may be tried.
 - Hypotension:
 - Rule out septicemia and hypovolemia.
 - Initiate dopamine or dobutamine when low cardiac output.
 - Neutralization of the toxin
- Human tetanus immune globulin (TIG):
 - 3,000–6,000 U IM for both adults and children
 - Administer *before* débridement of wound.
 - Neutralizes unbound toxins
 - No effect on toxin already bound in CNS
- Removal of remaining organism:
 - Limits the severity of the infection
 - Débridement removes any necrotic tissue.
 - Antibiotics are effective in eliminating *C. tetani* when used in conjunction with débridement
 - Metronidazole is the antibiotic of choice.

- Penicillin is a viable alternative.
- Prevention:
 - Primary vaccination series should be completed by age 18 mo; children receive the booster at ages 4, 11, and then every 10 yr after.
 - Diphtheria, pertussis, and tetanus vaccine for children <7 yr
 - Tetanus diphtheria (Td) can be used for children >7 yr and adults.
 - 1 dose of Tdap should be administered to everyone >11 yr of age if not received previously to address increase in pertussis.
 - Clinical tetanus does not confer immunity.
 - For clean, minor wounds:
 - Td should be given if unknown prior vaccination history or >10 yr since last booster.
 - For tetanus-prone wounds:
 - Td should be given if unknown vaccination history or >5 yr since last booster.
 - TIG should be given if unknown vaccination or patient has never received the primary series.

MEDICATION

- Benztropine: 1–2 mg IV
- Chlorpromazine: 10–50 mg IM
- Diazepam (benzodiazepine): 5–10 mg (peds: 0.2–0.4 mg/kg) IV
- Diphenhydramine: 50 mg IV
- Dobutamine: 2.5–15 µg/kg/min IV
- Dopamine: 2–20 µg/kg/min IV
- Doxycycline: 100 mg IV q12h
- Erythromycin: 500 mg IV q6h
- Labetalol: 20 mg (peds: 0.3–1 mg/kg/dose) IV q10min up to 300 mg PRN—start infusion 2 mg/min (peds: 0.4–1 mg/kg/h max. 3 mg/kg/h as needed)
- Metronidazole: 1 g (peds: 15 mg/kg) load, followed by 500 mg (7.5 mg/kg) IV q6h
- Penicillin G: 1.2 mU on 2 separate entries (peds: 100,000 IU/kg/24 h) IV q6h for 10 days
- Propranolol: 0.5–1 mg (peds: 0.01–0.1 mg/kg) IV
- TIG:
 - 250 IU IM
 - Administer in separate site from Td toxoid
 - For unimmunized or incompletely immunized in presence of tetanus prone wound
- Td 0.5 mL IM



FOLLOW-UP

DISPOSITION

Admission Criteria

All patients should be admitted to an ICU.

Discharge Criteria

None for suspected generalized tetanus

PEARLS AND PITFALLS

Aggressive management is indicated for tetanus-prone wounds.

ADDITIONAL READING

- American Academy of Pediatrics. *Report of the Committee on Infectious Diseases*. 29th ed. Elk Grove, IL: American Academy of Pediatrics; 2012.
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- McQuillan GM, Kruszon-Moran D, Deforest A, et al. Serologic immunity to diphtheria and tetanus in the United States. *Ann Intern Med*. 2002;136:660–666.



See Also (Topic, Algorithm, Electronic Media Element)

Immunizations



CODES

ICD9

- 037 Tetanus
- 771.3 Tetanus neonatorum

ICD10

- A33 Tetanus neonatorum
- A35 Other tetanus

THEOPHYLLINE POISONING

Navneet Cheema



BASICS

DESCRIPTION

- Theophylline causes:
 - Release of endogenous catecholamines resulting in stimulation of β_1 - and β_2 -receptors
 - Adenosine antagonism
 - Inhibition of phosphodiesterase (at supratherapeutic levels)
- Available in immediate- and sustained-release formulations
- Peak absorption is 60–90 min with immediate-release and 6–10 hr with sustained-release formulations
- *Acute overdose*:
 - Ingestion within an 8-hr interval in a patient with no prior theophylline use
- *Acute-on-chronic overdose*:
 - Single excessive dose in a patient previously receiving usual therapeutic doses for ≥ 24 hr
- *Chronic intoxication*:
 - Accumulation of theophylline > 20 $\mu\text{g}/\text{mL}$ associated with prior therapeutic use for ≥ 24 hr secondary to:
 - Drug–drug, drug–diet, or drug–disease interactions
 - Use of serial excessive doses

ETIOLOGY

- Acute ingestions require larger concentrations to achieve specific toxic effects compared with acute-on-chronic or chronic overdoses.
- Drug–drug interactions:
 - Inhibiting theophylline metabolism (leads to toxicity when started):
 - H_2 -receptor antagonists
 - Macrolide antibiotics
 - Fluoroquinolones
 - Allopurinol
 - Influenza vaccine
 - Interferons
 - Enhances theophylline metabolism (leads to toxicity when discontinued):
 - Carbamazepine
 - Barbiturates
 - Smoking
 - Rifampin

- Chronic theophylline accumulation:
 - Uncontrolled CHF
 - Liver disease (cirrhosis or severe hepatitis)
 - Acute viral infections



DIAGNOSIS

SIGNS AND SYMPTOMS

- Cardiovascular:
 - Sinus, atrial, and ventricular tachycardias:
 - Multifocal atrial tachycardia
 - Atrial fibrillation
 - Premature ventricular contractions
 - Ventricular tachycardia
 - Due to β_1 -receptor stimulation and adenosine antagonism
 - Hypotension:
 - Associated with theophylline $> 100 \mu\text{g/mL}$ (acute ingestion)
 - Due to vasodilatation induced by β_2 -receptor stimulation
 - May be refractory to fluids, positioning, and conventional vasopressors
- CNS:
 - Tremor
 - Mental status changes
 - Seizures:
 - 14% of chronic intoxications
 - 5% of acute intoxications
- GI:
 - Nausea, vomiting:
 - Protracted and may be refractory to antiemetics at usual doses
 - 75% of acute intoxications
 - 30% of chronic intoxications
 - Abdominal pain
 - Pharmacobezoars:
 - From sustained-release preparations in acute ingestions
 - Delays peak concentrations
- Metabolic:
 - Hypokalemia:
 - Typically decreases approximately to 3 mEq/L
 - Due to β -receptor stimulation
 - Hyperglycemia
 - Leukocytosis
 - Hypophosphatemia and hypomagnesemia

- Metabolic acidosis with increased serum lactate levels

ESSENTIAL WORKUP

- Serum theophylline concentration:
 - Finding of ≥ 20 $\mu\text{g}/\text{mL}$ confirms diagnosis.
- ECG and cardiac monitoring
- Detailed history to differentiate acute from acute-on-chronic from chronic intoxication

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Serum theophylline level:
 - Repeat every 2 hr until decreasing to confirm immediate absorption is complete and peak value has occurred.
 - Serious morbidity in acute overdose if ≥ 100 $\mu\text{g}/\text{mL}$
- CBC
- Electrolytes

Imaging

- KUB (kidneys, ureters, bladder):
 - Undissolved sustained-release tablets or pharmacobezoars may appear as radiopacities.
 - Bead-filled capsules may appear as radiolucencies.
- US of stomach may detect intact sustained-release dosage forms.

DIFFERENTIAL DIAGNOSIS

- Caffeine/ β -agonist bronchodilator overdose
- Amphetamines
- Sympathomimetics
- Anticholinergic agents
- Drug withdrawal syndromes
- Pheochromocytoma
- Thyrotoxicosis



TREATMENT

PRE HOSPITAL

Bring pill bottles/pill samples in suspected overdose.

INITIAL STABILIZATION/THERAPY

- ABCs:
 - Cardiac monitor

- Isotonic crystalloids as needed for hypotension
- Naloxone, thiamine, and dextrose (D₅₀W) as indicated for altered mental status
- Cardiovascular:
 - Initiate β -blockers or calcium channel blockers for rate control with supraventricular tachyarrhythmias (SVT).
 - Adenosine is antagonized by theophylline and may not be effective to treat SVT.
 - Administer isotonic crystalloid IV fluid resuscitation for hypotension:
 - With treatment failure, consider β -blocker to reverse theophylline-induced β_2 -receptor-stimulated vasodilation.
 - If vasopressors are needed, choose vasopressor that is not a β -agonist, such as phenylephrine.
 - Treat ventricular dysrhythmias conventionally.
- Seizures:
 - Administer benzodiazepines.
 - Phenytoin is contraindicated; it is usually ineffective and may paradoxically worsen seizures in theophylline intoxications.

ED TREATMENT/PROCEDURES

Decontamination

- Administer activated charcoal
- Multidose activated charcoal:
 - Especially with sustained-release products
 - Binds theophylline, which back-diffuses in to the small intestine
 - For mild to moderate toxicity
 - 25 g q2h until theophylline level ≤ 20 $\mu\text{g}/\text{mL}$
- Initiate whole-bowel irrigation with sustained-release products:
 - Administer 1–2 L/hr of polyethylene glycol until a clear, colorless rectal effluent or theophylline level ≤ 20 $\mu\text{g}/\text{mL}$
- Treat protracted vomiting with metoclopramide or 5-HT₃-receptor antagonists.
- Avoid syrup of ipecac.

Electrolyte Disturbances

- Treat hypokalemia in acute ingestions cautiously:
 - Relative hypokalemia owing to β -receptor-mediated intracellular shift of extracellular potassium.
 - Aggressive correction leads to potentially serious hyperkalemia as theophylline concentrations decrease.
- Most electrolyte imbalances respond to β -blocker therapy:
 - Generally not indicated; however, because of absence of associated morbidity and potential for β -blocker-induced bronchospasm in pulmonary

patients

Extracorporeal Elimination

Initiate hemodialysis or hemoperfusion if theophylline level:

- ≥ 90 $\mu\text{g/mL}$ and symptomatic in acute ingestions
- ≥ 40 $\mu\text{g/mL}$ and:
 - Seizures or
 - HTN unresponsive to IV fluid or
 - Ventricular dysrhythmias

MEDICATION

- Activated charcoal: 1 g/kg PO, if dose ingested is known, 10 g/1 g theophylline ingested, max. dose 100 g
 - Multidose-activated charcoal 25 g q2h until theophylline level ≤ 20 $\mu\text{g/mL}$
- Diazepam: 0.1 mg/kg IV q5–10min until seizures controlled, up to 30 mg
- Diltiazem: 0.25 mg/kg IV bolus; may repeat after 15 min, then 5–15 mg/h infusion for control of heart rate in patients with contraindication to β -blockade
- Esmolol: 500 $\mu\text{g/kg}$ IV bolus, followed by 50 $\mu\text{g/kg/min}$ infusion; increase by 50 $\mu\text{g/kg/min}$ increments to max. of 200 $\mu\text{g/kg/min}$
- Metoclopramide: 10 mg IV bolus; may repeat to max. of 1 mg/kg
- Ondansetron: 0.15 mg/kg IV bolus up to max. of 16 mg total
- Polyethylene glycol (high molecular weight): 1–2 L/h via nasogastric tube



FOLLOW-UP

DISPOSITION

Admission Criteria

ICU:

- Acute overdoses with serum theophylline concentrations ≥ 100 $\mu\text{g/mL}$
- Acute-on-chronic or chronic theophylline with either serum concentration ≥ 60 $\mu\text{g/mL}$ or patient > 60 yr old
- Seizures or hypotension refractory to fluids and vasopressors in a patient with serum theophylline concentration ≥ 40 $\mu\text{g/mL}$

Discharge Criteria

- 2 consecutive (≥ 2 hr apart) decreasing serum theophylline concentrations with most recent concentration < 30 $\mu\text{g/mL}$
- Mildly symptomatic or asymptomatic patient meeting above criterion and no evidence of suicidal intention

FOLLOW-UP RECOMMENDATIONS

- Follow up with medical toxicologist or primary care doctor
- If patient is on chronic theophylline, dosing regimen may have to be adjusted.

PEARLS AND PITFALLS

- Seizures are a major complication.
- Tachydysrhythmias are common in overdose.
- Multi-dose activated charcoal is beneficial in theophylline overdose.

A special thanks to Dr. Harry Karydes who contributed to the previous edition.

ADDITIONAL READING

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CODES

ICD9

975.7 Poisoning by antiasthmatics

ICD10

- T48.6X1A Poisoning by antiasthmatics, accidental, init
- T48.6X5A Adverse effect of antiasthmatics, initial encounter

THORACIC OUTLET SYNDROME

Adam J. Heringhaus • Daniel C. McGillicuddy

BASICS

DESCRIPTION

- The symptoms of thoracic outlet syndrome (TOS) are produced by compression of the brachial plexus, subclavian vein, or subclavian artery during their passage from the cervical area toward the axilla and proximal arm.
- Subdivided into 3 categories depending on the predominant symptoms:
 - Neurogenic thoracic outlet syndrome (NTOS):
 - Comprises 90–98% of adult patients
 - Female > male
 - True (1–3%): Those with objective findings
 - Disputed (90%): Those with no or limited objective findings
 - Venous thoracic outlet syndrome (VTOS):
 - 2–4% of patients
 - Arterial thoracic outlet syndrome (ATOS):
 - Least common, <1%
 - Male = female
- Vascular manifestations are more common in adolescents, seen in >50% of teens with TOS.
- Right extremity is more commonly affected.

ETIOLOGY

- Anatomic anomalies:
 - Bony anomalies include cervical rib, 1st thoracic rib, or clavicular abnormalities:
 - Cervical ribs occur in <1% of the population, ~70% in women, and most are asymptomatic.
 - Less commonly, fracture of the clavicle and trauma to the sternoclavicular and costoclavicular joints
 - Congenital bands or anomalous muscles
 - May play a role in neurologic and venous types but is almost always implicated in arterial type
- Neurogenic:
 - Often have a history of neck trauma, such as whiplash (hyperextension injuries) or with repetitive motion patterns
- Venous:
 - May be preceded by excessive activity, especially in adolescent athletes
 - Caused by acute thrombosis of the subclavian vein (also called Paget–

Schrötter disease) or by venous impingement

- Arterial:
 - Often develop spontaneously
 - Unrelated to trauma or work
 - May experience true claudication with overhead exercises
 - Almost always have a complete cervical rib or an anomalous 1st rib
 - Caused by subclavian artery aneurysm or subclavian/axillary artery impingement:
 - Arterial emboli that arise from either mural thrombus in the subclavian artery aneurysm or from thrombus forming distal to subclavian artery stenosis
- Descent of the shoulder girdle and sagging musculature can also predispose to TOS:
 - Aging
 - Obesity
 - Heavy breasts

DIAGNOSIS

SIGNS AND SYMPTOMS

- Neurogenic:
 - Classically, pain, paresthesia, and weakness of the hand, arm, and shoulder
 - May see wasting of the thenar eminence, also known as Gilliatt–Sumner hand
 - Analogous but not as severe as Erb–Duchenne syndrome
 - May also see Raynaud phenomenon, hand coldness, color change:
 - Not caused by ischemia, rather due to overactive sympathetic fibers that run on the circumference of the lower trunks of the brachial plexus
 - Similar symptoms can be seen in arterial TOS, so the 2 must be differentiated by evaluating for other signs and symptoms.
- Venous:
 - Swelling of the arm and cyanosis:
 - NTOS and ATOS do not exhibit arm swelling.
 - May see pain, aching of the arm
 - Hand paresthesia:
 - May be due to swelling as opposed to nerve compression
- Arterial:
 - Digital ischemia, claudication, pallor, coldness, paresthesia, and pain of the hand
 - Usually spares the shoulder and neck
 - Pallor and coldness are due to ischemia and not Raynaud

- Aneurysmal:
 - o Painless pulsating mass

History

- May be positional or exacerbated by repetitive use (i.e., working overhead)
- Usually insidious in onset and progressive
- Can occur or worsen suddenly after trauma or with acute clot

Physical-Exam

Provocative maneuvers can reveal NTOS (VTOS and ATOS often diagnosed with history and symptoms only):

- Roos, aka elevated arm stress test (EAST):
 - Arms abducted 90° from the thorax and elbows flexed at 90°
 - Shoulders braced slightly back of the frontal plane
 - Fists are open and closed for 3 min.
 - Early heaviness and fatigue of the arm
 - Gradual onset of hand numbness
 - Progressive aching through the arm and top of shoulder
 - Negative test is having only fatigue in forearms
- Adson test:
 - Arm down, patient rotates head toward extremity, looks up, and inhales.
 - Positive result is the alteration or obliteration of the radial pulse or change in the BP.
 - Not a reliable test, as many patients with NTOS have a negative test and many control patients have a positive test.
- Wright test:
 - Progressive hyperabduction and external rotation of affected arm while palpating pulse on ipsilateral side
 - Positive result if paresthesias or diminishing pulses
- None of the above is very sensitive nor specific.
- Difference of >20 mm Hg in BP is suggestive of compromise of subclavian artery in right clinical context

ESSENTIAL WORKUP

- Careful history and physical exam
- EKG to rule out cardiac ischemia

DIAGNOSIS TESTS & INTERPRETATION

Lab

Consider a coagulation workup for either venous or arterial TOS.

Imaging

- Perform as outpatient except in case of limb-threatening ischemia and/or suspicion of venous thrombus
- CXR:
 - Assess for anatomic abnormalities: 1st rib, cervical rib, clavicle deformity:
 - Without an abnormality, ATOS is very unlikely.
 - Pulmonary disease
- Cervical spine series:
 - Fracture
 - Scoliosis
- US can diagnose venous thrombosis
- Duplex scanning is the best way to screen for subclavian artery aneurysm or stenosis, which, if present, can lead to arteriography
- Arteriogram:
 - Usually used to help a surgeon plan reconstruction
 - Indications include:
 - Decreased radial pulse
 - BP is 20 mm Hg less than the opposite limb.
 - Suspected subclavian stenosis
 - Bruit or abnormal supraclavicular pulsations or pulsating mass
 - Peripheral emboli in the upper extremity
- Venography:
 - Indicated if edema, peripheral unilateral cyanosis, or distended thoracic and extremity veins
- NTOS:
 - No gold standard test: Diagnosis remains mostly clinical.
 - Electromyography and nerve conduction velocity tests are often normal.
- MRI may be required to assess for spinal cord disease, herniated cervical disk, or assessing for pancoast tumor.

DIFFERENTIAL DIAGNOSIS

- Cardiac ischemia
- Cervical spondylosis or disk disease
- Carpal tunnel syndrome or nerve entrapments
- Pancoast tumor; other neck/mediastinum malignancies
- Neuritis
- Myositis
- Raynaud disease
- Multiple sclerosis or degenerative spinal cord disease
- Shoulder inflammatory diseases: Arthritis, rotator cuff injury, bicipital tendonitis
- Atherosclerotic or thromboembolic disease



TREATMENT

ED TREATMENT/PROCEDURES

- Heparinization if signs of arterial or venous thrombosis
- Vascular surgery consult for signs of ischemia and for catheter-directed thrombolysis if needed:
 - Anticoagulation and thrombolysis followed by surgical decompression is required for thrombosis
- Initial management:
 - The majority improve with conservative treatment consisting of physical therapy and medications for symptomatic relief.
- Surgery reserved for failure of medical therapy:
 - Often required for vascular forms
 - People with NTOS often undergo more extensive evaluation and medical management prior to surgical intervention
 - 70–90% of patients experience some to complete relief postoperatively.

MEDICATION

- Cyclobenzaprine: 10 mg PO TID
- Diazepam: 5 mg PO TID
- Ibuprofen: 800 mg PO TID
- Methocarbamol: 1,000–1,500 mg PO TID
- Soothing liniments or ointments



FOLLOW-UP

DISPOSITION

Admission Criteria

- Ischemia
- Venous thrombosis
- Arterial thrombosis
- Arterial aneurysm or stenosis
- Intractable pain

Discharge Criteria

- Nonlimb-threatening neurologic findings
- Absence of arterial or venous thrombosis

FOLLOW-UP RECOMMENDATIONS

Vascular, neurologic, or orthopedic consultation is indicated according to the pathologic condition.

PEARLS AND PITFALLS

- 3 types of TOS: Neurogenic, arterial, and venous:
 - Neurogenic is most common in adults.
 - ATOS and VTOS are the more common types in children and adolescents.
- VTOS is the only type that has arm swelling and edema.
- Both NTOS and ATOS have hand coldness and pallor, but for different reasons.
- May have a history of repetitive use or trauma
- Exam or imaging may reveal a congenital abnormality such as a cervical rib.

ADDITIONAL READING

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Thank you to prior Authors Erin Horn, MD



See Also (Topic, Algorithm, Electronic Media Element)

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CODES

ICD9

353.0 Brachial plexus lesions

ICD10

G54.0 Brachial plexus disorders

THROMBOTIC THROMBOCYTOPENIC PURPURA

Hany Y. Atallah

BASICS

DESCRIPTION

- Thrombotic thrombocytopenic purpura (TTP) is a severe disorder of abnormal clotting affecting multiple organ systems.
- Classically characterized by pentad of:
 - Thrombocytopenia
 - Hemolytic anemia
 - Mild renal dysfunction
 - Neurologic signs
 - Fever
- Uncommon to see all 5 features in 1 patient; if present, severe end-organ damage or ischemia has likely taken place.
- Thrombocytopenia and hemolytic anemia are the most common features.
- Associated with acquired or congenital deficiency of plasma von Willebrand factor–cleaving protease (VWFcp)
- Patients who present with severe neurologic abnormalities with acute renal failure are best described by the comprehensive term TTP-HUS

Classic Course

- Acute onset
- Fulminant course lasting days to a few months
- Nearly always fatal without treatment:
 - >90% mortality without treatment
 - Reverses to >90% survival with modern treatment
- Clinical presentations include:
 - Idiopathic
 - Familial, chronic, or relapsing
 - Drug induced:
 - Allergic or immune mediated (quinine, ticlopidine, clopidogrel)
 - Dose-related toxicity (mitomycin C, cyclosporine)
 - Pregnancy, postpartum associated:
 - 10–25% of cases
 - Bone marrow transplantation associated
 - Infection
- More common in the 3rd–6th decades of life
- Uncommon in pediatric or geriatric populations
- Women affected about twice as frequently as men

ETIOLOGY

- Unknown primary stimulant; possibly systemic endothelial cell damage results in inactivation of coagulation pathway
- Platelet aggregation and fibrin deposition occurring in arterioles and capillaries leading to microthrombi and obstruction to blood flow
- Platelet aggregation leads to:
 - Consumption of platelets
 - Widespread microvascular hyaline thrombotic lesions
- Microvasculature obstruction with platelet aggregates leads to:
 - Red cell hemolysis
 - Accumulation of heme breakdown products
 - Anemia
- End-organ ischemia results from diffuse thrombosis in small vessels:
 - Most common in heart, brain, kidney, pancreas, and adrenal glands
- Deficiency of vWFcp causes failure of control of coagulation pathway.

RISK FACTORS

Genetics

- Some cases are genetic/familial.
- vWFcp was recently identified as new member of ADAMTS family and designated ADAMTS13.
- Mutations in ADAMTS13 gene cause autosomal recessive form of chronic relapsing TTP.

DIAGNOSIS

SIGNS AND SYMPTOMS

5 major clinical features: Classic pentad

- Thrombocytopenia:
 - Platelet count $< 20,000/\text{mm}^3$
- Microangiopathic and hemolytic anemia:
 - Hb $< 10 \text{ g/dL}$ ($< 6 \text{ g/dL}$ in 40%)
- Neurologic symptoms:
 - Presenting complaint in 60%, occur in 90%
 - Typically fluctuating
 - Headache
 - Altered mentation (confusion, stupor, coma)
 - Behavioral or personality changes
 - Focal sensory or motor deficits or aphasia
 - Seizures
 - Spontaneous intracranial hemorrhage

- Renal insufficiency:
 - Usually mild
 - Creatinine < 3 mg/dL
- Fever:
 - Occurs in acute episodes and prodromal syndromes
 - Fever is the least common feature
- Rare for all components of pentad to be present in the same individual

History

- General:
 - Weakness
 - Fatigue
 - Fever
 - Malaise
- Hemorrhage:
 - Easy bruising
 - Epistaxis
 - Menorrhagia
 - GI bleeding
 - Loss or change in vision
- GI complaints:
 - Anorexia
 - Diarrhea
 - Abdominal pain
- Neurologic:
 - Headache
 - Confusion
 - Seizure
 - Behavioral or personality changes
 - Focal sensory or motor deficits or aphasia

Physical-Exam

- Purpura
- GI hemorrhage
- Epistaxis
- Jaundice
- Shock
- Altered mental status
- Focal sensory or motor deficits
- Pulmonary infiltrates and edema
- Alteration of vision, retinal hemorrhage/detachment.
- Abnormalities of cardiac conduction

ESSENTIAL WORKUP

Clinical Diagnosis

- Because of success of treatment, base diagnosis on:
 - Identification of 2 major findings:
 - Thrombocytopenia
 - Microangiopathic hemolytic anemia
 - Exclude other major differential diagnoses.
- Comprehensive history and physical exam with directed lab testing
- Identify possible drug-associated disease and avoid re-exposure.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC/platelet count/reticulocyte count:
 - Anemia: Hemoglobin < 10 g/dL
 - Thrombocytopenia < 20,000/mm³
 - Increased reticulocyte count
- Coagulation studies:
 - Normal
- Peripheral blood smear:
 - Macroangiopathic changes
 - Schistocytes
 - Helmet cells
 - Nucleated RBCs
- Coombs test:
 - Negative direct Coombs test
- Electrolytes, BUN, creatinine, glucose:
 - Mild elevation of BUN, creatinine
 - Hyperkalemia owing to RBC lysis
- Lactate dehydrogenase (LDH):
 - Elevated 5–10 times due to hemolysis and tissue ischemia
- Bilirubin:
 - Increased unconjugated bilirubin
- Urinalysis:
 - Hematuria (microscopic to gross)
- ADAMTS13 assay may be used to distinguish chronic recurring TTP, TTP secondary to presence of ADAMTS13 inhibitor, and hemolytic-uremic syndrome (HUS):
 - ADAMTS13 deficiency does not detect all patients who may respond to plasma exchange transfusions.

Imaging

- CT head:
 - To rule out intracranial hemorrhage

Diagnostic Procedures/Surgery

- Biopsy:
 - Confirms diagnosis
 - Reveals hyaline lesions in small vessels
 - Contraindicated during fulminant presentation (hemorrhage risk)
- EEG:
 - To predict need for anticonvulsant therapy

DIFFERENTIAL DIAGNOSIS

- HUS:
 - Triad of thrombocytopenia, schistocytosis, and renal dysfunction
 - Neurologic symptoms unusual
 - Often preceded by infectious prodrome and diarrhea
- Disseminated intravascular coagulation (DIC):
 - Causes deposition of fibrin in microvasculature and not hyaline
 - Coagulation studies abnormal
- Idiopathic thrombocytopenic purpura (ITP):
 - No evidence of hemolysis
 - LDH and bilirubin normal
- Pregnancy-related thrombocytopenia:
 - Preeclampsia, eclampsia
 - Pregnancy-associated hemolysis
 - HELLP (hemolysis, elevated liver enzymes, and low platelets)
- Evans syndrome:
 - Autoimmune hemolytic anemia
 - Prominence of microspherocytes rather than schistocytes
 - Positive direct Coombs test
- Malignant hypertension
- Bacterial sepsis
- Subacute bacterial endocarditis
- Autoimmune disorders (e.g., systemic lupus erythematosus [SLE])
- Disseminated malignancy
- Heparin-associated thrombocytopenia
- Prosthetic valves or severely calcified aortic stenosis



TREATMENT

PRE HOSPITAL

- ABCs

- Evaluate for other possible causes of altered mental status (hypoglycemia, overdose)

INITIAL STABILIZATION/THERAPY

- ABCs
- 0.9% normal saline (NS) IV fluid resuscitation for shock or GI hemorrhage
- RBC transfusions:
 - _ For significant anemia or bleeding complications
- Platelet transfusions:
 - _ Reserve for life-threatening hemorrhage (e.g., CNS bleeds) or required invasive procedures
 - _ May aggravate the thrombotic, microvascular obstructive process and worsen the end-organ ischemia and shock

ED TREATMENT/PROCEDURES

- *Fresh frozen plasma* (FFP) or fresh unfrozen plasma:
 - _ Initiated as bridge to exchange transfusions on diagnosis of TTP
 - _ Success rate approaching 64%
 - _ Provides a platelet-antiaggregating factor absent or diminished in patient's own serum
 - _ Used prophylactically to prevent recurrence in chronic relapsing variant
- Plasma exchange transfusions:
 - _ Most important component of treatment
 - _ Combination of plasmapheresis and FFP infusion
 - _ Plasmapheresis removes:
 - Immune complexes responsible for endothelial damage and initiation of TTP
 - Circulating proaggregation factors promoting platelet aggregation
 - _ Perform daily until:
 - Platelet count normalizes
 - Neurologic symptoms improve
 - LDH normalizes
 - _ Improvement of renal function may lag behind other findings.
 - _ Taper frequency based on empiric judgment of response; may need to resume if relapse occurs.
 - _ Complications include:
 - Allergy or serum sickness
 - Secondary infection
 - Hypotension
- Corticosteroids:
 - _ Unproven therapeutic benefit
 - _ May limit immunologically mediated endothelial damage and decrease splenic sequestration of platelets and damaged RBCs

- Supportive benefit if adrenal glands damaged through hemorrhage or ischemia
- Antiplatelet or immunosuppressive drugs:
 - Aspirin and dipyridamole most commonly used
 - Use of sulfapyrazine, dextran, and vincristine has been reported.
 - Used with variable effectiveness
 - Can worsen bleeding complications
- Splenectomy:
 - Historically recommended
 - Of uncertain efficacy
- Dialysis:
 - For renal failure

MEDICATION

- Aspirin: 325–650 mg PO q4–6h
- Dipyridamole: 75–100 mg PO QID
- FFP:
 - Plasma infusion: 30 mL/kg/d (75–100 mL/h)
 - Plasma exchange transfusion: 3–4 L/d
- Methylprednisolone: 0.75 mg/kg q12h
- Prednisone: 1–2 mg/kg/d (high dose up to 200 mg/d)
- Rituximab: 375 mg/m² IV once per week for 4–8 doses
- Vincristine: 1.4 mg/m² once per week IV



FOLLOW-UP

DISPOSITION

Admission Criteria

- Newly diagnosed serious platelet disorder, especially with bleeding complications or altered mental status or renal dysfunction
- ICU admission for TTP with active bleeding or neurologic findings:
 - Transport to tertiary care center with appropriate specialty care facilities.

FOLLOW-UP RECOMMENDATIONS

Patients with known disease and found to be stable may follow up with a hematologist.

PEARLS AND PITFALLS

- TTP can be confused with HELLP syndrome in pregnant females.
- Because of the high mortality of untreated TTP, recognition of the disease and initiation of treatment is key.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Disseminated Intravascular Coagulation
- HELLP Syndrome
- Idiopathic Thrombocytopenia
- Renal Failure



CODES

ICD9

446.6 Thrombotic microangiopathy

ICD10

M31.1 Thrombotic microangiopathy

THUMB FRACTURE

Daniel R. Lasoff • Leslie C. Oyama



BASICS

DESCRIPTION

- Distal phalangeal fractures:
 - Blunt trauma, hyperextension of the thumb, axial loading of the thumb, and crush injuries.
 - *Tuft fracture* is a similar fracture in other digits, in which the distal phalanx is crushed and/or fragmented.
 - It may be open or closed and associated with nail bed injury.
 - Severe nail bed injury, intra-articular, displaced/angulated fractures, or tendon injuries warrant orthopedics' consultation.
 - Noncomplex tuft fractures can be splinted and treated as soft tissue injuries.
- Proximal phalangeal fractures and thumb metacarpal fractures:
 - Blunt trauma to the thumb:
 - Axial loading of the thumb with the metacarpophalangeal (MP) joint partially flexed, the hand closed or the thumb MP joint otherwise stabilized
 - Bennett fracture (type I):
 - Intra-articular fracture/dislocation at the base of the metacarpal where the ulnar aspect of the metacarpal maintains its attachment.
 - Rolando fracture (type II):
 - Comminuted Y- or T-shaped intra-articular fracture of the base of the 1st metacarpal.
 - Similar to a comminuted Bennett, these can be much more complex with multiple comminuted fractures.
 - Type III fractures
 - Extra-articular metacarpal fractures. Tend to be transverse or less commonly oblique.

ETIOLOGY

- Falls, hyperflexion, hyperextension
- Motor vehicle accidents
- Sports, especially downhill or alpine skiing
- Basketball
- Baseball
- Football
- Rugby

 **DIAGNOSIS****SIGNS AND SYMPTOMS**

- Pain, swelling, and deformity of the thumb
- Exam should include the thenar eminence for pain or deformity.
- The thumb may be rotated distal to the fracture site.
- The base of the thumb may appear radially deviated relative to the rest of the hand in the resting position.
- Occasionally, there may be damage to the thumb digital nerves.

Pediatric Considerations

- Fractures to the thumb sometimes occur in children.
- Consider nonaccidental trauma.
- Do not neglect appropriate pain management in children.

Physical-Exam

- Immobilize the thumb pending definitive evaluation.
- Neurovascular exam with 2-point discrimination.

ESSENTIAL WORKUP

Radiography as noted below

DIAGNOSIS TESTS & NTERPRETATION***Imaging***

- Plain radiography of affected areas
- Avoid testing stress of thumb MP joint, as in testing for gamekeeper thumb, until all plain radiography is complete.

DIFFERENTIAL DIAGNOSIS

- Extra-articular fracture of the base of the thumb metacarpal
- Scaphoid fracture
- Gamekeeper thumb: Ulnar collateral ligamentous injury

 **TREATMENT****PRE HOSPITAL**

- Dress open wounds.
- Immobilize hand and wrist with thumb in neutral position.
- Elevate and apply cold to reduce swelling.
- Age-appropriate social management

INITIAL STABILIZATION/THERAPY

Immobilize thumb pending definitive evaluation.

ED TREATMENT/PROCEDURES

- Thumb spica splint with the thumb in neutral position, as if holding a beverage can
- Splint instructions should be provided to patient.
- Angulated extra-articular fractures of the 1st metacarpal require reduction. Can tolerate up to 30° of angulation. Angulation > 30° requires another attempt at reduction or orthopedics' consultation.
- Distal phalangeal fractures require DIP splint in extension for 3–4 wk.

MEDICATION

Pain control with oral analgesic preparations



FOLLOW-UP

DISPOSITION

Admission Criteria

Open fracture, presence of multiple trauma, or other more serious injuries

Discharge Criteria

- Counsel the patient that there is a strong likelihood of the need for operative repair for 1st metacarpal injuries.
- Closed injuries: Referral, splinting, and explain frequent need for operative fixation

Issues for Referral

72-hr orthopedic referral

PEARLS AND PITFALLS

Due to tendon insertions, fractures at the base of thumb are often unstable and frequently require operative fixation

ADDITIONAL READING

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CODES

ICD9

- 816.00 Closed fracture of phalanx or phalanges of hand, unspecified
- 816.01 Closed fracture of middle or proximal phalanx or phalanges of hand
- 816.02 Closed fracture of distal phalanx or phalanges of hand

ICD10

- S62.509A Fracture of unsp phalanx of unsp thumb, init for clos fx
- S62.516A Nondisp fx of proximal phalanx of unsp thumb, init
- S62.523A Disp fx of distal phalanx of unsp thumb, init for clos fx

TIBIAL PLATEAU FRACTURE

Sarah V. Espinoza • Leslie C. Oyama



BASICS

DESCRIPTION

- Synonym: Tibial condylar fracture
- Fracture or depression of the proximal tibial articulating surface
- Valgus or varus force applied in combination with axial loading onto tibial plateau

Schatzker Classification of Plateau Fractures

- Type 1:
 - Split fracture of the *lateral* tibial plateau *without* depression of the plateau
- Type 2:
 - Split fracture *and* depression of *lateral* tibial plateau
 - Associated with lateral meniscus injury
- Type 3:
 - Central depression of the *lateral* plateau
 - Injuries may be unstable
- Type 4:
 - Split of the *medial* tibial plateau
 - Can cause damage to other structures:
 - Popliteal vessels
 - Peroneal nerve
 - MCL
 - Lateral meniscus
 - Lateral collateral ligament
 - Cruciate ligaments
 - Tibial spines
 - Compartment syndrome
- Type 5:
 - Bicondylar tibial plateau fracture
 - Same associated injuries as type 4
- Type 6:
 - *Bicondylar*, grossly comminuted fracture of the plateau
 - Diaphyseal–metaphyseal dissociation
 - Same associated injuries as types 4 and 5

ETIOLOGY

- Mechanism of injury:
 - Types 1 & 2 from a valgus force with axial loading, generally a low-energy

injury

- Associated with contact sports, twisting motions (e.g., skiing) or classically, pedestrians struck by a vehicle bumper
- Type 3 are low-energy injuries in osteopenic bone
- Types 4–6 are high-energy injuries usually from motor vehicle/cycle collisions and falls from height causing medial plateau fractures
 - Associated with neurovascular injuries
- Age associated
 - Type 1: Younger patients with cancellous bone of the plateau resists depression.
 - Types 2 & 3: Depression fractures seen in osteopenic older bones

Pediatric Considerations

Tibial plateau fractures are rare in children because of the dense cancellous bone of the tibial plateau

DIAGNOSIS

SIGNS AND SYMPTOMS

- Painful swollen knee
- Inability to bear weight
- Knee effusion (hemarthrosis)
- Active and passive range of motion limited
- Tender along the proximal tibia and joint line
- Possible varus or valgus deformity of the knee
- Possible joint instability due to associated ligamentous injury

History

- Hit to lateral knee
- Fall from a height with axial load
- Twisting injury

Physical-Exam

- Decision tools for the use of radiography:
 - Ottawa knee rules (highly sensitive): Knee radiographs are indicated if *any* of the following are present:
 - Age > 55 yr
 - Tenderness of the fibular head
 - Inability to flex to 90°
 - Isolated patellar tenderness
 - Inability to transfer weight for 4 steps both immediately after the injury and in the ED

- Limping is allowed.
- Pittsburgh knee rule (highly sensitive and specific): Knee radiographs are indicated in fall or blunt trauma when the following are present:
 - Age <12 or >55 yr
 - Inability to bear full weight for 4 steps in the ED
 - Limping is *not* allowed
 - Pittsburgh knee rule should be applied with caution to patients <18 yr old
- Neurovascular exam:
 - High-energy mechanism carries risk for neurovascular injury and compartment syndrome
 - Watch for unrelenting pain, muscle weakness, tense muscle swelling, hypesthesia or anesthesia, pain with passive stretch of muscles
 - Check popliteal, posterior tibial, and dorsalis pedis pulses
 - Check integrity of peroneal nerve:
 - Ankle and great toe dorsiflexion
 - Sensation in dorsal web space between great and 2nd toes

DIAGNOSIS TESTS & INTERPRETATION

Imaging

- Plain radiography:
 - Tibial plateau view:
 - Anteroposterior (AP) view angled at 10–15° of flexion to evaluate the tibial spines, fracture lines extending into the joint, and depressions
 - Sunrise view of the patella:
 - Useful in identifying fractures of the patella not visualized on AP or lateral views
 - Cross-table lateral view:
 - To evaluate the medial plateau and reveal lipohemarthrosis (fat–fluid level)
 - Oblique view:
 - To identify fractures not apparent on other films and provide more information on fracture patterns
 - Pay attention to areas of ligamentous attachment where avulsion fractures may take place:
 - Medial and lateral femoral condyles
 - Tibial spine (intercondylar eminence)
 - Fibular head
- CT used to reveal occult fracture(s) not seen on plain film & further characterize known fracture
- MRI used for identifying soft tissue injuries (ligamentous and meniscal injuries)
- Arteriography helpful in localizing the injured area but should not delay

revascularization and is indicated if:

- High-energy mechanism
- Schatzker type 4, 5, or 6 fracture
- Alteration in distal pulses
- Expanding hematoma
- Bruit
- Injury to anatomically related nerves

Diagnostic Procedures/Surgery

- Arthrocentesis to look for fat globules and bone marrow elements indicative of intra-articular fracture:
 - Indication to do procedure: Effusion present without fracture on plain radiographs
- Compartment pressure measurements are indicated if:
 - Pain not over fracture site
 - Pain on passive stretch
 - Paresthesias
 - Decreased distal pulses
 - Intracompartmental pressures > 30 mm Hg are an indication for emergent orthopedic consultation

DIFFERENTIAL DIAGNOSIS

- Knee dislocation
- Proximal fibular fracture
- Femoral condyle fracture
- Patellar fracture
- Tibial subcondylar fracture
- Tibial tuberosity fracture
- Tibial spine fracture
- Cruciate ligament tears
- Collateral ligament tears
- Meniscal tears

Pediatric Considerations

Include oblique views as part of routine radiography



TREATMENT

PRE HOSPITAL

Cautions:

- In high-energy mechanisms, associated major injuries take precedence
- Immobilize to prevent further neurologic or vascular injury

INITIAL STABILIZATION/THERAPY

- Stabilization of the multiple-injury trauma patient
- Long leg splint in full extension
- Ice
- Elevation
- Frank dislocations with vascular compromise may need immediate reduction in ED

ED TREATMENT/PROCEDURES

- Nonweight bearing
- Pain control
- Nondisplaced fractures or minimally displaced (< 8 mm) *lateral* plateau fractures without ligamentous injury:
 - Aspiration of hemarthrosis and injection of local anesthetic
 - Exam for ligamentous instability
 - If knee is *stable*:
 - Compressive dressing
 - Ice and elevation for 48 hr
 - No weight bearing/crutches
 - Knee is *unstable* if fracture is causing vascular injury or compartment syndrome
 - Urgent orthopedic consultation is warranted in the unstable knee
- Open fractures:
 - Remove contaminants
 - Apply moist sterile dressing
 - Assess tetanus immunity
 - Antibiotics
 - Early administration of antibiotic, within 2–3 hr
 - Orthopedics consult for early surgical débridement

MEDICATION

Open fractures: Aminoglycoside + Cephalosporin

- Cefazolin: 2 g IV (peds: 50 mg/kg)
- Gentamicin: 2–5 mg/kg IV (peds: 2.5 mg/kg)
- Tetanus toxoid if indicated
- Vancomycin: 1 g IV loading dose (peds: 10 mg/kg) if penicillin allergic



FOLLOW-UP

DISPOSITION

Admission Criteria

- Open fractures for débridement, irrigation, and IV antibiotics

- Comminuted, bicondylar fractures for traction
- High-energy mechanisms for observation of neurovascular status and development of compartment syndrome; may occur 24 or more after injury
- Pain control

Discharge Criteria

Nondisplaced or minimally displaced, stable fractures of the lateral plateau

FOLLOW-UP RECOMMENDATIONS

Orthopedic follow-up:

- Long leg splint with ice, elevation, and nonweight-bearing status of affected joint

PEARLS AND PITFALLS

- Consider popliteal artery injury with high-energy mechanisms of injury
- Lipoarthrosis (blood and fat globules) on arthrocentesis, is pathognomonic for intra-articular knee fracture
- Tibial plateau fractures, Segond fractures, and Salter–Harris 1 fractures are easily missed on plain knee radiographs

ADDITIONAL READING

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CODES

ICD9

- 823.00 Closed fracture of upper end of tibia alone
- 823.10 Open fracture of upper end of tibia alone

ICD10

- S82.143A Displaced bicondylar fracture of unsp tibia, init
- S82.143B Displaced bicondylar fx unsp tibia, init for opn fx type I/2
- S82.146A Nondisplaced bicondylar fracture of unsp tibia, init

TIBIAL/FIBULAR SHAFT FRACTURE

Stephen R. Hayden

BASICS

DESCRIPTION

Fracture Description

Tibia

- 80% have associated fibular fractures
- Open (24% are open) vs. closed
- Extent of soft tissue damage
- Gustilo–Anderson classification of open fractures:
 - Type I:
 - Wound < 1 cm
 - Little soft tissue damage
 - No crush injury
 - Type II:
 - Wound > 1 cm
 - Moderate soft tissue damage
 - Little or no devitalized soft tissue
 - Type III—severe soft tissue injury:
 - A—adequate soft tissue coverage of bone
 - B—tissue loss/periosteal stripping
 - C—neurovascular injury requiring surgery
- Anatomic location:
 - Proximal, middle, or distal 3rd
 - Articular extension
- Displacement
- Degree of shortening
- Angulation
- Configuration:
 - Spiral, transverse, or oblique
 - Comminuted, with butterfly fragment or multiple fragments

Fibula

- Proximal:
 - Associated with peroneal nerve injury
 - Disruption of ankle syndesmosis (Maisonneuve fracture)
- Middle
- Distal

Pediatric Considerations

- 3rd most common long bone fracture in children
- 2nd most common long bone fracture in nonaccidental trauma (usually apophyseal or metaphyseal corner)
- Nonphyseal fracture patterns:
 - Compression (torus): Distal metaphysis
 - Incomplete tension–compression (greenstick)
 - Plastic/bowing deformity of fibula may occur.
 - Complete fractures
- Physeal fracture patterns:
 - Tibial shaft fractures may extend to the physis in Salter–Harris II pattern.

ETIOLOGY

- High- vs. low-energy injury
- Amount of soft tissue injury is prognostic and determined by the degree of energy involved.
- Indirect force—frequently low-energy trauma:
 - Rotary and compressive forces often result in oblique and spiral fractures.
- Skiing, fall, child abuse
- Direct force—high-energy trauma:
 - Direct blow to leg often results in transverse and comminuted fractures.
- Pedestrian vs. auto, motor vehicle crash (MVC):
 - Bending force over a fulcrum often produces comminution with a wedge-shaped butterfly fragment.
- Skier's boot top, football tackle, MVC

Pediatric Considerations

- Bicycle spoke injury:
 - Foot and lower leg get caught between frame and wheel spoke
 - Crush injury is the primary problem.
 - Initial benign appearance of the soft tissues is often deceiving:
 - Full-thickness skin loss can occur in days.
 - Orthopedic surgery consultation should be obtained for all spoke-injury patients with associated fractures.
- Toddler fracture:
 - Spiral fracture involving the distal 3rd of the tibia with intact fibula secondary to rotational force (turning on planted foot)
 - Age range is 9 mo–6 yr, most often when learning to walk.
 - Fractures in midshaft or more transverse are suggestive of nonaccidental trauma.

SIGNS AND SYMPTOMS

History

- History of trauma
- Pain is usually immediate, severe, and well localized to the fracture site.

Physical-Exam

- Visible or palpable deformity at the fracture site
- Significant soft tissue damage with high-energy trauma
- Inability to bear weight if tibia involved:
 - _ May be able to walk if isolated fibular fracture
- Foot drop on affected leg from injury to the peroneal nerve as it wraps around the fibular head
- Compartment syndrome

Pediatric Considerations

- Rely on parents for historical information.
- Child may present limping with no obvious deformity.

ESSENTIAL WORKUP

- Careful assessment of soft tissues
- Careful neurovascular exam (compare with contralateral side)
- Examine for associated injuries.
- Completely expose patient and put into gown.
- Assessment for compartment syndrome

ALERT

- Compartment syndrome
- Occurs in 8% of diaphyseal fractures, more common in younger patients
- Relatively common complication of tibial fractures and may not appear until 24 hr after injury
- Pain disproportionate to that expected
- Patient may have swollen, tight compartment, but does not always have pain on palpation of compartment.
- Pain on passive stretch of foot, toes
- Sensory deficit
- Motor weakness is a late finding.
- Pulselessness is not a sign of compartment syndrome:
 - _ Palpable pulses are almost always present in compartment syndrome unless there is underlying arterial injury.
- 4 leg compartments: Anterior, lateral, deep posterior, and superficial posterior
- Anterior compartment:
 - _ Deep peroneal nerve

- Sensation of 1st web space
- Ankle and toe dorsiflexion
- Anterior tibial artery feeds dorsalis pedis artery
- Lateral compartment:
 - Superficial peroneal nerve
 - Sensation of dorsum of foot
 - Foot eversion
- Deep posterior compartment:
 - Tibial nerve
 - Sensation to sole of foot
 - Ankle and toe plantar flexion
 - Posterior tibial and peroneal arteries
- Superficial posterior compartment:
 - Branch of sural cutaneous nerve
 - Sensation to lateral foot

DIAGNOSIS TESTS & INTERPRETATION

Lab

Include creatine phosphokinase levels if concerned about compartment syndrome

Imaging

- Anteroposterior and lateral views of the leg, knee, and ankle
- Bone scan at 1–4 days for toddler fracture and stress fractures if radiographs unrevealing
- CT scan for complex fracture pattern to evaluate for rotational malalignment
- CT or MRI for pathologic fracture
- MRI for stress fractures may be necessary.

Diagnostic Procedures/Surgery

Compartment pressures:

- Pressures > 30 mm Hg are an indication for orthopedic consultation and fasciotomy.
- Delta P or difference between diastolic BP and compartment pressure < 20 is indicative of compartment syndrome
- Repeated pressure measurements over time, taken within 5 cm of fracture site, are necessary.

Pediatric Considerations

Oblique radiograph to detect nondisplaced fractures

DIFFERENTIAL DIAGNOSIS

- Stress fracture

- Pathologic fracture
- Osteomyelitis

Pediatric Considerations

- Sarcoma
- Pathologic fracture
- Osteomyelitis
- Nonaccidental trauma



TREATMENT

PRE HOSPITAL

- Look for associated injuries in high-energy mechanisms.
- Assess for neurologic or vascular compromise.
- Adequate immobilization is essential to prevent further injury.

INITIAL STABILIZATION/THERAPY

- Manage airway and resuscitate as indicated.
- Life-threatening injuries take precedence.
- Immobilize extremity.
- Apply ice
- Strict NPO
- Pain control

ED TREATMENT/PROCEDURES

- Closed fractures:
 - Gentle attempt at reduction if fracture is displaced (do not attempt multiple reductions; may increase risk for compartment syndrome).
 - Immobilization:
 - Well-padded long leg posterior splint
 - Knee in 10–20° of flexion
 - Avoid circumferential cast.
 - If pain persists after immobilization, suspect:
 - Compartment syndrome
 - Avoid elevation of leg in suspected compartment syndrome; it lowers perfusion to the extremity.
 - Nerve compression
 - Crutches
- Open fractures:
 - Remove contaminants and cover wound with moist, sterile dressing.
 - Antibiotics
 - Tetanus prophylaxis

- Immobilization with well-padded long leg posterior splint
- Immediate orthopedic surgery consultation for débridement and fracture fixation
- Isolated fibular fracture:
 - Usually treated symptomatically:
 - Padded splint
 - Elevation
 - Ice
 - No weight bearing until swelling resolves
 - Crutches if not bearing weight

MEDICATION

- Gram-positive cocci coverage for open fractures: Cefazolin 2 g loading dose then 1 g (peds: 50 mg/kg/d) IV/IM q8h
- Gustilo–Anderson type III, add gram-negative rod coverage: Gentamicin 3–5 mg/kg (peds: 2.5 mg/kg) IV q8h
- Farming accident, add *Clostridium* spp coverage: Penicillin G 10 million IU (peds: 250,000–400,000 IU/kg/d) IV q6h
- Tetanus 0.5 mL IM and tetanus immune globulin 250 U IM as indicated by the type of wound and the number of primary immunizations
- If penicillin allergic: Vancomycin 1 g (peds: 10 mg/kg) IV q12h



FOLLOW-UP

DISPOSITION

Admission Criteria

- Multiple trauma
- High-energy mechanism
- Soft tissue involvement
- Risk for compartment syndrome
- All open fractures
- Displaced, angulated, transverse, shortened, comminuted, and otherwise unstable fractures
- Intra-articular involvement
- Neurovascular compromise
- Inadequate pain control
- Pathologic fracture
- Nonaccidental trauma in children

Discharge Criteria

- Minimally displaced fracture with low-energy injury mechanism

- Close orthopedic follow-up
- Return parameters for compartment syndrome in a reliable patient
- If fracture is >48 hr old, compartment syndrome is unlikely to develop; if it has not occurred, discharge criteria may be more liberal.

FOLLOW-UP RECOMMENDATIONS

- Most pediatric fractures are treated with long leg cast for 4–6 wk.
- Nondisplaced and minimally displaced fractures in adults may be treated with long leg cast and closed reduction.
- Open contaminated fractures may be treated with external fixation and débridements.
- Treatment with intramedullary nail allows for early mobilization and weight bearing as tolerated.
- Kirschner wires are sometimes used in the treatment.

PEARLS AND PITFALLS

- High incidence of associated injuries in high-energy trauma:
 - Associated injuries commonly include:
 - Femoral fractures (“floating knee injury”)
 - Head trauma
 - Spine fractures
 - Deep venous thrombosis occurs in 10–25% of patients following tibial fracture.

ADDITIONAL READING

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CODES

ICD9

- 823.20 Closed fracture of shaft of tibia alone
- 823.22 Closed fracture of shaft of fibula with tibia
- 823.32 Open fracture of shaft of fibula with tibia

ICD10

- S82.209A Unsp fracture of shaft of unsp tibia, init for clos fx
- S82.209B Unsp fx shaft of unsp tibia, init for opn fx type I/2
- S82.409A Unsp fracture of shaft of unsp fibula, init for clos fx

TICK BITE

Jonathan A. Edlow

BASICS

DESCRIPTION

Even in high endemic areas for tick-borne diseases, the risk of infection with a tick-borne pathogen is very low. After a tick bite, patient concerns include:

- Tick removal
- Local effect of the bite
- Possibility of acquiring a tick-borne illness:
 - Fear of contracting Lyme disease
 - Desire to be tested or treated for Lyme

ETIOLOGY

- Specific tick-borne infections are discussed in other chapters.
- Tick bite can be from different species of ticks of 2 major types:
 - Soft ticks (*Ornithodoros*):
 - Cause tick-borne relapsing fever
 - Only feed for minutes and therefore almost never provoke a visit to the ED
 - Hard ticks—especially *Ixodes* and *Dermacentor*:
 - Feed for several days to a week and therefore, more likely to be noticed by patient and lead to an ED visit
- Lyme disease transmission:
 - Species of tick, stage of development, duration of attachment, and geography may all play a role in the possibility of developing Lyme disease.
 - Most cases of Lyme are associated with bites from nymphal *Ixodes scapularis* ticks.
 - Most cases of Lyme are transmitted only after the tick has been attached for 24–48 hr:
 - Degree of engorgement is a marker for duration of attachment.

DIAGNOSIS

SIGNS AND SYMPTOMS

Tick is attached to skin.

History

- The patient usually has made the diagnosis themselves, although sometimes they mistake the tick for skin tags or other skin lesions.

- Ask regarding duration of tick attachment, as this may influence the decision to prescribe antibiotic prophylaxis.

Physical-Exam

Directly examine the skin and the tick:

- Try to identify the tick species.
- Estimate degree of engorgement.

ALERT

- Some tick-borne infections are potentially fatal. Because there are no confirmatory diagnostic tests that are available in real time, they must be diagnosed based on history, physical, and epidemiologic context.
- Because the drug of choice for some of these infections—doxycycline—is not usually prescribed for empiric therapy for acutely ill febrile patients, ask about the potential for tick bites in the history of febrile patients and consider using this drug in the appropriate settings.

ESSENTIAL WORKUP

Accurate history and physical exam searching for presence of tick

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Testing for Lyme disease is *not* indicated:
 - Such antibody testing would only reflect prior exposure to *Borrelia burgdorferi*
 - No treatment implications whatsoever for the current bite

Diagnostic Procedures/Surgery

- Testing of the tick itself is not recommended.
- See treatment for tick removal.

DIFFERENTIAL DIAGNOSIS

- Tick-borne diseases in North America:
 - Lyme disease
 - Other Lyme-like diseases such as Southern tick-associated rash illness (STARI) and *Borrelia miyamotoi* infection
 - Babesiosis
 - Anaplasmosis (formerly ehrlichiosis)
 - Rocky Mountain spotted fever (RMSF)
 - Relapsing fever
 - Tularemia
 - Colorado tick fever

- Q-fever
- Tick-borne encephalitis (Powassan fever)
- Tick paralysis
- Additional tick-borne diseases found in Europe:
 - Tick-borne encephalitis
 - Boutonneuse fever (*Rickettsia conorii*)
 - Other spotted fever rickettsiae



TREATMENT

INITIAL STABILIZATION/THERAPY

Remove tick:

- Early removal reduces the likelihood of transmission of tick-borne infections.

ED TREATMENT/PROCEDURES

- Tick removal method:
 - Grasp the tick with very fine forceps, as close to the skin as possible, and gently lift up over 30–120 sec.
 - Most ticks will come out.
 - Do not to squeeze the tick, which could inject infectious materials into the patient's skin.
 - If mouthparts are left in the skin, although this could lead to local infection or foreign body reaction, it has no implications for transmission of tick-borne diseases.
- Another described method:
 - Inject an intradermal wheal of lidocaine with epinephrine beneath the tick.
 - Tick may crawl out of its own accord.
- Methods *not* to use include:
 - Burning the tick with a match
 - Covering it with petroleum jelly or other noxious agents
- Lyme disease prophylaxis:
 - Indicated if the tick is an engorged *I. scapularis* nymph is found within 72 hr of the bite, or if the physician decides to prophylax
 - Doxycycline 200 mg for 1 dose
 - For children, there is no studied single-dose regimen:
 - Prescribe amoxicillin (25–50 mg/kg) for 10 days in divided doses.
 - No data support prophylactic antibiotics for other tick-borne diseases.

Pediatric Considerations

- Several studies used 10 days of amoxicillin in children for prevention of Lyme disease.
- No patients in the treated groups developed Lyme or seroconverted.

- Tick paralysis is a rare disease but usually occurs in children, especially in girls with long hair; never diagnose Guillain–Barré syndrome without doing a thorough inspection of the entire body, especially the scalp, for ticks.

Pregnancy Considerations

Although there are no high quality data on antibiotic prophylaxis for Lyme disease in pregnant women, some authors recommend having a very low threshold for treating pregnant women with tick bites (using amoxicillin).

MEDICATION

- Amoxicillin: 25–50 mg/kg in div. doses TID for 14–21 days
- Doxycycline: 200 mg PO for 1 dose



FOLLOW-UP

DISPOSITION

Admission Criteria

- Tick bite with symptoms or signs of tick paralysis or in patients who have an established tick-borne disease which is severe (e.g., hypotension or sepsis)
- Tick bite leading to systemic infection sufficiently severe to require admission (e.g., RMSF, anaplasmosis, babesiosis [especially in a splenectomized patient]).

Discharge Criteria

All other patients, the vast majority, are safely discharged.

FOLLOW-UP RECOMMENDATIONS

- Follow-up with primary care physicians if there are issues regarding local bacterial infection from the bite (cellulites) or subsequent symptoms and signs of 1 of the tick-borne infections listed above.
- Seek medical attention in the event of a febrile illness and to report the history of the tick bite to that physician.
- Patients who have been bitten by ticks should be counseled about future tick bite prevention, including potential use of DEET to the skin and permethrin-treated clothing.

PEARLS AND PITFALLS

- Early tick removal reduces the likelihood of transmission of tick-borne infections.
- Lyme disease prophylaxis is indicated if the tick is an engorged *I. scapularis* nymph.
- Consider babesiosis in splenectomized patients presenting with fever.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Lyme Disease
- Rocky Mountain Spotted Fever



CODES

ICD9

- 066.1 Tick-borne fever
- 088.81 Lyme Disease
- 989.5 Toxic effect of venom

ICD10

- A68.9 Relapsing fever, unspecified

- A69.20 Lyme disease, unspecified
- T63.481A Toxic effect of venom of arthropod, accidental, init

TINEA INFECTIONS, CUTANEOUS

Aaron J. Monseau

BASICS

DESCRIPTION

- Superficial fungal infections of the hair, skin, or nails:
 - Usually confined to the stratum corneum layer
 - Among the most common diseases worldwide
- Requires keratin for growth, so does not involve mucosa
- Named for location of infection

ETIOLOGY

- Dermatophytes:
 - *Microsporum*
 - *Trichophyton*
 - *Epidermophyton*
 - *Malassezia furfur*, a yeast, is the etiologic agent of tinea versicolor (not a true tinea)
- Trauma or maceration of the skin may allow fungal entry into skin
- Transmission may be person to person, animal to person, or soil to person

Pediatric Considerations

- Fungi can be spread from toys and brushes
- Tinea unguium is rare in children and is associated with:
 - Down syndrome
 - Immunosuppression
 - Tinea pedis or capitis

DIAGNOSIS

SIGNS AND SYMPTOMS

- Tinea capitis:
 - Children are predominately affected
 - Most contagious dermatophytosis
 - Alopecia, dandruff-like scaling
 - Kerion:
 - Boggy, inflammatory mass that exudes pus and causes cervical lymphadenopathy
 - “Black dots” from infected hairs broken off at the scalp
- Tinea corporis (“ringworm”):

- Arms, legs, and trunk
- Sharply margined, annular lesion with raised margins and central clearing
- Hair follicle involvement may produce indurated papules and pustules
- Lesions may be single, multiple, or concentric
- Pets are often a vector
- Tinea cruris (“jock itch”):
 - Erythematous, scaly, margined patches involving the perineum, thighs, and buttocks
 - Associated with heat, humidity, and tight-fitting undergarments
 - Unlike the case in candidiasis, the scrotum and penis are spared
- Tinea pedis (“athlete’s foot”):
 - Scaling, maceration, fissuring between the toes
 - Risk factors:
 - Advanced age
 - Immunocompromised status
 - Hot, humid climates
 - Infrequent changing of socks
 - More common in adults than children
 - Most common tinea infection in US
 - “Trichophytid” reaction:
 - Vesicular eruption remote from infection
 - Involving hands, mimics dyshidrotic eczema
- Tinea unguium:
 - 1 type of onychomycosis
 - Yellow or brown discoloration with thickening and debris under the nails
 - Onycholysis: Loosening of the nail from bed
 - May involve the plantar surface of the foot
- Tinea versicolor (not true tinea):
 - Most common in warm months
 - Round or oval superficial brown, yellow, or hypopigmented macules that may coalesce
 - Upper trunk, arms, and neck
 - Facial involvement is common in children

History

- Time of onset from inoculation to visible skin changes is about 2 wk
- Main symptom is itching:
 - Hair loss with tinea capitis
- Participation in contact sports or contacts with similar skin disease

Physical-Exam

- Tinea capitis: Alopecia, broken hairs at scalp surface
- Tinea corporis: Areas of exposed skin typically involved with annular scaly plaques, raised edges, may have pustules and vesicles
- Tinea cruris: Erythematous lesions on groin and pubic region with central clearing and raised edges
- Tinea pedis: Scaling, maceration, and fissuring of toe webs, often only 1 foot affected
- Tinea unguium: Separation of nail plate from nail bed with thickened, discolored, broken nails

ESSENTIAL WORKUP

- Diagnose by clinical exam
- If diagnosis is in doubt, confirm with microscopy before starting oral antifungals because of possible side effects

DIAGNOSIS TESTS & INTERPRETATION

Lab

Fungal cultures are slow growing and should not be routinely done

Imaging

Generally not indicated

Diagnostic Procedures/Surgery

- Wood lamp is insensitive:
 - *Trichophyton*, the most common cause of tinea infections, does NOT fluoresce
 - *Microsporum* fluoresces bright green
 - *Malassezia* (tinea versicolor) fluoresces yellow to yellow-green
 - Erythrasma (nontinea corynebacterial infection) will fluoresce coral red
- Microscopy:
 - Cleanse area with 70% ethanol
 - Scrape active margin of lesion with no. 10 or no. 15 scalpel blades
 - Place scrapings on a glass slide, add a drop of 10–20% potassium hydroxide solution, and cover with a coverslip
 - The presence of septate hyphae confirms dermatophyte infection
 - Budding yeasts and short hyphae (“spaghetti and meatballs”) confirms *Malassezia*

Pediatric Considerations

- Methods to obtain fungal elements for culture or microscopy:
 - Brushing the hair with a toothbrush
 - Rolling a moistened cotton swab
 - Collecting skin cells with transparent tape

DIFFERENTIAL DIAGNOSIS

- Tinea capitis: Impetigo, pediculosis, alopecia areata, seborrheic dermatitis, atopic dermatitis, and psoriasis
- Tinea corporis: Impetigo, herpes simplex, Lyme disease, verruca vulgaris, psoriasis, nummular eczema, granuloma annulare, herald patch of pityriasis rosea, erythema multiforme, urticaria, seborrheic dermatitis, and secondary syphilis
- Tinea cruris: Impetigo, seborrheic dermatitis, psoriasis, candidal infection, irritant and allergic contact dermatitis, and erythrasma
- Tinea pedis: Scabies, erythrasma, *Candida*, allergic and contact dermatitis, and psoriasis
- Tinea unguium: Psoriasis, dermatitis, lichen planus, and congenital nail dystrophy
- Tinea versicolor: Vitiligo, secondary syphilis



TREATMENT

PRE HOSPITAL

Maintain universal precautions.

INITIAL STABILIZATION/THERAPY

Only in immunocompromised or septic patients

ED TREATMENT/PROCEDURES

- Improvement usually occurs within 1–2 wk of treatment; hair and nail tinea require longer treatment of 3–6 mo
- Topical antifungals do not penetrate hair/nails:
 - Use in conjunction with systemic agent for tinea capitis or unguium.
- Tinea capitis:
 - Terbinafine is now considered the drug of choice by most:
 - Pill form may be crushed in food
 - Newer oral antifungals, including terbinafine, itraconazole, and fluconazole, are preferred:
 - Retained in tissues longer
 - Allows for shorter treatment courses without a decrease in efficacy
 - Improved compliance
 - Selenium sulfide or ketoconazole shampoo reduces transmissibility
 - Kerion may respond more rapidly with addition of prednisone (peds: 1 mg/kg PO QD for 2 wk)

ALERT

Terbinafine may be less effective than griseofulvin against *Microsporum* species causing tinea capitis; however, *Trichophyton* species are the predominant causative organism in children:

- Tinea corporis, cruris, and pedis:

- Topical terbinafine or imidazoles (ketoconazole, miconazole, and clotrimazole) are 1st-line agents:
 - Topical terbinafine has been shown to be as effective as or more effective than the imidazoles, with a shorter course
- Oral therapy may be necessary for cases resistant to topical treatment or for immunocompromised patients
- Keep the area dry (talc powders) and frequently change socks and underclothes
- Tinea unguium:
 - Requires oral therapy and longer course than other tinea infections
 - Terbinafine had a slightly higher cure rate than imidazoles (ketoconazole, miconazole, and clotrimazole) or griseofulvin in a meta-analysis
 - Ciclopirox 8% nail lacquer approved for treatment but has low cure rates:
 - May enhance oral therapy
- Tinea versicolor:
 - Topicals are 1st-line therapy:
 - Selenium sulfide 2.5% shampoo was as effective as topical ketoconazole
 - Oral ketoconazole, itraconazole, or fluconazole have been used with cure rates up to 97% but are not as safe as topicals

MEDICATION

- Ciclopirox 8% nail lacquer: Apply to the affected nails daily, max. 48 wk; remove with alcohol every 7 days (peds: Same).
- Clotrimazole: Apply 1% cream to affected area BID for 4–6 wk (peds: Same).
- Fluconazole: Tinea unguium—150–300 mg/wk pulse therapy for 3–6 mo for fingernails, 6–12 mo for toenails; tinea corporis, cruris, and pedis: 150 mg PO weekly for 4–6 wk; tinea versicolor: 400 mg PO single dose (peds: 6 mg/kg/d for 3–6 wk for tinea capitis)
- Griseofulvin: Tinea capitis, corporis, cruris—500 mg PO QD for 4–6 wk (peds: 10–20 mg/kg up to 500 mg PO QD until the hair regrows, usually 6–8 wk)
- Itraconazole: Tinea capitis: Adults and peds: 3–5 mg/kg PO QD for 2–4 wk; tinea unguium: 200 mg PO QD for 3 mo; tinea versicolor: 400 mg PO QD for 3–7 days; contraindicated in CHF
- Ketoconazole: 2% topical cream QD for 4–6 wk; tinea capitis, corporis, cruris, pedis—200 mg PO QD for 4 wk (peds: 3.3–6.6 mg/kg PO QD for 4 wk); tinea versicolor—400 mg PO × 1 or 200 mg QD for 7 days (contraindicated with terfenadine and astemizole); soda increases absorption 65%
- Miconazole: Apply cream to affected area BID for 4–6 wk (peds: Same)
- Selenium sulfide: 2.5% shampoo to affected area for 10 min for 1–2 wk (peds: Same)
- Terbinafine: 1% topical cream BID for 4–6 wk for tinea pedis QD for tinea corporis and tinea cruris; tinea unguium—250 mg PO QD for 6 wk for fingernails, 12 wk

for toenails (peds: < 20 kg, 67.5 mg/d; 20–40 kg, 125 mg/d; > 40 kg, 250 mg/d at same interval as adult); tinea pedis: 250 mg PO per day for 2 wk; tinea capitis: 250 mg/d for 4 wk (dose by weight as for tinea unguium for 4 wk)

- Tolnaftate: Apply 1% cream/powder/solution to the affected area BID for 4–6 wk (peds: Same)

ALERT

- The oral antifungals may rarely cause hepatotoxicity; consider checking liver transaminases prior to initiating therapy

Pediatric Considerations

Topical preparations are preferred when possible

Pregnancy Considerations

- Few studies addressing the use of antifungal medications during pregnancy in humans
- Some of the imidazoles have shown adverse effects in animals – class C (fluconazole, itraconazole, ketoconazole)
- Clotrimazole, miconazole, and terbinafine are class B drugs
- Weigh risk: Benefit as elective antifungal therapy generally not recommended.

First Line

- Tinea capitis: Terbinafine
- Tinea corporis, cruris, pedis: Topical terbinafine or imidazoles (ketoconazole, miconazole, and clotrimazole)
- Tinea versicolor: Selenium sulfide shampoo and topical ketoconazole



FOLLOW-UP

DISPOSITION

Admission Criteria

- Invasive disease in immunocompromised host
- Kerion with secondary bacterial infection

Discharge Criteria

- Most patients may be managed as outpatients
- Children may return to school once appropriate treatment has been initiated

Issues for Referral

Patients started on oral antifungals should be referred for follow-up to monitor therapy and advised regarding symptoms of hepatitis

FOLLOW-UP RECOMMENDATIONS

- Monitor for bacterial superinfection, cellulitis, generalized invasive infection:
 - Especially in immunocompromised (diabetics, HIV patients)

PEARLS AND PITFALLS

- Tinea capitis is the most common pediatric dermatophyte infection
- Itching is the main symptom in most forms of tinea, with associated hair loss in tinea capitis
- Cellulitis frequently complicates of tinea pedis
- Relapse of tinea pedis/cruris is common
 - Patients should wash or replace contaminated socks/towels/footwear

ADDITIONAL READING

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CODES

ICD9

- 110.0 Dermatophytosis of scalp and beard
- 110.1 Dermatophytosis of nail
- 110.5 Dermatophytosis of the body

ICD10

- B35.0 Tinea barbae and tinea capitis
- B35.1 Tinea unguium
- B35.4 Tinea corporis

TOLUENE POISONING

Michael E. Nelson



BASICS

DESCRIPTION

- Prototypical volatile hydrocarbon
- Clear, colorless liquid with sweet odor

ETIOLOGY

- Abused for its euphoric effect
- Occupational exposures
- Used as organic solvent found in:
 - Oil paints and stains
 - Paint thinners
 - Glues, inks, dyes, correction fluid
 - Coolants
 - Petroleum products
 - Aerosolized household products
 - Degreasers
- Production and use of gasoline is largest source of exposure

Pediatric Considerations

- Prevalent in adolescent age group:
 - Inexpensive “high” with readily available sources
 - Many psychosocial problems
- May develop chronic neurologic dysfunction
- Mechanism:
 - Rapidly absorbed by inhalation
 - Readily crosses blood–brain barrier, reaching high concentrations in brain
 - May sensitize myocardium to dysrhythmogenic effect of catecholamines
 - Inhibits myocardial voltage-gated sodium channels and inward rectifying potassium channels
 - Alveolar excretion and liver metabolism
- Methods of intoxication:
 - Sniffing: Simple inhalation of substance directly from container
 - Huffing: Vapors inhaled through cloth saturated with substance
 - Bagging: Vapors inhaled from bag containing substance
- Toxic range:
 - 100 ppm: Impairment of psychomotor and perceptual performance
 - 500–800 ppm: Headache, drowsiness, nausea, weakness, and confusion,

potential lethal ranges

- >800 ppm: Convulsions, ataxia, staggering gait
- 10,000–30,000 ppm: Anesthesia within 1 min



DIAGNOSIS

SIGNS AND SYMPTOMS

- Acute:
 - Neurologic:
 - Depression
 - Euphoria
 - Ataxia
 - Dizziness
 - Seizures
 - Cardiac:
 - Fatal dysrhythmias
 - Pulmonary:
 - Chemical pneumonitis
 - Pulmonary edema
 - Electrolytes:
 - Hypokalemia
 - Hypocalcemia
 - Hyperchloremic metabolic acidosis, likely from hippuric acid metabolite
 - GI:
 - Abdominal pain
 - Nausea, vomiting
 - Hematemesis
 - Renal:
 - Distal renal tubular acidosis
 - Hematuria
 - Proteinuria
 - Musculoskeletal:
 - Diffuse weakness
- Chronic:
 - Neurologic:
 - Peripheral neuropathy (diffuse demyelination)
 - Leukoencephalopathy
 - Cerebral/cerebellar atrophy
 - Optic atrophy
 - Dementia
 - Cognitive/neurobehavioral abnormalities

- Cardiac:
 - Dysrhythmias
 - Dilated cardiomyopathy
- Renal:
 - Distal renal tubular acidosis
 - Renal failure
 - Fanconi syndrome
- Musculoskeletal:
 - Rhabdomyolysis
- Psychiatric:
 - Addiction/withdrawal

Pregnancy Considerations

- Fetal solvent syndrome reported from mothers who chronically abused toluene while pregnant, resembles fetal alcohol syndrome
- Infant more likely premature, low birth weight, microcephaly, and developmental delay

History

- Detailed history of sniffing, huffing, bagging, or other abuse of paints/solvents
- Occupational exposures

Physical-Exam

- Presence of agent on lips, nose, or clothes (metallic paint has highest concentration)
- Perioral eczematous dermatitis from chronic huffing or bagging
- Odor of agents

ESSENTIAL WORKUP

- Detailed physical exam
- CXR for suspected pneumonitis

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Electrolytes, BUN, creatinine, glucose:
 - Hypokalemia
 - Normal or high anion gap metabolic acidosis
 - Hyperchloremia
 - Impaired renal function
 - Severe hypocalcemia/hypophosphatemia
- Urinalysis:
 - Check for myoglobin (rhabdomyolysis)

- Hematuria and protein often present
- Creatinine kinase if suspect rhabdomyolysis
- Alcohol level—often coingestant
- Liver enzymes, prothrombin time (PT), partial thromboplastin time (PTT), INR, as may cause hepatotoxicity
- Urine for hippuric acid (metabolite of toluene):
 - Confirms exposure but does not correlate with systemic effects
- Serum levels only detectable for short time after exposure

Imaging

- EKG:
 - For atrial and ventricular dysrhythmias
- CXR:
 - Indicated if dyspnea or low oxygen saturation
 - Chemical pneumonitis
- CT head:
 - For altered mental status/chronic exposure
 - Cerebral/cerebellar atrophy, white matter hypodensity

Diagnostic Procedures/Surgery

CSF often unremarkable but may be indicated for altered mental status to rule out other etiologies

DIFFERENTIAL DIAGNOSIS

- Alcohol intoxication
- Other hydrocarbon abuse
- Other inhalants (nitrous oxide, difluoroethane, butane, etc.)
- Methanol
- Ethylene glycol
- Salicylate
- Heavy metal exposure
- Guillain–Barré syndrome
- Metabolic abnormalities



TREATMENT

PRE HOSPITAL

- Rapid onset of toxicity
- Death possible with sudden cardiac dysrhythmias (sudden sniffing death), often from catecholamine surge (e.g., eluding police)
- Topical decontamination as needed
- Forced emesis is not indicated:

- Decreased level of consciousness may lead to aspiration.

INITIAL STABILIZATION/THERAPY

- ABCs
- Supplemental oxygen
- Cardiac monitor
- 0.9% NS IV access
- Naloxone, thiamine, and check glucose if altered mental status

ED TREATMENT/PROCEDURES

- Treat cardiac dysrhythmias in standard fashion:
 - Consider β -blocker for tachydysrhythmias.
- Monitor respiratory status with pulse oximetry, CXR, and ABG if significant inhalation.
- Steroids not recommended for pneumonitis.
- Correct metabolic abnormalities:
 - Potassium
 - Calcium
 - Phosphate
- Acidosis resolves with IV fluids.
- If rhabdomyolysis, maintain high urine output.
- Gastric decontamination for oral ingestion rarely useful and may cause harm:
 - Charcoal does not bind hydrocarbons well and stomach distention may predispose to vomiting and aspiration.

MEDICATION

- Dextrose: D₅₀W, 1 amp: 50 mL or 25 g (peds: D₂₅W, 2–4 mL/kg) IV
- Naloxone (Narcan): 2 mg (peds: 0.1 mg/kg) IV or IM initial dose
- Thiamine (vitamin B₁): 100 mg (peds: 50 mg) IV or IM



FOLLOW-UP

DISPOSITION

Admission Criteria

- Altered mental status
- Dysrhythmias
- Hepatic dysfunction
- Renal failure
- Rhabdomyolysis
- Severe metabolic derangements
- Refractory hypokalemia

Discharge Criteria

After 4–6 hr of observation:

- Mental status at baseline
- No evidence of cardiac, metabolic, or neurologic derangement

FOLLOW-UP RECOMMENDATIONS

- Psychiatry referral for intentional/repeated ingestions and addiction counseling
- Cessation of use is most important intervention

PEARLS AND PITFALLS

- Myocardial sensitization to catecholamines:
 - Possibility of sudden dysrhythmia/death
 - Cardiac dysrhythmias have poor prognosis
- Monitor and replete electrolyte abnormalities.

ADDITIONAL READING

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The author would like to provide special thanks to the author of the prior edition, Matthew Valento.



CODES

ICD9

- 305.90 Other, mixed, or unspecified drug abuse, unspecified use
- 982.0 Toxic effect of benzene and homologues

ICD10

- F18.10 Inhalant abuse, uncomplicated
- F18.120 Inhalant abuse with intoxication, uncomplicated

- T52.2X1A Toxic effect of homologues of benzene, accidental (unintentional), initial encounter

TOOTHACHE

Franklin D. Friedman

BASICS

DESCRIPTION

- Tooth pain is caused by irritation of the root nerves located in pulpal tissue:
 - The pulp is the tooth's center and its neurovascular supply
- Other etiologies, both inside the mouth and referred to the oral cavity, may cause oral pain

ETIOLOGY

- Dental:
 - Dental caries (hard structures demineralized by bacteria)
 - Pulpitis (inflamed pulp secondary to infection)
 - Reversible pulpitis is mild inflammation of the tooth pulp caused by caries encroaching on the pulp
 - Irreversible pulpitis is the result of an untreated carious lesion causing severe inflammation of the pulp and severe, persistent, poorly localized discomfort
 - Periapical abscess (necrotic pulp and subsequent abscess)
 - Postextraction pain (dry socket, infection)
 - Cracked-tooth syndrome (pain, cold sensitivity, crack difficult to visualize)
- Periodontal disease:
 - Gingivitis and periodontitis (gingivitis with loss of periodontal ligament attachment)
 - Periodontal abscess (gum boil)
 - Pericoronitis (gingival inflammation from malerupted tooth)
 - Acute necrotizing ulcerative gingivitis (gingival pain, ulcers with/without pseudomembranes)
 - Denture stomatitis
 - Herpetic gingivostomatitis
 - Aphthous ulcers (canker sores)
 - Traumatic ulcers

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Tooth pain:

- May be referred to jaw, ear, face, eye, and neck (sensory distribution of 5th cranial nerve)
- Pain often associated with chewing, changes in temperature, and recumbency
- Malodorous breath
- Fever and chills
- Foul taste in mouth
- Associated symptoms
- Duration of symptoms
- Treatments that have already been tried

Physical-Exam

- Dental decay
- Facial swelling or erythema
- Trismus:
 - Decreased maximal interincisal opening (normal opening, 35–50 mm)
- Inspect and palpate lips, salivary glands, floor of the mouth, lymph nodes of the neck
- Assess voice changes
- Identify periodontal abscess
- Evaluate for deep-space infection
- Examine face for swelling, redness, tenderness, and increased warmth
- Examine neck for adenopathy and stiffness
- Teeth should be percussed for tenderness and mobility
- Teeth should be examined for fracture and missing teeth
- Dental numeric system used in adults:
 - Maxillary: Right to left 1–16; mandibular: Left to right 17–32 (peds: A–J and K–T)
 - Alternatively identification of teeth by their location is also appropriate (i.e., left rearmost, upper molar)

ESSENTIAL WORKUP

- Obtain appropriate medical and dental history
- Ask about drug allergies, especially antibiotics and analgesics, and current medications
- Assess need for pre-dental procedure antibiotic prophylaxis:
 - Rheumatic fever
 - Cardiac valve replacements
 - Orthopedic joint replacements
 - Mitral valve prolapse or valvular heart disease
- If physical exam conflicts with patient's history and intraoral source of pain is not apparent consider other sources of pain:
 - Nonodontogenic etiologies of pain

- Factitious pain/drug-seeking behavior

DIAGNOSIS TESTS & INTERPRETATION

Lab

- No lab tests needed except in patients with signs of systemic toxicity and those patients with perceptible deep-space infection
- As with any other infection with symptoms of systemic toxicity, consider CBC, blood cultures, markers of inflammation like ESR or CRP

Imaging

- Panoramic and periapical radiograph views if suspicion exists about dental infection or fracture
- CT or MRI to evaluate deeper infections

Diagnostic Procedures/Surgery

A local or regional dental nerve block may sometimes offer both therapeutic and diagnostic benefit

DIFFERENTIAL DIAGNOSIS

- Sinusitis
- Otitis media
- Pharyngitis
- Peritonsillar abscess
- Temporomandibular joint syndrome:
 - Usually presents with pain around the ear
- Trigeminal neuralgia
- Vascular headache
- Herpes zoster
- Cardiac ischemia

Pediatric Considerations

- Tooth eruption in a child or infant may cause oral pain, irritability, low-grade fever, diarrhea, and decreased food intake
- Facial swelling with fever and leukocytes $> 15,000/\text{mm}^3$ suggests a nonodontogenic source
- Children have a maximum of 20 deciduous teeth, 10 upper and 10 lower



TREATMENT

PRE HOSPITAL

- Maintain patent airway in patients with severe facial swelling or trismus

- The patient should be kept in a sitting position if possible

INITIAL STABILIZATION/THERAPY

- Airway management for deep-space infection and airway compromise
- Early pain management as indicated

ED TREATMENT/PROCEDURES

- Appropriate analgesia
- NSAIDs are *1st-line* therapy for uncomplicated dental pain
- Opiate analgesics are an alternative therapy
- Dental anesthetic field block:
 - Injected along the buccal surface of the affected tooth
 - Specific nerve block for multiple teeth
 - Long-acting anesthetic (e.g., bupivacaine)
- Antibiotics if dental infection is present:
 - *Penicillin is the antibiotic of choice* if patient is not allergic
 - Clindamycin for patients with penicillin allergy or for predominance of anaerobes
- Localized periapical and periodontal abscesses should be incised, drained, and irrigated:
 - Drain may be placed for 24 hr
- Saline rinses at home 4 times a day and dental referral in 24 hr

MEDICATION

- Antibiotics:
 - Ampicillin/sulbactam 1.5–3 g IM/IV q6h (peds: 300–600 mg/kg/d [max. 3 g] IV div. q6h)
 - Clindamycin: 150–450 mg PO q6h (peds: 15–30 mg/kg/24 h [max. 2 g] q6h):
 - IV dose 300–900 mg (peds: 25–40 mg/kg/24 h div. q8h)
 - Penicillin VK: 500 mg PO q6h (peds: 25–50 mg/kg/24 h [max. 3 g] q6h)
 - Penicillin G potassium aqueous: 4 mU IM/IV q4h (peds: 250,000–400,000 U/kg/d IM/IV div. q4–6h, max. 24 mU/d)
- Analgesics:
 - Acetaminophen: 500 mg PO/PR q4–6h (peds: 10–15 mg/kg/dose; do not exceed 5 doses/24 h); do not exceed 4 g/24 h
 - Acetaminophen and codeine no. 3: 1–2 tablets PO q4–6h (peds: elixir–codeine 12 mg/5 mL)
 - Oxycodone 5 mg ± with acetaminophen 325 mg: 1 or 2 tablets PO q6h (peds: 0.05–0.15 mg/kg (oxycodone) per dose [max. 5 mg]); not available in liquid preparation
 - Ibuprofen: 400–800 mg PO q8h (peds: 10 mg/kg PO q6h)
 - Ketorolac: 30 mg IV, 30–60 mg IM q6h (peds: 1 mg/kg/dose IM/IV)

- Morphine sulfate: 2–8 mg SC or IM/IV q2h (peds: 0.1 mg/kg/dose SC or IM/V q2h)

Pediatric Considerations

Teething infants may be helped by over-the-counter topical anesthetics and oral analgesics



FOLLOW-UP

DISPOSITION

Admission Criteria

- Suspicion of deep-space infections (e.g., Ludwig angina, retropharyngeal abscess)
- Facial cellulitis proximal to the eye
- Extensive trismus
- Inability to maintain nutrition and hydration
- Evidence of systemic toxicity

Discharge Criteria

Patients with toothache and localized dental infections can be discharged from the ED

Issues for Referral

Patients treated in the ED should be referred to a dentist or dental surgeon promptly

FOLLOW-UP RECOMMENDATIONS

Regular and routine dental evaluations

PEARLS AND PITFALLS

- Mistaking a deep infection for local infection
- Failing to identify source of referred pain to the mouth

ADDITIONAL READING

- Annino DJ Jr, Goguen LA. Pain from the oral cavity. *Otolaryngol Clin North Am.* 2003;6:1127–1135.
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- Van Meter MW, Dave AK. Oral Nerve Block. *Emedicine.* Available at



See Also (Topic, Algorithm, Electronic Media Element)

- [Aphthous Ulcer](#)
- [Facial Fracture](#)
- [Periodontal Abscess](#)
- [Peritonsillar Abscess](#)
- [Retropharyngeal Abscess](#)
- [Temporal–Mandibular Joint Injury/Syndrome](#)



CODES

ICD9

- [521.00 Dental caries, unspecified](#)
- [522.0 Pulpitis](#)
- [525.9 Unspecified disorder of the teeth and supporting structures](#)

ICD10

- [K02.9 Dental caries, unspecified](#)
- [K04.0 Pulpitis](#)
- [K08.8 Other specified disorders of teeth and supporting structures](#)

TORTICOLLIS

Andrew K. Chang • Robert Meyer



BASICS

DESCRIPTION

- Torticollis is a symptom, not a disease
- “Twisted neck” (L. *tortus*, twisted + *collum*, neck)
- A fixed or dynamic posturing of the head and neck
- Synonym(s): Cervical dystonia, wry neck

ETIOLOGY

Local

- Acute wry neck:
 - Develops overnight without provocation
 - Most prevalent
 - Self-limited, symptoms resolve in 1 to 2 wk
 - Cervical spine disease
 - Fracture
 - Dislocation, subluxation
 - Infections
 - Spondylosis
 - Tumor
 - Scar tissue–producing injuries
 - Ligamentous laxity in atlantoaxial region
- Inflammatory disease causing muscular damage:
 - Myositis
 - Lymphadenitis
 - Tuberculosis
 - Myasthenia gravis
 - Neuritis of the auriculotemporal branch of the trigeminal nerve
- Infections of surrounding soft tissues:
 - Nasopharyngeal abscess
 - Retropharyngeal abscess
 - Cervical adenitis
 - Tonsillitis
 - Meningitis
 - Mastoiditis
 - Sinusitis

Compensatory

- Tilt with essential head tremor (patient tilts head to suppress tremor)

- Ocular muscle palsy

Central

- Idiopathic spasmodic torticollis:
 - Female > male
 - Onset 31–60 yr old
- Dystonias:
 - Torsion dystonia
 - Generalized tardive dystonia
 - Wilson disease
 - λ-Dopa therapy
 - Acute (neuroleptic drugs)
 - Strychnine poisoning

Pediatric Considerations

Local

- Congenital:
 - Odontoid hypoplasia
 - Hemivertebrae
 - Spina bifida
 - Arnold–Chiari syndrome
 - Pseudotumor of infancy
 - Hypertrophy or absence of cervical musculature
- Otolaryngologic:
 - Vestibular dysfunction
 - Otitis media
 - Cervical adenitis
 - Retropharyngeal abscess
 - Pharyngitis
 - Mastoiditis
 - Esophageal reflux
 - Syrinx with spinal cord tumor
- Trauma:
 - Cervical fracture/dislocation
 - Clavicular fractures
 - Pneumomediastinum
- Juvenile rheumatoid arthritis

Compensatory

- Strabismus (4th cranial nerve paresis)
- Congenital nystagmus
- Posterior fossa tumor

Central

Dystonias:

- Torsion dystonia

- Drug induced
- Cerebral palsy



DIAGNOSIS

SIGNS AND SYMPTOMS

- Intermittent painful spasms of sternocleidomastoid (SCM), trapezius, and other neck muscles
- Head is rotated and twisted to 1 direction
- Pure flexion (anterocollis) or extension (retrocollis) is *rare*:
 - Represents symmetric involvement of muscles
- Symptoms usually aggravated by standing, walking, or stressful situations
- Usually does not occur with sleep

History

- Obtain a complete medication history
- The majority of antipsychotic medication–induced dystonic reactions occur between 12 and 23 hr
- Obtain a complete trauma history

Physical-Exam

- Head is rotated and twisted to 1 direction
- Neck movements vary from jerking to smooth
- The presence of fever supports an infectious or inflammatory etiology
- If the neurologic exam is focal, consider spinal cord or CNS disease
- Congenital form:
 - A firm, nontender enlargement of the SCM muscle visible at birth

ESSENTIAL WORKUP

- Geared toward diagnosing life-threatening etiologies above
- Distinguish torticollis from other causes of neck stiffness (meningismus)
- Cervical spine films to evaluate for fracture except patients with chronic paroxysmal episodes

DIAGNOSIS TESTS & NTERPRETATION

Lab

No specific tests helpful

Imaging

- CT or MRI of cervical spine if retropharyngeal abscess or tumor suspected
- High-frequency and color Doppler ultrasonography is the test of choice for

congenital muscular torticollis

- Plain films, lateral and AP view for acquired torticollis resulting from trauma
- CT scan of the neck or cervical spine in nontraumatic acquired torticollis

Diagnostic Procedures/Surgery

- Consider administering an anticholinergic medication if drug-induced etiology is suspected
- Consider performing the Tensilon test if myasthenia gravis is a consideration

DIFFERENTIAL DIAGNOSIS

- CNS infections
- Tumors of soft tissue or bone
- Basal ganglia disease
- Abscess of cervical glands
- Myositis of cervical muscles
- Cervical disk lesions
- Myasthenia gravis



TREATMENT

PRE HOSPITAL

- Ensure patent airway
- Cervical spine precautions for any history of trauma
- Support head

INITIAL STABILIZATION/THERAPY

Cervical spine immobilization if fracture is suspected

ED TREATMENT/PROCEDURES

- Drug (e.g., phenothiazine) induced:
 - Diphenhydramine or benztropine
- Acquired:
 - Soft collar and rest
 - Physical therapy
 - Massage
 - Local heat
 - Analgesics

MEDICATION

- Benztropine (for drug-related dystonia): 1–2 mg IM or slow IV, followed by 3–5 days PO
- Clonazepam (2nd-line drug): 0.5 mg PO TID
- Diphenhydramine (for drug-related dystonia): 25–50 mg IV or IM followed by 3–5

days PO q6–8h; (peds: 5 mg/kg/24 h div. q6h IV, IM, or PO)

- Trihexyphenidyl (a 1st-line drug): 2–5 mg/d PO, advance to 30 mg/d
- Valium: 2–5 mg IV, 2–10 mg PO TID (peds: 0.1–0.2 mg/kg/dose IV or PO q6h)
- Botulinum toxin is the 1st-line agent for treating nondrug-induced torticollis, though this is not typically administered in the ED setting



FOLLOW-UP

DISPOSITION

Admission Criteria

- Cervical spine fracture
- Diagnosis in doubt
- Infectious causes
- Toxic appearance
- Inability to maintain adequate fluid intake
- Lack of support system

Issues for Referral

Some patients who fail medical treatment may benefit from surgical treatment, such as accessory nerve ablation or deep brain stimulation

FOLLOW-UP RECOMMENDATIONS

- Outpatient referral to an orthopedist, neurologist, or neurosurgeon who uses botulinum toxin in his or her practice
- Physical therapy and consider chiropractic care
- Return to ED for weakness or worsening symptoms

PEARLS AND PITFALLS

Exclude infectious, inflammatory, traumatic, spinal cord and CNS causes of torticollis.

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CODES

ICD9

- 333.83 Spasmodic torticollis
- 723.5 Torticollis, unspecified
- 754.1 Congenital musculoskeletal deformities of sternocleidomastoid muscle

ICD10

- G24.3 Spasmodic torticollis
- M43.6 Torticollis
- Q68.0 Congenital deformity of sternocleidomastoid muscle

TOXIC EPIDERMAL NECROLYSIS

Andrew K. Chang • Andrew E. Chertoff

BASICS

DESCRIPTION

- One of the most fulminant and potentially fatal of all dermatologic disorders
- Skin sloughing at the dermal–epidermal interface results in the equivalent of a 2nd-degree burn
- Can affect up to 100% of total body surface area (BSA)
- May extend to involve:
 - GI mucosa
 - Respiratory mucosa
 - Genitourinary/renal epithelium
- Mechanism unclear, research indicates immunologic, cytotoxic, and delayed hypersensitivity may be involved as well as genetic susceptibility
- Current classification system proposes 3 categories within the spectrum of Stevens–Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN), distinct from erythema multiforme major and based on percentage of total BSA:
 - SJS: <10% of BSA
 - SJS–TEN overlap syndrome: 10–30% of BSA
 - TEN: >30% of BSA, can affect up to 100% BSA
- More common in older patients and immunocompromised patients
- Mortality rate is about 30%, usually due to secondary sepsis from *Staphylococcus aureus* and *Pseudomonas aeruginosa*
- Synonym(s):
 - Lyell syndrome
 - Fixed drug necrolysis
 - Epidermolysis necroticans combustiformis
 - Epidermolysis bullosa

ETIOLOGY

- Dose-independent drug reactions are the usual cause of TEN:
 - Drugs introduced within previous 1–3 wk are most likely candidates
 - Frequently implicated drugs include:
 - Sulfonamide and PCN antibiotics
 - Anticonvulsants (carbamazepine, phenytoin, phenobarbital, lamotrigine)
 - NSAIDs (oxicams, pyrazoles, sulindac),
 - Allopurinol
 - Corticosteroids

- Antiretroviral drugs
- Other rare causes: Infections, graft-versus-host disease, vaccinations, idiopathic cases (combined < 4%)

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Prodrome: Influenza like, one to several days of fever, malaise, pruritus, cutaneous tenderness, erythema, anorexia, myalgias, arthralgias
- Mucous membranes are commonly affected 1–3 days before skin lesions appear (oropharynx, eyes, genitalia, anus, esophageal and intestinal mucosae, respiratory epithelium) leading to conjunctivitis, esophagitis, pharyngitis, GI bleeding, vomiting, diarrhea, dysuria, cough, dyspnea

Physical-Exam

- Skin:
 - Rash usually begins on face (scalp usually spared) and trunk as erythematous macules, irregular target-like bullae, or diffuse, ill-defined erythema; initially may have pain at sites out of proportion to exam
 - Widespread epidermolysis, denuding of skin surfaces, flaccid bullae, and sheet-like sloughing of epidermis generally progress over 3–4 days but can progress rapidly over hours
 - Nikolsky sign: With lateral pressure, the skin denudes and sloughs from separation of epidermis from dermis
- Mucous membranes involved in > 90% of cases, initial swelling and erythema followed by blistering and ulceration
- Ocular lesions (pseudomembranes, synechiae or adhesions, keratitis, corneal erosions)

ESSENTIAL WORKUP

- Diagnosis is made clinically:
 - Based on history and characteristic skin and mucous membrane lesions

DIAGNOSIS TESTS & INTERPRETATION

Lab

- No confirmatory lab tests exist
- CBC: Normocytic anemia, leukocytosis, lymphopenia/neutropenia, and thrombocytopenia may be present
- ESR may be elevated as a result of systemic inflammation

- Serum chemistry: Electrolyte derangements if extensive fluid losses:
 - Prerenal azotemia
 - Serum bicarbonate < 20 associated with $40 \times$ higher mortality
- LFTs: Elevated transaminases, low total protein and albumin
- UA may show hematuria (urethral–mucosal erosion, glomerulonephritis) or casts (acute tubular necrosis)
- Wound/skin cultures and blood cultures
- Serum granulysin (an implicated cytotoxin)

Imaging

Chest radiograph should be obtained

Diagnostic Procedures/Surgery

- Severity of illness score for TEN (SCORTEN): Each risk factor earns 1 point, a higher score means a poorer prognosis:
 - Age > 40 yr
 - Malignancy
 - Tachycardia $> 120/\text{min}$
 - Initial percentage of epidermal detachment $> 10\%$
 - BUN > 27 mg/dL
 - Serum glucose level > 252 mg/dL
 - Serum bicarbonate level < 20 mEq/L
- Biopsy may be performed by consulting dermatologist to rule out autoimmune bullous diseases, staphylococcal scalded skin syndrome, and other diagnoses:
 - Results not immediately available to ED physician

DIFFERENTIAL DIAGNOSIS

- Stevens-Johnson syndrome (SJS)
- Erythema multiforme major (EMM):
 - Differentiation of SJS/TEN from EMM:
 - Immunopathologically distinct
 - *Etiology*: SJS/TEN is mainly drug-induced, mechanism uncertain; EMM both infection and drug-induced, mechanism type IV hypersensitivity
 - *Lesions*:
 - TEN: Widely distributed, mainly on the trunk and face, nonspecific, target-like lesions that often are confluent and too numerous to count, then desquamation
 - EMM: Limited in number, symmetric and acral distribution, typical target type (at least 3 concentric rings) with or without blisters
 - *Prognosis*: EMM is usually benign; recurrence of disease is common (30%)
- Staphylococcal scalded skin syndrome (SSSS):
 - Differentiation of TEN from SSSS:
 - *Age*: TEN: Primarily adults (but may occur in children); SSSS: Primarily

affects children

– *Etiology:*

- TEN most often represents an idiosyncratic, drug-induced, dose-independent reaction and hence does not require treatment with antibiotics
- SSSS results from infection and requires antibiotics

– *Pain:* TEN, painful; SSSS, painless

– *Mucous membranes:* Involved with TEN; spared with SSSS

– *Skin cleavage:* Dermal–epidermal junction in TEN; intraepidermally in SSSS (both can produce a positive Nikolsky sign)

- Autoimmune bullous diseases (pemphigus vulgaris, bullous pemphigoid)
- Scarlet fever
- Toxic shock syndrome
- Chemical or thermal scalds
- Hypersensitivity vasculitis
- Kawasaki syndrome



TREATMENT

PRE HOSPITAL

- Transport to facility with burn center
- Care during transport should be gentle to avoid skin trauma
- IV catheter should be avoided for short transport if hemodynamically stable (more sterile conditions in ED)
- Avoid using adhesive materials

INITIAL STABILIZATION/THERAPY

- If intubation or nasogastric tube is required, gentle technique must be used to minimize mucosal damage
- Meticulous sterile technique
- Peripheral IV line is preferred over central line to decrease risk of sepsis
- Cardiac monitor, pulse oximeter, nasogastric tube, Foley catheter

ED TREATMENT/PROCEDURES

- Identify and stop any causative medication
- Aggressive fluid resuscitation and electrolyte management as in burn care (Parkland formula):
 - Urine output should target a rate of 0.5–1 mL/kg/hr.
- Warming measures and frequent core temperature evaluation are important
- If available, cover with biologic dressings (e.g., Biobrane):
 - Reduces pain, decreases caloric and evaporative losses, and facilitates healing

- Antibiotic drops for eyes
- Petroleum jelly application to lips
- Prevention of peptic stress ulcers
- Topical antibiotics, including silver nitrate, are unproven but may be applied with the exception of silver sulfadiazine (sulfonamide derivative).
- Timely admission to burn unit/ICU
- Ophthalmology consultation is required for eye involvement (evaluation and removal of pseudomembranes and adhesions)

MEDICATION

There are no established treatment regimens; however, there are several suggested guidelines:

- Pain should be controlled with IV opiates
- Antibiotics should be used when documented signs of sepsis are present or for sudden deterioration in the clinical setting; coverage should include gram-positive, gram-negative (including *P. aeruginosa*), and aerobic organisms
- Antihistamines can be used for pruritus
- Anticoagulation should be considered while patients are nonambulatory for prevention of thromboembolic events
- Systemic corticosteroids continue to be controversial:
 - Retrospective studies show no benefit and suggest greater risk of death from infection
- IVIG should be started 48–72 hr after bulla formation but can be helpful after 72 hr
- The following experimental therapies are under investigation:
 - Plasmapheresis
 - Cyclosporine
 - Cyclophosphamide
 - N-acetylcysteine
 - Anti-TNF- α antibodies (i.e., Infliximab) but Thalidomide contraindicated (harm shown)



FOLLOW-UP

DISPOSITION

Admission Criteria

All patients with suspected TEN should be admitted to a burn unit (if burn unit is unavailable and transfer is not possible, then admit to ICU)

Issues for Referral

- Transfer to facility with burn unit has been shown to improve patient outcome
- Dermatology should be called to help confirm the diagnosis

- Ophthalmology should be called to evaluate and prevent corneal ulcerations and adhesions
- Surgery or plastic surgery should evaluate the need for wound débridement.
- Respiratory therapy should initiate pulmonary toilet in the setting of pulmonary mucosal sloughing.

PEARLS AND PITFALLS

- Burn units and ICUs offer the best management settings
- Remember to educate patients on medications (including combinations, medications, and structurally similar medications)
- Aggressive fluid hydration is essential

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See Also (Topic, Algorithm, Electronic Media Element)

Burns



CODES

ICD9

- 695.14 Stevens-Johnson syndrome-toxic epidermal necrolysis overlap syndrome
- 695.15 Toxic epidermal necrolysis

ICD10

- L51.2 Toxic epidermal necrolysis [Lyell]
- L51.3 Stevens-Johnson synd-tox epdrml necrolysis overlap syndrome

TOXIC SHOCK SYNDROME

Michelle J. Sergel • Kristopher Wnek



BASICS

DESCRIPTION

- Toxic shock syndrome (TSS) is a severe, acute life-threatening illness
- Etiologic organisms:
 - *Staphylococcus aureus*, more common (TSS)
 - Group A streptococcus or GAS, less common (Streptococcal TSS or STSS)
- *S. aureus* produce structurally similar toxins:
 - Toxic shock syndrome toxin (TSST-1)
 - Enterotoxin B (SEB)
 - Enterotoxin C (SEC)
- GAS pyrogenic exotoxins:
 - Exotoxin A (SPEA)
 - Exotoxin B (SPEB)
- Exotoxins act as superantigens causing overwhelming immune response:
 - Massive cytokine production
 - Induce fever directly at the hypothalamus or indirectly via interleukin-1 (IL-1) and tumor necrosis factor (TNF) production
 - Enhance delayed hypersensitivity
 - Suppress neutrophil migration and immunoglobulin
 - Enhance host susceptibility to endotoxins
- Massive vasodilation occurs
 - Serum protein and fluid shifts leading to hypotension

ETIOLOGY

- Initial cases described in young healthy menstruating females due to highly absorbent tampons
 - Changes made in tampon composition to decrease incidence
- Approximately one-half of reported TSS cases are nonmenstrual:
 - Surgical wounds
 - Postpartum wound infections
 - Mastitis
 - Septorhinoplasty
 - Sinusitis
 - Osteomyelitis
 - Arthritis
 - Burns
 - Nasal packing (nasal tampons)

- Cutaneous and subcutaneous lesions
- Nonmenstrual cases predominantly due to SEB and SEC producing *S. aureus*
- 30–50% of healthy adults and children carry *S. aureus* in the nasal vestibule, vagina, rectum and/or on the skin
- GAS infections often begin within 24–72 hr at the site of minor trauma, often without a visible in skin
- Despite increased incidence of Methicillin-resistant *S. aureus* (MRSA) infections, a recent study reported MRSA only accounting for 7% of cases

DIAGNOSIS

SIGNS AND SYMPTOMS

TSS Criteria for Diagnosis

- CDC case definition:
 - Fever $> 38.9^{\circ}\text{C}$ (102°F)
 - Hypotension (systolic BP < 90 mm Hg) or shock
 - Diffuse, blanching nonpruritic macular erythroderma rash
 - Subsequent desquamation 1–2 wk after the onset of illness (particularly involving palms and soles)
 - Multisystem involvement—*at least 3* of the following should be present:
 - GI: Profuse diarrhea or vomiting at onset of illness
 - Musculoskeletal: Severe myalgias or greater than a 2-fold increase in creatine phosphokinase (CPK)
 - Mucosal inflammation: Conjunctival, vaginal, or pharyngeal hyperemia
 - Renal: Increase in BUN or creatinine > 2 times normal upper limit or sterile pyuria without evidence of infection
 - Hepatic: Total bilirubin or transaminases > 2 times normal upper limit
 - Hematologic: Thrombocytopenia $< 100,000/\text{mm}^3$
 - CNS: Disorientation, confusion, or hallucinations
 - Negative results on the following tests, if obtained: Throat, or CSF cultures, rise in titer to Rocky Mountain spotted fever (RMSF), leptospirosis, or rubeola

Streptococcal TSS (STSS) Criteria for Diagnosis

- CDC case definition:
 - Isolation of GAS from a normally sterile site
 - Hypotension
 - Plus 2 or more of the following:
 - Renal impairment (creatinine > 2)

- Coagulopathy
- Liver involvement (> 2 times the upper limit of normal for transaminases or bilirubin)
- ARDS
- Erythematous macular rash, may desquamate
- Soft tissue necrosis

Other

- Tachycardia frequently present
- Can rapidly progress to multisystem dysfunction (ARDS or DIC)
- STSS often presents with diffuse or localized pain—abrupt in onset and severe
- Pain precedes physical findings
- Nearly 80% of patients with STSS have clinical signs of soft tissue infection

ESSENTIAL WORKUP

- Clinical diagnosis using diagnostic criteria in the absence of other attributable illness
- Thorough history and physical exam

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC:
 - Leukocytosis or leukopenia, marked bandemia common
- Electrolytes, BUN, creatinine, glucose:
 - Elevated BUN and creatinine common
- Calcium, magnesium:
 - Hypocalcemia/hypomagnesemia often present
- Urinalysis:
 - Normal or sterile pyuria without evidence of infection
- CPK:
 - 2-fold increase
- Hepatic function:
 - Elevated total bilirubin, AST, ALT
- Prothrombin time (PT), partial thromboplastin time (PTT), platelets:
 - Thrombocytopenia < 100,000 platelets/mm³
- Culture the site of injury/infection if possible
- Blood, urine, throat, and CSF cultures as indicated:
 - The case definition does not require a positive blood culture for *S. aureus*, but does for *Streptococcus* organisms.
- Serology for RMSF, rubeola, and leptospirosis
- Hepatitis B surface antigen

Imaging

- Chest x-ray – to rule out other sources of systemic illness
- Consider x-ray or CT scan if localized pain is concerning for abscess or necrotizing infection

DIFFERENTIAL DIAGNOSIS

- Staphylococcal scalded skin syndrome:
 - In children <5 yr of age
 - Initial macular rash followed by the formation of ill-defined bullae that can be rubbed off revealing a shiny, moist epidermis (positive Nikolsky sign)
- Scarlet fever:
 - Preceding streptococcal pharyngitis
 - Rash begins on the upper chest, neck, and back spreading to the remainder of the trunk, sparing the palms and soles
 - Hypotension absent
- Kawasaki disease:
 - Fever, conjunctival hyperemia, and erythema of the mucous membranes
 - Not associated with renal failure, hypotension, or thrombocytopenia
- Stevens–Johnson syndrome:
 - Severe multisystem involvement
 - Mucosal involvement of the mouth, conjunctivae, vagina, anus, and urethral meatus
- Leptospirosis:
 - Transmitted through contact with infected animals
 - Fever, headache, severe myalgias, and conjunctival suffusion
 - Truncal rash that only desquamates in children
- RMSF:
 - Rash is pink and macular, beginning on the wrists, palms, ankles, and soles spreading to the trunk and face
 - Petechiae appear after 4 days
- Meningococemia:
 - Meningismus present
 - Rash is petechial



TREATMENT

PRE HOSPITAL

- ABCs
- IV access
- IV fluids for hypotension

INITIAL STABILIZATION

- Again, ABCs
- Aggressive management of circulatory shock
 - IV fluids
 - Pressors

ED TREATMENT/PROCEDURES

Hypotension

- Aggressive fluid replacement
 - The 1st 24 hr may require 4–20 L of crystalloid and/or fresh frozen plasma (colloid)
 - *Caution:* Large amounts of IV fluids and pressors used to treat refractory hypotension can result in rapid onset pulmonary edema
 - Pressors (dopamine/norepinephrine) if fluid correction fails to restore normal arterial pressure

Infection Management

- Search for and treat the focus of infection
- Remove the source of infection (e.g., tampon, nasal or wound packing)
- Early surgical/gynecologic consultation if drainage or débridement of infectious sites necessary
- Antibiotics
 - Recommended to reduce recurrence, but have not been shown to alter the course of the initial infection
 - Clindamycin and linezolid are potent suppressers of bacterial toxin synthesis
 - Clindamycin or linezolid + vancomycin for TSS
 - Linezolid + vancomycin for TSS with extensive infection
 - If TSS due to known methicillin-susceptible *S. aureus* then clindamycin + oxacillin or nafcillin
 - Clindamycin + imipenem or meropenem or ticarcillin–clavulanate or piperacillin–tazobactam for STSS
- IV immunoglobulin (IVIG) treatment:
 - May be efficacious in streptococcal toxic shock, but no controlled trials have proven efficacy in staphylococcal TSS.
 - May initiate if no response to fluids, pressors, and antibiotics in patients with pulmonary edema and hypotension

MEDICATION

- Clindamycin: 600–900 mg (peds: 20–40 mg/kg/24 h) IV q6–8h
- Dopamine: 2–20 µg/kg/min IV, titrate to BP
- Linezolid: 600 mg (peds: 10 mg/kg/12 h) IV q12h
- Meropenem: 1 g IV q8h

- Nafcillin: 1.5 g (peds: 100 mg/kg/24 h) IV q4h
- Norepinephrine: 0.01–3 mcg/kg/min IV, titrate to BP
- Oxacillin: 1–2 g (peds: 50–100 mg/kg/24 h) IV q4h
- Piperacillin–tazobactam: 4.5 g q6h
- Ticarcillin–clavulanate: 3.1 g q4h
- Vancomycin: 30 mg/kg QD IV div. in 2 doses (peds: 40 mg/kg QD IV div. in 4 doses)
- Staphylococcal TSS: IVIG, 400 mg/kg over several hours
- Streptococcal TSS: IVIG 1 g/kg on day 1 then 0.5 g/kg on days 2 and 3



FOLLOW-UP

DISPOSITION

Admission Criteria

- Cases necessitate admission
- ICU admission for critically ill or those in shock

Discharge Criteria

None

Issues for Referral

Early surgical/gynecologic consultation if drainage or débridement is needed

FOLLOW-UP RECOMMENDATIONS

- Patients who are bacteremic are treated for a minimum of 14 days:
 - Depending on the clinical course
 - Continue treatment for 14 days from the last positive culture.
- Screening for *S. aureus* nasal carriage in patient with *S. aureus* TSS and eradication of the carrier state with mupirocin

PEARLS AND PITFALLS

- Consider the diagnoses of staphylococcal TSS and GAS TSS
- Ensure adequate supportive care for hypotension in TSS
- Prompt and aggressive exploration and débridement of suspected deep-seated infection
- Empiric broad-spectrum antibiotics including clindamycin or linezolid is recommended

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See Also (Topic, Algorithm, Electronic Media Element)

- Streptococcal Infections
- Kawasaki Disease
- Meningococemia
- Leptospirosis



CODES

ICD9

040.82 Toxic shock syndrome

ICD10

A48.3 Toxic shock syndrome

TOXOPLASMOSIS

Roger M. Barkin

BASICS

DESCRIPTION

- *Toxoplasma gondii*—intracellular protozoan parasite:
 - 3 forms:
 - Tachyzoite: Asexual invasive form
 - Tissue cyst: Persists in tissues of infected hosts during chronic phase
 - Oocyst: Contains sporozoites and produced during sexual cycle in cat intestine
- Transmission:
 - Ingesting tissue cysts or oocysts:
 - Ingesting undercooked meat
 - Vegetables contaminated with oocysts
 - Contact with cat feces, through cat or soil
 - Transplacental
 - Blood product
 - Organ transplantation

ETIOLOGY

- 70% of adults seropositive
- Asymptomatic in most immunocompetent patients
- Worldwide; cats are the common host
- Incubation is 7 days with a range of 4–21 days

DIAGNOSIS

SIGNS AND SYMPTOMS

4 types of infection

Immunocompromised Host

- CNS:
 - Subacute presentation (90%)
 - Encephalitis
 - Headache
 - Altered mental status
 - Fever
 - Seizures
 - Cranial nerve palsies
 - Spinal cord lesions

- Cerebellar signs
- Meningitis-like symptoms
- Movement disorders
- Neuropsychological symptoms:
 - Psychosis
 - Paranoia
 - Dementia
 - Anxiety
 - Agitation
- Pulmonary:
 - Pneumonitis
 - Prolonged febrile illness
 - Nonproductive cough
 - Dyspnea

Immunocompetent Host

- 90% are asymptomatic
- Lymphadenopathy, usually cervical
- Fever
- Malaise
- Mononucleosis-like syndrome with macular rash and hepatosplenomegaly
- Headache
- Sore throat
- Night sweats
- Maculopapular rash
- Urticaria
- Usually, self-limited process; resolves in 2–12 mo
- Rarely presents with pneumonitis or encephalitis

Ocular Toxoplasmosis

- Blurred vision
- Scotoma
- Pain
- Photophobia
- Retina:
 - Small clusters of yellow-white cotton-like patches
 - Chorioretinitis; affects 85% of young adults with untreated congenital infection

Congenital Toxoplasmosis

- Results from an asymptomatic acute infection during pregnancy
- 1st trimester:
 - Spontaneous abortion
 - Stillbirth
 - Severe disease up to 25% of the time
- 2nd or 3rd trimester:

- 50–60% chance of acquiring congenital toxoplasmosis
- 2% fatal
- Most asymptomatic at birth
- Delayed onset. 70–90% asymptomatic at birth:
 - CNS disease
 - Ocular disease (blindness months to years later)
 - Lymphadenopathy
 - Hepatosplenomegaly
 - At birth, may have maculopapular rash, lymphadenopathy, hepatomegaly, splenomegaly, jaundice, thrombocytopenia

ESSENTIAL WORKUP

- Diagnose via:
 - Isolation of organism:
 - Blood
 - CSF for encephalitis
 - Bronchoalveolar lavage for pneumonitis
 - Amniotic fluid
 - Aqueous humor
 - Detection of tachyzoites in tissues or body fluids
 - Demonstrating characteristic lymph node pathology
- Thorough ocular exam:
 - Retinal exam
 - Visual acuity

DIAGNOSIS TESTS & INTERPRETATION

Lab

- LDH > 600/UL associated with toxoplasmosis
- CBC:
 - Atypical lymphocytes
- ABG/pulse oximetry for pulmonary symptoms
- IgG antibodies:
 - High number of false-positive and false-negative results
 - Common tests:
 - Sabin–Feldman dye test
 - Indirect fluorescent antibody
 - Agglutination
 - Enzyme-linked immunosorbent assay test
- Immunoglobulin M (IgM) antibodies:
 - Absence excludes diagnosis in immunocompetent host
 - Reference labs may be helpful, such as Remington (650-853-4828 Toxoplasma Serology Laboratory) (www.pamf.org/serology)

- Diagnoses acute infection
- Appear in 5 days
- Disappear in weeks to months
- Neonatal testing differentiates from maternal infection

Imaging

- Chest radiograph for pulmonary symptoms:
 - Pneumonitis associated with reticulonodular pattern
- CT head with contrast:
 - Multiple bilateral hypodense ring-enhancing lesions
- MRI brain:
 - High signal abnormalities on T2-weighted images
- Serial fetal ultrasonography can be useful in exploring congenital infection of the CNS or other signs.

Diagnostic Procedures/Surgery

Brain biopsy for encephalitis—definitive diagnosis

DIFFERENTIAL DIAGNOSIS

- Cryptococcal meningitis
- CNS lymphoma
- *Pneumocystis carinii* pneumonia
- Cytomegalovirus retinitis
- Mycobacterial infection



TREATMENT

INITIAL STABILIZATION/THERAPY

- Treat seizures in standard fashion with diazepam and phenytoin.
- Initiate oxygen if hypoxia due to pneumonitis.

ED TREATMENT/PROCEDURES

Immunocompetent

Toxoplasmic lymphadenitis:

- No antibiotics unless symptoms severe and persistent
- Treat symptomatic patients with pyrimethamine and folinic acid plus sulfadiazine or clindamycin for 3–4 wk
- Clindamycin may be a useful alternative to sulfadiazine because of the side effects of the latter and in those who are hypersensitive to sulfa
- Pyrimethamine and sulfadiazine (Eon Labs 800-526-0225) is available as a combination drug.

- Corticosteroids may be useful for ocular complications and CNS disease.
- Reassess to determine if longer therapy needed.

Immunocompromised

- Confirmed acute infection by serology/symptoms:
 - Treat with pyrimethamine and folinic acid + sulfadiazine or clindamycin for 4–6 wk after resolution of symptoms.
 - Alternative medications:
 - Trimethoprim–sulfamethoxazole
 - Pyrimethamine and folinic acid + dapsone
- CNS symptoms + a lesion on CT or MRI:
 - Treat empirically with pyrimethamine and folinic acid + sulfadiazine or clindamycin.
 - Brain biopsy or CSF to confirm diagnosis
 - Administer anticonvulsants only if confirmed prior seizures:
 - Poorer outcome for patients on anticonvulsants
- Chronic asymptomatic infection:
 - No therapy required
 - Prophylaxis options for toxoplasmosis in AIDS and immunosuppressed patients:
 - Trimethoprim–sulfamethoxazole; lifelong prophylaxis should be considered in HIV patients after consultation.
 - Pyrimethamine (75 mg/wk) and dapsone (200 mg/wk) and leucovorin 10–25 mg with each dose pyrimethamine

Ocular

- Treat with pyrimethamine and sulfadiazine for 1 mo.
- May add clindamycin
- Administer systemic steroids with macular or optic nerve involvement.

Acute Acquired Infection in Pregnancy

- Initially treat with spiramycin pending confirmatory tests and consultation (FDA, Division of Special Pathogens and Transplant Drug Products 301-796-1600 or CDC at 404-718-4745).
- After the infection is documented, initiate treatment after consultation:
 - Spiramycin in the 1st 17 wk
 - Pyrimethamine and sulfadiazine after 17 wk
- Spiramycin may reduce congenital transmission but does not treat fetus if infection is in placenta; maternal therapy may decrease severity of congenital disease.
- Treat congenital infection with sulfadiazine, pyrimethamine, and folinic acid for 12 mo.
- Prevention of exposure in seronegative pregnant women is important when

contacting cats or their excrement.

MEDICATION

- Clindamycin:
 - 600 mg (peds: 20–40 mg/kg/24 h) IV q6h
 - 300 mg (peds: 8–20 mg/kg/24 h) PO q6h
 - Useful if patient hypersensitive to sulfa
- Dapsone: 50 mg PO per day or 200 mg PO per week (child > 1 mo: 2 mg/kg PO per day)
- Folinic acid: 5–25 mg PO daily in conjunction with pyrimethamine therapy
- Pyrimethamine: 100 mg BID on 1st day loading dose, then 25–50 mg PO per day
- Spiramycin: FDA authorization required
- Sulfadiazine: 500 mg–2 g (peds: 100–200 mg/kg/24 h div. BID) PO q6h
- Trimethoprim–sulfamethoxazole: 5 mg/kg of trimethoprim component IV or PO q12h



FOLLOW-UP

DISPOSITION

Admission Criteria

- Acute infection with severe systemic symptoms
- Immunocompromised patients with:
 - Toxoplasmosis encephalitis
 - Pneumonitis
 - Sepsis

Discharge Criteria

- Immunocompetent patients with:
 - Mild symptoms
 - Ocular
- Maternal/congenital infection with mild symptoms

Issues for Referral

Infectious disease consultant

ADDITIONAL READING

- American Academy of Pediatrics. *Red Book 2012 Report of the Committee on Infectious Diseases*. Elk Grove, IL: AAP; 2012.
- Centers for Disease Control and Prevention. Guidelines for prevention and treatment of opportunistic infections in HIV infected adults and adolescents.

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- Kaplan JE, Benson C, Holmes KK, et al. Guidelines for prevention and treatment of opportunistic infections in HIV-infected adults and adolescents: Recommendations from CDC, the National Institutes of Health, and the HIV Medicine Association of the Infectious Diseases Society of America. *MMWR Recomm Rep*. 2009;58:1–207.
- Sciammarella J. Toxoplasmosis. Available at <http://www.emedicine.com/emerg/topic601.htm>. Accessed on July 6, 2002.



CODES

ICD9

- 130.0 Meningoencephalitis due to toxoplasmosis
- 130.4 Pneumonitis due to toxoplasmosis
- 130.9 Toxoplasmosis, unspecified

ICD10

- B58.2 *Toxoplasma* meningoencephalitis
- B58.3 Pulmonary toxoplasmosis
- B58.9 Toxoplasmosis, unspecified

TRANSFUSION COMPLICATIONS

Philip Shayne



BASICS

EPIDEMIOLOGY

- Of 39 million hospital discharges in US, 5.8% (2.3 million) were associated with blood transfusions (2004).
- In 2011 there were 30 deaths in US fully attributable to transfusion complications.
- Some type of transfusion reaction occurs in 2% of units transfused within 24 hr of use.
- Noninfectious complications:
 - Febrile nonhemolytic reaction: RBCs 1 in 500 transfusions, platelets 1 in 900
 - Allergic reaction (nonanaphylactic): 1 in 3 to 1 in 300
 - Anaphylaxis: 1 in 20,000 to 1 in 50,000
 - Acute hemolytic reaction: 1 in 38,000 to 1 in 70,000
 - Delayed hemolytic reaction: 1 in 4,000 to 1 in 11,000
 - Transfusion-associated circulatory overload (TACO): 1 in 100, but as high as 10% in susceptible populations
 - Alloimmunization: 1 in 10 to 1 in 100
 - Graft-versus-host disease: 1 in 400,000; rare but has >90% mortality.
 - Transfusion-related lung injury (TRALI): 1 in 5,000 to 1 in 190,000; represents 13% of reported transfusion-related deaths
 - Iron overload: Unknown incidence, depends on volume of blood, often occurs after >100 RBC units
 - Hypocalcemia: Unknown incidence
 - Hyperkalemia: Unknown incidence
- Infectious complications:
 - Bacterial contamination: RBCs 1 in 65,000 to 1 in 500,000; platelets 1 in 1,000 to 1 in 10,000:
 - Most common bacterial agents: *Yersinia enterocolitica*, *Pseudomonas spp*, *Serratia spp*.
 - Leading cause of mortality among infectious complications; 17–22% of all cases
 - Hepatitis C: 1 in 1.6 million
 - Hepatitis B: 1 in 100,000 to 1 in 400,000
 - HTLV I and II: 1 in 500,000 to 1 in 3 million
 - HIV: 1 in 1.4 million to 1 in 4.7 million
 - HAV: 1 in 1,000,000
 - B19 parvovirus: 1 in 40,000; post-transfusion anemia rare with scattered case reports

- Parasites: *Babesia* and malaria: <1 in 1 million
- Parasites: *Trypanosoma cruzi*: 1 in 42,000
- Case reports of Epstein–Barr virus, Lyme disease, brucellosis, human herpesvirus, Creutzfeldt–Jakob disease

Acute Intravascular Hemolytic Transfusion Reaction

- Mortality and morbidity correlate with amount of incompatible blood transfused (symptoms can occur with exposure to as little as 5–20 mL)
- Occurs immediately from:
 - ABO incompatibility
 - Blood type identification error
 - Incompatible transfused cells immediately destroyed by antibodies
- Intravascular hemolysis causing activation of coagulation system, leading to inflammation, shock, and DIC
- Mediators (cytokines) released during inflammatory response
- Renal failure:
 - Cytokines cause local release of endothelin in kidney, causing vasoconstriction.
 - Leads to parenchymal ischemia and acute renal failure
- Respiratory failure owing to pulmonary edema/adult ARDS:
 - Free hemoglobin (Hb) causes vasoconstriction in pulmonary vasculature.

Other Transfusion-related Complications

- Hemolysis because of Rh incompatibility:
 - Mild, self-limiting
 - 1:200 U transfused
- Febrile nonhemolytic transfusion reaction:
 - Most common transfusion reaction, diagnosis of exclusion.
 - Temperature increases at least 1°C with chills within 6 hr
 - Antigen–antibody reaction to transfused blood components (WBCs, platelets, plasma)
 - Usually mild
 - Occurs more often with multiparous women or multiple transfusions
 - Recurs in 15% of patients
 - Acetaminophen may be used prophylactically; its use as premedication is controversial, though not harmful.
- Allergic transfusion reaction:
 - Occurs in 1% of transfusions
 - Usually seen with immunoglobulin A (IgA)–deficient patients
 - Urticaria alone is not a reason to stop transfusion.
 - Antihistamine may be used as therapy or prophylactically.
- Premedicating with acetaminophen and diphenhydramine found to have no effect

on incidence of transfusion reaction compared with placebo in some trials.

Delayed Reactions

- Infection:
 - HIV, hepatitis B, hepatitis C
 - Blood screened for viruses
 - Blood treated to inactivate viruses
 - Blood donors with recent history of travel or poor health are deferred from donating.
- Delayed extravascular hemolytic reaction:
 - Occurs 7–10 days after transfusion
 - Antigen–antibody reaction that develops after transfusion
 - Coombs test positive
 - Usually asymptomatic
 - Blood bank analysis detects antibody
- Electrolyte imbalance:
 - Hypocalcemia: Calcium binds to citrate
 - Hyper/hypokalemia: Citrate metabolized to bicarbonate, which drives potassium intracellularly; prolonged storage of blood may cause hemolysis and hyperkalemia
- Graft-versus-host disease:
 - Fatal in > 90%
 - Immunologically competent lymphocytes transfused into immunocompetent host
 - Host unable to destroy new WBCs
 - Donor WBCs recognize host as foreign and attack host's tissues.
- Anaphylactic reaction:
 - Can occur with < 10 mL of exposure
 - Generalized flushing, urticaria, laryngeal edema, bronchospasm, profound hypotension, shock, or cardiac arrest.
 - Treat with subcutaneous epinephrine, supportive hemodynamic and respiratory care.
- TRALI:
 - Symptoms typically begin with 6 hr of transfusion.
 - Acute onset of respiratory distress, bilateral pulmonary edema, fever, tachycardia, hypotension, with normal cardiac function
 - 3rd most common cause of fatal transfusion
 - Difficult to distinguish from ARDS and TACO; often misdiagnosed and underreported
 - Provide supportive care.
 - Disease is typically self-limited within 96 hr.
 - Mortality is 5–10%.
 - Diuretics contraindicated

Pediatric Considerations

Blood can be transfused through 22G peripheral catheter under pressure (but < 300 mm Hg) with minimal hemolysis.

DIAGNOSIS

SIGNS AND SYMPTOMS

- **General:**
 - Fevers
 - Chills
 - Burning at infusion site
 - Urticaria/pruritus/skin erythema
- **Pulmonary:**
 - Dyspnea
 - Bronchospasm
 - Respiratory distress/failure
- **Cardiovascular:**
 - Tachycardia
 - Hypotension
 - Substernal chest pain/tightness
- **GI:**
 - Nausea
 - Vomiting
 - Diarrhea
- **Hematologic:**
 - Bleeding
 - Hemoglobinuria
 - Oozing from surgical wounds
 - Jaundice
 - DIC
- **Miscellaneous:**
 - Low back pain
 - Renal failure (oliguria/anuria)
 - Classic triad of fever, flank pain, and red-brown urine of acute hemolytic reactions is rarely seen.

ESSENTIAL WORKUP

- Recognize clinical findings of transfusion reaction.
- Recheck identifying information of blood and patient compatibility.
- Recognize evidence of hypotension/shock, severe respiratory distress, sepsis, fever, and urticaria; intervene appropriately.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC
- Electrolytes, BUN, creatinine, glucose:
 - For electrolyte abnormalities
- PT, PTT
- Serum calcium
- Fibrinogen, fibrin degradation products
- Bilirubin (direct/indirect)
- Coombs test
- Hemoglobinemia:
 - Pink or red supernatant of plasma or serum indicates hemolysis.
- Urinalysis:
 - Hemoglobinuria: Dipstick-positive blood without RBCs on micro
- Lab findings indicating hemolysis:
 - Thrombocytopenia ($< 100,000$)
 - Fibrinogenopenia (< 150 mg/L)
 - Fibrin degradation products
 - Prolonged activated PTT (aPTT)
 - Spherocytosis
- Lab findings indicating hemolysis due to Rh incompatibility:
 - Positive Coombs test
 - Elevated indirect bilirubin
 - Post-transfusion Hb/hematocrit not showing expected rise

Imaging

Chest radiograph: Diffuse patchy infiltrates without cardiomegaly if TRALI.

Diagnostic Procedures/Surgery

ECG for dysrhythmia, sign of electrolyte abnormality

DIFFERENTIAL DIAGNOSIS

- Sepsis
- Anaphylaxis/allergic reaction to medication



TREATMENT

PRE HOSPITAL

Routine stabilization

INITIAL STABILIZATION/THERAPY

- Immediately stop infusion:
 - Severity of reaction proportional to amount of blood transfused

- ABCs
- Supplemental oxygen—intubation and mechanical ventilation if needed
- Recheck blood-identifying information—patient’s bracelet, blood labels, call blood bank

ED TREATMENT/PROCEDURES

- Hypotension:
 - 0.9% normal saline (NS) hydration with 2 large-bore IVs
 - Avoid Ringer lactate or solutions containing dextrose.
 - Trendelenburg position
 - Dopamine
- Prevention of renal failure:
 - Maintain urine output of 1 mL/kg/h
 - Adequate hydration
 - Furosemide or mannitol if oliguric
 - Dopamine infusion at 2 µg/kg/min
- Febrile reactions:
 - Antipyretics (acetaminophen/nonsteroidal anti-inflammatory drugs [NSAIDs])
 - Antihistamine (diphenhydramine + ranitidine) IV
 - Steroids (methylprednisolone)
- Allergic reactions:
 - Antihistamine (diphenhydramine + ranitidine) IV
 - Epinephrine for respiratory symptoms
 - Steroids (methylprednisolone)
- Redraw blood sample for repeat ABO/Rh typing, direct antiglobulin testing.
- Foley catheter to monitor urine output
- Replenish calcium if hypocalcemia develops.
- Treat DIC

MEDICATION

- Calcium gluconate: 10 mL of 10% (peds: 100 mg/kg/dose) solution slow IV push
- Dopamine: 2–20 µg/kg/min IV
- Diphenhydramine: 25–50 mg (peds: 1.25 mg/kg) IV or PO
- Ranitidine: 50 mg IV (peds: 1–2 mg/kg/dose max. 50 mg)
- Epinephrine (1 in 1,000): 0.3–0.5 mL (peds: 0.01 mL/kg) SC
- Methylprednisolone: 125 mg (peds: 2 mg/kg) IV



FOLLOW-UP

DISPOSITION

Admission Criteria

- Acute hemolytic transfusion reaction, pulmonary complications, anaphylaxis, sepsis:
 - Require ICU monitoring
- Delayed hemolytic transfusion reactions for evaluation/treatment
- Electrolyte abnormalities requiring cardiac monitoring

Discharge Criteria

Uncomplicated febrile or allergic reaction

PEARLS AND PITFALLS

- Blood transfusion is substantially over utilized and has significant associated risk, such as transfusion reactions, transmission of pathogens, and immune suppression.
- Maintaining body temperature during massive transfusion is crucial to correcting coagulopathy.
- Failure to properly compare patient identification to labeling on blood or failure to wait for fully cross-matched blood carries significant risks.
- Suspect acute intravascular hemolysis if patient develops hypotension, dark urine, or oozing from IV or other puncture sites.

ADDITIONAL READING

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- Goodnough LT, Levy JH, Murphy MF. Concepts of blood transfusion in adults. *Lancet*. 2013;381:1845–1854.
- Morton J, Anastassopoulos KP, Patel ST, et. al. Frequency and outcomes of blood products transfusion across procedures and clinical conditions warranting inpatient care: An analysis of the 2004 healthcare cost and utilization project nationwide inpatient sample database. *Am J Med Qual*. 2010;25:289–296.
- Spahn DR, Goodnough LT. Alternatives to blood transfusion. *Lancet*. 2013;381:1855–1865.
- Squires JE. Risks of transfusion. *South Med J*. 2011;104(11):762–769.



See Also (Topic, Algorithm, Electronic Media Element)

- Allergic Reaction
- Anaphylaxis
- Disseminated Intravascular Coagulation
- Sepsis



CODES

ICD9

- 780.66 Febrile nonhemolytic transfusion reaction
- 999.80 Transfusion reaction, unspecified
- 999.84 Acute hemolytic transfusion reaction, incompatibility unspecified

ICD10

- R50.84 Febrile nonhemolytic transfusion reaction
- T80.910A Acute hemolytic transfusion reaction, unspecified incompatibility, initial encounter
- T80.92XA Unspecified transfusion reaction, initial encounter

TRANSIENT GLOBAL AMNESIA

Kama Guluma

BASICS

DESCRIPTION

- Transient global amnesia (TGA) has the following features:
 - Episode of amnesia with abrupt onset
 - No focal neurologic signs or symptoms
 - Temporary, severe, anterograde amnesia:
 - Acute inability to form new memories
 - Permanent memory gap after the episode
 - Temporary short-range retrograde amnesia:
 - More recent memories at more risk
 - Previously encoded memories unavailable only temporarily
 - Gradually improves until only remaining memory deficit is the gap induced by the anterograde amnesia
- Incidence between 3 and 8 per 100,000 people:
 - 75% occur in people of 50–70 yr old
 - TGA rare < 40 yr
- Most attacks last between 1 and 8 hr (range 15 min–7 days)

ETIOLOGY

- Multimodal MRI, SPECT, and PET have shown some abnormalities of regional blood flow in selectively vulnerable hippocampal structures
- The exact etiology of TGA is unknown; speculation is controversial
- Speculated causes:
 - Vasoconstriction due to hyperventilation:
 - Psychogenic hyperventilation in setting of age-related cerebrovascular autoregulatory dysfunction
 - Hippocampal venous congestion with Valsalva:
 - Ultrasonography has suggested internal jugular vein incompetence
 - Migraine (in younger patients)
- No correlation between TGA and thromboembolic cerebrovascular disease has been found

DIAGNOSIS

SIGNS AND SYMPTOMS

Diagnostic criteria:

- Attack must be witnessed

- Acute onset of anterograde amnesia
- No alteration in consciousness
- No cognitive impairment except amnesia
- No loss of personal information (e.g., name, birth date, address, etc.)
- No focal neurologic symptoms
- No epileptic features
- No recent history of head trauma or seizures
- Attack must resolve within 24 hr
- Other causes of amnesia excluded

History

- Often precipitated by stressful condition:
 - Cough, Valsalva
 - Physical exertion
 - Sexual intercourse
 - Extreme fright or shock
 - Intense heat or cold
- Patient will likely feel something is wrong:
 - May ask “how did I get here?”
 - May be repetitive in questions
 - Will be generally aware of attack
- May have other subtle transient symptoms at onset, such as headache, dizziness, nausea
- Historical features helpful in excluding other diagnoses are:
 - Onset of attack witnessed, with no seizure activity or epileptiform features noted
 - No history of seizures in prior 2 mo
 - No history of recent traumatic brain injury
 - Acute anterograde amnesia with relatively preserved remote memory

Physical-Exam

- Marked anterograde amnesia
- Most cases ($\geq 90\%$ in case series) will demonstrate repetitive questioning
- Neurologic and general exam normal
- TGA patient *WILL NOT* be:
 - Somnolent
 - Inattentive
 - Globally confused
 - Confabulate
- TGA patient *WILL* be:
 - Oriented to name, birth date, address, phone number, date
 - Able to perform complex tasks and following complex commands

- Aphasia, apraxia, and agnosia are NOT findings consistent with TGA

ESSENTIAL WORKUP

- True TGA can be diagnosed with a careful history and physical exam alone
- If clinical diagnosis is certain, no other workup is essential

DIAGNOSIS TESTS & INTERPRETATION

Testing indicated only when the diagnosis is uncertain

Lab

- CBC, comprehensive chemistries including glucose, LFTs, NH₃, thyroid studies, and UA for organic–metabolic etiologies were implicated
- Tox screen, alcohol level for toxic etiologies were suspected

Imaging

- Consider MRI if indicated.
 - In true TGA, MRI may show a focal hippocampal DWI or T2 lesion that resolves over time
- Head CT for intracranial mass if indicated

Diagnostic Procedures/Surgery

- EEG for seizure or nonconvulsive status if suspected
- Lumbar puncture and CSF analysis for encephalitis if suspected

DIFFERENTIAL DIAGNOSIS

- Other entities may present somewhat similarly but will likely have historical or physical exam features that readily distinguish them from TGA:
 - Anterior choroidal artery or posterior cerebral artery or TIA:
 - Additional related neurologic signs such as hemianopia
 - Acute confusional state/Korsakoff syndrome/metabolic disorder:
 - Alcohol, medication, or toxin ingestion
 - Decreased attention or other findings of an encephalopathy
 - Impairment with serial 7s or spelling “world” backward
 - Able to lay down new memory if allowed time to encode
 - Complex partial seizures/epileptic amnesic attacks:
 - Witnessed epileptiform activity or features (e.g., blank stares, automatisms, lip-smacking, olfactory hallucinations)
 - Short duration (typically < 30 min; TGA lasts hours)
 - No repetitive questioning
 - Frequent and rapid recurrences
 - Psychogenic amnesia:
 - Younger patient with a known psychiatric stressor
 - Prominent retrograde amnesia

- Psychogenic memory loss for personal identification, name, birth date, etc.
- Temporal lobe brain lesion or encephalitis affecting the temporal lobe:
 - Has other associated neurologic symptoms (e.g., visual field cut, confusion)
 - Progressive and permanent amnesia
- Previously unrecognized Alzheimer dementia:
 - Memory loss for personal information such as date, phone number, address
 - Signs of additional global cognitive impairment



TREATMENT

PRE HOSPITAL

There are no considerations in true TGA that are specific to the pre-hospital environment

INITIAL STABILIZATION/THERAPY

There is no known effective therapy for TGA

ED TREATMENT/PROCEDURES

- TGA is a self-limited, relatively benign entity
- Observe the patient for improvement
- Assuming a true diagnosis of TGA, no acute treatment beyond reassurance of patient and family is indicated

MEDICATION

First Line

Not applicable

Second Line

Not applicable



FOLLOW-UP

DISPOSITION

Admission Criteria

- Admission for further observation for patients without significant improvement at the time of disposition
- Patients with uncertain diagnosis

- Patients showing a trend toward resolution but who have suboptimal social support at home

Discharge Criteria

- A clear diagnosis of TGA
- Resolving or resolved amnesia
- Good social support

Issues for Referral

- Recurrence rate of TGA is 8%
- Refer patients with recurrent episodes of TGA to a neurologist:
 - May benefit from ambulatory EEG to workup epilepsy

FOLLOW-UP RECOMMENDATIONS

Given median age of TGA patients (60 yr), follow-up with primary care provider for general cardiovascular risk factor modification may be beneficial:

- No follow-up specific to TGA is indicated
- See “Issues for Referral” for patient with recurrent episode of TGA

PEARLS AND PITFALLS

- TGA is a distinct and relatively benign entity:
 - Acute onset of isolated anterograde amnesia
 - Resolves spontaneously
- Be aware of subtle features that may suggest a more pathologic alternative diagnosis:
 - Short, recurrent episodes or automatisms in epilepsy
 - Cognitive impairment with encephalopathy
 - Subtle neurologic signs in encephalitis or TIA
- If there is uncertainty regarding the diagnosis, the highest yield tests are multimodal MRI and EEG

ADDITIONAL READING

- Bartsch T, Deuschl G. Transient global amnesia: Functional anatomy and clinical implications. *Lancet Neurol*. 2010;9:205–214.
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- Kirshner HS. Transient global amnesia: A brief review and update. *Curr Neurol Neurosci Rep*. 2011;11:578–582.
- Markowitsch HJ, Staniloiu A. Amnesic disorders. *Lancet*. 2012;380:1429–1440.



See Also (Topic, Algorithm, Electronic Media Element)

- Delirium

- Dementia



CODES

ICD9

- 437.7 Transient global amnesia
- 780.93 Memory loss

ICD10

- G45.4 Transient global amnesia
- R41.1 Anterograde amnesia
- R41.2 Retrograde amnesia

TRANSIENT ISCHEMIC ATTACK (TIA)

Casey Grover • Rebecca Smith-Coggins

BASICS

DESCRIPTION

- TIA – an episode of reversible neurologic deficit caused by a temporary decrease in blood flow to an area of the central nervous system (CNS)
- Classically described as symptoms lasting < 24 hr, but most TIA symptoms resolve in < 1 hr
- A warning for stroke, as 12–30% of strokes will be preceded by TIA

ETIOLOGY

- Transient decrease in perfusion to an area of the CNS, which can be caused by:
 - Thrombosis in medium to large arteries with atherosclerosis (25%)
 - Intracranial small vessel disease (25%)
 - Embolic cause from the heart (20%)
 - Miscellaneous, including arterial dissection, vasculitis, and hypercoagulable states (5%)
 - No clear predisposing vascular cause found (25%)

DIAGNOSIS

SIGNS AND SYMPTOMS

- Symptoms are determined by the vascular territories which are affected
- Large vessel TIA syndromes:
 - Anterior cerebral artery (ACA) – unilateral motor/sensory loss to leg > arm, disinhibition
 - Middle cerebral artery (MCA) – unilateral motor/sensory loss to face/arm > leg, aphasia if dominant hemisphere, neglect if nondominant hemisphere, homonymous hemianopsia
 - Posterior cerebral artery (PCA) – homonymous hemianopsia, may have alexia, prosopagnosia (can't recognize faces)
 - Anterior inferior cerebellar artery (AICA) – unilateral deafness, vertigo, tinnitus, vomiting, ipsilateral facial weakness and limb ataxia, contralateral decrease in pain and temperature sensation
 - Posterior inferior cerebellar artery (PICA) – unilateral palatal weakness, unilateral limb ataxia, unilateral Horner's syndrome, decreased pain/temperature sensation on contralateral body:
 - Wallenberg syndrome
 - Vertebrobasilar artery – ataxia, oculomotor palsies, facial paresis, loss of

consciousness, quadriplegia

- Carotid artery – unilateral motor/sensory loss to face/leg/arm, aphasia if dominant hemisphere, neglect if nondominant hemisphere, homonymous hemianopsia
- Small vessel TIA syndromes:
 - Amaurosis fugax – transient monocular blindness from occlusion of ophthalmic branch of internal carotid
 - Lacunar infarcts – occlusion of a deep penetrating artery of the brain. Usually produce pure motor or pure sensory deficits
 - Internal capsule – hemiparesis or dysarthria with clumsy hand
 - Corona radiata – hemiparesis
 - Pons – dysarthria with clumsy hand
 - Thalamus – sensory loss to 1 side of the body

History

- Historical features suggestive of TIA:
 - Sudden onset
 - Short duration (as >60% of events last <1 hr)
 - Negative symptoms – CNS is underperfused and therefore, not functioning, so TIA syndromes generally produce loss of neurologic function – i.e., weakness or aphasia
 - Symptoms are focal, related to specific vascular territory
- Historical features not suggestive of TIA:
 - Gradual onset
 - Positive symptoms – increased neurologic function in a particular area, such as convulsion, tingling, or twitching, suggests increased CNS activity as with migraine or seizure

Physical-Exam

- Detailed neurologic exam: Strength, sensation, coordination, gait, naming/speech, and visual fields
- Persistent neurologic deficits suggest acute stroke rather than TIA
- The National Institute of Health Stroke Scale (NIHSS) is a reliable and easily repeatable neurologic exam
(http://www.ninds.nih.gov/doctors/NIH_Stroke_Scale.pdf)

ESSENTIAL WORKUP

- Rapid history and physical exam including detailed neurologic exam
- Fingerstick glucose – hypoglycemia can produce focal neurologic deficits
- Noncontrast CT head – rule out hemorrhage
- If patients present with persistent deficits, obtain STAT neurologic consultation with concern for acute stroke rather than TIA

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Glucose
- Chemistry panel – check Na, renal function
- CBC – exclude anemia, polycytosis
- Troponin – rule out concomitant ACS or demand ischemia
- Hemoglobin A1C and fasting lipid panel for patients being admitted/observed

Imaging

- CT:
 - Upon arrival to ED, STAT noncontrast head CT to rule out CNS hemorrhage
- MRI:
 - Up to 50% of patients that clinically have a TIA will have evidence of infarction on MRI:
 - Goal is to have MRI in < 24 hr
 - Diffusion-weighted imaging (DWI) is the most sensitive protocol for detection of tissue infarction
- Vascular imaging:
 - Either with initial imaging or inpatient workup, perform vascular imaging of the head and neck:
 - Almost 50% of patients with TIA have stenosis or occlusion of large arteries
 - Carotid duplex ultrasound can be used to detect internal carotid stenosis
 - CT angiography:
 - Can be performed at the time of initial noncontrast head CT
 - Can be used to detect stenosis in intracranial and extracranial vessels
 - Requires contrast
 - MR angiography:
 - Can be used to detect stenosis in intracranial and extracranial vessels
 - Time of flight (TOF) sequences can provide angiographic images without contrast

Diagnostic Procedures/Surgery

- ECG – evaluate for thrombogenic rhythms such as atrial fibrillation
- Echocardiography in patients with no other cause for TIA – exclude existing thrombus and abnormal wall motion or aneurysms that cause thrombus

DIFFERENTIAL DIAGNOSIS

- Hypoglycemia
- Seizure
- Paralysis after seizure (Todd's paralysis)

- Atypical migraine
- Psychiatric disease
- Stroke
- CNS tumors or metastases
- Subdural hemorrhage
- Subarachnoid hemorrhage
- Multiple sclerosis
- Intracerebral hemorrhage
- Air embolism
- Vasculitis
- Arterial dissection

Pediatric Considerations

- Congenital heart disease
- Vasculitis
- Arterial dissection
- Sickle cell disease
- Neurocutaneous syndromes
- Vascular malformations
- Meningitis



TREATMENT

PRE HOSPITAL

- Rapid assessment of neurologic deficits
- Consider transport to a stroke center, when available, if deficits persist

INITIAL STABILIZATION/THERAPY

- IV access
- Cardiac monitoring
- Supplemental oxygen if hypoxic

ED TREATMENT/PROCEDURES

- Main goals in the management of TIA:
 - Improve perfusion to ischemic tissue
 - Prevent a subsequent stroke
- BP management:
 - BP should not be lowered acutely unless over 220/120 mm Hg
 - Hypertensive patients with TIA should have their BP lowered if stable at 24 hr after TIA
 - Key in patients upon discharge
 - 1st line – HCTZ or ACE inhibitor

- **Antiplatelet therapy:**
 - All patients, in the absence of contraindications, need antiplatelet therapy for stroke prevention
 - 1st line – aspirin (ASA):
 - Safe, cheap, effective
 - ASA allergy – clopidogrel, ticlopidine
 - ASA/dipyridamole may be more effective than ASA alone
- **Anticoagulation:**
 - Indicated for new onset atrial fibrillation or existing atrial fibrillation not on anticoagulants
 - Options include heparin/low-molecular-weight heparin with a transition to warfarin or dabigatran
 - The decision to anticoagulate is not emergent; discuss with admitting physician
- **Carotid endarterectomy (CEA):**
 - CEA within 2 wk after TIA in patients with >70% carotid stenosis reduces stroke risk by 10–15%
- **Lipid therapy:**
 - AHA guidelines recommend statin therapy for patients with TIA with a goal LDL of under 70 mg/dL
 - Key in patients upon discharge

MEDICATION

- **Antiplatelet agents:**
 - Aspirin 160–325 mg daily
 - Aspirin/dipyridamole 25 mg/200 mg daily
 - Clopidogrel 300 mg initially then 75 mg daily
- **Anticoagulation:**
 - Heparin 5,000–7,500 U IV bolus, followed by 1,000 U/h infusion OR 80 U/kg IV bolus then 18 U/kg/h
 - Warfarin dose is dependent on age and weight, but goal INR for atrial fibrillation is 2–3
 - Dabigatran 150 mg daily (normal renal function)
- **Acute BP management:**
 - Labetalol 20 mg IV bolus, followed by 20–80 mg IV every 10 min; max. cumulative dose of 300 mg
 - Nicardipine 5 mg/h infusion, increase by 2.5 mg/h every 5–15 min; max. dose of 15 mg/h



FOLLOW-UP

DISPOSITION

Admission Criteria

- There are no clear indications for admission or discharge
- Patients with TIA have variable short-term risk of stroke
- Goal of admission is to prevent subsequent stroke in high-risk patients
- Scoring systems have been developed to predict short-term risk of stroke and therefore can guide disposition
- Most common = ABCD2 score:
 - _ Age >60 = 1 point
 - _ BP >140/90 = 1 point
 - _ Clinical features:
 - Unilateral weakness = 2 points
 - Speech difficulty alone = 1 point
 - _ Duration:
 - >60 min = 2 points
 - 10–59 min = 1 point
 - <10 min = 0 points
 - _ Diabetes = 1 point
- ABCD2 score 0–3 = low risk of stroke (~1% at 7 days)
- ABCD2 score 4–5 = moderate risk for stroke (~6% at 7 days)
- ABCD2 score 6–7 = high risk for stroke (~12% at 7 days)
- Patients with moderate to high risk for short-term stroke = admission
- Patients with low risk for short-term stroke, but poor follow-up = observation unit

Pediatric Considerations

All children with TIA should be admitted for close neurologic observation, with strong consideration of ICU level care

Discharge Criteria

- No clear discharge criteria exist:
 - _ Low risk for short-term stroke, with good follow-up

Issues for Referral

- The risk of stroke after TIA is highest within 2 days of symptoms
- Discharged patients need to see neurology/primary care within 24–48 hr

FOLLOW-UP RECOMMENDATIONS

- Primary Care/Neurology – management of risk factors for cerebrovascular disease (hypertension, diabetes, etc.)
- Vascular surgery – for carotid stenosis. Follow-up within 1 wk, plan for possible CEA within 2 wk
- Cardiology – for those patients with cardiac cause of stroke, such as atrial fibrillation or cardiomyopathy

PEARLS AND PITFALLS

- Pearls:
 - Risk stratification scores (such as ABCD2) can help guide disposition
 - Patients with carotid stenosis need rapid vascular surgery follow-up
- Pitfalls:
 - Failure to recognize the subtle lacunar TIA syndromes, such as sensory loss
 - Failure to rapidly check a glucose in a patient with a focal neurologic deficit
 - Discharging patients with TIA without close outpatient follow-up

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CODES

ICD9

- 435.3 Vertebrobasilar artery syndrome
- 435.8 Other specified transient cerebral ischemias
- 435.9 Unspecified transient cerebral ischemia

ICD10

- G45.8 Oth transient cerebral ischemic attacks and related synd
- G45.9 Transient cerebral ischemic attack, unspecified
- G46.1 Anterior cerebral artery syndrome

TRANSPLANT REJECTION

Kyle R. Brown • Jeffrey N. Siegelman

BASICS

DESCRIPTION

Immune response to a graft's genetically dissimilar antigens resulting in rejection of the transplanted organ:

- **HLA incompatibility:**
 - _ Most common cause of rejection
 - _ Rejection of solid organ transplants
- **Blood group incompatibility:**
 - _ Much less of a risk to graft survival than HLA incompatibility
 - _ May result in hyperacute rejection of primarily vascularized grafts (kidney and heart)
- **3 phases of rejection:**
 - _ **Hyperacute:**
 - Immediate postoperative period
 - Antibody reaction to red cells or HLA antigens
 - Endothelial damage
 - Platelets accumulate, thrombi develop, and tissue necrosis occurs.
 - Rare with careful donor–recipient matching
 - _ **Acute:**
 - _ Within the 1st 3 mo postop
 - _ At any time if immunosuppressant (IS) medication is stopped
 - _ T-cell–dependent process. Inflammatory cells infiltrate allograft, release cellular and humoral factors, destroys graft
 - _ Presents with constitutional symptoms and signs of transplant organ insufficiency
 - _ **Chronic:**
 - _ Occurs over years
 - _ Results in gradual organ failure

EPIDEMIOLOGY

Incidence and Prevalence Estimates

- **Solid organ transplants:**
 - _ End of 2007: 183,222 living transplant patients
 - _ 27,281 organs transplanted in 2008
 - _ Most transplanted organs: Kidney (59%), liver (21%), heart (8%), lung (5%), pancreas (4%)
 - _ Most common diagnosis from visit to ED: Infection (36%), GI/GU pathology

(20%), dehydration (15%), electrolyte (10%), CV and pulmonary pathology (10%), injury (8%), rejection (6%). 60% required hospitalization

- Hematopoietic stem cell transplants:
 - 4,300 transplants in 2008
 - Acute graft-versus-host disease incidence: 20–80%.

ETIOLOGY

- Reduction or noncompliance with medication:
 - Medication interactions with cyclosporine, tacrolimus, or sirolimus:
 - Phenobarbital, phenytoin, carbamazepine, rifampin, isoniazid
- Kidney transplant rejection:
 - Early rejection caused by T and B lymphocytes, which attack microvasculature and impair graft perfusion; volume depletion, hypotension, infection
 - Chronic rejection caused by progressive nephrosclerosis of renal vessels, infection
- Liver transplant rejection:
 - Acute: 48% by 6 wk, 65% by 1 yr
 - Commonly follows reduction in the IS regimen
 - Chronic: <5%
 - 1 wk to 6 mo MC range to experience
- Cardiac transplant rejection:
 - Acute rejection:
 - 75–85% of patients within the 1st 3–6 mo due to T-cell-mediated response
 - Chronic rejection:
 - Accelerated atherosclerosis is the hallmark
 - Associated with change in IS therapy
- Lung transplant rejection:
 - Acute rejection develops early:
 - Can occur up to 6 times in the 1st year
 - Chronic rejection:
 - 25–40% of patients postop
 - MCC of death in 2nd postop year
 - Rejection caused by endothelial, vascular, and lymphocyte inflammation, recurrent acute rejection
- Bone marrow transplant rejection:
 - Acute graft-versus-host disease:
 - Immune attack of donor marrow on lung tissue
 - Chronic graft-versus-host disease:
 - 25–50% of patients
 - Marrow rejection:
 - MC in patients with plastic anemia who do not receive total body

radiotherapy or in patients receiving mismatched or unrelated transplants



DIAGNOSIS

SIGNS AND SYMPTOMS

- Renal transplant rejection:
 - Progressive systemic HTN
 - Decreased urine output
 - Swelling, fever, and tenderness:
 - Uncommon with IS therapy
- Liver transplant rejection:
 - Fever, RUQ pain, jaundice
- Heart transplant rejection:
 - Fever, dyspnea, chest pain, hypo- or hypertension, palpitations, nausea, vomiting, syncope, sudden death
 - Can be asymptomatic
- Lung transplant rejection:
 - Cough, dyspnea, fever, rales, and rhonchi
- Bone marrow transplant rejection:
 - Fever, wasting, mucositis, keratoconjunctivitis, dysphagia, cough, dyspnea, hypoxia, chest pain, abdominal pain, diarrhea, jaundice, rash, encephalopathy, seizures

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC
- IS medication levels:
 - Levels may not represent through if patient took medication prior to ED visit.
- Blood cultures
- Renal transplant rejection:
 - Electrolytes, BUN, creatinine, CRP
 - Urinalysis with micro:
 - Proteinuria may signal early rejection. Presence of leukocytes may be seen during rejection as well as with infection.
 - FENa helps differentiate rejection from iatrogenic causes
- Liver transplant rejection:
 - Coagulation panel, lipase, cultures (blood, urine, ascites), liver function tests
 - Late acute rejection presents with elevated bilirubin and transaminases.

- Heart transplant rejection:
 - Cardiac troponin
- Lung transplant rejection:
 - ABG, electrolytes, kidney function, CRP, liver function, bilirubin, LDH, CPK, EBV, CMV, cyclosporine levels
- Bone marrow transplant rejection:
 - ABG, liver function tests

Imaging

- CXR:
 - Acute lung rejection:
 - Diffuse infiltrates are seen early
 - Normal or unchanged > 1 mo after transplantation
 - Bone marrow transplant rejection:
 - Interstitial infiltrates, pleural effusion, pulmonary edema
- Renal US:
 - Indicated for suspicion of renal transplant rejection:
 - Hydronephrosis implies obstructive uropathy and may need urgent percutaneous nephrostomy.
- Liver transplant:
 - Hepatic US
 - CT abdomen
- Echocardiography:
 - Heart transplant
 - Assess for changes in cardiac output.
- MRI:
 - Renal transplant:
 - May be done with or without contrast
 - Consult transplant team before giving contrast

Diagnostic Procedures/Surgery

- Liver transplant rejection:
 - ERCP cholangiography
- Heart transplant rejection:
 - EKG:
 - Commonly demonstrates 2 P waves because the native sinus node is spared
- Lung transplant rejection:
 - Peak flow reduced FEV1
 - Early bronchoscopy and biopsy to differentiate infection from rejection

ESSENTIAL WORKUP

- Consider drug toxicity and infection as well as rejection.

- Ask about medication dose or compliance changes
- Low threshold for screening labs and imaging even with minimal signs and symptoms

DIFFERENTIAL DIAGNOSIS

- Infections:
 - Wide variety of bacterial, mycobacterial, fungal, viral, and parasitic pathogens can cause opportunistic infections in transplant patients.
- IS toxicity
- Drug interactions with IS medication
- Renal transplant rejection:
 - Any disorder that can affect the native kidneys can also occur in the transplant
 - Iatrogenic nephrotoxicity: Cyclosporine, tacrolimus, other medications
 - UTI/pyelonephritis:
 - Classic organisms as with native kidney infections
 - Tubulointerstitial nephritis caused by the BK-polyomavirus (incidence 3–5%)
 - Acute occlusion of the transplant renal artery or vein:
 - Acute occlusion usually occurs within the 1st post-transplant week (incidence 0.5–8%) and causes oliguria and acute renal failure.
 - Peritransplant hematoma
 - Urinary leak
 - Obstructive uropathy
 - Bleeding after renal graft biopsy
- Liver transplant rejection
 - Ascending cholangitis:
 - Possible from colonized postop biliary stent.
 - Cholestatic hepatitis from azathioprine
 - Methotrexate-induced hepatotoxicity
- Lung transplant rejection
 - MC bacterial infection in lung transplant is cytomegalovirus pneumonia.
 - MC fungal infection is Aspergillus.
 - Upper respiratory infection or bronchitis:
 - Mimic chronic lung rejection
 - Medication-induced pneumonitis



TREATMENT

PRE HOSPITAL

Avoid aggressive fluid resuscitation.

INITIAL STABILIZATION/THERAPY

- ABCs
- Shock state treated with IV fluids, and pressor agents.
- Treat hypertensive crisis like other hypertensive emergencies.

ED TREATMENT/PROCEDURES

ALERT

Always discuss with transplant service early unless unstable, especially when adding or changing medications.

ALERT

Caution with use of NSAIDs; there are many associated complications in these patients.

- Kidney, heart, lung, and liver rejection:
 - Administer high-dose steroids
 - Stress-dose corticosteroid coverage is also indicated in any ill-appearing transplant patient. Consult with transplant service early
 - Avoid blood transfusions because these need special screening to prevent transmission of disease.
- Heart transplant rejection:
 - Pressors and inotropics work as usual in the transplanted heart.
 - Atropine will have no effect on bradycardia because there is no vagal innervation.
 - Use dopamine, epinephrine drips, or external pacing to increase heart rate if bradycardia is symptomatic.
 - IV methylprednisolone: 1 gm/d for 3 days
- Lung transplant:
 - Treat for infection and rejection
 - IV methylprednisolone: 15 mg/kg daily × 3 days
- Graft-versus-host disease:
 - 1–2 mg/kg daily PO or IV steroids
 - For chronic, may need adjustments of IS therapy.
- Common IS regimens are cyclosporine, prednisone, and azathioprine or tacrolimus and prednisone.

MEDICATION

As directed by transplant team



FOLLOW-UP

DISPOSITION

Admission Criteria

- Admit transplant recipients with fever, shortness of breath, signs or symptoms of rejection, abdominal pain, or other signs of organ infection, pneumothorax, and respiratory failure.
- Admit to the ICU patients who are septic, in acute renal failure, or have cardiopulmonary compromise.

Discharge Criteria

Nontoxic patients in whom rejection or serious infection has been excluded may be discharged with close follow-up and in consultation with their transplant service.

Issues for Referral

Treatment decisions should be made in consultation with the patient's oncologist, transplant surgeon, or organ specialist.

FOLLOW-UP RECOMMENDATIONS

- The patient's transplant team should actively participate in the follow-up plan:
- All attempts at verbal communication with the covering transplant physician should be made while the patient is in the ED with any symptoms suggestive of rejection.

PEARLS AND PITFALLS

- Transplant patients presenting with minor complaints are at high risk for rejection and require an in-depth assessment in the ED, in conjunction with their transplant team.
- Patients with signs of possible transplant rejection should also be considered for infection and drug toxicity.
- A high percentage require admission

ADDITIONAL READING

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CODES

ICD9

- 996.80 Complications of transplanted organ, unspecified
- 996.81 Complications of transplanted kidney
- 996.82 Complications of transplanted liver

ICD10

- T86.11 Kidney transplant rejection
- T86.41 Liver transplant rejection
- T86.91 Unspecified transplanted organ and tissue rejection

TRAUMA, MULTIPLE

Daniel Davis • Alexander L. Bromfield

BASICS

DESCRIPTION

- Standardized approach for rapid assessment of the trauma patient
- Although presented as a sequential method for gathering information, many of these steps can be performed simultaneously.
- In general, injuries must be prioritized in order of severity to increase survival. Life-threatening injuries, particularly when abnormal vital signs are present, must be immediately addressed and treated before going on to the next level of care.
- With any change in the patient's status, the primary survey should be repeated.

ETIOLOGY

Variety of causes such as:

- Motor vehicle/motorcycle crashes
- Falls from heights
- Assault
- Airplane crashes
- Train derailments
- Results of mass-casualty weapons
- Terrorism

DIAGNOSIS

- Triage to a major trauma center is determined by local protocols.
- Injured patients with a need for surgical, neurosurgical, or orthopedic intervention should be transported to a major trauma center.
- Recent recommendations from the American College of Surgeons suggest that trauma victims with unstable vital signs should be taken to a Level I trauma center, where a larger volume of critically injured patients are seen.
- Primary survey should be performed at the scene and en route.

SIGNS AND SYMPTOMS

- Primary survey (ABCDE):
 - Airway, cervical spine:
 - Look, listen, and palpate from nose/mouth to trachea/bronchial tree.
 - Assess airway patency.
 - Evaluate gag reflex.
 - Cervical spine must be immobilized with significant mechanism of injury and either altered mental status or distracting injuries or with

signs and symptoms suggestive of neck injury.

- Ability to speak or effective movement of air with respiration indicates patency.
- Gurgling, stridor, wheezing, snoring, choking, or absence of air movement requires immediate intervention.
- Manage airway compromise before next step in primary survey.

– Breathing:

- Awake, alert patient with normal speech and good air movement suggests effective breathing.
- Symmetric chest wall rise/fall, equal breath sounds, normal respiratory rate, and oxygen saturation at 95% or more suggest effective breathing.
- Asymmetric chest movement, unequal breath sounds, abnormal respiratory rate, decreased oxygen saturation, inadequate air movement, or an obtunded patient suggests ineffective breathing.
- Decreased unilateral breath sounds, tracheal shift, hyperexpansion, hyperresonance to percussion, subcutaneous air, hypoxia, or hemodynamic compromise raises concerns about tension pneumothorax.
- Decreased breath sounds with dullness to percussion suggest hemothorax.
- Manage patients immediately with needle thoracostomy followed by tube thoracostomy.

– Circulation:

- Adequate circulating blood volume must be maintained.
- Primary assessment includes BP, heart rate, pulse quality, and end-organ function (e.g., mentation, urine output, capillary refill).
- Tachycardia and oliguria indicate early shock; hypotension is a late finding and necessitates a search for hemorrhage

– Disability:

- Assess level of consciousness, gross motor function, and pupillary size/reactivity.
- Glasgow Coma Scale is most commonly used; score of ≤ 8 indicates severe head injury/coma.
- Spinal cord injuries are grossly assessed by observing movement of all extremities.
- Pupillary size and reactivity to light measure brainstem function.

– Exposure:

- Patient should be undressed completely.

● Secondary survey:

- After the primary survey has been completed
- Patient stabilized at each level
- Complete physical exam from head to toe is performed.

- “Tubes and fingers in every body cavity”

History

The mechanism of injury, initial clinical presentation, suspected injuries, and treatment rendered should be elicited from EMS personnel.

Physical-Exam

Initial stabilization should begin simultaneously with essential workup.

ESSENTIAL WORKUP

- Primary and secondary surveys
- Cervical spine and chest radiographs are mandatory for victims of major trauma.
- Pelvic radiographs should be performed with clinical suspicion of pelvic trauma or with hemodynamic instability.
- Hemoglobin/hematocrit, ABG, blood type; a toxicology screen may also be considered.
- Urine dip for blood
- UA if dip shows positive result
- Urine-based pregnancy test for any female patient of childbearing age

DIAGNOSIS TESTS & INTERPRETATION

Lab

Baseline coagulation and chemistry studies with massive injury or hemorrhage

Imaging

- Loss of consciousness, post-traumatic amnesia (anterograde or retrograde), or persistent altered level of consciousness is indication for head CT.
- Significant blunt and penetrating chest trauma requires objective evaluation of the heart and great vessels with echocardiography, CT scan, angiography, or direct visualization.
- Blunt abdominal trauma requires objective evaluation using US, abdominal CT, or diagnostic peritoneal lavage, depending on patient's condition:
 - Hemodynamically stable patients should have an abdominal CT with IV contrast.
 - Unstable patients should have an abdominal ultrasound (FAST exam) or diagnostic peritoneal lavage.
 - Many centers now doing “Pan CT scan,” including head, neck, chest, abdomen/pelvis in a single pass with IV contrast
 - Pan CT lowers missed injury rate but involves significant radiation exposure
- Extremity injury:
 - Radiographs
 - Suspected vascular damage requires angiography or duplex ultrasound.

DIFFERENTIAL DIAGNOSIS

Some level of clinical suspicion should be maintained for other medical conditions leading to trauma (e.g., seizures, dysrhythmias).



TREATMENT

INITIAL STABILIZATION/THERAPY

- The initial treatment should parallel the primary survey with injuries treated before addressing the next assessment level.
- Airway with cervical spine control:
 - Jaw thrust, suctioning, and oropharyngeal or nasopharyngeal airways provide initial airway support.
- Rapid sequence intubation is the airway management option of choice for multiple trauma patients:
 - Insertion of an extraglottic airway (e.g., Combitube, laryngeal tube, or laryngeal mask airway) or cricothyroidotomy may be necessary.
 - Use of video laryngoscopy may allow endotracheal intubation with minimal impact on potential traumatic brain injury or an unstable cervical spine
- Breathing:
 - 100% oxygen and respiratory monitoring
 - Tension pneumothorax should be diagnosed clinically and decompressed on an emergency basis with a needle thoracostomy below the axilla or above the 2nd rib in the midclavicular line.
 - Tube thoracostomy should follow.
 - Open chest wounds should be covered with an adherent dressing and a tube thoracostomy performed.
 - Respiratory distress from flail segment or pulmonary contusion should prompt early intubation with mechanical ventilation and positive end expiratory pressure.
 - Hyperventilation should be avoided except with impending herniation or intracranial HTN resistant to other therapies; end-tidal carbon dioxide monitoring should be used.
- Circulation:
 - 2 large-bore IV lines with constant hemodynamic and cardiac monitoring should be placed.
 - A thoracotomy may be considered in a previously stable patient with penetrating chest trauma and an acute deterioration in status
 - A Foley catheter can be placed to help monitor urine output but should be withheld if blood is present at the urethral open until additional imaging can be performed
- Alternatives include central lines, venous cut-downs (e.g., saphenous or femoral), or intraosseous lines:

- Aggressive fluid replacement with 3 parts fluid for every 1 part circulatory volume loss remains most widely recommended care; adjust fluids based on ongoing assessment:
 - 2 L initial bolus in adults, 20 mL/kg in children
 - Whole blood or autotransfused blood for hemorrhagic shock or uncontrolled bleeding
- Pericardial tamponade requires emergent pericardiocentesis/pericardial window.
- External bleeding should be managed with direct pressure.
- Unstable pelvic fractures should be treated with pelvic binding
- Disability:
 - Head injury with Glasgow Coma Scale score of ≤ 8 should initiate treatment for elevated intracranial pressure with mannitol or hypertonic saline, rapid-sequence intubation, oxygenation, and controlled ventilation to a PCO_2 of 35 mm Hg.
 - Elevate head 20–30°, maintaining spine immobilization.

ED TREATMENT/PROCEDURES

- Definitive treatment is often surgical.
- Prompt stabilization, early recognition of the need for operative intervention, and appropriate trauma surgical consultation are paramount.

MEDICATION

Dictated by need for specific interventions

Pediatric Considerations

Intraosseous lines are an alternative to IV lines for fluids and medications. Lack of rib cervical spine fractures does not exclude spinal cord injury



FOLLOW-UP

DISPOSITION

Admission Criteria

- Most major trauma patients should be admitted for observation, monitoring, and further evaluation.
- Patients with significant injuries or hemodynamic instability should be admitted to an ICU.
- Patients requiring frequent assessments should be admitted to a monitored setting.

Discharge Criteria

Patients with minor trauma and negative objective workup/imaging may be observed in

the ED for several hours and then discharged.

Issues for Referral

The main indications for referral concern the availability of subspecialists, such as neurosurgeons, orthopedists/hand surgeons, otolaryngologists, plastic surgeons, or intensivists.

FOLLOW-UP RECOMMENDATIONS

Follow-up should be driven by the types of injuries and subspecialty care required.

PEARLS AND PITFALLS

- The ABCs of trauma remain the standard approach to guide the initial assessment and treatment of trauma patients.
- A high level of suspicion for occult injuries should be maintained, with a low threshold for obtaining objective imaging.
- Trauma systems are defined by an organized approach to accessing quality trauma and subspecialty care.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Specific Anatomic Injuries, Shock, Airway Management.



CODES

ICD9

- 952.9 Unspecified site of spinal cord injury without evidence of spinal bone injury
- 959.01 Head injury, unspecified
- 959.8 Other specified sites, including multiple injury

ICD10

- S09.90XA Unspecified injury of head, initial encounter
- S14.109A Unsp injury at unsp level of cervical spinal cord, init

- T14.90 Injury, unspecified

TRICHOMONAS

Herbert Neil Wigder • Erin Nasrallah



BASICS

DESCRIPTION

- Sexually transmitted disease (STD)
- Associated with high prevalence of other STDs
- Causes urogenital infections
- Sequelae:
 - May cause premature rupture of membranes or preterm labor in pregnancy
 - May cause low-birth-weight newborns
 - May facilitate transmission of HIV
- Prevalence:
 - 3–5 million cases per year in US
 - 35% of women treated in STD clinics
 - Overall prevalence 3.1%:
 - Prevalence in black women 13.3%
- Incubation 4–28 days
- May be asymptomatic

ETIOLOGY

Trichomonas vaginalis:

- Flagellated protozoan:
 - Commonly found in urethra, bladder, and Skene gland



DIAGNOSIS

SIGNS AND SYMPTOMS

OTHER

- Vaginitis:
 - Vaginal discharge is seen in < 30% of patients
 - Frothy yellow/green to gray/white
 - Vulvar itching and irritation
 - Vaginal odor
 - Symptoms same as with bacterial vaginosis (caused by Gardnerella vaginalis) and vulvovaginal candidiasis (caused by Candida albicans)
 - Dysuria and urinary urgency
 - Painful sexual intercourse
 - Often asymptomatic (50%)

- Cervix:
 - Diffuse erythema (10–33%)
 - Punctate hemorrhage—colpitis macularis or strawberry cervix (2%)
- Abdominal pain uncommon

Male

- Often asymptomatic (75%) or self-limited
- Male to male transmission is uncommon
- Nongonococcal urethritis:
 - 20% of nonspecific urethritis
 - Scant discharge
 - Dysuria and urinary urgency
- Complications:
 - Prostatitis
 - Epididymitis
 - Reversible sterility

Physical Exam

- Female:
 - Vaginal discharge:
 - Frothy yellow/green to gray/white
 - Odor
 - Red ulcerations—vaginal wall and cervix
- Male:
 - Scant discharge

ESSENTIAL WORKUP

- Treat empirically if high enough clinical suspicion
- Females: Wet mount (“Hanging-drop”):
 - 60–70% sensitive in symptomatic patients
 - Saline wet mount from cervical/vaginal vault smear:
 - Requires immediate evaluation of slide
 - Many polymorphonuclear leukocytes (PMNs)
 - Motile, pear-shaped, flagellated trichomonads (slightly larger than leukocytes; seen in 60%)
 - Specimen from spun urine less sensitive
 - Absence of trichomonads does not rule out *T. vaginalis* infection (only present in 60–70%)
 - Many EDs not equipped to perform wet mount
- Elevated vaginal pH (> 4.5) common:
 - Not specific
- Males: Wet mount insensitive
 - PCR reliable but not widely available

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Culture:
 - 95% sensitivity:
 - Prostate massage before collection increases sensitivity in males.
 - Do culture when trichomonads suspected but not confirmed by wet-mount microscopy
- Point-of-care tests:
 - High specificity (>97%) but variable sensitivities
- Polymerase chain reaction (PCR):
 - Expensive

DIFFERENTIAL DIAGNOSIS

- UTI
- Gonorrhea
- Chlamydia
- Bacterial vaginosis
- Candidal vaginitis
- Nonspecific vaginitis



TREATMENT

ED TREATMENT/PROCEDURES

- Female:
 - Metronidazole 2 g PO once:
 - 90–95% cure rate
 - Metronidazole 250 mg PO TID for 7 days (urethritis)
 - Tinidazole 2 g PO once:
 - 86–100% cure rate
 - Metronidazole gel, less effective:
 - Not recommended
- Pregnant:
 - Symptomatic:
 - Metronidazole (FDA category B)
 - Asymptomatic:
 - Treatment is controversial as it does not reduce incidence of premature rupture of membranes or preterm delivery
 - Metronidazole
- Males (urethritis):
 - Metronidazole 2 g PO once
 - Tinidazole 2 g PO once

- Metronidazole 250 mg PO TID for 7 days
- HIV positive
 - Consider 7-day course of treatment because of evidence of increased single dose treatment failure
- Treat sex partners to prevent reinfection
 - No sexual intercourse until both partners are asymptomatic and until after at least 1 wk after treatment completed
- Advise using latex condoms
- Avoid concomitant alcohol use with metronidazole:
 - No alcohol for 24 hr after last metronidazole as it precipitates Antabuse reaction



FOLLOW-UP

DISPOSITION

Discharge Criteria

All patients

PEARLS AND PITFALLS

- Typical treatment for nongonococcal urethritis (e.g., azithromycin, doxycycline) does not treat *T. vaginalis*.
- Vaginitis in females not responding to treatment for bacterial vaginosis might be due to *Trichomonas* infection.
- Nongonococcal urethritis in males not responding to azithromycin or doxycycline might be due to *Trichomonas*.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- [Gonococcal Disease](#)
- [Pelvic Inflammatory Disease](#)
- [Urethritis](#)
- [Vaginal Discharge/Vaginitis](#)



CODES

ICD9

- [131.01 Trichomonal vulvovaginitis](#)
- [131.02 Trichomonal urethritis](#)
- [131.9 Trichomoniasis, unspecified](#)

ICD10

- [A59.01 Trichomonal vulvovaginitis](#)
- [A59.03 Trichomonal cystitis and urethritis](#)
- [A59.9 Trichomoniasis, unspecified](#)

TRICYCLIC ANTIDEPRESSANT, POISONING

Steven E. Aks

BASICS

DESCRIPTION

- Primary mechanism of tricyclic antidepressant (TCA) toxicity:
 - Sodium channel blocking effect (quinidine-like effect)
 - Inhibition of norepinephrine reuptake
 - α -blockade
 - Anticholinergic effect
- Selective serotonin reuptake inhibitors (SSRIs):
 - Wider margin of safety than TCA
 - Less CNS/cardiovascular toxicity
- Nonselective serotonin reuptake inhibitors:
 - Serotonin and norepinephrine reuptake inhibitors (SNRIs)
 - Can cause cardiac dysrhythmias or seizures
 - Venlafaxine (Effexor)
 - See “Antidepressants, Poisoning.”

ETIOLOGY

- TCAs:
 - Amitriptyline
 - Nortriptyline
 - Imipramine
 - Doxepin
- Newer-generation antidepressants (nontricyclic):
 - Have different toxic profile than TCAs
 - See “Antidepressants, Poisoning.”
- Rapid deterioration may occur.

DIAGNOSIS

SIGNS AND SYMPTOMS

- Rapid deterioration may occur.
- Classic TCA compounds (imipramine, amitriptyline, nortriptyline)—greatest cardiovascular toxicity
- Newer agents (serotonergic agents)—less overall toxicity in overdose
- CNS:
 - Stimulation or depression
 - Stimulation:

- Tremulousness
- Agitation
- Fasciculation
- Seizures (resulting acidemia may lead to worsening cardiovascular toxicity)
- Depression:
 - Drowsiness
 - Lethargy
 - Coma
- Cardiovascular system:
 - Hypotension
 - Tachycardia:
 - Early; owing to blockade of norepinephrine reuptake and anticholinergic effects
 - Bradycardia:
 - Late; owing to catecholamine depletion state
 - ECG changes:
 - QRS widening ($> 100\text{--}120$ ms)
 - Rightward shift in terminal 40 ms in frontal plane axis (R wave > 3 mm in aVR)
 - Dysrhythmias:
 - Supraventricular tachycardia (SVT)
 - Ventricular arrhythmias
- Anticholinergic effects (less common):
 - Dilated pupils
 - Decreased bowel sounds
 - Urinary retention

History

Substance ingestion in patient with access to TCA

Physical-Exam

- CNS:
 - Stimulation or depression
- Cardiovascular:
 - Tachycardia
 - Mydriasis or midrange pupils
 - Decreased bowel sounds
 - Urinary retention (rare)

ESSENTIAL WORKUP

- ECG: Factors associated with TCA poisoning:
 - Sinus tachycardia (almost always present at some time after poisoning)

- QRS widening:
 - o >100 ms associated with seizure
 - o >160 ms associated with ventricular dysrhythmia
- QT prolongation
- PR prolongation
- Rightward shifting of terminal 40 ms QRS axis
- R-wave amplitude in aVR > 3 mm
- Continuous cardiac monitor

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC
- Electrolytes, BUN, creatinine, glucose
- ABG
- Urine toxicology screen:
 - Rule out other toxins.
- TCA levels:
 - Not useful
 - Do not correlate well with degree of toxicity
 - Qualitative screen appropriate to confirm ingestion if necessary

Imaging

Chest radiograph for aspiration pneumonia/pulmonary edema

DIFFERENTIAL DIAGNOSIS

- Drugs that cause coma:
 - Alcohols
 - Alcohol withdrawal
 - Anticholinergics
 - Lithium
 - Phencyclidine (PCP)
 - Opioids
 - Phenothiazines
 - Sedative hypnotics
 - Salicylates
- Cardiotoxic drugs:
 - Antidysrhythmics (category IA)
 - Digoxin toxicity
 - Sympathomimetics
 - Anticholinergics
- Drugs that cause seizures:
 - Alcohol withdrawal

- Anticholinergics
- Camphor
- Isoniazid
- Lindane
- Lithium
- Phenothiazines
- Sympathomimetics
- Toxic alcohols



TREATMENT

PRE HOSPITAL

- Do not be lulled into false sense of security with well-appearing patient:
 - Rapid onset of altered mental status, seizures, and dysrhythmias occur.
- Perform endotracheal intubation if any evidence of compromise.
- Secure IV access.
- Administer sodium bicarbonate if any evidence of QRS widening ($> 100\text{--}120$ ms):
 - 1 ampule in adults
 - 1–2 mEq/kg in children
- Ipecac contraindicated (risk for aspiration with development of depressed mental status or seizure)

INITIAL STABILIZATION/THERAPY

- ABCs:
 - Low threshold to intubate patients with altered mental status
- IV 0.9% normal saline (NS)
- Oxygen
- Cardiac monitor:
 - For wide-complex rhythm (QRS $> 100\text{--}120$ ms) bolus sodium bicarbonate
- Naloxone, thiamine, glucose (Accu-Chek) for altered mental status
- Flumazenil contraindicated in combined TCA/benzodiazepine overdose

ED TREATMENT/PROCEDURES

Cardiac Toxicity

- Initiate therapy for cardiac toxicity aggressively to prevent deterioration.
- QRS widening ($> 100\text{--}120$ ms):
 - Bolus with 1 amp (peds: 1–2 mEq/kg) of sodium bicarbonate; repeat if sudden increase in QRS width
 - Maintain arterial pH of 7.45–7.5 with hyperventilation.
 - Initiate sodium bicarbonate infusion if hyperventilation alone does not reach target pH.

- **Dysrhythmia:**
 - Sinus tachycardia requires no treatment.
 - Bolus 1–2 amps of sodium bicarbonate (1–2 mEq/kg in children) for sudden change in rhythm
 - Follow advanced cardiac life support (ACLS) protocol with addition of sodium bicarbonate boluses:
 - Lidocaine is 2nd-line agent after sodium bicarbonate.
 - Use of class IA (procainamide) and IC agents and physostigmine contraindicated

Hypotension

- 0.9% NS fluid bolus
- Norepinephrine:
 - Preferred pressor (over dopamine)
 - Counters α -blockade better
 - Dopamine requires higher doses.

Decontamination

- Gastric lavage:
 - For recent ingestion (< 1 hr)
 - Performed when airway has been secured in lethargic patient
- Administer activated charcoal with sorbitol.
- Ipecac contraindicated

Seizure

- Diazepam 1st-line followed by phenobarbital
- Neuromuscular paralysis with short-acting agent (rocuronium/vecuronium) for refractory seizures (monitor EEG)
- Sodium bicarbonate bolus to prevent acidosis

MEDICATION

First Line

- Sodium bicarbonate: 1–2 amps (50–100 mEq) IV push (peds: 1–2 mEq/kg)
- Activated charcoal slurry: 1–2 g/kg up to 90 g PO

Second Line

- Dextrose: D₅₀W, 1 amp: 50 mL or 25 g (peds: D₂₅W, 2–4 mL/kg) IV
- Diazepam (benzodiazepine): 5–10 mg (peds: 0.2–0.5 mg/kg) IV
- Dopamine: 2–20 μ g/kg/min IV infusion titrated to desired effect
- Intralipid fat emulsion 20%: 1.5 mL/kg IV followed by 0.25 mL/kg/min (experimental for patients refractory to bicarbonate). Call Poison Control Center

for guidance.

- Lorazepam (benzodiazepine): 2–6 mg (peds: 0.03–0.05 mg/kg) IV
- Naloxone (Narcan): 2 mg (peds: 0.1 mg/kg) IV or IM initial dose
- Norepinephrine: 4–12 µg/min (peds: 0.05–0.1 µg/kg/min) IV infusion titrated to desired effect



FOLLOW-UP

DISPOSITION

Admission Criteria

- Symptomatic patients observed > 6 hr
- Altered mental status
- Dysrhythmia or conduction delay
- Seizure
- Heart rate > 100 beats/min 6 hr after ingestion
- Coingestion requiring prolonged observation

Discharge Criteria

- Asymptomatic after 6-hr observation
- No alteration in mental status
- Normal ECG with heart rate < 100 beats/min
- Active bowel sounds; tolerated, activated charcoal
- Psychiatry clearance if there has been suicide attempt or gesture

Issues for Referral

Toxicology or poison center consultation for significant ingestions

FOLLOW-UP RECOMMENDATIONS

Psychiatry for suicide attempts

PEARLS AND PITFALLS

- The hallmark of TCA poisoning is rapid clinical deterioration.
- Vigilant monitoring for QRS widening beyond 120 ms is essential.
- Achieve target pH with hyperventilation in the intubated TCA overdose patient.
- Treat acute widening of the QRS beyond 120 ms with bolus bicarbonate.

ADDITIONAL READING

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- reuptake inhibitors, and atypical antidepressants. In: Erickson TB, Ahrens W, Aks SE, et al., eds. *Pediatric Toxicology*. New York, NY: McGraw-Hill; 2004:297–302.
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See Also (Topic, Algorithm, Electronic Media Element)

Antidepressant Poisoning



CODES

ICD9

969.05 Poisoning by tricyclic antidepressants

ICD10

- T43.011A Poisoning by tricyclic antidepressants, accidental, init
- T43.014A Poisoning by tricyclic antidepressants, undetermined, init

TRIGEMINAL NEURALGIA

Adam Z. Barkin



BASICS

DESCRIPTION

- The trigeminal nerve (cranial nerve [CN] V) innervates the face, oral mucosa, nasal mucosa, and cornea with its sensory fibers
- Trigeminal neuralgia is also known as tic douloureux:
 - _ Tic = spasmodic muscular contraction or movement
 - _ Douloureux = painful
- Usually occurs in patients >50 yr of age
- Facial pain syndrome recognizable by history alone
- Classical:
 - _ Paroxysmal attacks of unilateral (uncommonly bilateral) pain affecting 1 or more divisions of the trigeminal nerve
 - _ Has 1 of the following characteristics:
 - Superficial, sharp, or stabbing pain
 - Precipitated from trigger areas or factors
 - _ Lasts for <1 sec–2 min
 - _ Episodes are stereotyped in each individual
 - _ No clinically evident neurologic deficit
 - _ Not caused by another disorder
- Symptomatic:
 - _ Same as above but a causative lesion (not vascular compression) is identified
- Most common age group is 50–60 yr
- Females > males

ETIOLOGY

- Mechanism of pain production remains controversial; accepted theory suggests:
 - _ Demyelination of CN, leading to ectopic stimulation and pain:
 - Demyelination caused by tortuous or aberrant vascular compression of nerve root
 - 80–90% of classical trigeminal neuralgia have compression
 - Superior cerebellar artery is the most common (75%)
 - Anterior inferior cerebellar artery (10%)
- Secondary causes:
 - _ Herpes zoster
 - _ Multiple sclerosis
 - _ Space-occupying lesions:

- Cerebropontine angle tumor
- Aneurysm
- Arteriovenous malformation

DIAGNOSIS

SIGNS AND SYMPTOMS

- Brief, intense, recurrent sharp pain
- Often described as “electric like”
- Unilateral in the distribution of a branch of the trigeminal nerve:
 - Can occur in all 3 nerves: Maxillary > mandibular > ophthalmic
- More common on right side of face
- May occur without provocation, but triggers can be produced by talking, smiling, chewing, brushing teeth, shaving, or touching the face:
 - Touch and vibration are the most common stimulus
- Can occur infrequently or hundreds of times per day
- No pain between episodes, although chronic cases may complain of a continuous ache

History

- Rule out possible symptomatic causes with the following atypical features:
 - Abnormal neurologic exam
 - Abnormal oral/dental exam
 - Abnormal ear exam or hearing loss
 - Symptoms of dizziness, vertigo, visual changes, or numbness
 - Pain lasting > 2 min
 - Not in trigeminal nerve distribution

Physical-Exam

- Physical exam findings are normal; if abnormality found, consider other cause
- Carefully examine head and neck, with emphasis on CNs
- Patient’s report of pain following stimulation of a trigger point is pathognomonic

ESSENTIAL WORKUP

- Diagnosis is made clinically
- Clinical features to differentiate classical and symptomatic disease:
 - Age on onset < 50 yr
 - Sensory deficits
 - Bilateral involvement

DIAGNOSIS TESTS & INTERPRETATION

Lab

No specific lab tests apply

Imaging

- Patients with characteristic history and normal neurologic exam may be treated without further workup
- If dental problems are suggested, dental radiographs may be useful
- MRI brain/CT head may be useful if multiple sclerosis or tumor is suggested:
 - May be useful in initial presentation

DIFFERENTIAL DIAGNOSIS

- Multiple sclerosis
- Temporomandibular joint syndrome
- Glossopharyngeal neuralgia
- Compression of trigeminal root by tumors
- Dental problems/pain
- Cluster headache
- Postherpetic neuralgia
- Sinusitis
- Otitis media
- Temporal arteritis



TREATMENT

ED TREATMENT/PROCEDURES

- Appropriate pain relief
- Medical therapy:
 - Carbamazepine most commonly used
 - Other antiepileptics show some support as adjuvants for refractory pain.
- May need neurosurgical evaluation for treatment and/or exploration

MEDICATION

First Line

Carbamazepine: 200–800 mg/d PO BID

Second Line

- Gabapentin: Start 300 mg PO QD
- Lamictal: Start 25 mg PO QD
- Oxcarbazepine: 450–1,200 mg PO BID; start 300 mg PO BID
- Phenytoin: 300–400 mg/d div. QD–TID
- Valproic acid: Start 250 mg PO BID



FOLLOW-UP

DISPOSITION

Admission Criteria

- Trigeminal neuralgia with presence of other focal neurologic findings
- Positive CT or MRI studies may require emergent neurologic or neurosurgical consultation
- Refractory or recurrent trigeminal neuralgia not responding to outpatient pain management or anticonvulsant therapy:
 - May require admission for surgical intervention and ablation of the trigeminal nerve

Discharge Criteria

Patients without any focal neurologic findings and improved pain control in the ED may be managed as outpatients.

Issues for Referral

- Surgical therapy may be indicated for those who fail medical treatment
 - Pain relief in 85–90%
- Referral to a pain management center may be helpful in cases of refractory pain
- Anesthetic blocks of the trigeminal ganglion may be helpful

FOLLOW-UP RECOMMENDATIONS

- Follow up with PCP or neurologist for treatment
- Referral to a neurosurgeon may be indicated for refractory pain:
 - Percutaneous vs. open surgical treatment

PEARLS AND PITFALLS

- Unilateral, paroxysmal, and sharp/stabbing facial pain, following a portion of CN V distribution
- Trigger points are pathognomonic
- Do not miss an alternate (nonvascular) cause of nerve compression, such as CNS mass or aneurysm
- Carbamazepine is the most common treatment

ADDITIONAL READING

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- Wolfson AB, Hendey GW, Ling LJ, et al., eds. *Harwood-Nuss' Clinical Practice of Emergency Medicine.* 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2010.



CODES

ICD9

- 053.12 Postherpetic trigeminal neuralgia
- 350.1 Trigeminal neuralgia

ICD10

- B02.22 Postherpetic trigeminal neuralgia
- G50.0 Trigeminal neuralgia

TUBERCULOSIS

Vittorio J. Raho

BASICS

DESCRIPTION

- Tuberculosis (TB) is an infectious disease with protean manifestations, causing significant global morbidity and mortality.

Mechanism

- Infectious droplet nuclei are inhaled through the respiratory tract.
- Bacteria are dispersed through coughing, sneezing, speaking, singing.
- Primary TB/latent TB infection (LTBI):
 - _ Initial infection occurs when organisms enter the alveoli, become engulfed by macrophages, and spread via regional lymph nodes to the bloodstream.
 - _ Patients are usually asymptomatic.
 - _ May be progressive/fatal in immunocompromised hosts.
 - _ Positive reaction to purified protein derivative (PPD) indicates past exposure or infection.
 - _ Negative PPD does not rule out active TB.
 - _ May progress to active TB (5–10%).
- Reactivation TB:
 - _ LTBI becomes active TB.
 - _ Systemic (15%) and pulmonary (85%) symptoms.
- TB affects about one-third of the world's population (90 million new cases in the past decade worldwide, with about 30 million deaths).
- Centers for Disease Control and Prevention (CDC) statistics from 2011 show TB in US at an all-time low.
- TB rates in US have continued to decline since 1993.
- Increase in US foreign-born cases
- Still an estimated 10–15 million people are infected in US alone.

ETIOLOGY

- Infection with *Mycobacterium tuberculosis*, a slow-growing, aerobic, acid-fast bacillus resulting in disease.
- Humans are the only known reservoir.
- Recent TB epidemics:
 - _ HIV-infected patients
 - _ Multidrug-resistant TB (MDR-TB)
 - _ Extensively drug-resistant TB (XDR-TB):
 - High mortality, few effective drugs

SIGNS AND SYMPTOMS

- Depending upon site of infection; all human tissues have potential for infection.
- Pulmonary TB:
 - Cough
 - Fever, night sweats
 - Malaise, weight loss
 - Hemoptysis
 - Pleuritic chest pain
 - Shortness of breath
- Extrapulmonary TB:
 - CNS infections:
 - Meningismus
 - Cranial nerve defects, diplopia
 - Headache, fever, malaise
 - Confusion
 - Acute ischemic stroke
 - Pericarditis:
 - Pleuritic chest pain increased with recumbency
 - Renal infection:
 - Fever
 - Flank pain
 - Spinal TB (Potts disease):
 - Back pain/stiffness, point tenderness
 - Fever
 - Decreased range of motion
 - Cervical lymphadenitis (scrofula):
 - Unilateral, painless
 - May form draining sinus tracts
 - Miliary TB:
 - Multiorgan system involvement
 - Diffuse adenopathy
 - Hepatomegaly
 - Splenomegaly
 - Weight loss, fever

History

Predisposing factors and conditions for TB:

- HIV infection and other immunocompromised states (organ transplant, renal failure, diabetes)
- Drug and alcohol abuse

- Poverty, homelessness (living in shelters)
- Institutionalization (nursing homes, prisons)
- Immigration from an endemic area
- Positive PPD test/previous infection

Physical-Exam

- Fever
- Tachycardia
- Hypoxia
- Cachexia
- Abnormal breath sounds
- Cervical lymphadenopathy

ESSENTIAL WORKUP

- Diagnosis difficult due to the variety of clinical presentations.
- Chest radiography:
 - Most valuable test for active pulmonary TB
- Skin testing: PPD

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC
- Electrolytes, BUN, creatinine, glucose, LFTs
- Hyponatremia (due to syndrome of inappropriate antidiuretic hormone)
- ABGs for oxygenation/ventilation assessment
- Sputum staining for acid-fast bacilli (Ziehl–Neelsen stain):
 - Provides a quick presumptive diagnosis
- Sputum, CSF, blood, urine, or peritoneal fluid culture:
 - Gold standard for diagnosis of TB
 - Average time for positive culture is 3–6 wk.
 - DNA polymerase chain reaction (PCR) testing more rapid
- Lumbar puncture with CSF analysis:
 - For suspected TB meningitis
 - Elevated WBCs with lymphocyte predominance
 - Elevated protein
 - Low to normal glucose

Imaging

- Chest radiograph:
 - May be normal
 - In primary disease, parenchymal infiltrates with unilateral hilar adenopathy are the classic findings.

- Reactivation TB typically appears as cavitary lesions with or without calcification, usually in upper lung segments.
- Miliary TB shows bilateral disseminated 2-mm nodules throughout lungs.
- Chest radiograph may be nondefinitive in AIDS/immunocompromised patients.
- Unilateral pleural effusion in both primary and reactivation TB
- Tracheal deviation with scarring or atelectasis
- Ghon focus—calcified scar/healed primary focus of infection
- Ghon complex—primary infiltrate with associated unilateral hilar adenopathy
- Spine radiographs for Potts disease:
 - May be normal
 - Anterior wedging of 2 involved vertebral bodies and destruction of disk
- CT chest:
 - Better defines extent of disease

Diagnostic Procedures/Surgery

Skin testing:

- Inject 0.1 mL of PPD intradermally in the forearm.
- Positive test indicates prior or current infection with *M. tuberculosis*.
- Test results are read between 48 and 72 hr after administration.
- Interpretation of positive: > 5-mm induration:
 - Close contacts with TB patients
 - Positive chest radiographs for TB
 - HIV-positive
 - Organ transplant or other immunosuppression
- > 10-mm induration:
 - IV drug users
 - Immigrants from high-prevalence countries (within 5 yr)
 - Underlying disease (diabetes, renal failure, malignancies)
 - Healthcare workers
 - Prison inmates
 - Institutionalized (nursing home, homeless shelters)
- > 15-mm induration:
 - Low-risk individuals

DIFFERENTIAL DIAGNOSIS

- Bacterial pneumonia
- Bronchiectasis
- Coccidiomycosis
- Histoplasmosis
- Lung abscess
- Lung carcinoma

- Lymphoma
- *Pneumocystis carinii* pneumonia
- Pulmonary embolism
- Sarcoidosis



TREATMENT

PRE HOSPITAL

- Place patient in respiratory isolation (negative flow).
- Place a mask on the patient to prevent respiratory spread of the disease.
- Initiate treatment with an IV, oxygen, and pulse oximetry.
- Endotracheal intubation may be required in patients with severe hemoptysis or respiratory compromise.
- Providers should wear submicron particulate filter masks (N-95 designation).
- Inform close contacts.

INITIAL STABILIZATION/THERAPY

- ABCs:
 - Control airway as needed.
 - Administer oxygen as needed.
 - Place on patient cardiac monitor and pulse oximetry.
 - Establish IV access with 0.9% normal saline
- Isolate patients in negative pressure rooms with at least 6 air exchanges per hour.
- Protection for healthcare workers (N-95 masks)

ED TREATMENT/PROCEDURES

- *Isolation* and strict respiratory precautions
- Treatment is augmented due to increasing multidrug resistance.
- Any regimen must contain at least 2 drugs to which the TB bacillus is susceptible.
- CDC currently recommends initial therapy that includes 4 1st-line drugs.
- LTBI with normal chest x-ray given isoniazid (INH) for 9 mo or weekly combination of INH and rifapentine (RPT) for 12 wk.
- Consult infectious disease specialists when treating HIV patients on antiretroviral therapies.
- Add dexamethasone for TB meningitis.
- Surgical drainage for TB empyema may be necessary; consult thoracic surgeon.
- Directly observed therapy (DOT) may be necessary to ensure compliance in certain populations.
- Intermittent (biweekly) regimen may demonstrate higher patient compliance.

MEDICATION

First Line

- INH: 5 mg/kg, max. 300 mg (peds: 10–15 mg/kg, max. 300 mg) PO/IM per day:
 - Refractory seizures in overdose, treat with pyridoxine 5 g IV over 5 min or PO
 - Caution with alcohol coingestion, hepatitis
- Rifampin (RIF): 10 mg/kg, max. 600 mg (peds: 10–20 mg/kg, max. 600 mg) PO/IV per day
- Pyrazinamide (PZA): 20–25 mg/kg/d max. 2 g (peds: 15–30 mg/kg/d) or:
 - <55 kg: 1 g PO per day
 - 56–75 kg: 1.5 g PO per day
 - >75 kg: 2 g PO per day
 - Not recommended in pregnancy
- Ethambutol (ETB): 15–20 mg/kg, max. 1,600 mg (peds: 15–30 mg/kg, max. 1 g) PO per day or up to TID
 - Not recommended <13 yr old, requires visual testing
- RPT: 10 mg/kg, max. 900 mg (peds: Not recommended <12 yr old) PO once per week or 300 mg PO weekly for 10–14 kg, 450 mg PO weekly for 14.1–25 kg, 600 mg PO weekly for 25.1–32 kg, 750 mg PO weekly for 32.1–49.9 kg, 900 mg PO weekly for >50 kg
- Rifabutin: 5 mg/kg, max. 300 mg (peds: Unknown) PO per day

Second Line

(Less effective, more toxic)

- Streptomycin: 15 mg/kg/d, max. 1 g (peds: 20–40 mg/kg/d) IM/IV per day:
 - Teratogenic: Contraindicated in pregnancy
- Ethionamide: 0.5–1 g (peds: 10–20 mg/kg/d) PO div. QID
- Levaquin: 750 mg (peds: Contraindicated) PO/IV per day



FOLLOW-UP

DISPOSITION

Admission Criteria

- Respiratory compromise
- Suspicion of diagnosis
- Inability to comply with outpatient therapy
- Unavailable outpatient resources (no PCP)
- Involuntary admission for noncompliant outpatients occurs:
 - Be aware of respective state laws concerning involuntary admission (consult infectious disease specialist).

Discharge Criteria

- Without respiratory compromise
- Home isolation procedure compliance
- Ability and willingness to comply with long-term therapy
- Appropriate outpatient follow-up and treatment available
- Notification of the public health authorities is mandatory.

Issues for Referral

Referral to Department of Public Health for DOT

FOLLOW-UP RECOMMENDATIONS

- Sputum analysis periodically to document clearance
- Medication toxicity monitoring:
 - _ INH, RIF, PZA: Monitor liver function tests for hepatitis
 - _ PZA: Check uric acid levels
 - _ ETB: Eye testing for color blindness

PEARLS AND PITFALLS

- Early isolation and respiratory precautions
- Careful history to establish risk factors
- The chest x-ray and PPD are great diagnostic aids.
- Initial 4-drug regimen for active disease
- Nonadherent, active TB patients are considered a public health hazard:
 - _ Specific state laws are applicable in numerous areas.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Pneumonia, Adult
- Bronchiectasis
- Coccidiomycosis
- Histoplasmosis
- Lymphoma
- *Pneumocystis carinii* Pneumonia
- Pulmonary Embolism
- Sarcoidosis



CODES

ICD9

- 010.90 Primary tuberculous infection, unspecified, unspecified
- 011.90 Unspecified pulmonary tuberculosis, unspecified
- 795.51 Nonspecific reaction to tuberculin skin test without active tuberculosis

ICD10

- A15.7 Primary respiratory tuberculosis
- A15.9 Respiratory tuberculosis unspecified
- R76.11 Nonspecific reaction to skin test w/o active tuberculosis

TULAREMIA

Scott Bentz

BASICS

DESCRIPTION

- Tularemia is an acute febrile illness caused by the small aerobic gram-negative pleomorphic intracellular *coccobacillus Francisella tularensis*:
 - _ Organism is highly infectious.
 - _ Person-to-person transmission has not been reported.
- Humans become infected through different environmental exposures:
 - _ Bites from infected tick, deerfly, mosquito, or other infected insect
 - _ Direct contact with infectious animal tissue or fluid
 - _ Contact with or ingestion of contaminated food, water, or soil
 - _ Inhalation of infected aerosols (e.g., cutting grass with power mowers, which may aerosolize the organism)
- The 4 major strains of the bacterium have different virulence and geographic location:
 - _ 2 subspecies cause human infection in North America: *F. tularensis* subspecies tularensis (type A, more virulent) and *F. tularensis* subspecies holartica (type B, less virulent)
- Natural hosts:
 - _ Lagomorphs and other rodents
 - _ Found in species of wild animals (insects, rabbits, hares, ticks, flies, muskrats, beavers, mice), domestic animals (sheep, cattle, cats), ticks, and water and soil contaminated by infected animals
- Natural vectors:
 - _ Ticks
 - _ Biting flies
 - _ Mosquitoes
 - _ Wild rabbits
- Weaponization of tularemia was accomplished during the Cold War:
 - _ Because of its virulence and ability to be aerosolized, it remains a potential biologic agent for mass destruction.
- Lab technicians handling culture specimens are at high risk:
 - _ *F. tularensis* cultures should be manipulated only in a biosafety level 3 facility.
- Also known as “rabbit fever” or “deerfly fever”

ETIOLOGY

- Individuals who spend time outdoors in endemic areas are at higher risk:

- Farmers
- Hunters
- Forest workers
- Those who handle animal carcasses are at highest risk (taxidermists and butchers).
- Two-thirds of cases occur in males.
- Although tularemia can occur worldwide, it is endemic in the northern hemisphere:
 - Reported nationwide except in Hawaii
 - States with the highest incidence include Missouri, Arkansas, Kansas, South Dakota, and Oklahoma.
 - Few hundred cases annually in US, although probably underreported
 - Peak season is June–October.
- Mortality is 5–15%. Appropriately treated patients have mortality as low as 1%.

Pediatric Considerations

- 25% of cases occur in children 1–14 yr of age.
- Children who spend time outdoors in endemic rural areas are at highest risk.



DIAGNOSIS

SIGNS AND SYMPTOMS

- Tularemia has different presentations based on route of entry:
 - Primary route of entry is through skin; most often a cutaneous ulcer develops.
- Incubation is 3–5 days, range 1–14 days. Lesion usually begins as papule, often with fever.
- 6 forms of illness:
 - Ulceroglandular:
 - Most common presentation (70–80% of cases)
 - Inoculated cutaneously (scratch, abrasion, insect bite) with as few as 50 organisms
 - Initially, a local cutaneous papule at point of entry
 - Followed by tender regional adenopathy and constitutional symptoms to include fever, chills, myalgias, and headaches
 - Associated with pneumonia in 30% of cases
 - Glandular:
 - Rare form
 - Gains access to lymphatic system or bloodstream through inapparent abrasion
 - Tender regional lymphadenopathy with no local lesions
 - Oculoglandular:
 - Rare form

- Organism enters through a splash of infected blood/fluid to the eye or is introduced by eye rubbing after handling infectious materials (e.g., rabbit carcass).
- Edema, conjunctivitis, injection, chemosis with periauricular, submandibular, or cervical lymphadenopathy
- Pharyngeal:
 - Rare form
 - From ingestion of contaminated food or water
 - Severe throat pain with exudative pharyngitis and regional lymphadenitis
- Pneumonic:
 - Secondary to inhalation
 - Seen in sheep shearers, farmers, landscapers, and lab technicians
 - Fever, dry cough, and pleuritic chest pain develop.
 - Pneumonia can occur in 30% of patients with ulceroglandular tularemia
- Typhoidal:
 - Historically, the typhoidal form defined as devoid of skin or mucous membrane lesion or remarkable lymph node enlargement.
 - No known point of entry (probably oral or respiratory).
 - Only when no route of infection can be established may the term still be acceptable.
 - In North America, where type A is prevalent, fulminant manifestations are reported, including severe sepsis, meningitis, endocarditis, hepatic failure, and renal failure.
 - Septicemia associated with type A tularemia is usually extremely severe and potentially fatal. High fever, abdominal pain, and diarrhea may occur early in the course of disease.

History

- Exposure and epidemiologic risk factors can be helpful.
- Sudden fever, chills, headaches
- Progression of components of signs and symptoms may be useful in defining form of illness.

Physical-Exam

- Fever
- Tender, well-demarcated cutaneous ulcer
- Tender regional lymphadenopathy; lymph nodes can develop fluctuance and spontaneously drain.
- Exudative pharyngitis (with pharyngeal tularemia)
- Ulcerations of the conjunctiva with pronounced chemosis (with oculoglandular

tularemia)

DIAGNOSIS TESTS & INTERPRETATION

Lab

- No rapid diagnostic test available
- Routine lab studies nonspecific:
 - CBC can be normal.
 - ESR might be slightly elevated.
 - CSF: May have increased protein or mild pleocytosis
 - LFTs are often abnormal.
- Gram stain, cultures, and tissue biopsies:
 - Often negative
- Blood cultures usually negative because of specific growth requirements
- Enzyme-linked immunosorbent assay and polymerase chain reaction are available through reference labs.
- Serum antibody titers:
 - Typically do not reach diagnostic levels until ≥ 10 days after the onset of illness
 - A single titer of at least 1:160 for tube agglutination is diagnostic for *F. tularensis* infection.
 - May not be elevated before day 11 of illness and generally are diagnostic after 16th day.

Imaging

- Chest radiograph for:
 - Consolidative process, pleural effusions, and hilar adenopathy
- CT scan of chest for:
 - Severe pulmonary symptoms
 - Other possible etiologies of atypical pneumonia

DIFFERENTIAL DIAGNOSIS

- Ulceroglandular tularemia mimics include:
 - Tuberculosis
 - Cat scratch disease
 - Syphilis
 - Chancroid
 - Lymphogranuloma venereum
 - Toxoplasmosis
 - Sporotrichosis
 - Rat-bite fever
 - Anthrax
- Oculoglandular tularemia mimics include:

- Adenoviral infection
- Pharyngeal tularemia mimics include:
 - Diphtheria
 - Bacterial pharyngitis
 - Infectious mononucleosis
 - Adenoviral infection
- Typhoidal tularemia mimics include:
 - Salmonellosis
 - Brucellosis
 - Legionnaire disease
 - Q fever
 - Malaria
 - Disseminated fungal or mycobacterial infections
- Pulmonary tularemia mimics include:
 - Mycoplasmal infection
 - Legionnaire disease
 - Chlamydial infection
 - Tuberculosis



TREATMENT

PRE HOSPITAL

- Universal precautions
- Management of ABCs
- Treat dehydration/hypotension with boluses of normal saline.

INITIAL STABILIZATION/THERAPY

- ABCs
- Supplemental oxygen for hypoxia
- Fluid resuscitation with normal saline for intravascular volume depletion or septic shock
- Central line access for unstable patients
- Vasopressors for persistent hypotension

ED TREATMENT/PROCEDURES

- Fever control with acetaminophen
- Early administration of antibiotic therapy after obtaining cultures
- Antibiotic options:
 - 1st-line agents: Streptomycin or gentamicin continued for 10 days
 - Ciprofloxacin if community-acquired pneumonia is in the differential diagnosis of patients ≥ 18 yr of age
 - Tetracycline or doxycycline in those > 8 yr of age; or chloramphenicol:

- Continue for 14 days, since these drugs are only bacteriostatic.
- Associated with a higher rate of treatment failures than the previously mentioned antibiotics
- 3rd tier of treatment, since they are static
- *F. tularensis* is resistant to β -lactam drugs and carbapenems

Pediatric Considerations

Streptomycin and gentamicin are recommended as 1st-line agents.

MEDICATION

First Line

- Gentamicin: 5 mg/kg IV or IM q24h (peds: 2.5 mg/kg IV or IM q8h) \times 10 days
- Streptomycin: 1 g IM (peds: 15 mg/kg, not to exceed 2 g/d) q12h \times 10 days

Second Line

- Ciprofloxacin: 400 mg IV q12h \times 10 days
- Doxycycline: 100 mg (peds: If weight \geq 45 kg and child $>$ 8 yr, 100 mg; if weight \leq 45 kg and child $>$ 8 yr, 2.2 mg/kg) IV q12h for at least 14 days (longer treatment needed since doxycycline is bacteriostatic); max. 200 mg/d
- Chloramphenicol is usually avoided due to the possibility of adverse reactions. However, chloramphenicol may be considered in cases of tularemic meningitis due to its ability to cross the blood–brain barrier and reach higher concentrations in the CSF.



FOLLOW-UP

DISPOSITION

Admission Criteria

- ICU admission for advanced age, neutropenia, severe hypoxemia, hemodynamic instability, or patients presenting with typhoidal tularemia
- Inpatient floor bed admission for mild to moderate illness:
 - Isolation bed required only for the purpose of ruling out other etiology (e.g., tuberculosis)

Discharge Criteria

Outpatient therapy: Oral or IM therapy for mild illness with close follow-up

Issues for Referral

Critical care and infectious disease consultation to assist in assessment of differential considerations and manage life-threatening complications

FOLLOW-UP RECOMMENDATIONS

Infectious disease consultation to manage ongoing treatment and reduce subsequent exposures

PEARLS AND PITFALLS

- Patients presenting with high fever and regional lymphadenopathy, especially if there is an ulcer or conjunctivitis, should have tularemia in the differential.
- Epidemiology may be useful in pointing to this diagnosis.
- Definitive diagnosis ultimately based upon serology, which usually isn't positive until > 10 days of infection.
- Vaccine currently under review by FDA; not currently available in US
- Currently listed as category A (critical agent of concern) bioterrorism agent because of pathogenicity. It can be disseminated via dispersal in food, water, or air.

ADDITIONAL READING

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- Centers for Disease Control and Prevention. Available at www.cdc.gov/tularemia. Accessed on January 2011.
- Hofinger DM, Cardona L, Mertz GJ, et al. Tularemic meningitis in the United States. *Arch Neurol*. 2009;66(4):523–527.
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- Treat JR, Hess SD, McGowan KL, et al. Ulceroglandular tularemia. *Pediatr Dermatol*. 2011;28(3):318–320.
- World Health Organization Guidelines on Tularemia, 2007.



CODES

ICD9

- 021.0 Ulceroglandular tularemia
- 021.3 Oculoglandular tularemia
- 021.9 Unspecified tularemia

ICD10

- A21.0 Ulceroglandular tularemia
- A21.1 Oculoglandular tularemia
- A21.9 Tularemia, unspecified

TUMOR COMPRESSION SYNDROMES

Hany Y. Atallah

BASICS

DESCRIPTION

- Complications arising from the compression of neural or vascular structures by solid tumors or their direct infiltration of such structures
- Spinal cord compression:
 - Affects over 20,000 patients each year
 - Occurs in 5–14% of cancer patients
 - More than 50% of cases are metastases from lung, breast, or prostate cancer.
 - Vertebral metastases are far more common than epidural spinal cord compression (ESCC).
 - Approximately 20% of cases of ESCC represent the initial manifestation of malignancy.
- Other neurologic tumor compression:
 - Brachial plexus
 - Recurrent laryngeal nerve compression by mediastinal lymph nodes
- Superior vena cava (SVC) syndrome:
 - Obstruction of returning blood flow in the SVC by compression, infiltration, or thrombosis
 - Venous hypertension within the area ordinarily drained by the SVC
 - In severe cases, gradual elevation of the intracranial pressure (ICP), with altered mental status and coma
 - 60–85% caused by malignancy

ETIOLOGY

- Spinal cord compression:
 - Prostate cancer
 - Breast cancer
 - Lung cancer
 - Renal cell carcinoma
 - Multiple myeloma
 - Melanoma
 - Thyroid cancer
 - Lymphoma
 - Sarcoma
- Brachial plexus compression:
 - 0.4% of cancers

- 2–5% of those who receive radiation treatment
- Lung cancer
- Breast cancer
- SVC syndrome from tumor compression:
 - Lung cancer (most common):
 - Small cell lung cancer primarily
- Postirradiation fibrosis
- Lymphoma
- Breast cancer
- Testicular cancer
- See “Differential Diagnosis” for non malignant etiologies of the SVC syndrome.

Pediatric Considerations

In children with spinal cord compression, common causes are sarcoma, neuroblastoma, germ cell tumors, and lymphoma.

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Spinal cord compression:
 - History of malignancy
 - Back or neck pain:
 - Prolonged
 - Worse with rest
 - Most commonly affects the thoracic spine
 - Paresthesias
 - Difficulty ambulating
 - Constipation
 - Urinary retention
 - Urinary or fecal incontinence
 - Weight loss
- Brachial plexus compression:
 - Neuropathic pain involving the medial aspect of the upper extremity
- Intrathoracic vagal nerve compression:
 - Ipsilateral aching facial pain around the ear
- SVC syndrome:
 - Orthopnea
 - Dyspnea
 - Tightness of the shirt collar
 - Cough

- Chest pain
- Headache
- Facial swelling
- Head fullness
- Blurred vision
- Dizziness
- Syncope

Physical-Exam

- Spinal cord compression:
 - Loss of rectal tone
 - Loss of anal wink
 - Weakness in 60–85% of patients
 - Sensory findings less common
- Laryngeal nerve compression:
 - Hoarseness
 - Vocal cord paralysis
- Brachial plexus:
 - Ulnar paresthesias
 - Weakness and wasting of intrinsic hand muscles
 - Pan-plexopathy
 - Horner's syndrome
- SVC syndrome:
 - Periorbital edema
 - Conjunctival suffusion
 - Facial swelling
 - Facial plethora
 - Upper extremity edema
 - Findings exacerbated by recumbent or stooped-over position
 - Usually worse in the early morning hours
 - ICP may be elevated in severe cases:
 - Altered mental status
 - Coma
 - Papilledema

DIAGNOSIS TESTS & INTERPRETATION

Imaging

- Chest radiograph:
 - Spinal cord compression:
 - May identify a primary lung tumor
 - Helpful in excluding tuberculous spondylitis
 - SVC compression:

- Mass present in 10%
- Pleural effusion in 25%
- Plain spinal radiography
- Will show 85% of metastases causing compression
- A normal spine (or 1 showing just degenerative changes) on plain radiology does not exclude the diagnosis of possible cord compression.
- CT:
 - Contrast CT is more sensitive and specific than plain radiography and radionucleotide imaging in distinguishing benign from malignant disease in spinal compression syndrome
 - May identify mass and impingement in vena cava obstruction
- MRI:
 - Study of choice for spinal cord compression
 - Indicated in patients with back or neck pain and:
 - History of cancer
 - Bowel or bladder dysfunction
 - Lower extremity weakness
 - Sensory loss
 - Saddle anesthesia

Diagnostic Procedures/Surgery

- CT myelography:
 - Indicated for spinal cord compression when MRI is unavailable or contraindicated (pacemaker, metallic implants, severe claustrophobia)
- Minimally invasive techniques can often be used to establish a tissue diagnosis in cases of SVC syndrome.
- Occasionally an invasive procedure is required to obtain a tumor biopsy in patients with SVC syndrome:
 - Bronchoscopy
 - Mediastinoscopy
 - Scalene node biopsy
 - Limited thoracotomy
 - Video-assisted thoracic surgery (VATS)
- Radiation therapy (RT) can be done to shrink the tumor:
 - Should be done after tissue diagnosis is made, as RT can obscure tissue and make definitive diagnosis difficult.
- Endovascular stents can be used to achieve more rapid relief than can be achieved using RT.

DIFFERENTIAL DIAGNOSIS

Spinal Cord Compression

- Amyotrophic lateral sclerosis

- Arteriovenous malformations
- Epidural abscess
- Intervertebral disk disease
- Multiple sclerosis
- Neurologic diseases
- Osteoporotic vertebral fractures
- Primary bone tumors
- Spinal infarction
- Spondylitis
- Spondylosis
- Transverse myelitis

Superior Vena Cava Syndrome

- Pericardial tamponade
- Nephrotic syndrome
- Cor pulmonale
- Cirrhosis
- Nonmalignant etiologies of SVC syndrome:
 - Goiter
 - Pericardial constriction
 - Primary thrombosis
 - Idiopathic sclerosing aortitis
 - Tuberculous mediastinitis
 - Fibrosing mediastinitis
 - Histoplasmosis
 - Indwelling central venous catheters



TREATMENT

INITIAL STABILIZATION/THERAPY

- Early diagnosis and treatment are the keys to an improved outcome.
- Level of neurologic dysfunction on presentation is a key factor in the prognosis for spinal cord compression.
- Avoid IV line placement in upper extremities if severe SVC compression is present.

ED TREATMENT/PROCEDURES

Spinal Cord Compression

- Corticosteroids (dexamethasone):
 - Administer in ED.
 - Higher doses alleviate the pain more rapidly, but studies indicate no significant difference in outcome with regard to sphincter function or

ambulation between the dose schedules.

- Radiotherapy:
 - Definitive treatment modality
 - Pain medication with narcotics
 - Oncology, radiotherapy, and neurosurgical consultation for further management of tumor/malignancy
 - Consider empiric broad-spectrum antibiotics prior to the MRI if an epidural abscess is being considered.
- Urgent neurosurgical consultation

SVC Compression

- Manage the underlying malignancy with either radiotherapy or chemotherapy.
- Elevation of the head of the bed.
- Supplemental oxygen
- Administer steroids if there is respiratory compromise
- Judicious use of diuretics may transiently improve symptoms, but there is poor evidence to support efficacy.
- Urgent oncology referral
- Intravascular stents can relieve the obstruction more rapidly.

MEDICATION

- For ESCC there is limited evidence suggesting steroids are beneficial, but it is still generally considered to be part of the standard regimen of treatment
- For paresis or paraplegia high dose dexamethasone: 1 mg/kg loading dose, then halve the dose every 3 days
- For patients with minimal neurologic dysfunction dexamethasone 10 mg followed by 16 mg daily initially in divided doses with a gradual taper once definitive treatment is underway
- For SVC syndrome steroids can reverse symptoms from steroid responsive malignancies such as lymphoma or thymoma.
- In patients undergoing RT steroids are often prescribed to prevent swelling
- Furosemide (Lasix): No prior use—40 mg IVP; prior use—double 24 hr dose (80–180 mg IV)
- Hydrocodone/acetaminophen: 5/500 mg PO q4–6h
- Oxycodone/acetaminophen: 5/500 mg PO q4–6h



FOLLOW-UP

DISPOSITION

Admission Criteria

- Admission is advisable for all patients presenting with a tumor compression

syndrome.

- Transfer to a center with neurosurgical capabilities may be needed for patients with spinal cord compression.

Discharge Criteria

None

Issues for Referral

- Radiation oncology should be consulted for patients presenting with tumor compression.
- Early neurosurgical consultation for patients with spinal cord compression

PEARLS AND PITFALLS

- Average life expectancy among patients who present with malignancy-associated SVC syndrome is ~6 mo.
- Presentations may be subtle and compression syndromes should always be considered in patients with known malignancy and unexplained complaints.

ADDITIONAL READING

- Cole JS, Patchell RA. Metastatic epidural spinal cord compression. *Lancet Neurol*. 2008;7(5):459–466.
- Graham PH, Capp A, Delaney G, et al. A pilot randomized comparison of dexamethasone 96 mg vs 16 mg per day for malignant spinal-cord compression treated by radiotherapy: TROG 01.05 Superdex study. *Clin Oncol (R Coll Radiol)*. 2006;18:70–76.
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CODES

ICD9

- 239.9 Neoplasm of unspecified nature, site unspecified
- 336.9 Unspecified disease of spinal cord
- 459.2 Compression of vein

ICD10

- D49.9 Neoplasm of unspecified behavior of unspecified site
- G95.29 Other cord compression
- I87.1 Compression of vein

TYMPANIC MEMBRANE PERFORATION

Andrew K. Chang • Michelle M. Davitt

BASICS

DESCRIPTION

Perforations can be classified in several ways:

- **Duration:**
 - Acute (< 3 mo)
 - Chronic (> 3 mo)
- **Site:**
 - Pars tensa
 - Pars flaccida
- **Extent:**
 - Limited to 1 quadrant (< 25%)
 - 2 or more quadrants
 - Total perforation

ETIOLOGY

- **Infection (acute otitis media):**
 - Most common cause of an acute perforation
- **Blunt trauma (slap to the ear):**
 - Domestic violence, street fight
- **Penetrating trauma (Q-tip)**
- **Extrusion of tympanostomy tubes**
- **Rapid pressure change (diving, flying):**
 - Rupture usually occurs between 100 and 400 mm Hg (at a depth of 2.6 ft, there is a pressure differential of 60 mm Hg)
- **Extreme noise (blast)**
- **Lightning**
- **Acute necrotic myringitis (β -hemolytic streptococcus)**
- **Slag burns (welding or metalworking)**
- **Complications of surgical procedures:**
 - Myringotomy, tympanoplasty, tympanostomy tube insertion

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Ear pain (mild)

- Severe pain or complete hearing loss in the affected ear suggests additional injuries
- Tinnitus
- Vertigo (especially if perforation occurs in water)

Physical-Exam

- Loss of hearing (partial)
- Purulent or bloody discharge from ear canal
- Insufflation via pneumatic otoscope:
 - Small perforations may be evident only as an immobile tympanic membrane
 - Holding pressure for 15 sec (the fistula test) may cause nystagmus or vertigo if the pressure is transmitted through the middle ear and into a labyrinthine fistula
- Weber test (tuning fork on midline bone):
 - Sound should be equal or louder in the injured ear, consistent with decreased conduction
 - Sound localizing to the opposite side of injury indicates possible otic nerve injury
- Rinne test (tuning fork on mastoid process):
 - Usually normal (air conduction detected after bone conduction fades) or shows a small conductive loss

ESSENTIAL WORKUP

Clinical exam:

- Direct visualization of tympanic membrane with otoscope
- Test hearing in both ears
- Note any nystagmus with changes of position or pressure on the tragus occluding the canal (fistula sign)

DIAGNOSIS TESTS & INTERPRETATION

Lab

If an aural drainage is present, it may be desirable to culture the drainage

Imaging

Cranial CT:

- Obtain if clinically indicated to rule out temporal bone fracture

DIFFERENTIAL DIAGNOSIS

- Temporal bone fracture
- Serous otitis media
- Infectious otitis media

- Otitis externa
- Cerumen impaction
- Barotrauma
- Acoustic trauma
- Foreign body
- Child abuse



TREATMENT

INITIAL STABILIZATION/THERAPY

ABCs of trauma care:

- Immobilize cervical spine and investigate for intracranial injury when indicated

ED TREATMENT/PROCEDURES

- Remove debris from the ear canal:
 - Do not irrigate because this may force more debris into the middle ear
 - If the tympanic membrane is not visible because of impacted cerumen and suspicion for perforation is high, remove cerumen by manual disimpaction or suctioning
- If clinically indicated, obtain CT scan to rule out temporal bone fracture
- Prophylactic antibiotics are not indicated
- Prescribe antibiotics if there is evidence of infection or if water or contaminants may have entered the ear canal:
 - Amoxicillin
 - Augmentin
 - Cefixime, ceftriaxone
 - Azithromycin
 - Clindamycin
- Analgesics if needed for pain
- With the exception of fluoroquinolones, the use of ototopical medication is controversial because of the risk of ototoxicity:
 - Most advocate an antibiotic–cortisone otic medication whenever a discharge is present because this may treat or prevent an external canal infection and hasten the resolution of the middle-ear infection
 - Ototopical antibiotics provide a high concentration of antibiotic in the middle ear, potentially exceeding the MIC of organisms
 - Ototopical fluoroquinolones are the 1st-line therapy for chronic suppurative otitis media and in traumatic TM perforation with suspected entry of water into the middle ear (SCUBA, bathing, etc.)
- Urgent ENT consultation (indications):
 - Vertigo
 - Sensorineural hearing loss

- Severe tinnitus
- Active and significant bleeding
- Facial paralysis

MEDICATION

- Amoxicillin: 500 mg PO TID (peds: 80–90 mg/kg/24 h PO BID) for 7–10 days
- Augmentin: 875 mg (peds: 90 mg/kg/24 h) PO BID for 7–10 days
- Cefixime: 400 mg (peds: 8 mg/kg/24 h) PO QD for 7–10 days
- Ceftriaxone: 1–2 g IV/IM (peds: 50 mg/kg IM, max. 1 g) × 1 dose
- Azithromycin: 2 g (peds: 30 mg/kg, max. 1,500 mg) PO × 1 dose
- Clindamycin: 150–450 mg (peds: 30 mg/kg/24 h) PO QID for 7–10 days
- Ciprofloxacin/dexamethasone otic: 4 drops BID for 7–10 days
- Neomycin/polymyxin B/hydrocortisone otic suspension: 4 gtts in ear TID–QID (peds: 3 gtts TID–QID); max. 10 days; lie with affected ear upward for 5 min

First Line

- Amoxicillin and Augmentin are the primary antibiotic choices for acute otitis media with subsequent tympanic membrane perforation
 - Augmentin should be selected for patients with recurrent infections or those who have used antibiotics within 1 mo
 - Ciprofloxacin with dexamethasone otic drops are the medications of choice for chronic suppurative otitis media and traumatic tympanic membrane perforation with suspicion of water or contaminant entry into the middle ear (SCUBA, bathing, Q-tip)

Second Line

- Penicillin-allergic patients may be prescribed cephalosporins:
 - Ceftriaxone IM may be preferred in patients with vomiting or compliance issues
- Azithromycin or clindamycin may be used for patients with hypersensitivity type I allergic reaction to penicillin



FOLLOW-UP

DISPOSITION

Admission Criteria

- Associated injuries requiring admission
- Severe vertigo impairing ambulation

Discharge Criteria

Almost all patients will be discharged

Issues for Referral

- Arrange outpatient ENT follow-up within 1 wk:
 - After detailed exam and formal audiometric tests, most otolaryngologists practice “watchful waiting” because most tympanic membrane perforations heal spontaneously
 - Hearing loss increases with the size of the perforation and does vary appreciably with the location
 - Operative repair (patch or tympanoplasty) is reserved for the 10–20% that do not heal spontaneously

FOLLOW-UP RECOMMENDATIONS

- Provide detailed discharge instructions:
 - Occlude the ear canal with cotton coated in petroleum jelly or antibiotic ointment when showering to prevent entry of water into the middle ear, which can be painful and may cause further infection
 - Swim only with fitted earplugs
 - Avoid forceful blowing of the nose
- Expected outcome:
 - Most perforations heal spontaneously in a few days to several months; in 1 study of children, 70% closed within 1 wk and 94% closed within 1 mo
 - Spontaneous healing is associated with the perforation size, etiology, and whether it is dry or serosanguinous
 - Wet perforations tend to heal more quickly
 - Perforations caused by molten metal or electrical burns are less likely to heal spontaneously
 - Complications include:
 - Infection
 - Dislocation of ossicles
 - Perilymph leak
 - Cholesteatoma

PEARLS AND PITFALLS

- Acute otitis media is the most common cause of tympanic membrane perforation.
 - Small tympanic membrane perforations may be diagnosed only through insufflation by pneumatic otoscope
 - Debris, cerumen, or discharge should be suctioned or manually removed; irrigation is contraindicated in cases of suspected tympanic membrane perforation
 - Otological fluoroquinolones are the antibiotics of choice for chronic suppurative otitis media and traumatic tympanic membrane perforation with suspected penetration by foreign body, water, or contaminant
 - Most perforations heal spontaneously; however, care must be exercised to

prevent further introduction of infectious agents into the open middle ear

- Chronic otitis media is a recurrent infection of the middle ear and/or mastoid air cell tract in the presence of a tympanic membrane perforation
- Chronic suppurative otitis media is diagnosed when there is a persistent purulent drainage through a perforated tympanic membrane for > 6 wk
- Use neomycin/polymyxin B/hydrocortisone otic SUSPENSION (not solution) when treating otitis externa in the setting of TM perforation

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Barotrauma
- Otitis Media



CODES

ICD9

- 384.20 Perforation of tympanic membrane, unspecified
- 384.22 Attic perforation of tympanic membrane
- 872.61 Open wound of ear drum, without mention of complication

ICD10

- H72.10 Attic perforation of tympanic membrane, unspecified ear
- H72.90 Unsp perforation of tympanic membrane, unspecified ear
- S09.20XA Traumatic rupture of unspecified ear drum, initial encounter

ULTRAVIOLET KERATITIS

Yasuharu Okuda • Nicholas Genes



BASICS

DESCRIPTION

- Corneal epithelial damage caused by direct exposure to ultraviolet (UV) light.
- Also known as photokeratitis, UV conjunctivitis, snow blindness, and welder's flash.

ETIOLOGY

- Work-related exposures seen in welders, electricians, and mechanics
- Recreational exposures, including water sports, snow sports, and tanning booths
- Occurs with corneal absorption at 290 nm, the cutoff between UV-B and UV-C light
- UV light penetrates to epithelial nociceptor axons, destroying them and triggering pain from subendothelial nerve stimulation
- Related to intensity and duration of exposure



DIAGNOSIS

SIGNS AND SYMPTOMS

- Patients will present with bilateral eye pain, photophobia, redness, and tearing.
- No purulent discharge will be present.
- Associated facial edema, lid edema, erythema, and blepharospasm may be present.

History

- Elicit history of exposure to UV light 6–12 hr prior to complaint of pain.
- In addition to pain, complaints may include:
 - Photophobia
 - Tearing
 - Foreign-body sensation

Physical-Exam

- Visual acuity may be mildly diminished.
- Eye exam reveals chemosis, injection, tearing.
- Slit-lamp exam with topical ophthalmic anesthetics and fluorescein:
 - Multiple superficial punctate corneal lesions
 - Otherwise unremarkable

ESSENTIAL WORKUP

- Accurate history including:

- Type, timing, and duration of exposure
- Visual acuity
- Complete ocular exam including:
 - Extraocular movements
 - Exam of conjunctiva/sclera/cornea with fluorescein
 - Anterior chamber checking for cell and flare
 - Eversion of lids to check for foreign bodies

DIAGNOSIS TESTS & INTERPRETATION

Lab

Blood testing will not be necessary unless widespread severe sunburn is present.

Imaging

A careful history should obviate need for orbital US/CT/MRI for foreign body.

DIFFERENTIAL DIAGNOSIS

- Infection:
 - Bacterial or viral conjunctivitis
 - Corneal ulcers
- Allergic conjunctivitis
- Corneal abrasion
- Traumatic iritis
- Foreign bodies
- Acid, alkali, or thermal burns



TREATMENT

PRE HOSPITAL

When diagnosis is unambiguously established, pressure patching or applying mild pressure to eyes with closed lids may provide temporary relief.

ED TREATMENT/PROCEDURES

- Topical anesthetic to facilitate slit-lamp exam.
- Provide adequate oral analgesia as needed.
- Apply topical antibiotic ointment.
- Initiate short-acting cycloplegic agent.
- May apply eye patching for comfort (patching has not been shown to accelerate healing):
 - Soft double patching with mild pressure
 - If both eyes involved, either patch both eyes or patch the eye that is more severely affected.

MEDICATION

- Topical anesthetic agent (for ED only):
 - Tetracaine hydrochloride ophthalmic solution 0.5%: 1–2 drops into affected eye:
 - Do not prescribe for outpatient as this may impair healing and increase corneal ulcer formation.
- Oral analgesics:
 - Ibuprofen 10 mg/kg TID with meals
 - Acetaminophen with oxycodone 500 mg/5 mg, q4–6h PRN for breakthrough pain
- Topical antibiotic ointment:
 - Erythromycin ophthalmic ointment 0.5%, apply to affected eye QID
- Cycloplegic agent:
 - Scopolamine hydrobromide ophthalmic solution 0.25%: 1 or 2 drops into affected eye q6–8h
 - Cyclopentolate hydrochloride ophthalmic solution 0.5%: 1 or 2 drops into affected eye q6–8h



FOLLOW-UP

DISPOSITION

Admission Criteria

Consider admission in cases of severe decreased visual acuity, bilateral patching, or in situations when self-care and follow-up are difficult.

Discharge Criteria

Nearly all patients may be discharged from the ED following treatment with oral analgesics, topical antibiotics, cycloplegics, and/or patching:

- Lesions should heal completely in 24–72 hr.

FOLLOW-UP RECOMMENDATIONS

- Follow up with ophthalmologist within 24–48 hr to monitor healing and symptom resolution.
- Long-term UV damage to eye may result in pterygium and some forms of corneal degeneration, though association with UV keratitis episodes has not been demonstrated.

PEARLS AND PITFALLS

- Determining UV exposure 6–12 hr prior is the key to diagnosis and prevention:
 - The patient may not be aware of exposure

- Those at risk for occupational exposure must wear UV safety goggles, not glasses or lenses.
- Exquisitely painful but self-limited injury; risks from repeated exposures are not well defined.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Conjunctivitis
- Corneal Burn
- Red Eye



CODES

ICD9

- 368.13 Visual discomfort
- 370.24 Photokeratitis

ICD10

- H16.131 Photokeratitis, right eye
- H16.132 Photokeratitis, left eye
- H16.133 Photokeratitis, bilateral

URETHRAL TRAUMA

Amanda Jillian-Lamond Holden



BASICS

DESCRIPTION

- Blood at the urethral meatus, a palpable full bladder, inability to void, and/or gross hematuria are common findings with urethral trauma.
- Found in 14% of pelvic fractures
- High association with bilateral pubic rami fractures (aka, straddle fractures)
- Females: Urethral injuries are rare likely due to short, unexposed, and mobile urethras.
- Girls <17 yr old: Higher injury rate likely from a more flexible pelvic ring
- Bladder neck most commonly injured location.
- Males: The urethra is divided into 2 sections.
- Posterior urethra:
 - More commonly injured (~ 90%)
 - Prostatic portion
 - Membranous
- Anterior urethra:
 - Injuries are rare
 - Bulbar
 - Penile
- Posterior urethra injuries comprise up to 90% of trauma:
 - Type 1: Urethra stretched but not ruptured
 - Type 2: Prostatic/membranous portions disrupted (either partially or completely); urogenital diaphragm intact
 - Type 3: Urethral disruption both proximal and distal to the genitourinary diaphragm

ETIOLOGY

- Females:
 - Rare with pelvic fractures
 - Straddle injuries
 - Childbirth or vaginal surgery
 - Sexual trauma/abuse
- Males:
 - More common with pelvic fractures
 - More common with straddle injuries
 - Penetrating trauma, mutilation
 - Sexual activity/instrumentation

DIAGNOSIS

SIGNS AND SYMPTOMS

- Males:
 - Blood at the urethral meatus
 - Gross hematuria
- Females:
 - Blood in the vaginal vault
 - Gross hematuria

History

Trauma to pelvic area

Physical-Exam

- Exam of the torso and pelvis during the secondary survey may elicit pelvic pain.
- Triad of blood at the urethral meatus, inability to urinate, and a palpably full bladder
- Blood at the meatus found in 50% of cases.
- Urologic injury can be indicated by gross hematuria (any color to the urine other than clear or yellow)
- Digital rectal exam: “High-riding prostate” has a sensitivity of <50%. Do not rely on this finding to rule out urethral trauma if suspected.
- Bedside US: FAST exam, suprapubic views may reveal blood surrounding the bladder.

ESSENTIAL WORKUP

- Females:
 - Perform a detailed vaginal exam to exclude vaginal laceration or other etiologies of bleeding.
 - If injury is suspected, radiologic evaluation of urethra should be performed.
 - If not possible, suprapubic aspiration or cystostomy should be done.
- Male:
 - If injury is suspected, radiographic evaluation of urethral integrity should be performed before urinary catheter placement to prevent turning a partial urethral tear into a complete tear.
 - If not possible, suprapubic aspiration or cystostomy should be performed.

Pediatric Considerations

- If an exam of the male or female genitalia cannot easily be performed, exam under anesthesia should occur.
- An exam with procedural sedation or in the OR, in addition to being better tolerated by the patient, allows the physician to rule out sexual abuse and to

confirm that the injury is consistent with the history.

DIAGNOSIS TESTS & INTERPRETATION

Lab

Urinalysis, hematocrit, BUN, creatinine

Imaging

- Retrograde urethrography (RUG):
 - Water-soluble contrast is injected via a catheter-tipped syringe at the urethral meatus.
 - Extravasation of contrast and its relation to the prevesical space and urogenital diaphragm should be noted.
 - Proximity of the extravasation to the meatus and the bladder should be appreciated.
 - If the urethral tear is complete, there will be no contrast within the bladder and marked extravasation will occur.
 - A partial tear will demonstrate contrast material within the bladder with varying amounts of extravasation.
- Excretory urethrography should be performed to define proximal urethral tears.
- Cystography
- 40% of urethral injuries have concomitant bladder injuries.

Diagnostic Procedures/Surgery

Urethral trauma warrants urgent urologic consultation.

DIFFERENTIAL DIAGNOSIS

- Perineal and vaginal trauma
- Bladder injury
- Ureter or kidney trauma



TREATMENT

PRE HOSPITAL

Pre-hospital trauma protocols

INITIAL STABILIZATION/THERAPY

Stabilization of multiple traumas takes precedence.

ED TREATMENT/PROCEDURES

- Urethral contusions, lacerations, and avulsions are best managed by an experienced urologist.

- Bladder decompression is an important initial intervention. If urethral Foley catheter placement is not possible, suprapubic aspiration/cystostomy may need to be performed.

MEDICATION

Appropriate analgesia

First Line

Opioids:

- Morphine, dilaudid, or fentanyl, as needed per trauma protocols for pain



FOLLOW-UP

DISPOSITION

Admission Criteria

- Concurrent traumatic injuries
- Need for emergent operative management of urethral, penile, or bladder injuries
- Partial lacerations:
 - Managed with urethral or suprapubic drainage
- Complete lacerations:
 - Managed surgically or with suprapubic drainage alone:
 - Some are repaired with end-to-end anastomosis.

Discharge Criteria

Isolated urethral injuries frequently may be managed in the outpatient setting after appropriate urinary catheterization or suprapubic cystostomy with next-day urologic follow-up.

Issues for Referral

Urologic follow-up is necessary if patient is discharged from ED.

FOLLOW-UP RECOMMENDATIONS

Urologic follow-up is necessary for all patients with urethral injuries.

PEARLS AND PITFALLS

- Consult urology before attempting to insert a Foley in a trauma patient in whom urethral injury is highly suspected.
- Passing a Foley catheter against resistance could convert a partial tear to a complete tear.
- Failure to recognize a urethral injury can result in urinary incontinence and sexual

dysfunction.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

Pelvic Trauma



CODES

ICD9

- 665.50 Other injury to pelvic organs, unspecified as to episode of care or not applicable
- 867.0 Injury to bladder and urethra, without mention of open wound into cavity
- 867.1 Injury to bladder and urethra, with open wound into cavity

ICD10

- N36.8 Other specified disorders of urethra
- O71.5 Other obstetric injury to pelvic organs
- S37.33XA Laceration of urethra, initial encounter

URETHRITIS

Hany Y. Atallah



BASICS

DESCRIPTION

- Urethritis is inflammation of the urethra from any cause (usually infection).
- Associated with urethral discharge and dysuria
- Urethritis may develop after exposure to a partner with an STD, bacterial vaginosis, or UTI.
- Urethritis may also develop after orogenital contact.

ETIOLOGY

- STD; the most common causes are:
 - *Neisseria gonorrhoeae* (35%)
 - *Chlamydia trachomatis* (25–50%)
 - *Mycoplasma genitalium* and *Ureaplasma urealyticum* (30%)
- Rarer causes:
 - *Trichomonas vaginalis*
 - Candidal species
 - Herpes simplex virus
 - Adenovirus
 - Genital warts
 - Enteric bacteria (in the setting of insertive anal sex)
 - Alcohol
 - Systemic illnesses
 - Urethral foreign bodies



DIAGNOSIS

- Symptoms usually develop 1–2 wk after exposure but can take up to 4–6 wk.
- Initially minimal or absent in many patients

SIGNS AND SYMPTOMS

- Urethral discharge, dysuria
- Cloudy 1st portion of urine
- Pyuria
- Inguinal adenopathy may be present.

History

- Color, consistency, and quantity of urethral discharge.
- Associated symptoms of dysuria, urgency, frequency, hematuria, and

hematospermia

- Risk factors for STDs:
 - Recent new partner or multiple sexual partners
 - Symptoms of partner
 - Anal/oral practices
 - Young age
 - Lower socioeconomic status

Physical-Exam

- Urethral discharge
- Staining on undergarments
- Meatal crusting
- Genital lesions
- Lymphadenopathy
- Palpate testes, epididymis, and spermatic cord:
 - Masses or tenderness

ESSENTIAL WORKUP

- Urethral swabs for *N. gonorrhoeae* and *Chlamydia* species will confirm the diagnosis.
- DNA amplification, DNA probe, and testing of urine specimens via polymerase chain reaction (PCR) have shown good sensitivity and are acceptable tests
- A rapid plasma regain (RPR) or Venereal Disease Research Laboratory (VDRL) should be drawn because STDs frequently occur together.
- An HIV test should also be offered to the patient.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Gram stain and cultures from urethral swabs should be reviewed when the patient is re-evaluated by his or her physician after treatment.
- DNA amplification (ligase chain reaction [LCR] or PCR) can be used on 1st-void urine or urethral swab:
 - Equal efficacy for diagnosing *N. gonorrhoeae* and *Chlamydia* species
- UA should be performed after urethral swabs to identify UTIs.

DIFFERENTIAL DIAGNOSIS

- Chemical irritation from soaps or spermicides
- Epididymitis
- Orchitis
- Pelvic inflammatory disease
- Prostatitis
- Reactive arthritis (formerly Reiter syndrome)

- Urethral chancre (from syphilis)
- UTI

Pediatric Considerations

- Urethritis in children should arouse suspicion of child abuse.
- Because *N. gonorrhoeae* infects the entire vaginal vault in prepubescents, a speculum exam is not required:
 - External exam and cultures are sufficient.
- Potential complications:
 - Recurrent infections
 - Ascending UTIs, including pelvic inflammatory disease and epididymo-orchitis
 - Fallopian tube damage and infertility
 - Arthritis
 - Conjunctivitis, uveitis, and blindness



TREATMENT

INITIAL STABILIZATION/THERAPY

Most patients will not require significant stabilization.

ED TREATMENT/PROCEDURES

- Treatment may be given empirically based on probable etiology.
- Patients should be treated for both *N. gonorrhoeae* and *C. trachomatis*.

MEDICATION

- Gonorrhea:
 - Azithromycin 2 g orally once
 - Cefixime 400 mg PO once
 - Cefotaxime 500 mg IM once (administered with probenecid 1 g orally once)
 - Cefoxitin 2 g IM once (administered with probenecid 1 g orally once)
 - Cefpodoxime 400 mg PO once
 - Ceftizoxime 500 mg IM once
 - Ceftriaxone 250 mg (peds: 25–50 mg/kg) IM/IV once
 - Cefuroxime 1 g orally once
 - Ciprofloxacin 500 mg PO once
 - Gatifloxacin 400 mg PO once
 - Levofloxacin 250 mg PO once
 - Ofloxacin 400 mg PO once
 - Spectinomycin 2 g IM once
- Chlamydia:
 - Azithromycin 1 g (peds: 10 mg/kg day 1, 5 mg/kg days 2–5) PO once

- Doxycycline 100 mg PO BID for 7 days
- Erythromycin base 500 mg (peds: 40 mg/kg/d div. QID) PO QID for 7 days
- Erythromycin ethyl succinate 800 mg (peds: 30–50 mg/kg/d div. QID) PO QID for 7 days
- Levofloxacin 500 mg PO QD for 7 days
- Ofloxacin: 300 mg PO BID for 7 days
- *M. genitalium*:
 - Azithromycin 1 g (peds: 10 mg/kg day 1, 5 mg/kg days 2–5) PO once

Pregnancy Considerations

- Fluoroquinolones and doxycycline are contraindicated in pregnancy
- Azithromycin is safe and effective
- Repeat testing 3 wk after treatment is recommended to ensure cure.

ALERT

Increasing incidence of quinolone-resistant *N. gonorrhoeae* nationwide.



FOLLOW-UP

DISPOSITION

Admission Criteria

Patients should not require admission for urethritis unless there are other complaints or infections.

Discharge Criteria

All patients should be discharged with follow-up arranged at an outside clinic or with PCP.

Issues for Referral

- If child abuse is suspected, child protective services must be involved; the child should be admitted if a safe home situation cannot be ensured.
- Sexual partners should be evaluated.
- In many states, STDs require reporting.

FOLLOW-UP RECOMMENDATIONS

- All patients should follow up with primary care to ensure adequate treatment of the infection.
- All patients with suspected or confirmed urethritis should be referred for HIV testing.
- Patients should be given information regarding safe sexual practices.

PEARLS AND PITFALLS

- Always treat for both *N. gonorrhoeae* and *C. trachomatis* in suspected urethritis.
- There is increasing evidence suggesting that patients with recurrent urethritis should be evaluated for infection with other atypical organisms (doxycycline-resistant *U. urealyticum* or *M. genitalium*; *T. vaginalis*)
- Always consider other STDs in patients with urethritis.
- Ensure that patients will inform their sexual partners so that they can be treated as well.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Chancroid
- Epididymitis/Orchitis
- Gonococcal Disease
- Herpes, Genital
- Lymphogranuloma Venereum
- Pelvic Inflammatory Disease
- Prostatitis
- Syphilis
- UTIs, Adult
- UTIs, Pediatric
- Vaginal Discharge/Vaginitis



CODES

ICD9

- 098.0 Gonococcal infection (acute) of lower genitourinary tract
- 131.02 Trichomonal urethritis
- 597.80 Urethritis, unspecified

ICD10

- A54.01 Gonococcal cystitis and urethritis, unspecified
- A59.03 Trichomonal cystitis and urethritis
- N34.1 Nonspecific urethritis

URINARY RETENTION

Denise S. Lawe

BASICS

DESCRIPTION

- Acute urinary retention (AUR):
 - _ Sudden inability to void spontaneously
 - _ Occurs most frequently in men >60 yr old
 - _ Most common cause of AUR in the ED is benign prostatic hyperplasia (BPH)

ETIOLOGY

- Multiple diagnostic considerations, following list is not exhaustive
- Anatomic:
 - _ Penis:
 - Phimosis
 - Paraphimosis
 - Meatal stenosis
 - Foreign-body constriction
 - _ Urethra:
 - Tumor
 - Pelvic masses
 - Prolapse of pelvic organs
 - Foreign body
 - Calculus
 - Urethritis
 - Stricture
 - Meatal stenosis (can also be seen in females)
 - Hematoma
 - Vulvar edema after vaginal delivery
 - _ Prostate gland:
 - Benign prostatic hypertrophy
 - Carcinoma
 - Prostatitis
 - Contracture of bladder neck
 - Prostatic infarction
- Neurologic causes:
 - _ Motor/paralytic:
 - Spinal shock
 - Spinal cord syndromes
 - _ Sensory/paralytic:

- Diabetes
- Multiple sclerosis
- Spinal cord syndromes
- Drugs:
 - Antihistamines
 - Anticholinergics
 - Antispasmodics
 - Tricyclic antidepressants
 - α -Adrenergic stimulators
 - Narcotics
 - NSAIDs

DIAGNOSIS

SIGNS AND SYMPTOMS

- Lower abdominal or suprapubic discomfort
- Patients may appear restless or in distress
- Chronic urinary retention usually painless

History

- Past medical history:
 - History of urinary retention?
 - History of BPH or prostate cancer?
 - History of other cancer?
 - History of radiation treatment?
 - History of pelvic trauma?
- Any signs or symptoms of infection including an abscess?
- Any signs or symptoms of calculus?
- Any neurologic symptoms?
- History of or current IV drug abuse?
- Back pain?
- Complete list of all medications

Physical-Exam

- Vitals (Any evidence of infection? Shock?)
- Abdominal exam
- Rectal exam
- Genitourinary exam; consider pelvic exam in all women
- Thorough neurologic exam if appropriate
- In the trauma patient, evaluate for evidence of urethral injury

ESSENTIAL WORKUP

Due to the multiple causes of AUR a thorough history and physical exam are imperative, and will determine further workup

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Basic chemistry to assess renal function only if concerned for acute renal insufficiency (this usually does not occur in AUR)
- No benefit to PSA test in ED; usually elevated in setting of AUR
- Urinalysis if indicated on history or exam

Imaging

- Abdominal or pelvic US or CT abdomen/pelvis if concerned for mass, malignancy, abscess, bladder calculi, or other anatomic etiologic agent
- Neuro or spinal imaging if there is concern for an acute neurologic process

Diagnostic Procedures/Surgery

Postvoid residual: More than 200 mL is usually considered abnormal.

DIFFERENTIAL DIAGNOSIS

Chronic urinary retention



TREATMENT

PRE HOSPITAL

Address any life-threatening presentation

INITIAL STABILIZATION/THERAPY

- Identify and treat any life-threatening presentation
- Prompt bladder decompression:
 - Try placement of 14–18F urinary catheter
 - If unable to pass a 14–18F catheter and there is a history of prior transurethral procedure or known stricture, downsize to a 10–12F
 - In men with no prior instrumentation and unable to pass catheter, consider a 20–22F catheter with a coude tip
 - If unable to pass a catheter, then either suprapubic aspiration as a temporizing measure or placement of suprapubic catheter is indicated
- Defer catheterization of the ureter in the trauma patient suspected of having a ureteral injury (gross hematuria, high-riding prostate on rectal exam, blood at the meatus) until a retrograde urethrogram has been done

ED TREATMENT/PROCEDURES

- Drain bladder and monitor urine output:
 - Rapid decompression following catheter placement may result in transient gross hematuria, rarely clinically significant
 - Postobstructive diuresis:
 - Can be a complication of AUR in the catheterized patient
 - No randomized trials comparing rapid and intermittent bladder decompression
 - It is generally now felt that rapid bladder decompression is safe provided that supportive care is available if hypotension develops
- Probably best to observe for 2–3 hr after bladder decompression to ensure that a postobstructive diuresis does not cause clinical deterioration
- Place leg catheter bag before discharge if catheter is to remain indwelling
- Educate patient and family on catheter care.
- Although commonly used, prophylactic antibiotics are not indicated for patients with an indwelling urinary catheter and no evidence of infection
- Start patients with BPH on an α -blocker
- Consider stopping any medication that may be contributing to AUR
- Treat constipation if appropriate

MEDICATION

- Prazosin HCl (Minipress) for treatment of BPH: Initially 1 mg PO BID to TID, slowly increase to 20 mg/d in div. doses
- Tamsulosin (Flomax) is an α -1 antagonist used to treat BPH: 0.4 mg PO QD after the same meal daily; may increase to 0.8 mg PO QD
- Alfuzosin (Uroxatral) is an α -blocker used to treat BPH: 10 mg PO daily after the same meal each day
- Terazosin (Hytrin) facilitates urinary flow in the presence of BPH: Start 1 mg PO QHS, max. 20 mg/d



FOLLOW-UP

DISPOSITION

Admission Criteria

- Significant postobstructive diuresis requiring IV fluids or pressors
- Sepsis
- Obstruction related to spinal cord compression
- Consider in patient with obstruction due to malignancy or mass
- Any process requiring acute urologic or surgical intervention

Discharge Criteria

Most patients can be discharged

FOLLOW-UP RECOMMENDATIONS

Most patients will need follow-up for ongoing evaluation and management of AUR as well as catheter management

PEARLS AND PITFALLS

- Carefully evaluate for evidence of a mass or malignancy as the cause of AUR.
- Carefully evaluate for evidence of spinal cord compression as the cause of AUR.
- Take a thorough drug history including over-the-counter medications, especially if no other clear reason for AUR.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

UTIs



CODES

ICD9

- 598.9 Urethral stricture, unspecified
- 600.91 Hyperplasia of prostate, unspecified, with urinary obstruction and other lower urinary symptoms (LUTS)
- 788.20 Retention of urine, unspecified

ICD10

- N35.9 Urethral stricture, unspecified
- N40.1 Enlarged prostate with lower urinary tract symptoms
- R33.9 Retention of urine, unspecified

URINARY TRACT FISTULA

Denise S. Lawe

BASICS

DESCRIPTION

Urinary tract fistulas can form between any part of the urinary tract and structures in the thoracic cavity, the abdominal cavity, the pelvis, and the skin.

ETIOLOGY

- Colovesical fistula:
 - Usually complication of primary GI disease such as diverticular disease (most common), Crohn's disease, or colon carcinoma
 - Iatrogenic (postsurgical or radiation treatment most common)
 - Urethral disruption from trauma
 - More common in males
- Vesicovaginal, urethrovaginal, and ureterovaginal fistulas:
 - Vesicovaginal fistula is the most common acquired fistula of the urinary tract
 - Etiology varies with geography (developed vs. developing countries):
 - In developed countries it is usually due to injury to the structures during surgery, pelvic pathology, radiation therapy, or injuries incurred in the healing process. Radiation-induced fistulas may not present for months to years after exposure.
 - In developing countries it is usually due to obstructed labor and obstetric trauma.

DIAGNOSIS

SIGNS AND SYMPTOMS

- Colovesical fistula:
 - Chronic or recurrent UTIs
 - Suprapubic pain
 - Abnormal urine: Pneumaturia, fecaluria, hematuria, malodorous urine, debris in the urine (food particles)
- Vesicovaginal fistula:
 - If after surgical procedure, may present on removal of urinary catheter or 1–3 wk post procedure
 - Usually painless
 - Constant urine leakage from the vagina (may be confused with urinary incontinence)

- Perineal skin irritation due to urine leakage
- Urethrovaginal fistula:
 - Symptoms largely dependent on size and location of fistula
 - May be asymptomatic or with continuous vaginal urine drainage
- Ureterovaginal fistula:
 - Usually a history of recent surgery, particularly a complicated hysterectomy
 - Abdominal or flank pain, fever, and ileus. If these symptoms present, likely due to urinoma or renal obstruction
 - Intermittent urine leakage from vagina

History

- A thorough past medical, surgical, and obstetric history to determine risk factors
- Description and timing of presumed urinary discharge: Intermittent or positional usually due to ureterovesical fistula; continuous flow more likely to be from vesicovaginal fistula.
- Characteristics of presumed urinary discharge
- Associated symptoms

Physical-Exam

- Colovesical fistula:
 - There might be findings consistent with the primary GI disease; otherwise physical exam is frequently unremarkable
- Vesicovaginal, urethrovaginal, ureterovaginal fistulas:
 - Speculum exam may reveal a small reddened area of granulomatous tissue at site of the fistula opening. May also see pooling of the urine in the vaginal vault.

ESSENTIAL WORKUP

Must evaluate for associated urinary infection, renal obstruction, or acute emergencies related to primary disease processes (e.g., complications from a malignancy or Crohn's disease).

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Urinalysis:
 - Colovesical fistula:
 - WBCs, bacteria, and debris
 - Vesicovaginal, urethrovaginal, and ureterovaginal fistulas:
 - WBCs, bacteria
- BUN and creatinine:
 - If renal obstruction is present, might be abnormal.

Imaging

- Usually not an emergent need to image and would discuss with specialist
- Colovesical fistula:
 - CT of the abdomen and pelvis with contrast
- Vesicovaginal, urethrovaginal, and ureterovaginal fistulas:
 - Cystoscopy with retrograde pyelography or IVP

Diagnostic Procedures/Surgery

- Usually an outpatient workup
- Colovesical fistula:
 - Oral administration of activated charcoal will result in black particles in urine, which can be diagnostic.
- Vesicovaginal, urethrovaginal, ureterovaginal fistulas:
 - Double-dye test: (1) Tampon is placed in vagina, (2) oral phenazopyridine is administered, (3) methylene blue or indigo carmine is instilled into the bladder, (4). if, after an hour, tampon is yellow-orange at the top, ureterovaginal fistula is suggested. Midportion blue discoloration suggests vesicovaginal fistula. Distal blue discoloration suggests urethrovaginal fistula.

DIFFERENTIAL DIAGNOSIS

- Colovesical fistula:
 - Recurrent UTI
 - Other causes of pneumaturia:
 - UTI with gas-forming organism such as clostridia
 - Fermentation of diabetic urine
 - Recent urinary tract instrumentation
- Vesicovaginal, urethrovaginal, and ureterovaginal fistulas:
 - Urinary incontinence
 - Normal vaginal discharge
 - Vaginitis



TREATMENT

INITIAL STABILIZATION/THERAPY

Treat urosepsis (rare) with IV fluid bolus, pressors, and IV antibiotics as appropriate.

ED TREATMENT/PROCEDURES

- Colovesical fistula:
 - Evaluate for complications from patient's primary disease.
 - Obtain cultures if there are signs of UTI.
 - Initiate antibiotic if infection is found.

- Urgent urologic referral for further management and possible surgical treatment.
- Vesicovaginal, urethrovaginal, and ureterovaginal fistulas:
 - Consider placing urinary catheter
 - Initiate antibiotics if a UTI is present.
 - Urgent referral to urologist and gynecologist for further care



FOLLOW-UP

DISPOSITION

Admission Criteria

- Sepsis
- Inability to take oral antibiotics if needed
- Acute emergencies from primary GI disease or malignancies

Discharge Criteria

- No evidence of sepsis
- Able to tolerate oral antibiotics if UTI present

FOLLOW-UP RECOMMENDATIONS

Urogenital specialist (Urology or Gynecology) follow-up is required.

PEARLS AND PITFALLS

- Suspect a urinary tract fistula in the patient with the appropriate risk factors (usually a complicated recent pelvic surgery) and recurrent UTIs
- In the presence of urinary tract fistula, malignancy is always an important diagnostic consideration
- Urine leakage from the vagina may be confused with urinary incontinence

ADDITIONAL READING

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- Vasavada SP. (2011, Sep 14). Vesicovaginal and ureterovaginal fistula. Retrieved from www.emedicine.com.
- Wein AJ. Urinary tract fistula. In: *Campbell-Walsh Urology*. 9th ed. Philadelphia, PA: Saunders; 2007.



See Also (Topic, Algorithm, Electronic Media Element)

UTIs, Adult



CODES

ICD9

- 596.1 Intestinovesical fistula
- 599.1 Urethral fistula
- 619.0 Urinary-genital tract fistula, female

ICD10

- N32.1 Vesicointestinal fistula
- N36.0 Urethral fistula
- N82.0 Vesicovaginal fistula

URINARY TRACT INFECTIONS, ADULT

Paul A. Szucs • Barnet Eskin



BASICS

DESCRIPTION

- Colonization of urine with uropathogens and invasion of genitourinary (GU) tract
- Defined as urinary symptoms with $\geq 10^2$ to 10^5 CFU/mL of uropathogen and ≥ 10 WBC/mm³
- Lifetime risk of UTI in women is $> 50\%$
- Uncomplicated cystitis:
 - Females aged 13–50
 - Symptoms < 2 –3 days
 - Not pregnant
 - Afebrile (temperature $< 38^\circ\text{C}$)
 - No flank pain
 - No costovertebral angle tenderness (CVAT)
 - Fewer than 4 UTIs in past year
 - No recent instrumentation or previous GU surgery
 - No functional/structural GU abnormality
 - Not immunocompromised
 - Neurologically intact
- Complicated cystitis:
 - Do not meet above criteria
 - Male gender
 - Patients with anatomic, functional, or metabolic abnormalities of GU tract
 - Postvoid residual urine
 - Catheters
 - Resistant pathogens
 - Recent antimicrobial use
- Uncomplicated pyelonephritis:
 - Renal parenchymal infection
 - Dysuria, frequency, urgency
 - Fever, chills, myalgias, nausea, vomiting
 - Flank, back, or abdominal pain
 - CVA tenderness
 - Leukocytosis (common)
- Complicated pyelonephritis:
 - Renal parenchymal infection
 - Temperature $> 40^\circ\text{C}$
 - Urosepsis with septic shock

- Intractable nausea, vomiting
- Diabetes, other immunosuppression
- Pregnancy (especially latter half)
- Concomitant obstruction or stone
- Asymptomatic (occult)

ETIOLOGY

- Mechanism:
 - Organisms colonize periurethral area and subsequently infect the GU tract.
- Risk factors:
 - Population:
 - Newborn, prepubertal girls, young boys
 - Sexually active young woman
 - Postmenopausal woman, elderly males
 - Behavior:
 - Sexual intercourse, spermicides, diaphragms
- Elderly females/postmenopausal state
- Less efficient bladder emptying, bladder prolapse, alteration of bladder defenses
- Increased vaginal pH
- Contamination due to urinary or fecal incontinence (Enterobacteriaceae)
- Instrumentation:
 - Elderly males due to prostatic hypertrophy and instrumentation
- Organisms:
 - *Escherichia coli* (80–85%)
 - *Staphylococcus saprophyticus* (10%)
 - Other (10%): *Klebsiella*, *Proteus mirabilis*, *Enterobacter* spp., *Pseudomonas aeruginosa*, group D streptococci

DIAGNOSIS

SIGNS AND SYMPTOMS

- Lower tract infection: Cystitis:
 - Dysuria, frequency, urgency, hesitancy
 - Suprapubic pain
 - Hematuria
- Upper tract infection: Pyelonephritis:
 - Symptoms of cystitis:
 - Fever, chills
 - Flank pain and/or tenderness
 - Nausea, vomiting, anorexia
 - Leukocytosis
 - Up to 50% of patients with cystitis may actually have pyelonephritis:
 - Symptom duration > 5 days, homelessness, and recent UTI are risk

factors for upper tract infection

- Elderly or frail patients:
 - Altered mental status
 - Anorexia
 - Decreased social interaction
 - Abdominal pain
 - Nocturia, incontinence
 - Syncope or dizziness

ESSENTIAL WORKUP

- Urinalysis (dipstick test, microscopy)
- Females: Rule out pregnancy, urethritis, vaginitis, pelvic inflammatory disease (PID)
- Males: Rule out urethritis, epididymitis, prostatitis; inquire about anal intercourse/HIV.
- Urologic evaluation in young healthy males with 1st UTI is *not* routinely recommended.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Rapid Urine Screen:
 - Dipstick (leukocyte esterase + nitrite) most effective when urine contains 10^5 CFU/mL
 - Lab specimen unnecessary if pyuria and bacteriuria confirmed by dipstick
 - Leukocyte esterase: Positive likelihood ratio (LR+) ~5, negative likelihood ratio (LR-) ~0.3
 - Nitrite: LR+ ~30, LR- ~0.5
- Urinalysis/microscopy:
 - Obtain if rapid urine screen is unavailable or negative in patients with presumed UTI.
 - 10 WBC/mm³ in clean catch midstream urine indicates infection.
 - Bacteria detected in unspun urine indicates $>10^5$ CFU/mL. (LR+ ~20, LR- ~0.1)
- Indications for urine culture:
 - Complicated UTIs
 - Negative rapid urine screen or microscopy in patients with presumed UTI
 - Persistent signs and symptoms after 2–3 days of treatment
 - Recurrence (relapse vs. reinfection)
 - Recently hospitalized patients
 - Nosocomial infections
 - Pyelonephritis

Geriatric Considerations

- Asymptomatic bacteriuria (including positive cultures) occurs in 20% of women >65 yr, 50% of women >80 yr and generally should *not* be treated.
- Consider treating symptomatic geriatric patients for 5–10 days to decrease risk of recurrent or persistent bacteriuria.
- Fluoroquinolones may cause CNS side effects.

Imaging

- Indicated for complicated upper tract disease (see Pyelonephritis)
- Helical CT, renal ultrasound, or IV pyelogram if concomitant stone or obstruction suspected

Diagnostic Procedures/Surgery

Patients with significant hematuria, recurrent UTI with same uropathogen, or symptoms of obstruction *need* urologic evaluation to identify structural or functional abnormality.

DIFFERENTIAL DIAGNOSIS

- Appendicitis
- Diverticulitis
- Epididymitis
- Nephrolithiasis
- PID/cervicitis
- Prostatitis
- Pyelonephritis
- Urethritis
- Vulvovaginitis



TREATMENT

INITIAL STABILIZATION/THERAPY

Urosepsis/septic shock:

- Manage airway and resuscitate as indicated
- IV crystalloid and vasopressors as needed
- Early goal-directed therapy

ED TREATMENT/PROCEDURES

Stable Patients

- For uncomplicated UTIs in women for most antibiotics, 3 days of therapy:
 - More effective than single dose
 - Clinically as effective as 5–10-day course with fewer side effects
- Resistance varies by place and changes over time:

- In North America, 40–50% of *E. coli* are resistant to ampicillin; 3–17% to fluoroquinolones and is increasing.
- Resistance to trimethoprim–sulfamethoxazole (TMP/SMX) is increasing (up to 30%).
- Nitrofurantoin: In some studies, nitrofurantoin resistance is less than for other more widely used antibiotics.
- Culture resistance may not correlate with clinical effect because urine antibiotic concentrations are much higher than those used in laboratory testing. However, symptom resolution may be delayed a few days in patients with resistant bacteria.
- Antibiotics of choice:
 - Nitrofurantoin
 - TMP/SMX
 - Fluoroquinolones 2nd-line treatment in women:
 - Sulfonamide intolerance
 - All quinolones equally effective (~95% susceptibility rates) but side effects vary
 - High frequency of antimicrobial resistance related to recent treatment
 - Live in areas with unknown or >20% resistance to TMP/SMX
 - Oral cephalosporins may be reasonable alternatives in specific circumstances:
 - Require 7-day treatment regimens
 - Amoxicillin–clavulanate not as effective as ciprofloxacin, probably due to failure to eradicate vaginal *E. coli*
 - Diabetic women have increased risk of bacteriuria with *Klebsiella* spp.
 - Treat dysuria with phenazopyridine.
 - Treat pain with appropriate analgesics.
- Cranberry juice or tablets/products:
 - Prevents specific *E. coli* from adhering to uroepithelial cells but probably does not lower UTI recurrence rate in women with history of recurrent UTIs
 - Evidence suggests ineffective for treatment
- Treatment of upper tract disease—*rule of 2s*:
 - 2 L of IV crystalloid
 - 2 tablets of oxycodone/acetaminophen
 - 2 g of ceftriaxone or 2 mg/kg of gentamicin
 - If fever drops by 2°C and patient can retain 2 glasses of water
 - Discharge with fluoroquinolone for 2 wk.
 - Follow up in 2 days.

Pregnancy Considerations

- Treat asymptomatic bacteriuria in pregnancy with 4–7-day course of antibiotics:
 - Nitrofurantoin:
 - May cause birth defects if used in 1st trimester

- Contraindicated in G6PD-deficiency
- Amoxicillin (not 1st-line treatment due to high rate of resistance)
- Fosfomycin (safe and effective)
- TMP/SMX:
 - SMX should be avoided late in pregnancy as kernicterus can result.
 - TMP should be avoided in 1st trimester (folic acid antagonist; possible birth defects).
- Quinolones should be avoided:
 - CNS reactions
 - Blood dyscrasias
 - Effects on collagen formation

MEDICATION

- Amoxicillin: 500 or 875 mg PO q12h
- Cefixime: 400 mg PO q24h
- Cefpodoxime: 400 mg PO q12h
- Ceftazidime: 1–2 g IV q8–12h
- Ceftriaxone: 1–2 g IV/IM q24h
- Cefuroxime: 250–500 mg PO q12h
- Cephalexin: 250–500 mg PO q6h
- Ciprofloxacin: 100–500 mg PO q12h
- Doripenem: 500 mg IV q8h
- Fosfomycin: 3 g single dose
- Gentamicin: 2 mg/kg IV or IM q8h
- Levofloxacin: 250 mg PO q24h
- Nitrofurantoin macrocrystals 100 mg PO q12h
- Norfloxacin: 400 mg PO q12
- Ofloxacin: 200 mg PO q12h or 400 mg IV q12h
- Phenazopyridine: 200 mg PO TID for 2 days:
 - For symptomatic treatment of dysuria
 - May turn urine and contact lenses orange
- TMP/SMX: 160 mg/800 mg PO q12h or 10 mg/kg/d IV div. q6–8–12h



FOLLOW-UP

DISPOSITION

Admission Criteria

- Inability to comply with oral therapy
- Toxic appearing, unstable vital signs
- Pyelonephritis:
 - Intractable symptoms

- Extremes of age
- Immunosuppression
- Urinary obstruction
- Consider if coexisting urolithiasis
- Significant comorbid disease
- Outpatient treatment failure
- Late in pregnancy

Discharge Criteria

- Well appearing, normal vital signs
- Can comply with oral therapy
- No significant comorbid disease
- Adequate follow-up (48–72 hr) as needed
- Healthy patients with uncomplicated pyelonephritis who respond to treatment in ED according to rule of 2s
- Pyelonephritis in early pregnancy with good follow-up may be treated as outpatients

Issues for Referral

Recurrent UTIs require workup for underlying pathology.

FOLLOW-UP RECOMMENDATIONS

Follow-up for UTIs should start with primary care physician.

PEARLS AND PITFALLS

- For women who have more than 2 episodes of acute cystitis in 6 mo or 3 episodes in 1 yr, consider long-term (6–12 mo) prophylactic antibiotics or postcoital prophylaxis
- Pregnant women should be screened and treated for asymptomatic bacteriuria (ASB) because 20–40% of women with ASB progress to pyelonephritis.
- ASB in pregnant women associated with increased risk of preterm birth, low birth weight, and perinatal mortality.
- Treat ASB in renal transplant recipients, patients who have recently undergone a urologic procedure, and neutropenic patients.
- Risk factors for acute cystitis in men: Increased age, uncircumcised, HIV infection (low CD4 counts), anatomic abnormalities (BPH or urethral strictures), and sexual activity (especially insertive anal intercourse).
- 25% of male GU complaints are attributable to prostatitis. TMP/SMX or fluoroquinolones are 1st-line treatment.
- In patients with indwelling catheters, pyuria is less strongly correlated with UTI than in patients without catheters.

ADDITIONAL READING

- Gupta K, Hooton TM, Naber KG, et al. International clinical practice guidelines for the treatment of acute uncomplicated cystitis and pyelonephritis in women: A 2010 update by the Infectious Diseases Society of America and the European Society for Microbiology and Infectious Diseases. *Clin Infect Dis*. 2011;52:e103–e120.
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- St. John A, Boyd JC, Lowes AJ, et al. The use of urinary dipstick tests to exclude urinary tract infection. *Am J Clin Pathol*. 2006;126:428–436.



See Also (Topic, Algorithm, Electronic Media Element)

- Pyelonephritis
- UTI, Pediatric



CODES

ICD9

- 590.80 Pyelonephritis, unspecified
- 595.9 Cystitis, unspecified
- 599.0 Urinary tract infection, site not specified

ICD10

- N12 Tubulo-interstitial nephritis, not spcf as acute or chronic
- N30.90 Cystitis, unspecified without hematuria
- N39.0 Urinary tract infection, site not specified

URINARY TRACT INFECTIONS, PEDIATRIC

Suzanne Z. Barkin

BASICS

DESCRIPTION

- Bacteria colonize via retrograde contamination of rectal or perineal flora:
 - Infants—often hematogenous spread
 - Older children—vesicoureteral reflux (VUR) major risk
- UTI is defined by culture of a single organism of $> 10,000/\text{mL}$ on a catheterized or suprapubic specimen. Other collection techniques are not routinely used in young children for definitive diagnosis.
- In infants 0–3 mo old, UTI is associated with a 30% incidence of sepsis.
- Predisposing factors:
 - Poor perineal hygiene
 - Short urethra of female
 - Female $>$ male
 - Infrequent voiding
 - Constipation
 - Sexual activity
 - Male circumcision probably reduces risk

ETIOLOGY

- UTI found in 4–7% of febrile infants
- Bacterial agents:
 - *Escherichia coli* accounts for 80%
 - *Klebsiella pneumoniae*
 - *Staphylococcus aureus*
 - *Enterobacter* species
 - *Proteus* species
 - *Pseudomonas aeruginosa*
 - *Enterococcus* species

DIAGNOSIS

ALERT

UTIs in children may be difficult to diagnose without lab confirmation.

SIGNS AND SYMPTOMS

History

- Often nonspecific
- Neonates:
 - Manifestations of sepsis
 - Feeding difficulties
 - Irritability, listlessness
 - Fever, hypothermia
- 1 mo–3 yr of age:
 - Fever
 - Irritability
 - Vomiting, diarrhea
 - Abdominal pain
 - Poor feeding, failure to thrive
- Hematuria
- In girls <2 yr, an increased risk is associated with those having ≥ 3 factors (<12 mo old, white, temperature $\geq 39^\circ\text{C}$, absence of other source of fever, fever ≥ 2 days)
- Children >3 yr of age:
 - Dysuria
 - Frequency
 - Enuresis
 - New onset of urinary incontinence
 - Pain: Abdominal, suprapubic, back, costovertebral angle (CVA)
 - Fever
 - Hematuria
 - Malodorous cloudy urine
 - Systemic toxicity: High fever and chills with CVA tenderness
- Complications:
 - Recurrent UTI
 - Pyelonephritis
 - Chronic renal failure:
 - Scarring probably may be reduced by early detection and intervention
 - Perinephric abscess
 - Bacteremia/sepsis
 - Urolithiasis

Physical-Exam

- Vital signs, esp. temperature and blood pressure
- Toxicity
- Growth parameters
- Abdomen: Tenderness, esp. CVA pain
- GU: Genitalia

ESSENTIAL WORKUP

- UA with microscopic RBC and WBC counts and Gram stain for bacteria:
 - UA alone has low diagnostic sensitivity in infants.
 - Causes of pyuria besides UTI include chemical (bubble bath) or physical (masturbation) irritation, dehydration, renal tuberculosis, trauma, acute glomerulonephritis, respiratory infections, appendicitis, pelvic infection, and gastroenteritis.
 - Leukocyte esterase correlates with presence of pyuria.
 - Positive nitrite test indicates presence of bacteria capable of fixing nitrate. False-negative tests common
 - Gram stain of urinary sediment is more reliable than dipstick methods of diagnosis and superior to traditional UA.
 - Up to 80% of UAs in neonates with documented UTIs may be normal.
- Urine culture:
 - Specimen should be cultured within 30 min or refrigerated.
 - False-negative results may be caused by dilution, improper culture medium, recent antimicrobial therapy, fastidious organisms, bacteriostatic agent in urine, and complete obstruction of ureter.
- Clean-catch and bag specimens
 - Clean catch in cooperative male children
 - Plastic bag collection adequate for UA (70% contamination rate).
 - Clean the perineum (females) and glans (males) before application.
 - Can be used as a screening tool to rule out an infection if patient is not placed on antibiotics empirically and follow-up culture possible if the initial assessment is suggestive of infection.
- Catheterization is the preferred technique to obtain urine because contamination is common with bag collection and clean catch:
 - Bladder catheterization:
 - Acceptable in all infants
 - Higher success rate than suprapubic aspiration
 - Aseptic technique essential
 - Discarding the 1st 1–2 mL of urine before collecting specimen reduces contamination.
- Suprapubic aspiration is used on rare occasion and does provide a good specimen:
 - Most useful in infants
 - Full bladder optimal
 - Uncommonly used
 - Ultrasound may be useful adjunctive measure to improve yield.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC and blood culture for young children with fever or nonspecific symptoms and no source on exam. Consider additional evaluation as appropriate.

- Electrolytes, BUN, creatinine:
 - Check if there is dehydration, pyelonephritis, or recurrent infection.

Imaging

- Children requiring radiologic evaluation:
 - Infants < 3 mo of age
 - Males (increased association with anomaly) with 1st UTI
 - Clinical signs and symptoms consistent with pyelonephritis
 - Clinical evidence of renal disease
 - Some suggest that girls < 3 yr of age with a 1st UTI should be studied.
 - Females > 3 yr of age
 - 1st UTI in patients who have a family history of UTIs, abnormal voiding pattern, poor growth, HTN, urinary tract anomalies, or failure to respond promptly to therapy
 - 2nd UTI
- Voiding cystoureterogram (VCUG):
 - UTI is often associated with VUR and other genitourinary abnormalities and identified by VCUG. The importance of identifying VUR has been questioned.
- Renal/bladder ultrasound (US):
 - Ultrasonography is useful in excluding obstructive lesion and identifying children with solitary/ectopic kidney and some patients with moderate renal damage/scarring:
 - Renal/bladder US is indicated to identify anatomic abnormalities. Should be done in children < 2 yr with 1st febrile UTI, children with recurrent febrile UTIs, children with a UTI and family history of GU disease, poor growth, or hypertension as well as those children who do not respond as anticipated to antibiotics.
 - Nuclear cystogram (DMSA) may be substituted for VCUG in females. Its role is being clarified.
 - Further evaluation with nuclear medicine studies depends upon the grade of VUR and response to treatment

DIFFERENTIAL DIAGNOSIS

- Infection:
 - Vulvovaginitis
 - Viral cystitis
 - Urethritis (*Neisseria gonorrhoeae* or *Chlamydia trachomatis*)
 - Glomerulonephritis
 - Appendicitis
- Trauma:
 - Chemical irritation/cystitis
 - Perineal

- Sexual abuse
- Genitourinary
- Masturbation
- Foreign body
- Nephrolithiasis
- Diabetes



TREATMENT

INITIAL STABILIZATION/THERAPY

- Treat infants < 3 mo old presumptively for sepsis if febrile and/or toxic until blood and other appropriate cultures are final.
- Airway intervention for septic/acidotic infants with depressed respiratory drive
- Bolus of 20 mL/kg 0.9% NS for dehydration, hypovolemia, or sepsis; may repeat

ED TREATMENT/PROCEDURES

- Initiate IV antibiotics in all febrile infants < 3 mo with UTI:
 - Ampicillin and gentamicin in neonates
 - Cephalosporins after 4–8 wk of age
- Outpatient oral antibiotic for 10–14 days for children discharged. Should reflect local resistance patterns. Once sensitivity is known, antibiotic may need to be changed:
 - Amoxicillin
 - Amoxicillin/clavulanate
 - Cephalexin
 - Trimethoprim–sulfamethoxazole (TMP–SMX)
 - Many suggest 3rd-generation cephalosporin (cefixime, cefdinir) as 1st-line drug in treatment of children without GU anomaly because of changing resistance patterns. Oral therapy is generally adequate although close follow-up is essential to monitor clinical response and sensitivity of the etiologic organism.
 - Recent UTI may provide information related to sensitivities in children with recurrent UTIs
 - Length of treatment in children with afebrile UTI may be shortened to 5 days in children > 2 yr. The short course is still not generally recommended in children with febrile UTI.

MEDICATION

First Line

- Amoxicillin: 40 mg/kg/24 h PO q8h
- Amoxicillin/clavulanate: 40 mg/kg/24 h PO q8h

- Ampicillin: 100 mg/kg/24 h IV q6h
- Cefdinir 14 mg/kg/24 h PO QD
- Cefixime 16 mg/kg/24 h PO on 1st day followed by 8 mg/kg/24 h PO QD
- Ceftriaxone: 50–75 mg/kg/24 h q12–24h IV or IM
- Cephalexin: 50 mg/kg/24 h PO q6–12h
- Gentamicin: 2.5 mg/kg/dose IV q8h if full-term and age >7 days; 2.5 mg/kg/dose IV q12h if full-term and age 0–7 days (special dosing regimens in infants <36 wk postconceptual age)
- TMP–SMX (Bactrim or Septra suspension): 5 mL liquid (of 40/200 per 5 mL) per 10 kg per dose PO BID



FOLLOW-UP

DISPOSITION

Admission Criteria

- Infants <3 mo
- Dehydration
- Ill appearance/toxicity/sepsis
- Suspected pyelonephritis
- Urinary obstruction
- Vomiting, inability to retain medications
- Failure to respond to outpatient therapy
- Immunocompromised patient
- Renal insufficiency
- Foreign body (indwelling catheter)
- Pregnant patient

Discharge Criteria

- Sufficiently hydrated
- Low risk for sepsis or meningitis
- Nontoxic
- Able to take oral antibiotics; compliant

Issues for Referral

- Patients needing admission often require a pediatrician, urologist, or infectious disease consultant, esp. if there is VUR, renal anomaly, impaired renal function, recurrent infection, or hypertension.
- Good follow-up is mandatory.

FOLLOW-UP RECOMMENDATIONS

Monitoring of urine for sterility, further evaluation for underlying pathology, and

following growth pattern

PEARLS AND PITFALLS

- UTI may require lab confirmation of clinical suspicion. Signs and symptoms are often nonspecific.
- Febrile infants with UTI may be bacteremic.
- Neonates with UTI may have normal urinalysis.

ADDITIONAL READING

- American Academy of Pediatrics, Subcommittee on Urinary Tract Infection. Urinary tract infection: Clinical practice guidelines for the diagnosis and management of the initial UTI in febrile infants and children 2 to 24 months. *Pediatrics*. 2011;128:595–610.
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See Also (Topic, Algorithm, Electronic Media Element)

UTI, Adult



CODES

ICD9

- 041.49 Other and unspecified Escherichia coli [E. coli]
- 593.70 Vesicoureteral reflux unspecified or without reflux nephropathy
- 599.0 Urinary tract infection, site not specified

ICD10

- B96.20 Unsp Escherichia coli as the cause of diseases classd elswhr
- N13.70 Vesicoureteral-reflux, unspecified
- N39.0 Urinary tract infection, site not specified

URTICARIA

Fred A. Severyn



BASICS

DESCRIPTION

- Cutaneous mast and basophil cellular release of inflammatory mediators, primarily histamine:
 - Increased vascular permeability and pruritus
- Edema of the epidermis as well as the upper and middle dermis:
 - More common in children and young adults
 - More common in women
 - More common in the atopic patient
- 40% of patients with urticaria will have a component of angioedema:
 - Affects deeper subdermal and/or submucosal sites

Pediatric Considerations

- Urticaria is often the result of reactions to foods and infections
- Swelling of distal extremities and acrocyanosis may be prominent in infants
- Bullae may form in the center of the wheal, especially on legs and buttocks

ETIOLOGY

Acute:

Presumptive trigger may be found, but majority of cases are idiopathic

Course of < 6 wk

- Drugs:
 - Few have recurrent urticaria on later antigenic challenge
- Foods or additives
- Herbal medications, vaccines, opiates
- Insect bites and stings
- Connective tissue diseases
- Endocrine disorders, especially Hashimoto's thyroiditis
- Cancers, especially lymphoproliferative
- Hormonal imbalance, pregnancy, menstrual cycle, exogenous estrogens
- Infections:
 - Viral (including hepatitis, HIV)
 - Viral URI most common associated infection
 - Bacterial
 - Fungal
 - Parasitic
- Inhaled or contact allergen

- Emotional stress
- Physical urticaria—> 20 identified types, including:
 - Dermographism:
 - Most common physical form
 - Reaction to skin pressure
 - Linear wheals under tight clothing
 - Areas scratched with a firm object
 - Cholinergic:
 - Monomorphic wheals 2–3 mm
 - Bright red flare and intense pruritus
 - A response to elevated core temperature:
 - Hot bath
 - Fever
 - Exercise
 - Other rare forms:
 - Cold-induced (may be fatal in cold immersions)
 - Sun exposure
 - Aquagenic

Chronic:

Course of >6 wk

- 75% idiopathic in nature
- Autoimmune disease spectrum
- Immune complex–induced
- Often an unrecognized recurring physical urticaria
- May be due to occult or subclinical infection or systemic disease

 **DIAGNOSIS**

SIGNS AND SYMPTOMS

History

- Prior history
- Familial history
- Alleviating and/or aggravating factors
- Time course of current presentation:
 - Often helpful to circle lesions to document their duration
 - Fever and systemic symptoms
 - Arthralgias and myalgias
 - Weight loss and lymphadenopathy
 - Hypotension, flushing, headache
 - Swelling of mucosal sites
 - Respiratory distress or airway symptoms:

- May be part of an anaphylactic reaction

Physical-Exam

- Focus on signs of systemic allergic reaction or infection
- Airway—angioedema, airway compromise, inability to handle secretions, abnormal phonation, stridor
- Breathing—wheezing
- Circulation—systemic signs of anaphylaxis, such as hypotension
- Abdomen—hepatosplenomegaly, pregnancy
- Dermal—associated edema, associated petechiae, or purpura:
 - Generalized, transient, pruritic, well-circumscribed skin eruptions
 - May include palms or soles
 - May include bullae or purpuric lesions
 - Lesions are of various sizes and shapes, haphazard in distribution, and may become confluent
 - Wheals usually resolve in 3–4 hr
 - New lesions evolve as old ones resolve
- Lymphadenopathy
- Dermographism:
 - Scratch skin with a tongue blade; observe for linear wheal
- Cholinergic:
 - Exercise challenge to raise core temperature or induce sweating
- Expose to sunlight
- Cold-induced:
 - Place an ice cube on skin for 5 min
- Aquagenic:
 - Apply tap water at differing temperatures
- Significant mucosal edema:
 - Suspect angioedema
 - Severe reaction with hypotension
 - Suspect anaphylaxis
- Prolonged, painful, or nonblanching lesions:
 - Suspect urticarial vasculitis

ESSENTIAL WORKUP

- Complete history and physical exam
- Lesion appearance, location, timing, duration
- Identify as acute vs. chronic time-course
- Associated symptoms, triggers
- Coexisting diseases, allergies, medications
- Evaluate for sources of infection and signs of systemic diseases

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Acute urticaria: No labs needed
- Chronic urticaria:
 - Evaluate for infection or systemic disease:
 - CBC with differential, ESR, and/or CRP
 - Thyroid-stimulating hormone and thyroid functions
 - Urinalysis, liver function tests
- Skin biopsy if urticarial vasculitis suspected (not done in ED)

Imaging

- Acute cases: Not needed
- Chronic cases:
 - Directed at search for occult infection

Diagnostic Procedures/Surgery

Skin biopsy—for chronic urticaria or urticarial vasculitis

DIFFERENTIAL DIAGNOSIS

- Angioedema:
 - Can be life-threatening
 - May have component of abdominal symptoms
 - Hereditary or acquired
- Cutaneous vasculitis
- Serum sickness
- Erythema multiforme
- Bullous pemphigoid
- Juvenile rheumatoid arthritis
- Erythema marginatum
- Dermatitis herpetiformis
- Systemic mastocytosis
- Henoch–Schonlein purpura



TREATMENT

PRE HOSPITAL

- Cautions:
 - Systemic allergic reactions can rapidly progress if not treated with early epinephrine
- Severe reaction:
 - Manage airway, oxygen
 - IM epinephrine
 - Parenteral or inhaled β -agonist for bronchospasm

- IV crystalloid and vasopressors as needed

INITIAL STABILIZATION/THERAPY

Remove offending agent if possible

ED TREATMENT/PROCEDURES

- Largely symptomatic except in severe reactions
- Treatment aimed at stimulus, effector cells, inflammatory mediators, and target receptors
- β -Agonist (parenteral or inhaled):
 - Severe hives, angioedema, systemic features
- H_1 -receptor antagonist (1st or 2nd generation):
 - Mainstay of treatment
- H_2 -receptor antagonist:
 - May be beneficial as adjunct to H_1 blocker when no response to H_1 blocker alone
- Corticosteroid (oral):
 - Severe or refractory cases
- Avoid NSAIDs and opiates:
 - May exacerbate condition
- Concurrent use of ketoconazole or macrolides alters hepatic metabolism of antihistamine; use with caution

MEDICATION

- β -Agonists:
 - Epinephrine (1:1,000 solution): 0.1–0.5 mg IM q10–15min PRN (peds: 0.01 mg/kg, IM [max. single dose not to exceed 0.3 mg] q15min PRN)
 - IV epinephrine 0.1–0.25 mg (1:10,000 sol) IV over 5–10 min q5–15min then 1–4 μ g/min IV ONLY if anaphylactic shock
 - Albuterol (0.5% solution): 0.5 mL nebulized q20min PRN (peds: 0.01–0.05 mL/kg per dose [max. 0.5 mL/dose] nebulized q20min PRN bronchospasm)
 - Terbutaline: 0.25 mg SC q15–30min PRN (max. 0.5 mg q4h); (peds: <12 yr old; 0.005–0.01 mg/kg [max. 0.4 mg/dose] SC q15–20min \times 3 PRN bronchospasm)
- H_1 -receptor antagonist (1st generation—lipophilic and sedating)
 - Diphenhydramine: 25–50 mg PO, IV, or IM q6h (peds: 1 mg/kg q6h [max. 300 mg/24 h])
 - Hydroxyzine: 25–50 mg PO or IM q6h (peds: 2 mg/kg/24 h PO div. q8h or 0.5–1 mg/kg IM q4–6h PRN)
- H_1 -receptor antagonist (2nd generation—less sedating and preferred):
 - Cetirizine: Adult and peds \geq 6 yr old: 5–10 mg PO QD (peds 2–6 yr old: 2.5 mg QD to BID)

- Loratadine: 10 mg PO BID (peds 2–6 yr old: 5 mg PO QD)
- Fexofenadine: 60 mg PO BID or 180 mg PO QD (peds 6–12 yr old: 30 mg PO BID)
- H₂-receptor antagonist (suggested dosage):
 - Famotidine: 20 mg IV q12h or 20–40 mg PO QHS (peds: 1 mg/kg/d div. QID [max. 40 mg/24 h])
 - Ranitidine: 150 mg PO BID (peds: Neonate: 2–4 mg/kg/24 h PO div. q8–12h or 2 mg/kg/24 h IV div. q6–8h; infants and children: 4–5 mg/kg/24 h PO div. q8–12h or 2–4 mg/kg/24 h IV or IM div. q6–8h)
- Corticosteroid:
 - Methylprednisolone: 125 mg IV (peds: Start at 2 mg/kg × 1)
 - Prednisolone: 50 mg PO QD for 3 days (peds: 0.5–2 mg/kg/24 h [max. 80 mg/24 h] div. QD to BID for 3–5 days)
 - Prednisone: 40 mg PO QD or 20 mg PO BID for 3–5 days (peds: 1–2 mg/kg/24 h [max. 80 mg/24 h] div. QD to BID for 3–5 days)
- Antileukotrienes:
 - Montelukast: 10 mg PO QD
 - Zafirlukast: 20 mg PO BID

First Line

- H₁-receptor antagonist, 2nd generation
- Corticosteroids
- β-Agonists:
 - Albuterol if bronchospasm present
 - Epinephrine for severe or systemic signs

Second Line

- Antileukotrienes
- H₁-receptor antagonist, 1st generation
- H₂-receptor antagonist, data weak



FOLLOW-UP

DISPOSITION

Admission Criteria

- Systemic allergic reaction with:
 - Respiratory distress or failure
 - Refractory hypotension or shock
- Severe case with dysfunction of health-related quality of life
- Other comorbidities

Discharge Criteria

- Normal vitals
- Absence of other condition requiring admission
- Adequate ability of caregivers at home to monitor for further exacerbations

FOLLOW-UP RECOMMENDATIONS

Follow with PCP, especially if lasting >6 wk

PEARLS AND PITFALLS

- If severe presentation, there is often a biphasic course. Rebound may occur in 4–6 hr
- Chronic urticaria often has a systemic cause

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Angioedema
- Erythema Multiforme
- Vasculitis



CODES

ICD9

- 708.0 Allergic urticaria
- 708.1 Idiopathic urticaria
- 708.9 Unspecified urticaria

ICD10

- L50.0 Allergic urticaria
- L50.1 Idiopathic urticaria
- L50.9 Urticaria, unspecified

UVULITIS

James P. Brewer

BASICS

DESCRIPTION

Uvulitis refers to any inflammatory condition involving the uvula. Uvulitis can be separated into 2 broad categories:

- Infectious:
 - Bacterial
 - Viral
 - Candidal
- Traumatic or noninfectious

EPIDEMIOLOGY

Incidence and Prevalence Estimates

- Exact incidence is unknown owing to limited reporting
- Once thought to be rare but may in fact be more common (e.g., viral etiologies)
- Children (age 5–15) more often affected than adults due to prevalence of group A streptococcal infections in this age group
- Noninfectious causes more common than infectious causes in adult population

ETIOLOGY

- Infectious:
 - Bacterial:
 - Group A streptococcal infection (GAS), most common
 - *Haemophilus influenzae* type b (Hib)
 - Other bacterial infections (*Fusobacterium nucleatum*, *Prevotella intermedia*, *Streptococcus pneumoniae*)
 - Viral:
 - Not well reported but suspected in mild/transient cases
 - Known to cause uvular lesions however rare in isolation
 - Coxsackie virus (other enteroviruses)
 - Herpes simplex virus
 - Varicella-zoster virus
 - Epstein–Barr virus
 - Candidal infections
- Noninfectious:
 - Trauma/procedure related
 - Inhalation/ingestion of chemical or thermal irritants

- Vasculitis
- Allergic
- Angioedema:
 - Hereditary
 - Medication induced (e.g., Angiotensin-converting enzyme inhibitor [ACEi], Angiotensin receptor blocker [ARB])

DIAGNOSIS

SIGNS AND SYMPTOMS

Dependent upon etiology and associated structural involvement (pharyngitis, epiglottitis, laryngitis, etc.)

History

- Generally rapid in onset (< 4–6 hr) depending on etiology
- All types:
 - Foreign-body sensation
 - Sore throat
 - Dysphagia
 - Odynophagia
 - Dyspnea
- Infectious:
 - Fever (reported)
- Noninfectious:
 - Trauma or recent procedure
 - New medication exposure (ACEi)
 - Caustic or thermal ingestion
- Prior event of tongue, lip, or mouth swelling
- Immunization history in pediatric population
- Medical comorbidity leading to immune compromise

Physical-Exam

- Ranging from limited and well appearing to severe and marked distress
- General:
 - “Toxic” appearance
 - Muffled or “hot-potato” voice
 - Drooling
 - Stridor
 - Gagging
 - Respiratory distress
- HEENT:
 - Erythematous or pale uvula

- Uvular edema
- Exudate (present on uvula or oral pharynx)
- Cervical lymphadenopathy
- Pharyngitis
- Associated findings:
 - Fever
 - Hypoxia
 - Urticaria
 - Wheezing

ESSENTIAL WORKUP

- Evaluation and stabilization of airway as needed
- Determine infectious vs. noninfectious etiology
- Initiate treatment based on suspected etiology (antibiotics, steroids, antihistamine, etc.)
- Consultation with otolaryngologist as warranted

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Rapid GAS antigen
- Surface mucosa bacterial culture
- CBC:
 - Leukocytosis suggesting bacterial infection
 - Eosinophilia suggesting allergic etiology
- Complement testing:
 - Elevated C4 level suggesting esterase deficiency
 - C1 esterase immunochemical assay

Imaging

- Used to rule out other conditions in the differential diagnosis when clinical suspicion exists or when physical exam is limited
- Lateral neck x-ray to visualize and evaluate the epiglottis or for foreign-body aspiration
- CT scan soft tissue neck with IV contrast to evaluate for space occupying fluid collection, cellulitis, deep tissue involvement

Diagnostic Procedures/Surgery

- As warranted and in consultation with otolaryngology when severity of disease warrants:
 - Fiberoptic nasopharyngeal endoscopy
 - Cricothyrotomy
 - Uvular aspiration/decompression

DIFFERENTIAL DIAGNOSIS

- Pharyngitis
- Peritonsillar abscess
- Retropharyngeal abscess
- Epiglottitis
- Angioedema
- Aspirated foreign body



TREATMENT

PRE HOSPITAL

- Rapid assessment of airway, definitive management as warranted
- Supplemental oxygen
- Peripheral IV access
- Assessment of patient surroundings, potential ingestions/inhalants
- Per pre-hospital protocol, IM epinephrine injection, nebulized β -agonist, or racemic epinephrine
- Rapid/emergent transport

INITIAL STABILIZATION/THERAPY

- Initial focus on managing ABCs
- Rapid assessment of airway and need for definitive management
- Peripheral IV access
- Cardiac and oxygen saturation monitoring
- Continued pre-hospital therapy or initiate respiratory therapy:
 - Supplemental oxygen
 - Nebulized β -agonists or racemic epinephrine
- Definitive airway:
 - Endotracheal intubation:
 - Rapid sequence
 - Delayed sequence/awake
 - Fiberoptic assist and indirect laryngoscopy
 - Cricothyrotomy in severe cases
- Early consultation with otolaryngology as warranted

ED TREATMENT/PROCEDURES

- Basic ED treatment is focused on rapid reversal of inflammatory conditions (allergic, angioedema)
- Oral therapy vs. parenteral dependent upon severity of condition

MEDICATION

- Severe conditions (airway compromise):
 - Epinephrine, 1:1,000: 0.3–0.5 mg (peds: 0.01 mg/kg) SQ or IM q30min × 3 doses
 - Diphenhydramine: 25–50 mg (peds: 1–2 mg/kg) IV
 - Methylprednisolone: 125 mg (peds: 0.5–1 mg/kg) IV q4h
- Suspected infectious etiology:
 - Empiric parenteral antibiotic to cover most common etiologies (GAS and Hib)
 - Several options based on patient profile/allergy:
 - Ceftriaxone: 1–2 g (peds: 50 mg/kg) IV (max. dose 2 g/d)
 - Clindamycin: 300 mg (peds: 25–40 mg/kg) IV q8h
 - Empiric oral antibiotic options:
 - Penicillin V: 500 mg (peds: <27 kg 250 mg, >27 kg 500 mg) PO BID–TID × 10 days
 - Amoxicillin: 875 mg (peds: 50 mg/kg/d PO div. q8h) PO q8h × 10 days
 - Clindamycin: 300 mg (peds: 25–40 mg/kg) PO QID × 10 days
- Suspected hereditary angioedema:
 - Anabolic steroid:
 - Danazol: 200 mg PO BID–TID
 - Purified C1 inhibitor concentrate:
 - Berinert: 20 U/kg IV × 1
 - Cinryze: 1,000 U IV
 - Selective bradykinin B₂-receptor antagonist:
 - Icatibant: 30 mg SC × 1
 - Reversible inhibitor of plasma kallikrein:
 - Ecallantide: 30 mg SQ × 1 (as 3–10 mg injections)
 - Fresh frozen plasma:
 - Generally not for acute attacks



FOLLOW-UP

DISPOSITION

Disposition dependent upon severity of condition and response to therapy

Admission Criteria

- Severe airway obstruction warranting definitive airway and ventilatory management
- Need for surgical intervention
- Indication of systemic bacterial infection and need for parenteral antibiotics
- Moderate to severe conditions not responsive to treatment:
 - Hypoxia or oxygen requirement

- Ongoing respiratory compromise
- Inability to tolerate oral intake
- Intractable pain
- Significant comorbid illness
- Poor social conditions limiting outpatient care

Discharge Criteria

- Rapid reversal of condition
- Observation in the ED for 4–6 hr without recurrent symptoms
- No respiratory compromise
- Able to tolerate oral medications and liquids
- Close follow-up available within 24–48 hr
- Access to prescription medications

Issues for Referral

History of recurrent angioedema warrants adjustment of medication, possible referral to Otolaryngology

FOLLOW-UP RECOMMENDATIONS

- Severe infectious etiologies warrant close follow-up with primary physician (24–48 hr) to ensure improvement
- For suspected angioedema, immediately discontinue use of ACEi and ARB

PEARLS AND PITFALLS

- Uvulitis can be caused by several etiologies ranging from infection to hereditary disorder
- Treatment should be directed toward the suspected etiology based on history and exam
- Uvulitis in isolation rarely causes respiratory compromise. If severe respiratory distress, look for additional causes (epiglottitis, anaphylaxis, retropharyngeal abscess, etc.)
- Emergent definitive airway management should be anticipated with tools, medications, and other resources kept near the patient at all times
- Early consultation with otolaryngology when anticipated

ADDITIONAL READING

- Buyantseva LV, Sardana N, Craig TJ. Update on treatment of hereditary angioedema. *Asian Pac J Allergy Immunol*. 2012;30:89–98.
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CODES

ICD9

528.3 Cellulitis and abscess of oral soft tissues

ICD10

K12.2 Cellulitis and abscess of mouth

VAGINAL BLEEDING

Carla C. Valentine



BASICS

DESCRIPTION

- Common presenting complaint to EDs
- Most cases have benign etiology
- Some patients may have potentially life-threatening conditions
- Key principles in evaluating women with vaginal bleeding:
 - Any woman capable of childbearing might be pregnant
 - Menstrual and sexual histories do not rule out pregnancy

ETIOLOGY

PREGNANCY RELATED

- Early pregnancy:
 - Ectopic pregnancy (occurs in 2% of pregnancies)
 - Abortion:
 - Threatened, incomplete, complete, missed, inevitable, septic
 - Molar pregnancy
 - Trauma
- Later pregnancy:
 - Placenta previa
 - Placental abruption
 - Molar pregnancy
 - Labor
 - Trauma
- Immediate postpartum period:
 - Postpartum hemorrhage
 - Uterine inversion
 - Retained placenta
 - Endometritis

NONPREGNANT PATIENTS

- Dysfunctional uterine bleeding (DUB)
- Structural abnormalities:
 - Uterine fibroids
 - Cervical/endometrial polyps
 - Pelvic tumors
- Atrophic endometrium:
 - Most common cause of postmenopausal bleeding
- Rare for systemic disorders to present solely with vaginal bleeding:

- Von Willebrand disease
- Idiopathic thrombocytopenic purpura
- Trauma
- Foreign bodies
- Infections

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Light headedness
- Fatigue
- Weakness
- Thirst
- Duration of bleeding
- Quantity:
 - Average tampon holds ~ 5 mL
 - Average pad holds ~ 5–15 mL
- Last menstrual period
- Home pregnancy tests
- Prior ectopic pregnancy
- Passage of clots or tissue
- Menstrual history
- Family history
- Trauma

Physical-Exam

- Vital signs
- Cardiopulmonary exam
- Abdominal exam (gravid uterus, masses)
- Pelvic exam:
 - Source of bleeding
 - Evidence of trauma
 - Cervical os open or closed
- Change in mental status may occur with significant blood loss and/or hypotension

ESSENTIAL WORKUP

- Qualitative pregnancy test:
 - Point-of-care urine-based pregnancy test preferred
- Pelvic exam:
 - Essential for all women with vaginal bleeding

- Assess whether cervical os open or closed
- Delay pelvic exam pending US result in late pregnancy:
 - Evaluate for placenta previa
- Defer exam if patient is near term with possible rupture of fetal membranes
- Pregnancy test mandatory for all patients with childbearing potential
- Early pregnancy:
 - Blood type and Rh
 - US to confirm intrauterine pregnancy (IUP)
 - Quantitative β -human chorionic gonadotropin (HCG)
 - Hematocrit
 - Type and cross-match:
 - Ectopic pregnancy
 - Low hematocrit levels
 - Hemodynamic instability
 - UA
- Later pregnancy:
 - Type and Rh
 - Fetal heart tones
 - US indications:
 - No fetal heart tones
 - No documented IUP
 - Unknown placental lie
 - Hematocrit if significant bleeding
 - Type and cross-match if placenta previa/abruption or low hematocrit levels
 - DIC panel if placental abruption:
 - Platelets, PT, PTT, Fibrinogen, fibrin split products
- Early postpartum:
 - US for retained products
 - Hematocrit
 - β -HCG if concern for retained tissue

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Qualitative and/or quantitative HCG
- Hematocrit for women with significant bleeding
- Type and Rh
- Platelet count for suspected thrombocytopenia
- PT/PTT for suspected coagulopathy
- Send any passed tissue or clot for pathology evaluation

Imaging

- Bedside US may be indicated based on presentation, pregnancy status, and other

considerations:

- US and discriminatory zone:
 - _ Transabdominal US:
 - Should detect gestational sac if HCG > 6,500 mIU/mL
 - _ Transvaginal US:
 - Should detect gestational sac if HCG > 1,000–1,500 mIU/mL

DIFFERENTIAL DIAGNOSIS

- DUB
- Ectopic pregnancy
- Menorrhagia
- Menometrorrhagia
- Threatened miscarriage
- Placental abruption
- Placenta previa
- Postpartum hemorrhage
- Leiomyoma
- Pelvic masses and tumors
- Postcoital bleeding
- Traumatic injury
- Thyroid dysfunction
- Bleeding disorders



TREATMENT

PRE HOSPITAL

- Establish IV 0.9% NS with 1–2 L fluid bolus for significant bleeding or hypotension
- Administer high-flow oxygen in pregnant or unstable patients
- In later pregnancy:
 - _ Place patient in left lateral recumbent position to prevent occlusion

INITIAL STABILIZATION/THERAPY

- Manage airway and resuscitate as indicated
- Place cardiac/pulse oximeter monitors
- Oxygen for significant bleeding or unstable patient
- Establish 2 large-bore IVlines and initiate fluid bolus (1–2 L) for hypotensive patients
- Type and cross-match:
 - _ Transfuse blood if continued hypotension from blood loss despite IV fluid resuscitation
 - _ Conjugated estrogens (Premarin) 25 mg IV slowly over 10–15 min q4–6h until bleeding stops for uncontrolled menorrhagia:

- Not to exceed 4 doses

ED TREATMENT/PROCEDURES

- If unstable with surgical condition, arrange for transfer of the patient to the OR as soon as possible
- RhoGAM for vaginal bleeding, pregnancy, and Rh-negative mother

EARLY PREGNANCY

- If US reveals an ectopic pregnancy:
 - Methotrexate according to standards at treating institution
 - Definitive treatment is surgery
- If US reveals an IUP without concerns of heterotopic pregnancy (1/2,600–1/30,000):
 - Discharge patient with arranged obstetric follow-up with precautions for a threatened miscarriage
- US indeterminate for IUP or ectopic with β -HCG greater than institutional discriminatory zone:
 - Cannot exclude ectopic pregnancy
 - If hemodynamically stable with little bleeding, repeat measurement of β -HCG and outpatient obstetric follow-up within 48 hr
 - Strict return parameters
- US indeterminate for IUP or ectopic with β -HCG level less than institutional discriminatory zone:
 - Patient stable with low risk for ectopic pregnancy may be discharged
 - Repeat measurement of β -HCG level and obstetric follow-up within 48 hr
 - Patient may still have an ectopic pregnancy
- Complete abortion:
 - Discharge patient if stable without significant ongoing bleeding
- Incomplete abortion:
 - Obstetric consultation is required
 - Dilation and curettage vs. expectant management
- Missed abortion:
 - Expectant management initially
- Septic abortion:
 - IV antibiotics and admission
- Molar pregnancy:
 - Chemotherapy
 - Very responsive in early stages of disease

LATER PREGNANCY

- Placenta previa:
 - Obstetric consultation for possible admission
- Placental abruption:
 - Induction of labor if large
 - Can lead to fetal/maternal death

- May require cesarean section

IMMEDIATE POSTPARTUM

- Uterine inversion:
 - Prevent by avoiding strong traction on umbilical cord after delivery
 - Replace uterus immediately
 - Occasionally requires operative management
- Postpartum hemorrhage:
 - Extraction of placenta if retained
 - Hysterectomy if uncontrolled life-threatening bleeding

EARLY POSTPARTUM

- Retained tissue:
 - Dilation and curettage
- Endometritis:
 - IV antibiotics

NONPREGNANT

- Menses:
 - NSAIDs and supportive care
- DUB:
 - <35–40 yr of age:
 - If known anovulatory DUB:
 - Medroxyprogesterone (Provera)—warn patient about withdrawal bleeding
 - Oral contraceptive pill daily for 7 days
 - Patients >35–40 yr of age:
 - US for any masses palpated during physical exam
 - Gynecologic referral
 - Uterine sampling necessary before initiation of hormonal treatment
 - Evaluate for endometrial cancer

STRUCTURAL ABNORMALITIES

- Pap smear/biopsy for cervical lesions
- US for workup of pelvic masses
- Fibroids or uterine tumors
- Conservative management or lumpectomy/hysterectomy

MEDICATION

- Conjugated estrogens 25 mg IV slowly over 10–15 min q6h until bleeding stops(not to exceed 4 doses)
 - If no response after 1–2 doses re-evaluation needed
- Known anovulatory DUB:
 - Medroxyprogesterone 10 mg PO per day for 1st 10 days of menstrual cycle (warn patient about withdrawal bleeding)
 - Norethindrone and ethinyl estradiol (Ortho-Novum) 1/35 BID for 7 days
- MICRhoGAM 50 µg IM if <12 wk pregnant

- RhoGAM 300 µg IM if > 12 wk pregnant



FOLLOW-UP

DISPOSITION

Admission Criteria

- Ectopic pregnancy not meeting methotrexate discharge criteria
- Uterine inversion
- Septic abortion
- Placental abruption
- Postpartum hemorrhage
- Endometritis
- Unstable DUB
- Newly diagnosed molar pregnancy

Discharge Criteria

- Stable vital signs
- Confirmed IUP
- Ectopic pregnancy meeting institutional methotrexate discharge criteria
- Pregnant patient with low risk for ectopic pregnancy:
 - No findings of IUP on US
 - Levels of β-HCG below discriminatory zone
- Nonpregnant patients with vaginal bleeding that are hemodynamically stable

Issues for Referral

Obstetric/gynecologic referral

FOLLOW-UP RECOMMENDATIONS

- Obstetric referral within 48 hr for 1st-trimester vaginal bleeding without identified IUP
- OB/GYN referral for patients with menorrhagia for continued evaluation, workup, and treatment

PATIENT EDUCATION

Ectopic precautions: Return immediately for increasing abdominal pain, vaginal bleeding more than 1 pad per hr for 3–4 hr, fever >100.4°F, syncope, or dizziness. Patients should not be left alone until the diagnosis of ectopic pregnancy can be safely ruled out. Family and friends should also be instructed on the warning signs and symptoms of ruptured/bleeding ectopic pregnancies.

PEARLS AND PITFALLS

- Pregnancy test for all women of reproductive age
- If there is 1st-trimester vaginal bleeding, evaluate for ectopic pregnancy

ADDITIONAL READING

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- Tsai MC, Goldstein SR. Office diagnosis and management of abnormal uterine bleeding. *Clin Obstet Gynecol.* 2012;55:635–650.



See Also (Topic, Algorithm, Electronic Media Element)

- Vaginal Bleeding in Pregnancy
- Threatened Abortion
- Placental Abruption
- Placenta Previa
- Ectopic Pregnancy



CODES

ICD9

- 623.8 Other specified noninflammatory disorders of vagina
- 640.90 Unspecified hemorrhage in early pregnancy, unspecified as to episode of care or not applicable
- 641.80 Other antepartum hemorrhage, unspecified as to episode of care or not applicable

ICD10

- O20.9 Hemorrhage in early pregnancy, unspecified
- O46.90 Antepartum hemorrhage, unspecified, unspecified trimester
- N93.9 Abnormal uterine and vaginal bleeding, unspecified

VAGINAL BLEEDING IN PREGNANCY

Paul Ishimine

BASICS

DESCRIPTION

- Major cause of maternal/fetal morbidity and mortality
- Early pregnancy hemorrhage (≤ 20 wk):
 - Occurs in 30% of all pregnancies
 - 50% lead to spontaneous abortion
- Late pregnancy hemorrhage (> 20 wk):
 - Occurs in 3–5% of all pregnancies
- Risk factors:
 - Advanced maternal age
 - Substance abuse
 - Pelvic inflammatory disease (PID)
 - Previous cesarean section
 - Previous termination of pregnancy
 - Previous dilation and curettage (D&C)
 - Previous ectopic pregnancy
 - Increased parity
 - Multiple gestation
 - Preeclampsia
 - Hypertension
 - Trauma
 - Use of assisted reproductive technology
- Genetics:
 - 50–60% of miscarriages due to chromosomal abnormalities

ETIOLOGY

- Vaginal
- Cervical
- Uterine
- Uterine–placental interface
- Hematologic dysfunction

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Intensity and duration of bleeding:
 - Amount (clots, number of pads)
 - Color (dark or bright red)
 - Painful or painless
 - Watery, blood-tinged mucus
 - Life-threatening conditions may present with only minimal bleeding
- Last normal menstrual period
- Passage of tissue
- Estimated duration of gestation
- Gravidity/parity
- Fever
- Last intercourse
- Intrauterine device use
- Previous obstetric–gynecologic complications
- Syncope or near-syncope
- Previous obstetric–gynecologic complications
- Spontaneous abortion: Classically crampy, diffuse pelvic pain
- Ectopic pregnancy: Classically sharp pelvic pain with lateralization
- Placenta previa: Classically painless bright red hemorrhage
- Placental abruption: Classically painful dark red hemorrhage

Physical-Exam

- Vital signs:
 - Tachycardia
 - Hypotension
 - Orthostatic changes
 - Signs of hemodynamic instability may be absent due to pregnancy-related physiologic increase in blood volume
- Fetal heart tones:
 - Fetal cardiac activity seen on transvaginal US at 6.5 wk
 - Auscultated with hand-held Doppler past 10 wk gestation
 - Normal fetal heart rate: 120–160 beats/min
- Abdominal exam:
 - Uterine size:
 - 12 wk: Palpable in abdomen
 - 20 wk: Palpable at umbilicus
 - Peritoneal signs
 - Firm or tender uterus in late pregnancy suggests abruption
- Pelvic exam—only in early pregnancy:
 - Evaluate source and intensity of bleeding
 - Determine patency of cervical os (use finger and only in first trimester):
 - Threatened abortion: os closed
 - Inevitable abortion: os open

- Incomplete abortion: os open or closed
- Complete abortion: os closed
- Embryonic demise (missed abortion): os closed
- Products of conception (POC) may be noted in incomplete or completed abortion:
 - POC in the cervical os can result in profuse bleeding
- Evaluate uterine size, tenderness
- Evaluate for uterine fibroids or adnexal masses
- Late pregnancy: Do not perform pelvic exam unless in controlled OR setting:
 - Severe hemorrhage may ensue
 - Placenta previa or vasa previa must be ruled out by US prior to pelvic exam

ESSENTIAL WORKUP

- CBC
- Type and screen
- Quantitative HCG in early pregnancy
- Urinalysis
- US:
 - Transvaginal US provides more information than transabdominal US in early pregnancy

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC:
 - Dilutional “anemia” is a normal physiologic change in pregnancy:
 - Blood volume expands by 45%
- Qualitative beta-human chorionic gonadotropin (β -hCG)
- Quantitative β -hCG:
 - Imperfect correlation with US findings
 - Detectable 9–11 days following ovulation
- Blood typing and Rh typing:
 - Cross-match if significant bleeding
- Disseminated intravascular coagulation (DIC) panel in embryonic demise, placental abruption
- Blood cultures with septic abortion
- Suspected POC to lab for identification of chorionic villi

Imaging

- US:
 - Essential diagnostic modality:

- Confirms intrauterine pregnancy (IUP)
- Detects gestational sac at 5 wk (usually with β -hCG \geq 1,000–2,000 IU), yolk sac at 6 wk, and cardiac activity at 5–6 wk of gestation
- Essentially rules out ectopic pregnancy by showing IUP (except in women at high risk for heterotopic pregnancy)
- Proves ectopic pregnancy by showing fetal pole outside uterus
- Suggests ectopic pregnancy by detecting free fluid in cul-de-sac or adnexal mass
- Detects retained POC
- Demonstrates “snowstorm” appearance within uterus with gestational trophoblastic disease

Diagnostic Procedures/Surgery

- **Culdocentesis:**
 - Limited use
 - Identifies free fluid in cul-de-sac
- **D&C or vacuum aspiration:**
 - Indicated if suspected incomplete or septic abortion, embryonic demise, gestational trophoblastic disease, or anembryonic gestation to evacuate retained POC
- **Laparoscopy/laparotomy:**
 - Indicated for unstable patients
 - Definitive diagnosis and treatment of ectopic pregnancy

DIFFERENTIAL DIAGNOSIS

- **Early pregnancy (< 20 wk):**
 - Implantation bleeding
 - Threatened abortion
 - Complete, incomplete, inevitable, embryonic demise (missed abortion), and septic abortion
 - Ectopic pregnancy
 - Heterotopic pregnancy
 - Gestational trophoblastic disease (molar pregnancy)
 - Subchorionic hemorrhage
 - Anembryonic gestation (blighted ovum)
 - Infection (e.g., cervicitis)
 - Trauma
 - Cervical and vaginal lesions (e.g., polyps, ectropion, carcinoma)
 - Bleeding disorders
- **Late pregnancy (> 20 wk):**
 - Placental abruption (30%)
 - Placenta previa (20%)
 - Bloody show (associated with cervical insufficiency or labor)

- Vasa previa
- Cervical/vaginal trauma or pathology
- Uterine rupture (uncommon)
- Infection (e.g., cervicitis)
- Trauma
- Cervical and vaginal lesions (e.g., polyps, ectropion, carcinoma)
- Bleeding disorders



TREATMENT

PRE HOSPITAL

- Unstable vital signs warrant aggressive resuscitation
- In late pregnancy, position patient on left side to decrease uterine compression of inferior vena cava (IVC)
- Consider preferential transport of a woman with late pregnancy to a facility with obstetric capabilities

INITIAL STABILIZATION/THERAPY

- Airway management
- Oxygen
- Pulse oximetry
- Cardiac monitor
- 2 large-bore IV lines
- Blood transfusion as indicated
- Continuous fetal monitoring in later pregnancy

ED TREATMENT/PROCEDURES

- All women with early pregnancy vaginal bleeding must be evaluated for ectopic pregnancy (preferably by transvaginal US)
- Administer Anti-Rh₀ (D) immune globulin if patient is Rh-negative
- Suspected ectopic pregnancy:
 - Unstable: Consider bedside US with emergent OB/GYN consultation for laparoscopy/laparotomy
 - Stable: Perform US:
 - If confirmatory or suggestive of ectopic pregnancy, obtain OB/GYN consultation for surgery or methotrexate therapy
 - If inconclusive, obtain OB/GYN consultation and arrange for repeat β -hCG testing in 2 days
- Threatened abortion:
 - Emergent OB/GYN consultation for heavy/uncontrolled bleeding
 - Arrange OB/GYN follow-up for minimal bleeding
- Inevitable/incomplete/missed (embryonic demise) abortion:

- POC in the cervical os can result in profuse bleeding
- If POC cannot be removed with gentle traction, obtain emergent OB/GYN consultation
- Arrange OB/GYN follow-up if bleeding minimal
- Complete abortion:
 - Emergent OB/GYN consultation for heavy/uncontrolled bleeding
 - Arrange OB/GYN follow-up if bleeding minimal
- Septic abortion:
 - Initiate broad-spectrum antibiotic therapy
 - Emergent OB/GYN consultation for D&C
- Late pregnancy vaginal bleeding:
 - Hemodynamic stabilization:
 - Fluid resuscitation
 - Positioning of patient onto left side or displacement of uterus laterally to relieve compression by IVC
 - DIC:
 - Associated with late pregnancy bleeding
 - Especially with placental abruption
 - Treated with blood products
 - Immediate obstetric consultation and rapid transfer to obstetric unit

MEDICATION

First Line

- Anti-Rh₀ (D) immune globulin: < 12 wk–50 µg IM; > 12 wk–300 µg IM
- Methotrexate:
 - Variable dosing regimens
 - Only recommended for hemodynamically stable women with unruptured ectopic pregnancy with low β-hCG
- Antibiotics for septic abortion:
 - Multiple acceptable antibiotic regimens
 - Must provide polymicrobial coverage

Second Line

Misoprostol has been used in completed abortion to facilitate uterine evacuation in completed miscarriage



FOLLOW-UP

DISPOSITION

Admission Criteria

- Early pregnancy vaginal bleeding with:
 - Unstable vital signs or significant bleeding
 - Ruptured ectopic pregnancy
 - Incomplete abortion (open os)
 - Septic abortion
- All patients with late pregnancy vaginal bleeding need to be admitted to a labor and delivery unit

Discharge Criteria

- Stable patients with threatened abortion complete abortion, embryonic demise, or anembryonic gestation
- Asymptomatic, hemodynamically stable patient with small, unruptured ectopic (or suspected ectopic) pregnancy after OB/GYN consultation
- Controlled bleeding from vaginal/cervical source

Issues for Referral

- Patients with embryonic demise, anembryonic gestation, or gestational trophoblastic disease need to be referred for uterine evacuation if D&C not performed in ED
- Women with threatened, inevitable, complete, or missed (embryonic demise) abortion should have OB/GYN follow-up within 24–48 hr

FOLLOW-UP RECOMMENDATIONS

- Discharge instructions:
 - No strenuous activity, tampon use, douching, or intercourse
 - Seek medical advice for increased pain, bleeding, fever, or passage of tissue
- All pregnant women with vaginal bleeding during pregnancy who are discharged from the ED require follow-up care
- Women with threatened abortions, known or suspected ectopic pregnancy require repeat β -hCG testing and repeat exams in 2 days

PEARLS AND PITFALLS

- Failure to check Rh status in pregnant women with vaginal bleeding
- Failure to give Anti-Rh₀ (D) immune globulin in Rh-negative women with vaginal bleeding
- Placenta previa or vasa previa must be ruled out by US prior to pelvic exam in late pregnancy

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Abortion, Spontaneous
- Ectopic Pregnancy
- Hydatidiform Mole
- Placental Abruption
- Placenta Previa
- Postpartum Hemorrhage



CODES

ICD9

- 634.90 Spontaneous abortion, without mention of complication, unspecified
- 640.90 Unspecified hemorrhage in early pregnancy, unspecified as to episode of care or not applicable
- 641.80 Other antepartum hemorrhage, unspecified as to episode of care or not applicable

ICD10

- O03.9 Complete or unspecified spontaneous abortion without complication
- O20.9 Hemorrhage in early pregnancy, unspecified
- O46.90 Antepartum hemorrhage, unspecified, unspecified trimester

VAGINAL DISCHARGE/VAGINITIS

Elizabeth M. Foley • Carrie Tibbles

BASICS

DESCRIPTION

- Vaginitis is vulvovaginal inflammation with or without abnormal vaginal discharge.
 - Common symptoms: Itching, burning, irritation, pain.
 - Abnormal discharge is defined as an increased amount or change in color.
- Some amount of vaginal discharge is normal.
 - Glands in the cervix produce clear mucus that may turn white or yellow when exposed to air.

ETIOLOGY

- Bacterial vaginosis (BV):
 - The most common cause
 - Loss of normal *Lactobacillus* sp. (e.g., antibiotics)
 - Inability to maintain normal vaginal pH
 - Overgrowth of normally present bacteria such as *Gardnerella vaginalis*, *Mycoplasma hominis*, *Mobiluncus* sp., *Prevotella* sp., and Peptostreptococcal
- Bacterial infections:
 - *Trichomonas vaginalis* (Trichomoniasis)
 - Group A strep
 - *Staphylococcus aureus*
- Fungal infections:
 - *Candida* sp. most common
 - Often underlying immune dysfunction:
 - Diabetes
 - HIV
- Chemical irritants
- Foreign body
- Atrophic vaginitis:
 - Caused by estrogen deficiency
- Hypersensitivity
- Collagen vascular disease
- Herpes simplex virus (HSV):
 - Vulvovaginitis
 - Cervicitis
- Lichen sclerosis (atrophic)
- Fistula

DIAGNOSIS

SIGNS AND SYMPTOMS

- Abnormal discharge
- Vaginal or vulvar irritation
- Localized pain
- Dyspareunia
- Erythema
- Edema
- Dysuria
- Pruritus
- Excoriations
- Abnormal odor
- Can be asymptomatic

History

- Description and duration of symptoms
- Description of discharge, if any
- Timing with regard to menses
- Sexual history of patient and partners
- Sexual practices
- Hygienic practices
- Use of oral contraceptives and/or antibiotics
- Likelihood of pregnancy
- Other symptoms (e.g., abdominal pain; must rule out pelvic inflammatory disease [PID])

Physical-Exam

- Abdominal exam to assess for tenderness
- Inspection of vulva, vaginal os, perineal area
- Speculum and bimanual exam

ESSENTIAL WORKUP

- Pelvic exam
- Saline and KOH wet prep of vaginal discharge

DIAGNOSIS TESTS & NTERPRETATION

Lab

- β -human chorionic gonadotropin (β -hCG)
- pH of discharge with Nitrazine paper:
 - Normal in premenopausal adults: < 4.5

- >4.5: BV, trichomoniasis
- pH normal in candidiasis
- Saline wet prep of discharge:
 - Clue cells: BV
 - Motile flagellated protozoa: Trichomoniasis
 - Presence of polymorphonuclear leukocytes
- Potassium hydroxide (KOH) wet prep of discharge:
 - Pseudohyphae, budding yeast: Candidiasis
- KOH prep “Whiff” test:
 - Amine or “fishy” odor suggests BV, trichomoniasis.
- Trichomonas Rapid Test:
 - Point-of-care test
 - Immunochromatographic dipstick
- PIP test card for BV:
 - Point-of-care test
 - Detects proline aminopeptidase
- Nucleic acid probe test for *Trichomonas*, *G. vaginalis*, and *Candida albicans*
- Gram stain:
 - Large, gram-positive rods: Lactobacilli (normal flora)
 - Small, gram-variable coccobacilli and curved rods: *Gardnerella*, *Prevotella*, *Mobiluncus* (BV)
- Vaginal culture:
 - *Gardnerella*: Not routinely recommended
 - *Candida*: Recommended for recurrently symptomatic patients
 - *Trichomoniasis*: Gold standard
- Endocervical swab for gonorrhea (culture—Thayer–Martin media; DNA probe; amplification techniques—PCR/LCR) and chlamydia (DNA probe or amplification techniques—PCR/LCR)
- Viral cultures for HSV, DFA, or Tzanck smear for multinucleated giant cells if ulcers or vesicles are present
- Urinalysis/urine culture if c/o dysuria
- Rule out sexually transmitted infections:
 - GC/Chlamydia testing
 - Consider RPR to rule out syphilis.
 - Discuss HIV testing.

Imaging

N/A unless fistula is suspected.

DIFFERENTIAL DIAGNOSIS

- UTI
- PID
- Dermatitis

- Discharge from cervicitis can be mistaken for vaginitis
- *Chlamydia trachomatis*
- *Neisseria gonorrhoeae*



TREATMENT

ED TREATMENT/PROCEDURES

- BV:
 - _ Metronidazole vaginal gel daily × 5 days *or*
 - _ Metronidazole 500 mg PO BID × 7 days *or*
 - _ Clindamycin vaginal cream × 7 days *or*
 - _ Clindamycin ovules PV daily × 3 days
 - _ Rx before certain gynecologic procedures
 - _ Advise against alcohol intake if taking metronidazole for 24 hrs after treatment.
 - _ Routine treatment of male sex partner: NO
 - _ *Lactobacillus* not found to be more effective than placebo
- Candidiasis:
 - _ Single-dose oral fluconazole *or*
 - _ Intravaginal imidazole drug × 7 days
 - _ Routine treatment of male sex partner: NO
- Chemical irritant:
 - _ Avoid irritant
 - _ Use sitz baths, cotton underwear.
- Foreign body:
 - _ Removal of foreign body
 - _ Sedation may be required for removal
 - _ Give appropriate antibiotics if infection present
- Chlamydia cervicitis:
 - _ Azithromycin 1 g PO in single dose (for cervicitis, not adequate for PID) *or*
 - _ 7 days of doxycycline, ofloxacin, levofloxacin, or erythromycin
 - _ Treat for presumed concurrent gonococcal infections.
 - _ Routine treatment of male sex partners: YES
- Gonococcal cervicitis:
 - _ Ceftriaxone 250 mg IM × 1 AND azithromycin 1 g PO × 1 *or* doxycycline 100 mg BID × 7 days.
 - _ Oral cephalosporins (cefixime) no longer recommended.
 - _ Treat for presumed concurrent chlamydial infection.
 - _ Routine treatment of male sex partners: YES
- HSV:
 - _ Acyclovir, famciclovir, or valacyclovir for 7–10 days for initial attack; 5 days for recurrences

- Lidocaine jelly for topical relief
- Rule out other causes of genital ulcers. Offer RPR, HIV testing, and counseling.
- Routine treatment of male sex partners: Only if symptomatic; however, patient and partner may shed virus asymptotically.
- Lichen sclerosis:
 - Referral to gynecologist for estrogen cream and further treatment
- Trichomoniasis:
 - Metronidazole 2 g PO once *or*
 - Tinidazole 2 g PO once *or*
 - Metronidazole 500 mg PO BID for 7 days (avoid ethanol)
 - Routine treatment of male sex partners: YES
- All sexually transmitted causes:
 - Advise patient to avoid sexual contact with partner until partner is evaluated and treated when appropriate.
 - Educate regarding STDs/safer sex/HIV/hepatitis vaccines

Pregnancy Considerations

- BV:
 - Treat symptomatic women with oral metronidazole or clindamycin
 - Insufficient evidence for screening or treatment of asymptomatic pregnant women
- Candidiasis:
 - Only topical azole drug recommended in pregnancy; no oral fluconazole.
- Chlamydia cervicitis:
 - Azithromycin is the 1st-line choice for treating chlamydia in pregnant patients
 - Do not treat with doxycycline, ofloxacin, or levofloxacin.
- Trichomoniasis:
 - Metronidazole given early in pregnancy shown to increase preterm birth.
 - Give 2 g single-dose metronidazole, preferably after 37 wk gestation.

Pediatric Considerations

- Ask about new irritants: Bubble bath, soap, and laundry detergent.
- Consider sexual assault/abuse.

MEDICATION

- Acyclovir: 200 mg PO 5 times per day × 10 days or 400 PO TID × 10 days (for initial attack); 200 mg PO 5 times per day × 5 days or 400 PO TID × 5 days (for recurrent attack)
- Azithromycin: 1 g PO × 1
- Butoconazole 2% cream: 5 g PV × 3 days
- Butoconazole SR 2% cream: 5 g PV × 1

- Ceftriaxone: 125 mg IM or 250 mg IM × 1
- Ciprofloxacin: 500 mg PO × 1
- Clindamycin 2% cream: 1 applicator PV QHS × 7 days
- Clindamycin: 300 mg PO BID × 7 days
- Clotrimazole 1% cream: 5 g PV × 7–14 days
- Clotrimazole: 100 mg vaginal tablet × 7 days
- Doxycycline: 100 mg PO BID × 7 days (class D)
- Erythromycin ethyl succinate: 800 mg PO QID × 7 days
- Erythromycin base: 500 mg PO QID × 7 days
- Famciclovir: 250 mg PO TID × 7–10 days (for initial attack); 125 mg PO BID × 5 days (for recurrent infection)
- Fluconazole: 150 mg PO × 1
- Levofloxacin: 500 mg PO per day × 7 days
- Metronidazole: 500 mg PO BID for 7 days
- Metronidazole 0.75% gel: PV daily × 5 days
- Miconazole: 1,200 mg PV × 1
- Miconazole: 200 mg PV QHS × 3 days
- Miconazole 2% cream: 5 g PV QHS × 7 days or 100 mg supp. PV QHS × 7 days
- Nystatin 100,000 unit vaginal tablet: Nightly × 14 days
- Terconazole: 80 mg supp QHS × 3 days
- Terconazole 0.8% cream: 5 g PV × 3 days
- Terconazole 0.4% cream: 5 g PV × 7 days
- Tinidazole: 2 g PO daily × 1 day
- Tioconazole 6.5% cream: 5 g PV × 1
- Valacyclovir: 1 g PO BID × 7–10 days (for initial attack); 500 mg PO BID × 3–5 days or 1 g PO per day × 5 days (for recurrent attack)



FOLLOW-UP

DISPOSITION

Admission Criteria

- Disseminated gonococcal infection
- Sepsis secondary to foreign body
- PID toxicity
- Pain control, consequent inability to urinate or pass stool (HSV)

Discharge Criteria

Most can be discharged. Follow-up in ~1 wk is suggested.

Issues for Referral

- Vaginal discharge and vaginitis can be safely managed as an outpatient by the

patient's primary physician or gynecologist:

- Suggested follow-up in 1 wk

FOLLOW-UP RECOMMENDATIONS

- Recommend good hygiene
- Advise patient to return to the ED or see her doctor if:
 - Symptoms do not resolve in 3–5 days
 - Abdominal pain or cramping
 - Fever or chills
 - Pain during sexual intercourse
 - Lower back or flank pain
 - Difficulty urinating or urinary frequency

PEARLS AND PITFALLS

- pH of BV is often > 4.5
- Candidiasis often presents right before menses and can be precipitated by antibiotic use, DM, and immunosuppression.
- Trichomoniasis often presents after menses and has similar risk factors as other sexually transmitted diseases, including number of sexual partners and sexual practices.
- Partner treatment required for gonococcal and chlamydial infection, trichomoniasis.

ADDITIONAL READING

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CODES

ICD9

- 131.01 Trichomonal vulvovaginitis
- 616.10 Vaginitis and vulvovaginitis, unspecified
- 627.3 Postmenopausal atrophic vaginitis

ICD10

- A59.01 Trichomonal vulvovaginitis
- N76.0 Acute vaginitis
- N95.2 Postmenopausal atrophic vaginitis

VALVULAR HEART DISEASE

Liudvikas Jagminas



BASICS

DESCRIPTION

- **Mitral stenosis:**
 - Obstruction of diastolic blood flow into the left ventricle (LV)
- **Mitral regurgitation:**
 - Inadequate closure of the leaflets allows retrograde blood flow into the left atrium (LA).
 - Acute: Pressure overload in LA and pulmonary veins causing acute pulmonary edema
 - Chronic: LV volume overload with dilatation and hypertrophy with LA enlargement
- **Aortic stenosis:**
 - Obstruction of LV outflow with increased systolic gradient
 - Progressive increase in LV systolic pressure and concentric hypertrophy
- **Aortic regurgitation:**
 - Acute LV pressure and volume overload leading to left-heart failure and pulmonary edema
 - Chronic volume overload with LV dilation and hypertrophy

Pregnancy Considerations

Pregnancy is associated with significant hemodynamic changes that can aggravate valvular heart disease and increase the risk of thromboembolic events.

Geriatric Considerations

- Degenerative valvular disease is most common (aortic stenosis and mitral regurgitation)
- Aortic valve replacement is the most common surgical procedure

ETIOLOGY

- **Mitral stenosis:**
 - Rheumatic fever
 - Cardiac tumors
 - Rheumatologic disorders (lupus, rheumatoid arthritis)
 - Myxoma
 - Congenital defects: Parachute valve
- **Mitral regurgitation (acute):**
 - Ruptured papillary muscle (infarction, trauma)

- Papillary muscle dysfunction (ischemia)
- Ruptured chordae tendineae (trauma, endocarditis, myxomatous)
- Valve perforation (endocarditis)
- Weight-loss medications (fenfluramine, dexfenfluramine)
- Aortic stenosis:
 - Congenital aortic stenosis: Male > female (4:1)
 - Congenital bicuspid valve (1–2%)
 - Rheumatic aortic stenosis
 - Calcific aortic stenosis
- Aortic regurgitation:
 - Infective endocarditis
 - Rupture of sinus of Valsalva
 - Acute aortic dissection
 - Chest trauma
 - Following valve surgery
 - Bicuspid aortic valve
 - Rheumatic fever
 - Weight-loss medications (fenfluramine, dexfenfluramine)
 - Collagen vascular or connective-tissue diseases
 - Systemic lupus erythematosus
 - Marfan syndrome
 - Pseudoxanthoma elasticum
 - Ankylosing spondylitis
 - Ehlers–Danlos syndrome
 - Polymyalgia rheumatica

DIAGNOSIS

SIGNS AND SYMPTOMS

- Mitral stenosis:
 - Malar flush (“mitral facies”)
 - Prominent jugular A-waves
 - Right ventricular lift
 - Loud S1
 - Opening snap
 - Low-pitched diastolic rumble
 - Exertional dyspnea
 - Fatigue
 - Palpitations
 - Paroxysmal nocturnal dyspnea
 - Orthopnea
 - Hemoptysis

- Systemic emboli
- Pulmonary edema
- Mitral regurgitation:
 - Acute pulmonary edema
 - Jugular venous pressure (JVP) exhibits cannon A-waves and giant V-waves.
 - Harsh blowing apical crescendo–decrescendo murmur radiating to the axilla
 - Palpable thrill at apex
 - S3 and S4
 - Palpitations
 - Atrial fibrillation
 - Dyspnea
 - Orthopnea
 - Nocturnal paroxysmal dyspnea
 - Peripheral edema
 - Systemic emboli
 - Normal JVP
 - Left ventricular hypertrophy (LVH)
 - Apical high-pitched pansystolic murmur
 - Decreased or obscured S1
 - Widely split S2
 - S3
- Aortic stenosis:
 - Exertional angina
 - Syncope (during exercise)
 - CHF (initially diastolic failure, then systolic)
 - Sudden death secondary to ventricular fibrillation
 - Harsh crescendo–decrescendo (diamond-shaped) systolic murmur at aortic focus radiating to carotids
 - Absent aortic component of S2
 - Delayed upstroke in peripheral pulse (pulsus parvus et tardus)
 - S4 gallop
 - Ejection click
- Aortic regurgitation:
 - Fatigue
 - Dyspnea on exertion
 - Paroxysmal nocturnal dyspnea
 - Orthopnea
 - Syncope
 - Acute pulmonary edema
 - High-pitched blowing decrescendo diastolic murmur at aortic area
 - Accentuated A2 heart sound
 - Wide pulse pressure
 - Corrigan pulse (collapsing pulse)

- Duroziez sign (to-and-fro murmur)
- De Musset sign (head bobbing with systole)
- Quincke pulse (nail bed pulsations)
- Austin Flint murmur (soft diastolic rumble)

ESSENTIAL WORKUP

- History and symptoms
- Thorough cardiopulmonary exam
- ECG

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Blood cultures
- Presumed endocarditis
- CBC:
 - Anemia

Imaging

- CXR:
 - Mitral stenosis:
 - Enlarged LA
 - Pulmonary vascular congestion (Kerley B lines)
 - Prominent pulmonary arteries
 - Mitral regurgitation:
 - LV and LA enlargement in chronic cases
 - Pulmonary edema and normal LV and LA dimensions in acute cases
 - Aortic stenosis:
 - LVH
 - Aortic calcification
 - Dilation of ascending aorta
 - Pulmonary congestion and cardiomegaly
 - Aortic regurgitation:
 - Acute = normal cardiac silhouette and pulmonary edema
 - Chronic = enlarged LV and dilated aorta
- ECG:
 - Quality assessment of valvular structures
 - Measurements of flow through valves
 - Identification of regurgitation
 - Ventricular dilatation or hypertrophy
- Spiral CT scan:
 - To exclude aortic dissection with acute aortic regurgitation

Diagnostic Procedures/Surgery

EKG:

- Mitral stenosis:
 - LA enlargement (broad notched P-waves)
 - RV hypertrophy
 - Right axis deviation
 - Atrial fibrillation
 - Acute mitral regurgitation:
 - Left atrial enlargement
 - LVH
 - Left axis deviation
- Aortic stenosis:
 - LVH most common
 - Atrial fibrillation
 - Interventricular conduction delay
 - Complete AV block
- Aortic regurgitation:
 - Acute = LV strain
 - Chronic = LVH and strain

DIFFERENTIAL DIAGNOSIS

See Etiology.



TREATMENT

PRE HOSPITAL

Avoid vasodilators in aortic stenosis.

INITIAL STABILIZATION/THERAPY

- ABCs
- Administer oxygen.
- Monitor and measure pulse oximetry.
- IV access

ED TREATMENT/PROCEDURES

- Mitral stenosis:
 - Treat symptoms of CHF.
 - Rate control if in atrial fibrillation
 - Digoxin
 - β -blockers
 - Heparin (if new-onset atrial fibrillation)
 - Diuretics
 - Endocarditis prophylaxis/education

- Mitral regurgitation:
 - Differentiate between acute and chronic MR:
 - Acute:
 - Afterload reduction (nitroglycerin, morphine, or sodium nitroprusside)
 - Diuresis
 - Intra-aortic balloon pump (temporizing for urgent surgery)
 - Chronic:
 - Diuresis
 - Nitrates
 - Hydralazine
 - ACE inhibitor
 - Digoxin
 - β -adrenergic blocker (ventricular rate control)
 - Calcium antagonist (ventricular rate control)
 - Heparin (if atrial fibrillation)
 - Endocarditis prophylaxis/education
- Aortic stenosis:
 - Gentle diuresis if CHF
 - Mild hydration if hypotensive and not in CHF
 - Avoid nitrates and afterload reduction.
 - Digoxin
 - Intra-aortic balloon pump (temporize for surgery)
 - Endocarditis prophylaxis/education
- Aortic regurgitation:
 - Chronic:
 - Preload and afterload reduction
 - Digoxin
 - Diuretics
 - Endocarditis prophylaxis/education
 - Acute:
 - Preload and afterload reduction
 - Intra-aortic balloon pump
 - Urgent surgery

MEDICATION

- Atenolol: 0.3–2 mg/kg/d PO, max. 2 mg/kg/d (peds: 1–2 mg/kg/dose PO daily suggested)
- Digoxin: 0.5 mg bolus IV, then 0.25 mg IV q2h up to 1 mg; 0.125–0.375 mg/d PO
- Diltiazem: 0.25 mg/kg IV over 2 min (repeat in 15 min PRN with 0.35 mg/kg) then 5–15 mg/h
- Enalapril: 1.25 mg IV q6h; PO 2.5–10 mg BID (peds: 0.1–0.5 mg/kg/d PO div. q12–24h, max.: 0.58 mg/kg/d or 40 mg/d)

- Esmolol: IV: 500 µg bolus, then 50–400 µg/kg/min
- Furosemide: 20–80 mg/d PO/IV/IM; titrate up to 600 mg/d for severe edematous states (peds: 1 mg/kg IV/IM slowly under close supervision; not to exceed 6 mg/kg)
- Heparin: 80 U/kg IV bolus, then 18 U/kg/h drip, adjust to maintain partial thromboplastin time 1.5–2 × control (INR 2–3)
- Hydralazine: 10–25 mg IV q2–4h (peds: 0.1–0.5 mg/kg IM/IV q4–6h; max. 20 mg/dose)
- Metoprolol: 5 mg IV q2min × 3 doses; then 50 mg PO q6h × 48 hr
- Nitroglycerin: Start at 20 µg/min IV and titrate to effect (up to 300 µg/min); SL 0.3–0.6 mg PRN; USE NON-PVC tubing. Start at 5 µg/min, titrate up by 5 µg/min every 3–5 min until desired effect. Topical 1/2–2 in of 2% q6h (peds: 0.25–0.5 µg/kg/min IV, increase by 0.5–1 mg/kg/min; max. 20 µg/kg/min)
- Phentolamine: 5 mg bolus IV, then 1–2 mg/min IV infusion
- Propranolol IV: 1–3 mg at 1 mg/min
- Sodium nitroprusside IV: 0.5 µg/kg/min; increase in increments of 0.5 to 1 µg/kg/min q5–10min up to 10 µg/kg/min
- Amoxicillin: 2 g PO 1 h before the procedure; alternatively, 3 g PO 1 h before the procedure, followed by 1.5 g PO 6 h after the initial dose:
 - Pediatric dose: 50 mg/kg PO 1 h before procedure
- Ampicillin: 2 g IV/IM 30 min before the procedure (peds: 50 mg/kg IV/IM 30 min before the procedure)
- Clindamycin: 600 mg PO 1 h before procedure (peds: 20 mg/kg PO 1 h before procedure; not to exceed 600 mg)



FOLLOW-UP

DISPOSITION

Admission Criteria

- New-onset atrial fibrillation
- CHF/pulmonary edema
- Hemodynamically unstable
- Acute mitral or aortic regurgitation
- Cardiac ischemia
- Angina
- Syncope
- Arrhythmias

Discharge Criteria

- Hemodynamic stability
- Unchanged ECG

- Resolution of CHF symptoms with diuresis
- Chronic mitral regurgitation

Issues for Referral

- For patients who are candidates for outpatient management, close follow-up with a cardiologist to assess severity of valvular disease and need for cardiac surgery
- Educate patient about risks of valvular heart disease and need for antibiotic prophylaxis with dental and medical procedures.

PEARLS AND PITFALLS

In patients with chest pain and aortic stenosis, nitrates are contraindicated.

ADDITIONAL READING

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- Rahimtoola SH. The year in valvular heart disease. *J Am Coll Cardiol.* 2013;61(12):1290–1301.
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CODES

ICD9

- 394.0 Mitral stenosis
- 424.0 Mitral valve disorders
- 424.90 Endocarditis, valve unspecified, unspecified cause

ICD10

- I05.0 Rheumatic mitral stenosis
- I34.0 Nonrheumatic mitral (valve) insufficiency
- I38 Endocarditis, valve unspecified

VARICELLA

Michael J. Bono

BASICS

DESCRIPTION

- Commonly known as chickenpox
- Most common in late winter and early spring
- Vaccine has reduced incidence by 85%
- Adults have a 15 times greater risk for death from varicella than children

ETIOLOGY

- DNA virus:
 - _ Latency in cranial nerve ganglia, dorsal root ganglia, and autonomic ganglia with periodic reactivation
 - _ Presents as herpes zoster or shingles decades after primary infection
 - _ Virus is transmitted by respiratory route and direct contact with skin lesions
 - _ Humans are only known reservoir

DIAGNOSIS

SIGNS AND SYMPTOMS

- Varicella causes a spectrum of disease
- Classic childhood illness:
 - _ Usually affects children ages 1–9
 - _ Low-grade fever (100–103°F), headache, malaise, usually precedes rash by 1–2 days
 - _ Pruritus, anorexia, and listlessness
 - _ 10–21 day incubation period
 - _ Infectious from 48 hr before vesicle formation until all vesicles are crusted, typically 3–7 days after onset of rash
 - _ Classic exanthem:
 - Lesions begin on the face, spreading to the trunk and extremities
 - Papules, vesicles, or pustules, on erythematous base
 - Lesions in varying stages of evolution, which is hallmark of Varicella
 - “Dewdrop on rose petal”
 - Vesicles 2–3 mm in diameter
 - Duration of vesicle formation 3–5 days
 - May involve conjunctival, oropharyngeal, or vaginal mucosa
 - Skin superinfection with group A streptococcus or staphylococcus in 1–4% of healthy children

- Adolescents and adults:
 - Similar presentation to children but greater risk of severe disease:
 - Extracutaneous manifestations in 5–50%, particularly pneumonia
- Immunocompromised patients:
 - HIV, transplant patients, leukemia patients at highest risk for disseminated form
 - Patients on chemotherapy, immunosuppressants, and long-term corticosteroid therapy at high risk
 - More numerous lesions that may have hemorrhagic base
 - Healing may take longer
 - Pneumonia common in these patients
- Pregnant patients:
 - Prevalent in young expectant women
 - More severe disease presentation:
 - Risk to fetus greatest in 1st half of pregnancy
 - Risk to mother greatest if infection in 2nd half of pregnancy
 - Perinatal disease can occur from 5 days predelivery to 48 hr postdelivery
- Congenital varicella syndrome
- Occasionally follows maternal zoster infection
- Limb hypoplasia or paresis
- Microcephaly
- Ophthalmic lesions
- Extracutaneous manifestations:
 - Pneumonitis:
 - 25 times more common in adults
 - Highest risk in adult smokers and immunocompromised children
 - Occurs 3–5 days after onset of rash
 - Signs: Continued eruption of new lesions, and new-onset cough
 - Tachypnea, dyspnea, cyanosis, pleuritic chest pain, and hemoptysis
 - Cerebellar ataxia:
 - May develop 5 days after rash
 - Ataxia, vomiting, slurred speech, fever, vertigo, tremor
 - Cerebritis:
 - Develops 3–8 days after start of rash
 - Duration about 2 wk
 - Progressive malaise
 - Headache, meningismus, vomiting, fever, delirium, seizures
 - Reye syndrome risk

Geriatric Considerations

- Increased risk of extracutaneous manifestations
- Lower immunity allows for reactivation as herpes zoster

Pediatric Considerations

- No aspirin for treatment of fever, possible association with Reye syndrome:
 - Acetaminophen—is recommended antipyretic treatment
- Parents need to be cautioned regarding risk for secondary bacterial infection and possible progression to sepsis

Pregnancy Considerations

- Pregnant women with no childhood history of varicella and no antibodies to varicella zoster virus (VZV) require varicella zoster immunoglobulin (VZIG)
- Varicella pneumonia in pregnancy is medical emergency, associated with life-threatening respiratory compromise and death (mortality can be 10–45%)
- Likely to occur in 3rd trimester

History

- Thorough history:
 - _ Fever, systemic symptoms
 - _ Immunization history
 - _ Immunocompetent vs. immunocompromised

Physical-Exam

- Thorough physical exam:
 - _ Characterize rash spread and extent
 - _ Evaluate for any extracutaneous manifestations

ESSENTIAL WORKUP

- History and physical exam are sufficient in uncomplicated cases
- Pneumonitis:
 - _ CXR shows 2–5 mm peripheral densities, may coalesce and persist for weeks
- Reye syndrome:
 - _ Ammonia level peaks early
 - _ LFTs will be elevated
 - _ PT, PTT
- Cerebritis:
 - _ Lumbar puncture demonstrates lymphocytic pleocytosis and elevated levels of protein

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Viral culture (results in 3–5 days), polymerase chain reaction (PCR), or direct fluorescent antibody using skin scrapings from crust or base of lesion
- Serologic tests for varicella antibodies
- PCR is diagnostic method of choice, but uncomplicated patients need no labs

Imaging

Not generally indicated unless there is concern for extracutaneous manifestations

Diagnostic Procedures/Surgery

Liver biopsy definitive test for Reye syndrome

DIFFERENTIAL DIAGNOSIS

- Impetigo
- Disseminated herpes
- Disseminated coxsackievirus
- Measles
- Rickettsial disease
- Insect bites
- Scabies
- Erythema multiforme
- Drug eruption (especially Stevens–Johnson syndrome)



TREATMENT

PRE HOSPITAL

- Nonimmune transport personnel must avoid respiratory or physical contact with patients
- Transport personnel who have varicella or herpes zoster should not come in contact with immunocompromised or pregnant patients

INITIAL STABILIZATION/THERAPY

- Airway management and resuscitate as indicated:
 - Protect airway if obtunded

ED TREATMENT/PROCEDURES

- Generally, acetaminophen and antipruritics are the keys to treating classic childhood illness
- Closely cropped nails and good hygiene help prevent secondary bacterial infection
- Infants/children ≤ 12 yr of age:
 - Acyclovir:
 - Recommended in children taking corticosteroids, long-term salicylate therapy, or chronic cutaneous or pulmonary diseases
 - Modest benefit, reduces lesions by 25% and fever by 1 day
 - Should be given within 24 hr of symptom onset
 - NOT recommended in uncomplicated Varicella in healthy children
 - Prophylaxis with VZIG in susceptible patients:
 - Immunocompromised children at high risk for complication with

- significant exposure
 - Susceptible children in the same household as person with active chickenpox or herpes zoster
 - In 2012 FDA extended period for VZIG administration to 10 days after exposure
 - VZIG in short supply, difficult to obtain
- Adolescents/adults:
 - Acyclovir now recommended in adults with uncomplicated varicella initiated within 24 hr to decrease progression to disseminated disease
 - Symptomatic treatment with antipyretics and antipruritics
- Pregnant women:
 - If exposed to Varicella, no childhood history of varicella, no antibodies to VZV, need VZIG
 - 80–90% immune from prior infection, need antibody testing prior to administration of VZIG
 - Acyclovir or Valacyclovir prophylaxis especially during 2nd or 3rd trimesters:
 - Safe during pregnancy (category B)
 - IV acyclovir for pneumonitis/other complications:
 - Respiratory, neurologic, hemorrhagic rash, or continued fever > 6 days
- Immunocompromised patients:
 - IV Acyclovir recommended, poor PO bioavailability
 - PO valacyclovir better bioavailability, approved in 2008 for lower risk immunocompromised patients
 - Should be started within 24 hr of onset to maximize efficacy
 - Foscarnet for acyclovir-resistant disease
 - Prophylaxis with VZIG for the susceptible immunocompromised patient
- Extracutaneous:
 - IV acyclovir or foscarnet if resistant
- Vaccine:
 - Children:
 - Routine vaccination for all susceptible children at 12 mo and older, 2 doses
 - Adolescents and adults:
 - Age 13 and older without history of varicella need vaccine
 - 2 doses separated by 4–8 wk
 - Recommended in high-risk groups: Health care workers, family member of immunocompromised person, susceptible women of childbearing age, teachers, military, international travelers
 - Post exposure prophylaxis:
 - Susceptible patients 12 mo or older, given with 72–120 hr, with 2nd dose at age appropriate interval

- Will produce immunity if not infected
- Immunocompromised persons:
 - Most immunocompromised persons should not be immunized

MEDICATION

- Acyclovir:
 - Uncomplicated: *Adults*: 800 mg PO QID for 5 days; *Adolescents* (13–18 yr old): 20 mg/kg per dose QID for 7 days; *Peds*: 20 mg/kg suspension PO QID for 5 days [max. 800 mg PO QID]
 - Immunocompromised: *Adults*: 10 mg/kg IV q8h infused over 1 hr, or 800 mg PO 5 times a day for 7 days. *Peds*: 10–12 mg/kg IV q8h infused over 1 hr, or 500 mg/m²/day IV q8h for 7–10 days
- Valacyclovir: 1 g PO TID for 5–7 days
- Famciclovir: 500 mg PO TID for 7 days
- Foscarnet: *Adults*: 90 mg/kg q12h IV over 90–120 min for 2–3 wk; *Peds*: 40–60 mg/kg q8h over 120 min for 7–10 days; Foscarnet is not FDA approved
- Hydroxyzine: *Adults*: 25–50 mg IM or PO q4–6h. *Peds*: 0.5 mg/kg q4–6h suspension (supplied as 10 and 25 mg/5 mL)
- Diphenhydramine: *Adults*: 25–50 mg IV, IM, or PO q4h. *Peds*: 5 mg/kg/d elixir
- VZIG: *Adults*: 625 IU IM. *Peds*: 1 vial per 10 kg IM to a max. of 5 vials [each vial contains 125 IU]



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients with pneumonia require admission:
 - ICU for respiratory observation or support
- Immunocompromised patients: ICU vs. ward, depending on severity of illness
- All admitted patients must be kept in isolation

Discharge Criteria

- Immunocompetent children without evidence of Reye syndrome or secondary bacterial infection
- Adults with no evidence of extracutaneous disease

FOLLOW-UP RECOMMENDATIONS

Patients who are discharged need close follow-up with PCP to assure resolution without complications

PEARLS AND PITFALLS

- Patients with varicella are infectious from 48 hr before vesicle formation until all vesicles are crusted
- Immunocompromised patients with Varicella need careful consideration and admission in most cases
- Varicella pneumonia is medical emergency, particularly in pregnancy

ADDITIONAL READING

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- van Lier A, van der Maas N, Rodenburg GD, et al. Hospitalization due to varicella in the Netherlands. *BMC Infect Dis*. 2011;11:85.



See Also (Topic, Algorithm, Electronic Media Element)

Herpes Zoster



CODES

ICD9

- 052.9 Varicella without mention of complication
- 053.9 Herpes zoster without mention of complication
- 053.21 Herpes zoster keratoconjunctivitis

ICD10

- B01.9 Varicella without complication
- B02.9 Zoster without complications
- B02.31 Zoster conjunctivitis

VARICES

Galeta C. Clayton

BASICS

DESCRIPTION

- Increased portal venous pressure results in portal–systemic shunts.
- Shunts at gastroesophageal junction result in fragile submucosal esophageal varices.

ETIOLOGY

- 10–30% of all cases of upper GI bleeding
- 90% of upper GI bleeding in patients with cirrhosis
- Variceal hemorrhage occurs in 30% of patients with cirrhosis:
 - 50% will stop bleeding spontaneously
 - 30% mortality per episode
 - 70% have recurrent bleeding
- In adults:
 - Cirrhosis due to alcoholism or chronic hepatitis
 - Storage disease: Wilson or hemochromatosis
 - Middle East: Schistosomiasis
- In children:
 - Intrahepatic obstruction from biliary cirrhosis
 - Biliary atresia
 - Cystic fibrosis
 - β -antitrypsin deficiency
 - Hepatitis

DIAGNOSIS

SIGNS AND SYMPTOMS

- General:
 - Weakness and fatigue
 - Tachycardia
 - Tachypnea
 - Hypotension
 - Cool, clammy skin; prolonged capillary refill
- Abdominal:
 - Significant active upper GI bleeding:
 - Hematemesis
 - Hematochezia

- Melena
 - 20–40% of total blood volume loss possible
- Abdominal pain
- Stigmata of severe hepatic dysfunction:
 - Jaundice
 - Spider angiomata
 - Palmar erythema
 - Pedal edema
 - Hepatosplenomegaly
 - Ascites
- History of portal hypertension:
 - Most commonly alcoholic cirrhosis
 - Others, including:
 - Primary biliary cirrhosis
 - Schistosomiasis
 - Budd–Chiari syndrome
 - Severe CHF
 - Sarcoidosis
- Cardiovascular:
 - Chest pain/shortness of breath
- CNS:
 - Syncope
 - Confusion and agitation initially
 - Lethargy and obtundation later

Pediatric Considerations

- Massive hematemesis: Typical initial presentation:
 - Hypotension may be a late finding.

History

- Gastroesophageal varices are present in 50% of patients with cirrhosis and correlate with severity of disease.
- The most important predictor of hemorrhage is size of the varices. Other factors include number of varices, severity of hepatic disease and endoscopic findings.
- Patients with PBC develop varices and variceal hemorrhage early in their course of disease, even prior to development of cirrhosis.

Physical-Exam

- Vitals signs may be normal or may show tachycardia (early) and hypotension (late).
- Altered mental status with encephalopathy or poor perfusion
- Active hematemesis

- Stigmata of alcoholic liver disease:
 - Ascites
 - General edema
 - Jaundice

ESSENTIAL WORKUP

- Gastric tube placement:
 - Determines whether patient is actively bleeding
 - Decompresses stomach that may aid in hemostasis. Possible role in reducing aspiration risk
 - Facilitates endoscopic exam
 - Will not increase or cause esophageal variceal bleeding
- Emergent endoscopy

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Type and cross-match 6–8 U:
 - Significant transfusion requirements
- ABG for:
 - Acidosis
 - Hypoxemia
- CBC:
 - Hematocrit is an unreliable indicator of early rapid blood loss.
 - Perform serial CBCs to follow blood loss.
- Electrolytes, BUN, creatinine, glucose:
 - Evaluate renal function.
 - BUN:creatinine ratio > 30 suggest significant blood in GI tract.
- PT/PTT/INR and platelets:
 - Coagulopathy
 - Prolonged bleeding times
 - Thrombocytopenia

Imaging

- Chest radiograph (portable) for aspiration/perforation
- ECG for myocardial ischemia

DIFFERENTIAL DIAGNOSIS

- Bleeding/perforated peptic ulcer
- Erosive gastritis
- Mallory–Weiss syndrome
- Boerhaave syndrome
- Aortoenteric fistula

- Gastric varices
- Gastric vascular ectasia



TREATMENT

PRE HOSPITAL

- Airway stabilization
- Treat hypotension 0.9% normal saline infusion bolus through 2 large-bore 16G or large IV lines.
- Cardiac and pulse oximetry monitoring

INITIAL STABILIZATION/THERAPY

- ABCs with early aggressive airway control/intubation:
 - _ Early intubation = easier intubation
 - _ For AMS or massive hemoptysis
 - _ Facilitates emergency endoscopy
- Establish central IV access with invasive intravascular monitoring for hypotension not responsive to initial fluid bolus.
- Replace lost blood as soon as possible:
 - _ Initiate with O-negative blood until type-specific blood available.
 - _ 10 mL/kg bolus in children
 - _ Fresh-frozen plasma and platelets may be required.
- Place gastric tube nasally (awake) or orally (intubated)
- Controversy:
 - _ Overly aggressive volume expansion may lead to rebound portal HTN, rebleeding, and pulmonary edema.
 - _ Transfusion goal is Hb = 8.
 - _ rFVIIa may decrease hemostasis failure rates in Child–Pugh class B/C patients

Pediatric Considerations

- Initiate intraosseous access if peripheral access unsuccessful in unstable patient.
- Most bleeding in children stops spontaneously.
- Vital sign changes may be a late finding in children:
 - _ Subtle changes in mental status, capillary refill, mild tachycardia, or orthostatic changes may indicate significant blood loss.
 - _ Overaggressive correction in infants can quickly lead to significant electrolyte abnormalities.

ED TREATMENT/PROCEDURES

- Emergent endoscopy required for active bleeding:
 - _ Use pharmacologic and tamponade devices as temporizing measures.

- **Endoscopy**
 - Emergent with active bleeding in nasogastric tube
 - Procedure of choice in acute esophageal bleeding
 - Esophageal band ligation equivalent to sclerotherapy with fewer complications:
 - May be difficult to visualize in cases of massive bleeding
 - Sclerotherapy with massive bleeding
 - Gastric varices are not amenable to endoscopic repair due to high rebleeding rate:
 - Treat pharmacologically.
 - Administer antibiotics at time of procedure to decrease risk for spontaneous bacterial peritonitis:
 - Fluoroquinolone or ceftriaxone
- **Pharmacological Therapy**
 - Somatostatin is 1st-line therapy where available (not widely available in US) due to greater efficacy and fewer side effects when compared to octreotide
 - Octreotide is 1st-line therapy where somatostatin not available:
 - Complications include hyperglycemia and abdominal cramping.
 - Vasopressin replaced by octreotide/somatostatin secondary to high incidence of vascular ischemia
- **Balloon Tamponade**
 - Initiate in massive uncontrollable bleed.
 - Sengstaken–Blakemore and Minnesota tubes
 - Applies direct pressure but risks esophageal perforation and ulceration
 - Temporary benefit only with massive uncontrolled bleeding in the hands of experienced clinician
- **Refractory Bleeding Therapy**
 - Interventional radiology:
 - Transjugular intrahepatic portosystemic shunt procedure.
Recommended for refractory gastric varices or for patients who are poor surgical candidates
 - Surgical options:
 - Portacaval shunt
 - Variceal transection
 - Stomach devascularization
 - Liver transplantation

MEDICATION

- Ceftriaxone: 2 g (peds: 50–75 mg/kg/24 h) IV q24h in Child–Pugh class B/C or in quinolone-resistant areas
- Cefotaxime: 2 g (peds: 50–180 mg/kg/24 h) IV q8h
- Erythromycin 250 mg IV:

- Shown to aid in gastric clearing for better visualization during endoscopy
- Norfloxacin 400 mg PO q12 or Ciprofloxacin 500 mg IV q12 if cannot tolerate PO (contraindicated in peds)
- Octreotide: 50 µg bolus, then 50 µg/h infusion for 5 days
- Somatostatin: 250 µg IV bolus followed by 250 µg/h IV infusion for 5 days

First Line

- Somatostatin or octreotide (if somatostatin not available)
- Norfloxacin PO or ciprofloxacin IV

Second Line

- Erythromycin
- Ceftriaxone



FOLLOW-UP

DISPOSITION

Admission Criteria

- ICU admission for actively bleeding varices
- Recent history of variceal bleeding
- High risk for early rebleeding:
 - Age >60 yr, renal failure, initial hemoglobin count <8

Discharge Criteria

Nonbleeding varices

Issues for Referral

- Continued hemorrhage requiring surgery or higher level of care
- Liver transplant

FOLLOW-UP RECOMMENDATIONS

- Timely outpatient GI follow-up:
 - Will need annual surveillance endoscopies
- Medication and lifestyle modifications

PEARLS AND PITFALLS

- Intubate early, especially in patients with hepatic encephalopathy or hemodynamic instability.
- Begin prophylactic antibiotics prior to endoscopy. Improves survival
- In US, octreotide has replaced vasopressin owing to better side-effect profile. If

vasopressin is required, use IV nitroglycerin infusion concomitantly to reduce end-organ ischemia.

- Control the airway prior to placement of balloon tamponade device, which provides only a temporizing measure prior to surgery or TIPS
- Hematochezia in a hemodynamically unstable patient is an upper GI bleed until proven otherwise.
- Consult your GI specialists early, since endoscopy is the 1st-line diagnostic and therapeutic procedure.

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See Also (Topic, Algorithm, Electronic Media Element)

- Cirrhosis
- Gastrointestinal Bleeding



CODES

ICD9

- 456.0 Esophageal varices with bleeding
- 456.1 Esophageal varices without mention of bleeding
- 456.21 Esophageal varices in diseases classified elsewhere, without mention of bleeding

ICD10

- I85.00 Esophageal varices without bleeding
- I85.01 Esophageal varices with bleeding
- I85.10 Secondary esophageal varices without bleeding

VASCULITIS

Richard S. Klasco

BASICS

DESCRIPTION

- Injury to the walls of blood vessels from inflammation:
 - Ischemia and necrosis
 - Aneurysms and hemorrhage
 - Immunopathologic mechanisms:
 - Deposition of circulating antigen–antibody complex and complement fixation
 - Cell-mediated hypersensitivity
 - Granulomatous tissue reaction from persistent inflammation and formation of epithelioid giant cells
- The vasculitides represent a wide group of disorders:
 - Multisystem disease with constitutional symptoms and inflammatory lab indices
 - Secondary to another disorder or trigger, or primary if vasculitis is the principal feature and the cause is unknown
 - Multiple factors determine presentation:
 - The size of the affected blood vessels
 - The specific distribution, severity, and duration of the inflammation
 - Degree of permeability or occlusion of the affected vessels
- 1 out of 2,000 adults has some form of vasculitis

ETIOLOGY

- Classification is evolving and is increasingly based on presence or absence of antineutrophil cytoplasmic antibodies (ANCA).
- Traditional classification is based on vessel size.
- Large vessel vasculitides:
 - Temporal (giant cell) arteritis:
 - Granulomatous arteritis of the aorta and its major branches often involving the temporal artery
 - Patients > 50 yr
 - Takayasu arteritis:
 - Granulomatous inflammation of the aorta and its major branches
 - Usually occurs in patients < 50 yr
- Medium vessel vasculitides:
 - Polyarteritis nodosa (PAN):
 - Small- and medium-sized arteritis

- Common distribution includes vessels supplying the muscles, joints, intestines, nerves, kidneys, and skin
- Most common in middle age
- Kawasaki disease (mucocutaneous lymph node syndrome):
 - Coronary arteries are often involved and involves large-, medium-, and small-sized arteries
 - Usually occurs in children
- Isolated CNS vasculitis
- Small vessel vasculitides:
 - Granulomatosis with polyangiitis (Wegener granulomatosis):
 - Necrotizing vasculitis affecting small- to medium-sized vessels
 - Granulomatous inflammation of the upper and lower respiratory tract and glomerulonephritis
 - Microscopic polyangiitis:
 - Necrotizing affecting small vessels
 - Glomerulonephritis is very common.
 - Pulmonary capillaritis often occurs.
 - Churg–Strauss syndrome (allergic granulomatosis):
 - Small- and medium-sized arteries
 - Mainly lungs, GI, and nerves
 - Can also involve heart, skin, and kidney
 - Henoch–Schönlein purpura:
 - Most patients <20 yr
 - Buerger disease (thromboangiitis obliterans):
 - Hypersensitivity vasculitis:
 - Recurring inflammation and thrombosis of small and medium arteries and veins of the hands and feet
 - Typically between 20 and 40 yr and male
 - Secondary vasculitides:
 - Bacterial infections:
 - Streptococcal, tuberculous, staphylococcal, Lyme disease, leprosy
 - Viral infections:
 - Hepatitis B or C, CMV, HSV, HIV
 - Rickettsial infections
 - Drug related
 - Connective tissue disease:
 - Systemic lupus erythematosus
 - Rheumatoid arthritis
 - Behçet disease
 - Malignancy:
 - Hairy cell leukemia
 - Lymphoma

- Mixed cryoglobulinemia
- Goodpasture syndrome
- Serum sickness

DIAGNOSIS

SIGNS AND SYMPTOMS

- Systemic complaints are common early in the presentation of vasculitis, before vascular-related complications occur:
 - Fever, fatigue, weight loss, diffuse aches and pains
- Signs of arterial insufficiency:
 - Ischemic pain:
 - Angina, abdominal angina, claudication, jaw claudication
 - Neurologic ischemia:
 - Headache, TIA, stroke, visual and sensorineural hearing loss, hallucinations, neuropathy, vision loss
 - Renal ischemia:
 - Severe or resistant HTN
 - Dermatologic ischemia:
 - Classic skin findings include palpable purpura.
 - Nodular lesions, ulcers, livedo reticularis, and digital ischemia may also be seen
 - Oligoarthritis
 - Ocular ischemia:
 - Diplopia, retinal hemorrhages, scleritis, and episcleritis
 - Respiratory tract:
 - Sinusitis, epistaxis, nasal and oral ulcerations, strawberry tongue
 - GI ischemia:
 - Hematochezia, melena, hematemesis, peritonitis, hepatitis
 - Cardiac:
 - Coronary artery aneurysms, myocarditis, pericarditis, valvular disease, CHF

History

- Suspect vasculitis with general systems and signs of arterial insufficiency:
 - Claudication, angina, abdominal angina, or TIA, in a young patient
 - Prolonged systemic illness with multiorgan dysfunction
 - History of glomerulonephritis, peripheral neuropathy, or autoimmune disease
- Diagnostic clues to the etiology:
 - Age, gender, ethnicity, travel history
 - Specific complaints that suggest the size of the involved vessel and organs

- Recent infections
- Connective tissue disorders
- Medications that may cause vasculitis:
 - Levsamisole (as a cocaine adulterant), phenytoin, carbamazepine, isoniazid, methimazole, minocycline, penicillamine, propylthiouracil, sulfasalazine

Physical-Exam

Classify vasculitis:

- Large arteries:
 - Diminished pulses and bruits over several large arteries
 - BP discrepancy > 10 mm Hg between left and right limbs
 - Pulse discrepancy > 30 mm Hg between the left and right limbs
 - Cool extremities due to claudication and ulceration
- Medium and small arteries:
 - Palpable purpura (nodules, ulcers, livedo papules)
 - Skin ulcers
 - Digital ischemia

ESSENTIAL WORKUP

- History and physical exam
- CBC, ESR, CRP, urinalysis, BUN, creatinine

DIAGNOSIS TESTS & NTERPRETATION

Lab

- CBC:
 - Leukocytosis
 - Eosinophilia
 - Anemia
- Creatinine
- LFT
- CRP
- ESR
- ANA
- ANCA
- Complement
- CPK
- Urinalysis:
 - Proteinuria and hematuria

Imaging

- CXR:

- PAN usually has a nonspecific patchy alveolar infiltration.
- CT scan:
 - Sinus CT for suspected granulomatosis with polyangiitis (Wegener)
- CTA:
 - Coronary artery aneurysms in Kawasaki
- Echocardiography:
 - Coronary artery aneurysms in Kawasaki
- MRI and MRA:
 - Positron emission tomography (PET) scan for suspected Takayasu and Kawasaki
- ECG:
 - Indications:
 - Suspected Takayasu and Kawasaki
- US:
 - Temporal artery US for suspected giant cell arteritis
 - Use pretest probability in interpretation of results
- Arteriography

Diagnostic Procedures/Surgery

- EKG:
 - Pericarditis, conduction disturbances
- Endoscopy, sigmoidoscopy, and colonoscopy for GI tract involvement
- Tissue biopsy

DIFFERENTIAL DIAGNOSIS

- Endocarditis
- Adverse drug reaction
- Viral infections (e.g., enterovirus)
- Scarlet fever
- Staphylococcal scalded skin syndrome
- Toxic shock syndrome
- Stevens–Johnson syndrome
- Rocky Mountain spotted fever
- Leptospirosis
- Antiphospholipid antibody syndrome
- Disseminated intravascular coagulation
- Cholesterol emboli
- Calciphylaxis



TREATMENT

INITIAL STABILIZATION/THERAPY

Stabilization of cerebrovascular complications

ED TREATMENT/PROCEDURES

- Treatment for vasculitis is determined by the underlying cause or the specific disease and is best initiated by rheumatology.
- Kawasaki: Aspirin, IVIG
- Giant cell arteritis: Corticosteroids
- PAN: Steroids, cyclophosphamide
- Takayasu arteritis: Corticosteroids, methotrexate, azathioprine, cyclophosphamide
- Wegner granulomatosis: Corticosteroids:
 - Cyclophosphamide, azathioprine may be substituted
 - Plasma exchange may be helpful in severe disease.

MEDICATION

- Azathioprine: 2 mg/kg/d PO
- Cyclophosphamide:
 - IV: 0.5–1 mg/m² body surface area
 - PO: 2 mg/kg/d (up to 4 mg/kg) (peds: dose as per consultant)
- IVIG: 1–2 g/kg IV
- Methylprednisolone: 0.25–1 mg/d IV
- Methotrexate: 7.5–15 mg/wk PO
- Prednisolone: 1 mg/kg/d PO
- Prednisone: 40–60 mg/d (peds: 1–2 mg/kg/d) PO



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients with evidence of severe disease and end-organ dysfunction should be admitted.
- Consult for procedures to revascularize ischemic organs.

Discharge Criteria

Less-symptomatic patients without evidence of end-organ involvement

Issues for Referral

- Any patient suspected of vasculitis and being managed as an outpatient should be referred as soon as possible to a rheumatologist for the definitive diagnosis and treatment.
- Consult appropriate specialties based on the severity of the end-organ damage.

FOLLOW-UP RECOMMENDATIONS

Stress the need for close follow-up with general symptoms to confirm the diagnosis and initiate therapy that will be life-saving on a long-term basis.

PEARLS AND PITFALLS

- Drug therapy may be toxic; do not prescribe without specialist consultation.
- Patients may be immunosuppressed and at risk for opportunistic pathogens.
- Do not miss subacute bacterial endocarditis as a mimic of vasculitis.
- Temporal (giant cell) arteritis does not occur before age 50 yr.
- Nodular lesions are the skin changes most likely to yield a diagnosis of vasculitis.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Erythema Nodosum
- Henoch–Schönlein Purpura
- Hepatitis
- Reiter Syndrome
- Systemic Lupus Erythematosus



CODES

ICD9

- 446.0 Polyarteritis nodosa
- 446.5 Giant cell arteritis

- 447.6 Arteritis, unspecified

ICD10

- I77.6 Arteritis, unspecified
- M30.0 Polyarteritis nodosa
- M31.6 Other giant cell arteritis

VENOUS INSUFFICIENCY

Cameron R. Wangsgard • Bo E. Madsen

BASICS

DESCRIPTION

- Inadequacy of the venous valves that causes impaired venous drainage leading to edema of the lower extremities.
- A chronic condition of lower extremity vascular incompetence.
- Normal blood flow in the venous system is unidirectional from the superficial veins to the deep veins.
- Unidirectional flow is maintained by contraction of leg muscles and by valves within the veins.
- Damage to the valves, e.g. following DVT, causes them to become rigid and they lose their ability to prevent retrograde blood flow properly.
- Decreased venous return from lower extremities causes increased pressure and distention of the veins, which in turn causes separation of the valve leaflets.
- Increased pressure transmitted into the dermal microcirculation results in extravasation of macromolecules and red blood cells causing inflammatory injury resulting in ulcer formation, skin changes, and poor ulcer healing.

ETIOLOGY

- Primary valve incompetence (most common)
- Deep vein thrombosis (DVT)
- Risk factors include advanced age, family history, smoking, sedentary lifestyle, obesity, lower extremity trauma, prior DVT, and pregnancy

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Asymptomatic phase:
 - Venous dilation ranging from venous flares to small varicosities
- Symptomatic phase:
 - Ankle and calf swelling
 - Varicose veins
 - Skin discoloration/hyperpigmentation
 - Ulcer formation
 - Lipodermatosclerosis
 - Dull ache/pain in the legs:

- Worsened by prolonged standing
- Resolves with leg elevation
- Burning sensation
- Pruritus
- Night cramps

Physical-Exam

- Varicose veins
- Ankle- and calf-dependent edema
- Ulcers, most often situated over the malleoli or medial portion of calf:
 - Must have preserved peripheral pulses to ensure ulcers are due to venous insufficiency and not arterial insufficiency
- Red, purple discoloration of skin
- Telangiectasias
- Reticular veins
- Stasis dermatitis
- Brownish hyperpigmentation
- Sclerosis, induration, and atrophy of skin
- Bacterial infection:
 - Surrounding cellulitis
 - Rapidly growing ulcer
 - Purulent drainage from ulcer
 - Increased pain
 - Fever
 - Lymphangitis
- Other etiologies than venous insufficiency, proximal to the lower extremity, should be suspected in the following settings:
 - History of heart failure
 - History of liver disease
 - Leg edema and ulcers in a patient with ascites
 - Periorbital edema
 - Orthopnea
 - Positive hepatojugular reflex
 - Jugular venous distention

ESSENTIAL WORKUP

The physical exam is essential to the diagnosis.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Lab tests add little to the physical exam unless other causes need to be excluded.
- Cardiac markers, brain natriuretic peptide, albumin, and tests of renal function

can be sent if considering other causes of leg edema.

Imaging

- Doppler auscultation (DopA):
 - Used to assess for arterial flow to the lower extremities in differentiating venous insufficiency from arterial insufficiency
 - Used to estimate blood flow, as well as the presence or absence of reflux in a given vein.
 - Indications are diameter > 3 mm, signs/symptoms of chronic venous insufficiency, the presence of a painful vessel, or concern for arterial insufficiency (no distal pulses, history of peripheral vascular disease, decreased ankle–brachial index)
- Duplex US (DUS):
 - Combines Doppler and gray-scale imaging and shows vascular anatomy, soft-tissue features, detection and quantification of reflux and the source of it
 - Can assess for DVT, valvular incompetence, and retrograde flow.
 - Can be used for diagnosis, as part of the treatment (duplex-guided sclerotherapy and endovenous ablation) and for postoperative evaluation.
- Photoplethysmography (PPG):
 - Assesses venous hemodynamics and venous refilling time with and without leg muscle contraction.
 - Used to measure vein outflow and inflow, as well as muscle pump adequacy.
- Venography:
 - Expensive and invasive
 - Despite still often being considered the gold standard, duplex ultrasonography has been found to be more sensitive and specific in predicting the clinical severity of venous insufficiency.

Diagnostic Procedures/Surgery

Ankle–brachial index:

- Should be measured if arterial insufficiency is suspected

DIFFERENTIAL DIAGNOSIS

- Venous valvular incompetence
- Deep venous thrombosis (DVT)
- Arterial insufficiency
- Lymphatic disorders or obstruction
- Soft-tissue infections (diabetic foot ulcers)
- Trauma (compartment syndrome, vascular or lymphatic disruption, inflammatory response)
- Ruptured Baker cyst

- Pyoderma gangrenosum
- Congestive heart failure
- Pulmonary hypertension
- Renal disease (nephrotic syndrome, renal failure resulting in hypervolemia)
- Liver disease (ascites)
- Vasculitis or autoimmune disorders (polyarteritis, hypothyroidism with myxedema, systemic lupus erythematosus)
- Pregnancy (both normal pregnancy and preeclampsia/eclampsia)
- Medications (NSAIDs, calcium channel blockers)



TREATMENT

INITIAL STABILIZATION/THERAPY

- Leg elevation to above the level of the heart
- Control bleeding with direct pressure.

ED TREATMENT/PROCEDURES

- Leg elevation above the level of the heart
- Compression stockings
- Barrier creams (white petroleum jelly or Zinc oxide) with wound dressings applied to ulcers
- Anticoagulants if confirmed DVT
- Antibiotics if signs of infection, specifically cellulitis or infected ulcer
- Aspirin (improves ulcer healing rate)
- Topical steroids for stasis dermatitis, pruritis, dry skin
- Antihistamines for pruritus

MEDICATION

- Aspirin: 325 mg once daily PO
- Augmentin: 875 mg BID PO
- Benadryl: 12.5–25 mg QID PO
- Cephalexin: 500 mg QID PO
- Dicloxacillin: 500 mg QID PO
- Coumadin: Dose per prothrombin time/INR
- Lovenox: 1 mg/kg SC BID



FOLLOW-UP

DISPOSITION

Admission Criteria

- Arterial insufficiency

- Evidence of cellulitis, lymphangitis, or osteomyelitis may require admission, specifically in a patient that is immunocompromised (on steroids, receiving chemotherapy or radiation therapy, history of diabetes)
- DVT in the following setting:
 - Thrombosis is extensive
 - There is concern for pulmonary embolism
 - The patient is at high risk for bleeding with anticoagulation
 - Outpatient management with low molecular weight heparin and/or close follow up is not appropriate or available
- Treatment of an underlying etiology of lower extremity swelling other than primary venous insufficiency or the patient's other comorbid conditions warrant admission

Discharge Criteria

- Lower extremity pulses are present
- No evidence of bleeding or compartment syndrome
- DVT has been ruled out with DUS or patient is low risk (See Well's Criteria for DVT)
- No evidence of bacterial infection requiring admission
- Appropriate follow-up/referral arranged
- Patient has been given instructions for wound care, dressing changes, and the use of compression stockings

Issues for Referral

The patients should be referred to their primary care physician. They should be referred to a vascular surgeon if there is concern for peripheral vascular disease.

FOLLOW-UP RECOMMENDATIONS

- Home health care or close follow-up with outpatient care provider for ulcer management
- Immediate surgical procedures are not required for varicose veins.
- Vein stripping, vein ligation, sclerotherapy and endovenous thermal ablation are options for cases refractory to medical management:
 - These do not improve healing of ulcers but reduces ulcer recurrence
 - All these methods cause irreversible changes to the venous system of the lower extremity which can result in recurrence of edema and can increase risk for DVT in the future.

PEARLS AND PITFALLS

- In patients with no palpable pulses, extremity pain, ulcerations, or risk factors for peripheral artery disease, ensure that arterial insufficiency is not the underlying cause before assuming venous insufficiency.

- Compression therapy is contraindicated in patients with peripheral vascular disease and venous insufficiency presenting with overlying cellulitis.

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See Also (Topic, Algorithm, Electronic Media Element)

Deep Venous Thrombosis



CODES

ICD9

- 453.40 Acute venous embolism and thrombosis of unspecified deep vessels of lower extremity
- 454.9 Asymptomatic varicose veins
- 459.81 Venous (peripheral) insufficiency, unspecified

ICD10

- I82.409 Acute embolism and thrombosis of unspecified deep veins of unspecified lower extremity
- I83.90 Asymptomatic varicose veins of unspecified lower extremity
- I87.2 Venous insufficiency (chronic) (peripheral)

VENTILATOR MANAGEMENT

Ruth L. Lamm

BASICS

DESCRIPTION

- Mechanical ventilation is machine generated flow of gas into and out of the lungs that acts as a substitute for normal respiratory function

Basic Concepts: Physiology and Pulmonary Mechanics

- Mechanical ventilation is *positive pressure ventilation* indicating that forced gas delivery generates positive pressure during inspiration
- Negative pressure ventilation:
 - _ Natural respiratory pattern
 - _ At rest (functional residual capacity) surface tension of alveoli is balanced by elastic recoil of chest wall; alveoli pressure equals atmospheric pressure at this point
 - _ In inspiration, lungs expand causing alveolar pressure to become negative compared with atmospheric pressure and air travels down pressure gradient into lungs
 - _ Exhalation is normally passive, but can be made active with the use of accessory muscles in the setting of airway obstruction/increased airway resistance
- Minute ventilation (MV):
 - _ Total volume of breaths in 1 min
 - _ Breaths in 1 min is respiratory rate (RR)
 - _ Standard breath is called tidal volume (TV)
 - _ $MV = TV \times RR$: Each component can be adjusted to control ventilation
- Oxygenation is controlled with adjusting fraction of inspired oxygen (FiO_2) and positive end-expiratory pressure (PEEP)
- Compliance:
 - _ Describes lung distensibility
 - _ Defined as change in volume with given change in pressure
 - _ Decreased lung compliance can be caused by problems with the lung parenchyma (i.e., pneumonia, ARDS) or problems with the chest wall/pleura (i.e., abdominal distension)
 - _ Lung compliance determines *plateau pressure*:
 - Plateau pressure is the steady state pressure; represents the attenuated pressure that is distributed to the small airways and alveoli during positive pressure ventilation
 - Goal ≤ 30 mm Hg
- Resistance:

- Defined as change in pressure with given flow
- Main determinant is airway radius
- Increased resistance can be caused by problems with the airways (i.e., bronchospasm), problems with the endotracheal tube (i.e., secretions), or problems with ventilator tubing
- Resistance determines *peak pressure*:
 - Peak pressure is the pressure seen in the larger airways before delivered volume is distributed to smaller airways and alveoli
 - Also determined by TV delivered
 - Goal ≤ 40 mm Hg

DIAGNOSIS

SIGNS AND SYMPTOMS

- Indications for mechanical ventilation:
 - Failure to oxygenate:
 - Diffusion defect (i.e., pulmonary edema, pneumonitis, pneumonia)
 - Severe ventilation/perfusion mismatch (i.e., PE, severe hypoventilation)
 - Severe shock:
 - Shock = oxygen supply does not meet oxygen demand by tissues
 - Mechanical ventilation can help improve shock states in 2 ways:
 - Increased oxygen delivery
 - Reducing overall oxygen demand by replacing organ system with high oxygen requirement
- Failure to ventilate:
 - Obtundation/sedation
 - Loss of ability to control diaphragm or intercostals (i.e., high spinal cord injury)
 - Severe myopathy
 - Dysfunctional chest wall (i.e., flail chest, increased abdominal pressures leading to decreased chest wall excursion, obesity)
 - Increased dead space (large PE, airway obstruction)
 - Metabolic acidosis (creates need for higher MV to compensate)
- Other:
 - Patient safety/need for evaluation
 - Predicted deterioration in clinical course
- *Ventilation strategy should specifically address the indication for mechanical ventilation!*
 - Example: In the setting of severe acidosis a preferred mode would be one where you could control MV closely

- Example: In severe pulmonary edema controlling the MV is not as important as ensuring oxygen delivery

Incidence and Prevalence Estimates

HISTORY & PHYSICAL EXAM

- Focus on underlying etiology for respiratory failure
- Exam on mechanical ventilation should include assessing oxygen saturation, evaluating end-tidal CO₂ (ETCO₂) with capnometry and capnography (see below), auscultating lung sounds/air movement, observing chest wall rise, palpating for abdominal distension
- ETCO₂:
 - Capnometry is the quantitative partial pressure of ETCO₂.
 - Capnography is the graphic representation of the changes in ETCO₂ with respiratory cycle
 - Normal lungs have a small degree of ventilation/perfusion mismatch as well as anatomic dead space. As a result, ETCO₂ is usually around 2–5 mm Hg lower than PaCO₂
 - Capnometry will be affected by: Amount of dead space or ventilation/perfusion mismatch; changes in metabolic CO₂ production (although ratio between PaCO₂ and ETCO₂ will not change); venous return (also will not affect ratio)
 - Evaluation of the ETCO₂ waveform can be very useful:
 - Can help assess response to bronchodilator therapy as waveform in airway obstruction has a steeper upslope instead of a plateau given the prolonged expiratory phase
 - Can help assess adequacy of CPR (will see return of waveform with good compressions)
 - Can help assess cause of tachypnea or dyssynchrony
- Monitor hemodynamic status closely with mechanical ventilation

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Arterial blood gas (ABG):
 - Should be checked within 15–30 min of initiation of mechanical ventilation and repeated with any change in clinical status
 - pH and PaCO₂ will help assess ventilation
 - PaO₂ will assess oxygenation
 - With ability to assess continuous oxygen saturation and ETCO₂ need for frequent ABGs, even after change in ventilator settings, may be eliminated

or reduced

- Serum chemistries including basic electrolytes with bicarbonate, liver function, renal function may help assess acid/base status which will affect ventilation strategy
- Hemoglobin/hematocrit will help describe state of oxygen delivery

Imaging

Imaging may include bedside US, chest x-ray, and chest CT to assess for endotracheal tube placement and pathophysiology of the lung and chest wall

DIFFERENTIAL DIAGNOSIS

See indications for mechanical ventilation above



TREATMENT

PRE HOSPITAL

Respiratory support per local EMS protocol

INITIAL STABILIZATION/THERAPY

- Cardiac monitor
- BP monitoring
- Pulse oximetry
- End-tidal CO₂ monitoring when available

ED TREATMENT/PROCEDURES

Critical actions include: Choosing appropriate ventilatory mode; assessing and adjusting ventilator settings; standard postintubation care; treatment of the underlying process.

- Postintubation care is of utmost importance. Includes: Sedation and/or analgesia; confirmation of tube placement; adjustment of ventilator settings based on clinical condition and ABG; establishing ETCO₂ gradient if using capnometry; elevating head of bed; placement of NG or OG tube.
- Settings common to most modes include:
 - RR:
 - In all modes, but will be set by patient in more spontaneous modes
 - Normal starting rates can vary from 12–20
 - Consider underlying pathophysiology before arbitrarily setting rate (i.e., elevated ICP, severe asthma)
 - Fraction of inspired oxygen (FiO₂):
 - Oxygen concentration in gas mixture
 - Usually start out with FiO₂ of 1 (100%) but wean down quickly after confirmation of stable oxygenation with prompt ABG

- PEEP:
 - Pressure that is applied to end expiration to maintain alveolar recruitment
 - Significant increase in work of breathing is required to open up collapsed alveoli
 - Collapsed alveoli do not participate in gas exchange, creating ventilation/perfusion (V/Q) mismatch and difficulty oxygenating and ventilating
 - By stenting open more alveoli, increased PEEP can improve oxygenation, especially at lower TVs (although be careful of high PEEP and overdistension which can lead to significant alveolar injury)
 - With normal chest wall compliance, basic starting PEEP will be 5–10 mm Hg
 - In setting of low chest wall compliance (obesity, anasarca, abdominal distension) may need to start with higher PEEP, around 10–15 mm Hg
- Inspiratory:expiratory ratio (I:E): Will alter flow rates. Allows for optimal mechanics in disease specific situations: E.g., increase E fraction in obstructive airway disease to prevent “breath stacking.” Normal ratio ~ 1:2.

Basic modes of ventilation:

- Early, classic modes of ventilation allowed for only simple ventilator/patient interaction and limited control of small number of variables
- *Continuous mandatory ventilation (CMV):*
 - CMV is the classic mode where only 1 variable can be set
 - Allows for NO interaction between the patient and the ventilator—all breaths are fully controlled breaths
 - Breathes are delivered only at a set rate—time is the trigger for every breath
 - Breathes are defined only by the control:
 - Volume controlled (also known as volume cycled) CMV: Delivers set volume with each breath and guarantees certain MV
 - Pressure controlled (also known as pressure cycled) CMV: Delivers constant flow of gas until set inspiratory pressure reached which guarantees peak pressures will be reasonable
 - The variable that is not set cannot be controlled (i.e., may have very high peak inspiratory pressures in order to deliver a certain TV or may dangerously hypoventilate in order to keep safe airway pressures)
- *Assist-control (AC):*
 - Similar to CMV in that all breaths are the same controlled breaths based on machine determined variables
 - In AC, patient can trigger a breath, but the same machine controlled breath is delivered
 - Spontaneous breath trigger is either the reduction in airway pressure or the

increase in air flow as patient initiates breath

- *Intermittent mandatory ventilation (IMV)*:
 - Delivers controlled breath at set RR
 - Patient may breath spontaneously between these breaths; however:
 - Spontaneous breaths are not supported
 - Can lead to breath stacking as ventilator does not take patient's spontaneous breaths into consideration
 - In some IMV modes, spontaneous breaths can be pressure supported, but this is not the rule
- *Synchronous intermittent mandatory ventilation (SIMV)*:
 - Same as IMV, but ventilator tries to synchronize patient's spontaneous breaths with those set by RR
 - Lowers risk of breath stacking
- *Pressure support ventilation (PSV)*:
 - Ventilator augments patient's spontaneous breaths with set amount of pressure
 - If support is adequate to meet needed driving pressure and patient is able to initiate breaths, often most comfortable mode
- Most modern ventilators and newer modes allow for much more complex interaction between ventilator and patient as well as increased control of multiple variables:
 - Newer modes are quite variable and are dependent on patient specifics.
 - May be dynamic combination of more traditional types of breaths as described below
 - Can often tailor breath delivery to optimize mechanics in specific disease process
- Risks of mechanical ventilation:
 - Ventilator-induced lung injury (VILI): Overdistension caused by high pulmonary pressures leads to inflammation and alveolar injury
 - Derecruitment injury: Inflammation and injury caused by repetitive opening and collapse of alveoli; can be reduced with appropriate use of PEEP
 - Barotrauma outside lungs due to cyclical reinflation (i.e., pneumothorax, pneumoperitoneum, subcutaneous emphysema)
 - Oxygen toxicity
 - Decreased venous return and subsequent drop in cardiac output/BP due to elevated intrathoracic pressures
 - Increased V/Q mismatch due to altered pattern of gas delivery (alveoli that usually do not get significant gas delivery in natural breathing will be responsible for more gas exchange without any augmented blood supply AND overdistension of alveoli may cause compression of alveolar blood supply)
 - Loss of upper airway defenses against infection
 - Associated risks of sedation (delirium, increased immobility, prolonged

- illness, etc.)
- Associated risks of immobility (severe myopathy, thrombosis, prolonged illness, etc.)
- Stress ulcer formation
- Problems related to endotracheal tube or tracheostomy such as tracheomalacia or vocal cord paralysis

MEDICATION

- Sedation and analgesia strategies should prioritize pain control, target the lowest level of sedation possible, and utilize intermittent bolus therapy before resorting to infusion
- Oversedation and benzodiazepines are both associated with risk of critical illness delirium
- Propofol: 0.3–1 mg/kg IV loading dose, maintenance initiated at 5–50 µg/kg/min IV infusion. Causes vasodilation and associated hypotension. Especially with bolus loading dose. Risk of propofol infusion syndrome with prolonged infusions.
- Dexmedetomidine: 0.2–1.4 µg/kg/h. Can be used with loading bolus of 1 µg/kg. Does not cause respiratory depression. Can be associated with significant bradycardia.
- Ketamine: Load 1–3 mg/kg with maintenance 1–2 mg/kg/h. Potential benefit is avoiding hemodynamic instability seen with many other agents. Benzodiazepine dosing prior to emergence can help prevent emergence nightmares. There is controversy about using ketamine in patients with elevated intracranial pressures, but it may actually help maintain cerebral perfusion pressure in mechanically ventilated patients.
- Fentanyl: Bolus 0.5–1.5 µg/kg IM or slow IV. Infusion rates start at 1 µg/kg/h. Consider prior opiate exposure when dosing.
- Albuterol: 2.5–5 mg/5 mL saline q4h via in-line endotracheal delivery
- Ipratropium bromide: 0.5 mg/2.5 saline q4h vial in-line endotracheal delivery



FOLLOW-UP

DISPOSITION

Admission Criteria

ICU admission required for all intubated patients

PEARLS AND PITFALLS

- Physiology can help you troubleshoot the vent. Remember that you control ventilation by adjusting the TV and RR and that you control oxygenation by adjusting PEEP and FiO₂. Peak pressure is determined by airway resistance.

Elevated peak pressures can be caused by problems such as bronchospasm, secretions, or kinked tubes. Plateau pressure is determined by lung and chest wall compliance. Elevated plateau pressures can be caused by problems such as ARDS, pulmonary fibrosis, obesity, or edema.

- Knowing the indication for mechanical ventilation is key to choosing the most appropriate and least harmful mode of ventilation and ventilator settings
- It is important to understand whether a breath is controlled or assisted, what triggers a breath, and how the breath is given in order to understand modes of ventilation. Most modern modes of ventilation are a complex combination of different types of breaths based upon goals set by the clinician or interactions with the patient.
- ARDS requires low TV ventilation and open lung ventilatory strategies can be used for severe cases
- Remember to allow time for full expiration for patients with obstructive airway disease

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See Also (Topic, Algorithm, Electronic Media Element)

- Dyspnea
- Respiratory Distress



CODES

ICD9

V46.11 Dependence on respirator, status

ICD10

Z99.11 Dependence on respirator [ventilator] status

VENTRICULAR FIBRILLATION

Marie-France Petchy • Richard E. Wolfe



BASICS

DESCRIPTION

- Ventricular fibrillation (VF) is completely disorganized depolarization and contraction of small areas of the ventricle without effective cardiac output.
- Cardiac monitor displays absence of QRS complexes and T-waves with the presence of high-frequency, irregular undulations that are variable in both amplitude and periodicity.

ETIOLOGY

- Damaged myocardium creates sites for re-entrant circuits:
 - Myocardial damage may be caused by multiple factors including ischemia, necrosis, reperfusion, healing, and scar formation
- Most often a result of severe myocardial ischemia:
 - 7% of patients with STEMI develop sustained VF, 80–85% occurring in the 1st 24 hr
- Complication of cardiomyopathy:
 - Up to 50% of patients with dilated cardiomyopathy suffer an episode of VF.
 - In hypertrophic cardiomyopathy, unexpected sudden death occurs with reported frequency of up to 3%/yr.
- Nonischemic causes of ventricular tachycardia may evolve into VF:
 - Drug toxicities (cyclic antidepressants, digitalis)
 - Electrolyte or acid–base abnormalities
 - Congenital and acquired prolonged QT syndromes.
 - Short QT syndrome
 - Brugada syndrome
- Premature ventricular complexes (PVCs) with R-on-T phenomenon
- Other less common causes of VF:
 - Electrocution
 - Hypoxia
 - Hypothermia
 - Blunt chest trauma
 - Iatrogenic myocardial irritation from pacemaker placement or pulmonary artery catheter
- Idiopathic VF (5–10%)

Pediatric Considerations

- Primary ventricular dysrhythmias are extremely rare in children.

- VF usually results from a respiratory arrest, hypothermia, or near drowning.

DIAGNOSIS

SIGNS AND SYMPTOMS

- Loss of consciousness
- Seizure
- Transient gasping followed by apnea
- Absent pulse and heart sounds
- Death if the rhythm remains untreated:
 - VF is the initial rhythm in ~5–70% of patients sustaining sudden cardiac death in the pre-hospital setting

ESSENTIAL WORKUP

- AED or manual defibrillator to confirm and treat a shockable rhythm
- Cardiac monitor

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Lab tests are not useful during resuscitation
- After return of spontaneous circulation (ROSC): Electrolytes including calcium and magnesium, BUN, creatinine, troponin, ABG, lactic acid level, and toxicology screen

Imaging

- After ROSC
- To identify cause of VF:
 - EKG
 - Cardiac US
 - CXR, also to monitor placement of an endotracheal tube (ETT)

DIFFERENTIAL DIAGNOSIS

- Asystole:
 - Fine VF may mimic asystole in a single lead.
 - Check rhythm in another lead for fine fibrillations

TREATMENT

ALERT

- Early defibrillation of VF is the most important determinant of survival, and each minute without defibrillation reduces survival by 7–10%.

- Single shock defibrillation strategy is the current standard
- Supraventricular tachycardia or VT with a pulse may degenerate into VF if cardioverted without synchronization.
- In a hypothermic cardiac arrest with VF, follow standard defibrillation strategy while rewarming the patient
- Do not defibrillate any conscious patient.

Controversies

- Escalating biphasic energy levels have been shown to improve conversion of VF:
 - Almost all automated external defibrillators (AED) and manual defibrillators commercially available are biphasic
 - Biphasic defibrillators are recommended because less energy is required
- Some study raised questions on the benefit of epinephrine in cardiac arrest
- The benefit of amiodarone or lidocaine in post cardiac arrest after ROSC is uncertain
- The benefit of procainamide as a 2nd-line antiarrhythmic remains controversial and is no longer included in the AHA guidelines

PRE HOSPITAL

- Promptly recognize cardiac arrest
- Follow initial stabilization/therapy
- Ideally, transport to the closest facility delivering comprehensive post cardiac arrest treatment

INITIAL STABILIZATION/THERAPY

- Use AED or manual defibrillator as soon as available
- Perform early CPR starting with chest compressions until defibrillator is ready
- Defibrillator confirms shockable rhythm
- Initiate SCREAM acronym
- **Shock:**
 - Immediate defibrillation with 1 shock
 - Biphasic energy level:
 - Follow manufacturer's recommendations (e.g. 120–200 J) for 1st shock; if unknown, use maximum available
 - Same or higher energy for subsequent shocks
 - 360 J monophasic for 1st and subsequent shocks
 - May repeat q2min until rhythm changes
- **CPR:**
 - Immediately resume CPR after each shock for 2 min starting with chest compressions
 - 30:2 compression–ventilation ratio if no advanced airway in place
 - ≥ 100 compressions per minute
 - Minimize CPR interruptions

- **Rhythm** check after every 2 min of CPR
- Secondary ABCD survey to try and determine underlying cause while resuscitation in progress
- Establish IV/IO access
- **Epinephrine** if defibrillation is unsuccessful:
 - Start after 2nd shock
 - May repeat q3–5min
 - Vasopressin may replace 1st or 2nd dose of epinephrine
- **Antiarrhythmic medications** if refractory VF:
 - Start after 3rd shock
 - Amiodarone
 - Lidocaine if amiodarone is not available
 - Magnesium for torsade de pointes
 - May consider a continuous infusion of the antiarrhythmic agent associated with ROSC
- Advanced airway management:
 - Should not delay initial CPR and defibrillation
 - Resume CPR with continuous chest compressions ≥ 100 /min and 1 ventilation every 6–8 sec
 - Use capnography to monitor ETT position, optimize quality of CPR, and detect ROSC

Pediatric Considerations

- Defibrillation sequence: Monophasic 2 J/kg, 2–4 J/kg, 4 J/kg
- May consider 4–10 J/kg or adult maximum dose for subsequent shocks

ED TREATMENT/PROCEDURES

- Post cardiac arrest care
- Identify and treat the cause of the VF arrest recognizing that the most likely cause is acute myocardial infarction:
 - Provide percutaneous coronary intervention when indicated
- Maintain SpO₂ $\geq 94\%$ and PETCO₂ at 35–40 mm Hg
- Treat SBP < 90 mm Hg
- Maintain body temperature at 32–34°C
- Treat hyperglycemia > 180 mg/dL (> 10 mmol/L)

MEDICATION

- Epinephrine: 1 mg IV/IO bolus, may repeat dose q3–5min
- Vasopressin: 40 U IV/IO bolus single dose
- Amiodarone: 300 mg in IV/IO bolus, may repeat 150 mg IV/IO bolus once:
 - Amiodarone infusion after ROSC: 1 mg/min for 1st 6 hr then 0.5 mg/min for 18 hr. Max. cumulative dose 2.2 g/24 h
- Lidocaine: 1–1.5 mg/kg IV/IO bolus, may repeat 0.5–0.75 mg/kg IV bolus q5–

- 10min; 3 doses max. or max. cumulative dose 3 mg/kg:
 - Lidocaine infusion after ROSC: 1–4 mg/min (30–50 µg/kg/min)
- Magnesium sulfate: 1–2 g in 10 mL D₅W IV/IO bolus
- Follow each medication with a 20 mL NS flush.

Pediatric Considerations

- Epinephrine: 0.01 mg/kg IV/IO, may repeat q3–5min; max. cumulative dose 1mg
- Amiodarone: 5 mg/kg IV/IO, may repeat 5 mg/kg; max. cumulative dose 15 mg/kg/d
- Lidocaine: 1 mg/kg IV/IO:
 - Lidocaine infusion 20–50 µg/kg/min
- Magnesium sulfate: 25–50 mg/kg IV/IO up to 2 g
- Follow each medication with a 3–5 mL NS flush.



FOLLOW-UP

DISPOSITION

Admission Criteria

All patients who survive need admission to the ICU/CCU.

Discharge Criteria

No patient who suffers a VF arrest may be discharged from the ED.

Issues for Referral

Patients with episodes of VF occurring >48 hr post-MI may need referral to electrophysiology.

PEARLS AND PITFALLS

ACC/AHA guidelines recommend that patients with an acute myocardial infarction should have their serum potassium maintained above 4 mEq/L to prevent ventricular dysrhythmias

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

2010 ACLS Guidelines



CODES

ICD9

427.41 Ventricular fibrillation

ICD10

I49.01 Ventricular fibrillation

VENTRICULAR PERITONEAL SHUNTS

Richard S. Krause

BASICS

DESCRIPTION

- Ventricular peritoneal (VP) shunts are usually placed for hydrocephalus:
 - Conduit between CSF and peritoneal cavity (or right atrium)
- **Obstruction:** Shunt malfunction impairs drainage of CSF:
 - Increases intracranial pressure (ICP)
 - Rate of increase in ICP determines severity
 - 30–40% mechanical malfunction rate in 1st year
- **Overdrainage syndrome:**
 - Upright posture increases CSF outflow
 - Decreases ICP
 - Produces postural headache and nausea (as after lumbar puncture)
- **Infection:**
 - Shunt is a foreign body
 - *Staphylococcus epidermidis* and other *Staphylococcus* species in 75% of infections
 - Gram-negative organisms also implicated
 - Multidrug-resistant *Staphylococcus aureus* (MRSA) has been reported
 - Most occur soon after placement
 - Shunt removal usually required
- **Slit ventricle syndrome:**
 - Prolonged overdrainage causes decreased ventricular size
 - Intermittent increases in ICP occur owing to proximal obstruction

Pediatric Considerations

- Complications more common in children, especially neonates
- If cranial sutures are open, CSF may accumulate without much ICP increase
- Produces relatively nonspecific signs and symptoms:
 - Drowsy
 - Headache
 - Nausea and Vomiting

ETIOLOGY

- Shunt may be needed to treat increased ICP due to:
 - Congenital malformations
 - Idiopathic intracranial hypertension (pseudotumor cerebri)
 - Post CVA

- Tumor or other mass lesions
- Post head trauma
- Subarachnoid hemorrhage
- Scarring at base of brain after bacterial meningitis



DIAGNOSIS

SIGNS AND SYMPTOMS

- Shunt obstruction:
 - Headache, nausea
 - Malaise, general weakness, irritability
 - Decreased level of consciousness (LOC)
 - Increased head size or bulging fontanelle
 - Seizures: New-onset or increased frequency
 - Autonomic instability
 - Decreased upward gaze
 - Apnea
 - Papilledema—rare
- Overdrainage syndrome:
 - Headache, focal neurologic signs, malaise, seizures, coma
 - Signs and symptoms often postural
- Rapid overdrainage may cause upward shift of the brainstem, leading to signs and symptoms of herniation: Apnea, bradycardia, decreased LOC
- Shunt infections:
 - Fever (may be absent)
 - Meningeal signs
 - Local signs of infection (erythema, swelling, tenderness)
 - Peritonitis (can cause retrograde CSF infection)
 - Infections usually occur soon after shunt placement (about 80% \leq 6 mo)
- Slit ventricle syndrome:
 - Episodic headache
 - Alternating periods of normal behavior and lethargy
 - Headache, nausea, and vomiting

History

- Timing of shunt placement
- Reason for shunt
- Recent instrumentation/revision

Physical-Exam

- Altered mental status
- Focal neurologic deficit

- Fever
- Erythema or tender shunt

ESSENTIAL WORKUP

- Suspected shunt malfunction:
 - Manipulation of the pumping chamber:
 - Chamber should compress easily and refill within 3 sec
 - Failure to compress easily implies distal obstruction
 - Failure to fill implies proximal obstruction
 - *Up to 40% of malfunctioning shunts compress/fill normally*
 - Head CT
 - Shunt series:
 - Radiographs of skull, chest, abdomen
 - Aids in diagnosis of disconnection, malposition, or kinking of shunt components
- Suspected infection:
 - Aspiration of CSF from shunt reservoir (in consultation with neurosurgeon):
 - May be performed using sterile technique and 23G butterfly needle
 - Slowly aspirate 5–10 mL CSF for the studies noted in the next section

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Electrolytes, renal function, and glucose
- Anticonvulsant levels
- CBC
- Suspected infection:
 - Analysis of CSF from the shunt reservoir:
 - Send for culture, cell count, Gram stain, glucose, and protein levels
 - CSF analysis may have normal early result, especially with prior antibiotic treatment
 - Blood cultures

Imaging

- Head CT: To compare ventricular size and evaluate catheter position:
 - Enlarged ventricles: Shunt malfunction
 - Smaller ventricles: Overdrainage
 - Most useful when compared with previous scan
 - Diagnose subdural hematoma
- US: Used in children with open fontanelle to evaluate position of shunt tip and assess ventricular size

Diagnostic Procedures/Surgery

- If symptoms of shunt malfunction are present but CT scan is not diagnostic, shunt tap is the next test:
 - Shunt manometry: High pressure > 20 cm H₂O implies distal shunt obstruction
 - Also used to evaluate CNS infection

DIFFERENTIAL DIAGNOSIS

- Seizure disorder (idiopathic, toxic, metabolic)
- Infections:
 - CNS infection not related to the shunt
 - Systemic infections
- Metabolic abnormalities:
 - Hypoglycemia
 - Hyponatremia
 - Hypoxia
- Intoxication/poisoning
- Head trauma



TREATMENT

PRE HOSPITAL

- Patients with shunt malfunction are at risk for apnea and respiratory arrest
- Oxygen should be applied with close monitoring of respiratory status
- When increased ICP is suspected, transport patient with head elevated to 30°

INITIAL STABILIZATION/THERAPY

- Signs of impending herniation:
 - Rapid-sequence intubation and controlled ventilation to Pco₂ ~ 35 mm Hg
 - Consider pretreatment with lidocaine (pediatric: Plus atropine)
 - Thiopental or etomidate for induction
 - Succinylcholine may increase ICP a few mm Hg, although this may not be clinically significant
 - Use only pretreatment dose of nondepolarizing agent if depolarizing agent chosen
 - Nondepolarizing agent (rocuronium) may be preferable
- Forced pumping of shunt chamber:
 - Flush the device with 1 mL of saline solution to remove distal obstruction
 - Allow slow drainage of CSF from the reservoir to achieve pressure < 20 cm H₂O
- IV mannitol to lower ICP
- *Ventricular puncture* and CSF drainage is a procedure of last resort if less invasive procedures unsuccessful and neurosurgeon unavailable

- *Status epilepticus*: Treated with benzodiazepines (lorazepam)

ED TREATMENT/PROCEDURES

- Early neurosurgeon consultation
- Shunt malfunction:
 - Elevate head of bed to 30°
 - Medical management with diuretics (mannitol, furosemide) may be appropriate in certain mild cases
- Overdrainage syndrome:
 - Maintain patient's supine position
 - Correct volume depletion
- Shunt infection:
 - Systemic antibiotics:
 - Vancomycin *plus* cefotaxime or gentamicin if gram-negative suspected

MEDICATION

- Adult and pediatric doses:
 - Atropine: 0.02 mg/kg IV (min. 0.1 mg)
 - Cefotaxime: 1–2 g (peds: 50 mg/kg) IV/IM q8–12h
 - Furosemide: 1 mg/kg IV
 - Gentamicin: 2–5 mg/kg IV
 - Lidocaine: 1 mg/kg IV
 - Mannitol: 1 g/kg IV
 - Rocuronium: 1 mg/kg IV
 - Succinylcholine: 1.5 mg/kg IV
 - Vancomycin: 15 mg/kg loading dose IV
 - Vecuronium: 0.08-0.1 mg/kg IV



FOLLOW-UP

DISPOSITION

Admission Criteria

Patients with shunt complications usually require neurosurgical consultation and admission. An ICU or other monitored setting is often needed.

Discharge Criteria

When shunt malfunction is ruled out, disposition depends on alternate diagnosis and patient condition.

PEARLS AND PITFALLS

- Avoid “tunnel vision” in a patient with a shunt and consider other causes for the

presentation

- Severe constipation may cause increased intra-abdominal pressure and decrease drainage resulting in increased ICP:
 - Treatment of constipation may ameliorate the apparent “shunt malfunction”

ADDITIONAL READING

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CODES

ICD9

- V45.2 Presence of cerebrospinal fluid drainage device
- 996.63 Infection and inflammatory reaction due to nervous system device, implant, and graft
- 996.75 Other complications due to nervous system device, implant, and graft

ICD10

- T85.09XA Other mechanical complication of ventricular intracranial (communicating) shunt, initial encounter
- T85.79XA Infect/inflm reaction due to oth int prosth dev/grft, init
- Z98.2 Presence of cerebrospinal fluid drainage device

VENTRICULAR TACHYCARDIA

Daniel C. McGillicuddy • Emily M. Mills



BASICS

DESCRIPTION

- A wide complex tachydysrhythmia with a quasirandom signal (QRS) > 120 and a rate > 100
- Rapid and regular depolarization of the ventricles independent of the atria and the normal conduction system
- Re-entry:
 - Structural heart disease most common
 - Seen in dilated cardiomyopathy, ischemia, and infiltrative heart disease, previous MI, scarring
 - May be pharmacologically induced
 - Usually produces a regular and monomorphic rhythm
- Triggered automaticity:
 - Minority of ventricular tachycardia (VT)
 - Caused by repetitive firing of a ventricular focus
- Torsades de pointes:
 - Polymorphic form of VT
 - Alternating electrical polarity and amplitude
 - Prolongation in repolarization necessary
 - Usually pharmacologically induced
- Regardless of the mechanism, all VT may degenerate to ventricular fibrillation (VF).

ETIOLOGY

- Wide complex tachycardia:
 - 80% likelihood of being VT
 - 20% supraventricular tachycardia (SVT) with a baseline left bundle branch block (LBBB) or aberrancy
- Wide complex tachycardia and a history of MI:
 - $> 98\%$ likelihood of being VT
 - Age > 35 : 80% risk of VT
 - Age < 35 : 75% risk of SVT
- Incidence of nonsustained VT:
 - 0–4% in the general population
 - Up to 60% of patients with dilated cardiomyopathy
- Associated with increased risk for sudden cardiac death (SCD)

SIGNS AND SYMPTOMS***History***

- Asymptomatic
- Syncope/near syncope
- Lightheadedness/dizziness
- Shortness of breath
- Palpitations
- Chest discomfort/pain
- Diaphoresis
- Cannon A-waves
- Hypotension
- CHF
- Beat-to-beat variability of systolic BP
- Variability in heart tones, especially S1

Physical-Exam

- Establish presence of pulses, mental status and vital sign abnormalities.
- Auscultation of heart will reveal tachycardia.

ESSENTIAL WORKUP

- EKG:
 - Most important initial test to differentiate VT from SVT with aberrancy or LBBB
- VT definition:
 - ≥ 3 consecutive QRS complexes with a ventricular rate over 100 bpm and a QRS duration > 120 msec
- Torsades de pointes:
 - Polymorphic VT that rotates its axis every 10–20 beats
- Criteria to determine VT:
 - Atrial ventricular (AV) dissociation (present in 60–75%)
 - Fusion beats (P-wave partially activates ventricle in advance of next VT cycle), capture beats (P-wave totally activates ventricle)
 - Uniform morphology (except in the case of torsades)
 - Extreme axis deviation (-90° to $+180^\circ$)
 - QRS > 140 msec, with right bundle branch block (RBBB) morphology; or QRS > 160 msec, with LBBB morphology, but > 160 suggests VT regardless of bunch branch morphology
 - QRS concordance in the precordial leads
 - RBBB pattern V1 with $R > R'$ is VT 50:1.

- LBBB pattern with Q or QS pattern in V 6 is VT 50:1.
- Brugada criteria defines VT in wide complex tachycardia:
 - 99% sensitivity, 97% specificity
 - Only need to meet 1 criterion
 - AV dissociation
 - R-S interval absent in all precordial leads
 - QRS onset to the nadir of S >100 msec in any precordial lead
 - V1 R-wave >30 msec; R-S interval >70 msec, slurred, notched S
 - Wide QRS with LBBB in precordium
- Indicators of SVT with aberrancy include:
 - Normal-axis QRS <140 msec
 - Absence of Q-waves
 - RBBB in V1 with rsR' triphasic pattern
 - AV nodal blockade: Slowing of impulse conduction velocity seen with antiarrhythmic drugs is more pronounced at faster rates, so may result in wide complex SVT (SVT with aberrancy)

DIAGNOSIS TESTS & NTERPRETATION

Lab

- Cardiac enzymes
- Electrolytes, BUN, creatinine, glucose
- Magnesium level
- Calcium level
- Digoxin level if toxicity suspected

Imaging

CXR:

- Cardiomegaly or other cardiac anomalies may be apparent.

ECHO:

- Structural disease may be identified.

Diagnostic Procedures/Surgery

Esophageal pacing catheters:

- May be able to detect atrial activity to establish AV dissociation and therefore diagnose VT
- Catheters can then be used to overdrive pace if refractory to cardioversion/antiarrhythmics.

DIFFERENTIAL DIAGNOSIS

- SVT with aberrancy or baseline LBBB
- Proarrhythmia secondary to antidysrhythmia medications; suspect if:
 - VT morphology is different than previous episodes of VT

- Medications have recently been started or changed
- QT interval is >440 msec.
- Torsades de pointes
- If VT continues to recur after cardioversion



TREATMENT

PRE HOSPITAL

- Cautions:
 - Transport stable patients suspected of being in VT without attempting to convert them.
 - *Synchronized* cardioversion for unstable patients with a pulse
 - Defibrillation for pulseless VT
- Controversies:
 - Lidocaine:
 - No benefit in the prevention of VT in patients with isolated premature ventricular contractions, regardless of the frequency

INITIAL STABILIZATION/THERAPY

Pulseless VT: Defibrillate immediately and follow the VF treatment plan.

ED TREATMENT/PROCEDURES

- Unstable patient:
 - Definition:
 - Chest pain
 - Hypotension
 - Evidence of worsening heart failure
 - Initiate immediate synchronized cardioversion with 100 J, quickly progressing to 200 J, 300 J, and 360 J if no response.
 - If the VT is polymorphic, begin cardioversion at 200 J.
 - Sedate the patient before cardioversion if at all possible.
 - If unable to terminate the VT, administer lidocaine and repeat the cardioversion.
 - Antitachycardia overdrive pacing if torsades
 - After successful return of sinus rhythm, begin amiodarone.
- Stable patient, monomorphic VT:
 - Normal cardiac function at baseline:
 - Procainamide *or* sotalol; may also consider amiodarone or lidocaine
 - Avoid sotalol if evidence of prolonged QT or known long QT syndrome.
 - Impaired cardiac function at baseline:
 - Amiodarone bolus, then infusion *or* lidocaine, then synchronized

cardioversion

- Stable patient, polymorphic VT:
 - Normal QT interval at baseline:
 - Correct electrolyte abnormalities.
 - Treat ischemia if present.
 - Then begin 1 of the following: b2-blockers, lidocaine, amiodarone, procainamide, or sotalol.
 - Prolonged QT Torsades de pointes:
 - Correct electrolytes.
 - Magnesium sulfate *or* overdrive pacing *or* 1 of the following: Isoproterenol, phenytoin, lidocaine
 - Isoproterenol is used to overdrive the tachycardia if the patient has no history of coronary artery disease or long QT syndrome.
 - Temporizing measure until external pacing available
 - Impaired cardiac function at baseline
 - Amiodarone bolus *or* lidocaine bolus then synchronized cardioversion

Pediatric Considerations

- Primary cardiac arrest and VT are rare in children.
- Usually secondary to hypoxia and acidosis
- VT is tolerated for longer periods in children than adults and is less likely to degenerate to VF.
- Infants in VT most commonly present with CHF.
- VT in children results from:
 - Cardiomyopathy
 - Congenital structural heart disease
 - Congenital prolonged QT syndromes
 - Coronary artery disease secondary to vasculitis
 - Toxins, poisons, drugs
 - Severe electrolyte imbalances, especially of potassium

MEDICATION

First Line

- Procainamide: 3–6 mg/kg over 5 min, may repeat every 5–10 min to max. total dose of 15 mg/kg. Do not exceed 100 mg/dose or 500 mg in 30 min (peds: 15 mg/kg IV/IO over 30–60 min).
- Amiodarone: 150 mg IV bolus over 10 min, may repeat; arrest dose is 300 mg IV/IO max. cumulative dose 2.2 g IV/24 h; followed by 1 mg/min for 6 hr, then 0.5 mg/min for 18 hr. (peds: 5 mg/kg IV or IO over 20–60 min, max. 15 mg/kg/d)
- MgSO₄: 2 g in D₅W over 5–10 min followed by infusion of 0.5–1 g/h IV, titrate to control torsades (peds: 25–50 mg/kg IV/IO over 10 min, max. dose 2 g)

Second Line

- Lidocaine: 1–1.5 mg/kg bolus IV push 1st dose, 0.5–0.75 mg/kg 2nd dose, and q5–10min for a max. of 3 mg/kg; tracheal administration 2–4 mg/kg; maintenance infusion 1–4 mg/min if converted. Not recommended for ACS induced VT (peds: 1 mg/kg bolus with infusion 20–50 µg/kg/min)
- Adenosine: 6 mg IV push followed by 12 mg IV push if needed in 1–2 min (peds: 1 mg/kg, max. 6 mg). Note: Does not convert VT, do not use if unstable or irregular WCT.
- Isoproterenol: 2–10 µg/min, titrate to heart rate (peds: 0.1 µg/kg/min). Note: Do not give with epinephrine, may precipitate VT/VF (no longer part of ACLS protocol), do not give if prolonged QT.
- Sotalol: 100 mg IV over 5 min. (peds: Use not recommended for initial management). Note: Do not give if prolonged QT.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Admit sustained VT to a critical care setting.
- Admit nonsustained VT and a history of MI or dilated cardiomyopathy for electrophysiologic studies.

Discharge Criteria

- Rare patients with nonsustained VT and a previous evaluation that revealed no structural heart disease can be discharged:
 - At low risk for SCD
- Patients with automatic internal cardiac defibrillators that are well functioning can also be discharged.

Issues for Referral

All patients discharged with VT should be followed by a cardiologist within 48 hr.

FOLLOW-UP RECOMMENDATIONS

Patients should follow-up with a cardiologist.

PEARLS AND PITFALLS

- Search for contributing factors such as toxins, metabolic abnormalities, trauma, hypothermia, thrombosis.
- Unstable VT requires early cardioversion.
- Administer postresuscitation maintenance medications to prevent recurrence.

- Watch for bradycardia and GI toxicity after amiodarone administration.
- Discontinue any proarrhythmic drugs
- Consider b2-blockade for ischemia-induced VT and polymorphic VT.

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

2010 AHA Guidelines for CPR and ECC



CODES

ICD9

427.1 Paroxysmal ventricular tachycardia

ICD10

I47.2 Ventricular tachycardia

VERTEBROBASILAR INSUFFICIENCY

Andrew K. Chang

BASICS

DESCRIPTION

- Vertebrobasilar (VB) vascular system feeds the posterior region of the brain, which includes the brainstem, cerebellum, and inner ear
- 2 vertebral arteries (VA) derive from subclavian arteries and give rise to the anterior spinal artery and then join to form the basilar artery
- Arteries supplying the brainstem and cerebellum originate from the VB system before it branches into the 2 posterior cerebral arteries (PCA), such that a wide variety of focal neurological deficits arise from VB circulatory dysfunction
- Vertebrobasilar insufficiency (VBI) results in inadequate perfusion of VB arterial circulation from thrombotic, embolic, or low-flow states

ETIOLOGY

- Mechanism:
 - _ Thrombosis:
 - VB ischemia due to underlying VB atherosclerosis and clot formation
 - _ Embolus:
 - VB ischemia due to embolization of clot from proximal location
 - _ Low-flow states:
 - Hypoperfusion of VB system from systemic (e.g., cardiogenic shock) or localized (e.g., subclavian steal) reduction in blood flow
 - _ Less common etiologies:
 - Fibromuscular dysplasia
 - Hypercoagulable states
- Ischemic mechanisms causing VB insufficiency can herald and lead to VB territory infarcts
- Severe episodes of VB hypoperfusion or loss of circulation can lead to:
 - _ “Locked-in” syndrome:
 - Quadriplegia (eyelid or eye movement only) with intact consciousness
 - _ “Top-of-basilar” syndrome:
 - Pontine and cerebellar dysfunction with diminished level of consciousness

DIAGNOSIS

SIGNS AND SYMPTOMS

All history and physical exam items may present intermittently

History

- Dizziness/vertigo (“mild,” “nonviolent”; may be isolated finding)
- Onset usually abrupt and spontaneous rather than position induced
- May have a flurry of spells within a few weeks time
- “Drop attack”
- Headache
- Nausea/vomiting
- Paresis/paresthesia
- Seizure
- Syncope
- Neurologic symptoms localizing to the posterior circulation:
 - Visual changes (double vision, blurry vision, blindness)
 - Numbness of the face or extremities
 - Weakness in arms or legs
 - Clumsiness in arms or legs
 - Confusion or loss of consciousness
 - Difficulty with speech
 - Difficulty swallowing
 - Pain in neck or shoulder

Physical-Exam

- Brainstem:
 - “Crossed” findings (i.e., ipsilateral facial and contralateral body deficits)
 - Altered mental status or responsiveness
 - Decreased respiratory drive
 - Horner's syndrome (enophthalmos, ptosis, miosis, anhidrosis)
 - Internuclear ophthalmoplegia
 - Nystagmus (especially nonfatigable, vertical/rotatory)
 - Paresis/paresthesias
- Cranial nerves:
 - Extraocular muscle paresis (e.g., diplopia)
 - Pupillary abnormalities
 - Facial paresthesia
 - Facial muscle paresis
 - Hearing abnormalities
 - Dysphagia
 - Dysarthria
- Cerebral cortex (PCA circulation):
 - Visual disturbances (e.g., homonymous hemianopsia)
- Cerebellar:
 - Ataxia
 - Dysmetria
 - Gait abnormality

- Cardiovascular:
 - Carotid/VA bruit
 - Irregular/asymmetric/weak pulses

ESSENTIAL WORKUP

- Emergent head CT (noncontrast) to evaluate for hemorrhage (parenchymal, subarachnoid, traumatic), large acute infarcts, prior pathology
- Thorough neurologic and cardiac exam
- Neurology consultation
- 12-lead ECG for arrhythmias and myocardial ischemia
- CTA and/or MRA for imaging of the posterior circulation

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC:
 - Anemia, thrombocytopenia; polycythemia, thrombocytosis
- Coagulation studies (PT/PTT):
 - Hypo- and hypercoagulable states; baseline values for anticoagulant and fibrinolytic therapies
- Electrolytes, BUN/creatinine, glucose
- Cardiac markers for concurrent myocardial ischemia
- Urinalysis
- ESR for systemic vasculitides
- Rapid plasma reagin
- Thyroid stimulating hormone
- Lipid profile

Imaging

- Emergent head CT (noncontrast)
- Head and neck CT angiogram (CTA) for evaluation of posterior circulation and possible acute vascular intervention
- Chest radiograph; consider chest CTA for cardiopulmonary and great vessel pathology
- MRI/magnetic resonance angiography (MRA) for improved characterization of ischemic lesion and cerebrovascular circulation (e.g., congenital VB anomalies, exclusion of VA dissection)
- Echocardiography for intracardiac embolic source
- Cervical Doppler US
- Transcranial Doppler US

Diagnostic Procedures/Surgery

- Neuroangiography for diagnosis

- Directed intra-arterial thrombolytic therapy/angioplasty/stenting/embolectomy are still under investigation)

DIFFERENTIAL DIAGNOSIS

- CNS:
 - CVA (hemorrhagic or ischemic):
 - Cerebral
 - Cerebellar
 - Brainstem
 - Multiple sclerosis
 - Migraine syndromes
 - Seizure (focal)
 - Traumatic injury/postconcussive
 - Tumor
 - Vascular malformation hemorrhage (arteriovenous malformation, subarachnoid)
 - Brainstem herniation
- Peripheral nervous system:
 - Vestibular neuritis
- Ear, nose, throat:
 - Cerebellopontine angle tumor
 - Ear canal pathology (foreign body, tumor)
 - Labyrinthitis/otitis media
 - Ménière disease
 - Benign paroxysmal positional vertigo
- Cardiovascular:
 - Arrhythmia
 - Myocardial ischemia/infarct
 - Aneurysm/dissection (VA, basilar artery, subclavian artery, aorta)
 - Hypovolemia
 - Vasculitides
- Endocrine:
 - Adrenal insufficiency
 - Hypothyroidism
- Hematologic:
 - Anemia
 - Coagulopathy/hypercoagulable state
- Infectious:
 - Encephalitis/meningitis
 - Otitis media/mastoiditis
 - Septic shock
 - Syphilis
- Metabolic:

- Hypoglycemia; hyperglycemia
- Electrolyte imbalance
- Toxicologic:
 - Ataxia: Alcohols, lithium, phenytoin
 - Salicylism
 - Serotonin syndrome
 - Iatrogenic



TREATMENT

PRE HOSPITAL

- ABCs
- Fingertick glucose measurement
- Naloxone if indicated
- Notification:
 - Urgent contact with receiving facility if airway compromise or hemodynamic instability

INITIAL STABILIZATION/THERAPY

- ABCs
- Administer oxygen
- Place on cardiac monitor and pulse oximeter
- Establish IV access with 0.9% normal saline

ED TREATMENT/PROCEDURES

- Cerebrovascular perfusion management:
 - Supportive care
 - Supine position
 - Antiplatelet agent if no hemorrhagic source
 - Anticoagulation:
 - Consider in consultation with neurology if significant risk factors for embolic source, unstable or progressive ischemic symptoms
 - Ideal BP targets not well defined; maintain BPs within patient's expected range (i.e., account for chronic hypertension)
- If hypotensive: Fluid resuscitation; vasopressors or blood as indicated
- If hypertensive: Administer titratable antihypertensive medications for severe HTN (mean arterial pressure > 140 mm Hg, systolic BP > 220 mm Hg, diastolic BP > 130 mm Hg) or hemorrhage/aneurysm/dissection, myocardial or other end-organ dysfunction
- GI:
 - NPO (rehydrate with IV fluids; maintain normoglycemia)
 - Antiemetics

- Consultation:
 - Neurology
 - Vascular interventional radiology for neuroangiography

MEDICATION

- Aspirin: 325 mg PO
- Clopidogrel: 75 mg PO
- Warfarin (dose for atrial fibrillation): 2–5 mg PO loading dose
- Heparin (dose for atrial fibrillation): 50–60 U/kg IV bolus, then IV infusion at 12–18 U/kg for target PTT 50–70 sec
- Labetalol: 20–40 mg IV over 2 min, then 40–80 mg IV q10min (max. 300 mg IV)
- Meclizine: 25 mg PO q8–12h
- Naloxone: 0.4–2 mg IM/IV q2–3min PRN
- Nitroprusside: 0.25–10 µg/kg/min IV infusion (max. 10 µg/kg/min)
- Ondansetron: 4 mg IV
- Promethazine: 12.5–25 mg PO/PR/IV q6–8h
- Ticlopidine: 250 mg PO BID



FOLLOW-UP

DISPOSITION

Admission Criteria

- ICU admission for:
 - Altered mental status with airway issues
 - Concurrent hemodynamic instability
 - Malignant cardiac arrhythmias
- Admit to hospital to identify or exclude etiologies of VB ischemia and to prevent recurrence or progression to VB circulation cerebrovascular accident, especially in the following populations:
 - Elderly
 - Inability to ambulate
 - Inability to tolerate oral intake
 - Inability to arrange (expeditious) outpatient follow-up
 - New or changing neurologic deficit
 - Persistent dizziness
 - Syncope
 - Vascular risk factors

Discharge Criteria

- Consider discharge with outpatient follow-up in populations with the following:
 - None of above indications to consider admission

- Alternative explanation for symptomatology

Issues for Referral

- VB ischemia-related referrals as arranged/recommended by admitting team
- Arrange expeditious referrals with PCP or appropriate specialist (e.g., neurology, otorhinolaryngology, vascular surgery) as indicated for alternative explanation for symptomatology

FOLLOW-UP RECOMMENDATIONS

- VB ischemia-related follow-up as arranged/recommended by admitting team
- Urgency and nature of other follow-up as determined by alternative explanation of symptomatology

PEARLS AND PITFALLS

- Always consider VB insufficiency for dizziness, vertigo, mental status changes, syncope, and overlapping/atypical neurologic presentations
- VBI more likely to occur in patients with spontaneous vertigo lasting a few minutes with accompanying neurologic symptoms and who have cardiovascular risk factors
- Start antithrombotic/antiembolic treatments for VB insufficiency in the absence of contraindications

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CODES

ICD9

435.3 Vertebrobasilar artery syndrome

ICD10

G45.0 Vertebro-basilar artery syndrome

VERTIGO

William E. Baker

BASICS

DESCRIPTION

- Dizziness, 3–4% of ED visits, difficult symptom to diagnose, describes a variety of experiences, including:
 - Vertigo
 - Weakness, fainting
 - Lightheadedness
 - Unsteadiness
- Vertigo, a hallucination of movement:
 - Spinning or turning
 - Sensation of movement between the patient and the environment
 - Oscillopsia (illusion of an unstable visual world)
 - Most patients have an organic etiology.
- Maintenance of equilibrium depends on interaction of 3 systems:
 - Visual
 - Proprioceptive
 - Vestibular
- Any disease that interrupts the integrity of above systems may give rise to vertigo.
- Peripheral vertigo:
 - Often, severe symptoms
 - Intermittent episodes lasting seconds to minutes, occasionally hours
 - Horizontal or horizontal–torsional nystagmus (also positional, fatigues, and suppressed by fixation)
 - Normal neurologic exam
 - Sometimes associated hearing loss or tinnitus
- Central vertigo:
 - Usually mild continuous symptoms
 - All varieties of nystagmus (horizontal, vertical, rotatory)
 - No positional association
 - Presence of neurologic findings most of the time

ETIOLOGY

Peripheral

- Acute peripheral vestibulopathy (APV):
 - Vestibular neuritis (most common):
 - Single acute attack continuous rotational vertigo

- Constant for several days
- Present even when still
- No hearing deficits
- Highest incidence in 3rd–5th decade
- Acute labyrinthitis:
 - Similar to vestibular neuritis but:
 - Associated with hearing deficit
 - May be viral (common), serous, acute suppurative, toxic, or chronic
- Benign paroxysmal positional vertigo (BPPV):
 - Most common cause recurrent vertigo
 - Posterior canal, 85–95% of BPPV cases
 - Lateral semicircular less common
 - Probable cause is loose particles (otoliths) in semicircular canals
 - Can be secondary to other entities including trauma and APV
- Ototoxic drugs:
 - Aminoglycosides
 - Antimalarials
 - Erythromycin
 - Furosemide
- Ménière disease:
 - Episodic vertigo, hearing loss, and tinnitus
- Acoustic neuroma:
 - Tumor of Schwann cells enveloping the 8th cranial nerve (CN VIII)
 - Develops into central cause
 - Progressive unilateral hearing deficits and tinnitus
 - May also involve CN V, VII, or X
- Trauma:
 - Rupture of tympanic membrane, round window, labyrinthine concussion, or development of perilymphatic fistula can all have severe symptoms.
- Otitis media and serous otitis with effusion
- Foreign body in ear canal

Central

- Vertebrobasilar artery insufficiency:
 - Disturbances may be transient or exacerbated by movement of the neck.
- Cerebellar infarction
- Cerebellar hemorrhage:
 - Neurosurgical emergency
 - Sudden onset of headache, vertigo, vomiting, and ataxia
 - Visual paralysis to affected side
 - Ipsilateral CN VI paralysis
- Multiple sclerosis:

- Onset between 20–40 yr
- All forms of nystagmus
- May have abrupt onset of severe vertigo and vomiting
- History of other vague and varying neurologic signs or symptoms
- Brainstem hypertensive encephalopathy
- Trauma:
 - Vertiginous symptoms common after whiplash injury
 - Postconcussive syndrome or damage to labyrinth or CN VIII secondary to basilar skull fracture
 - Vertebral artery injury has been seen after chiropractic manipulation.
- Temporal lobe epilepsy:
 - Associated with hallucinations, aphasia, trancelike states, or convulsions
 - More common in younger patients
- Vertebrobasilar migraines:
 - Prodrome of vertigo, dysarthria, ataxia, visual disturbances, or paresthesias followed by headache
 - Often a family history of migraines or similar attacks
- Tumor
- Subclavian steal syndrome:
 - Exercise of an arm causing shunting of blood from vertebral and basilar arteries into the subclavian artery, resulting in vertigo or syncope
 - Secondary to a stenotic subclavian artery
 - Diminished unilateral radial pulse or differential systolic BP between arms
- Hypoglycemia



DIAGNOSIS

SIGNS AND SYMPTOMS

Sensation of motion, spinning, disorientation in space, or disequilibrium

History

- Does true vertigo exist?
- Timing of onset:
 - Gradual (hours–days): Probably neuritis
 - Sudden and fixed symptoms (seconds–minutes) consider stroke (but see BPPV below)
 - Multiple prodromal episodes in months, especially weeks prior (TIAs): Stroke more likely
 - Repeated intense episodes provoked/exacerbated by head movements: BPPV more likely but could be TIA
 - Episodic attacks with auditory symptoms: Consider Ménière
- Stroke risk factors including age > 50 and vascular risks

- Severity of symptoms: Imbalance out of proportion to vertigo, consider stroke
- Modifiers: Head movement, BPPV more likely
- Associated symptoms:
 - Hearing loss (new unilateral): Labyrinthitis, Ménière (with tinnitus), rarely, but possibly stroke
 - Neurologic symptoms (central cause):
 - Unilateral limb weakness
 - Dysarthria
 - Headache
 - Ataxia
 - Numbness of the face
 - Hemiparesis, headache
 - Diplopia/visual disturbances
 - Has there been head or neck trauma?
- Past medical history/ROS:
 - Stroke risk factors
- Medication history

Physical-Exam

- Extraocular movements:
 - Nystagmus (direction defined by fast component)
 - Unilateral, horizontal, some rotational component in (unilateral) APV, worse with gaze in the direction of nystagmus (fast away from lesion, linear slow phase)
 - Worse with occlusive ophthalmoscopy (cover 1 eye, examine optic disc with ophthalmoscope): APV more likely
 - Bilateral direction suggests central etiology, as does pure vertical or torsional nystagmus. If direction changes with gaze, central cause.
- Head impulse test (HIT) for unilateral vestibular loss (smartphone with slow motion video app promising aide for such testing):
 - Face patient, grasp head with both hands
 - Patient to look at your nose (or camera)
 - Rapidly rotate head 10–20° then back to midline:
 - Normal: Maintains gaze
 - Abnormal: Lag in maintaining gaze and corrective saccade back to nose/camera
 - Rotation to left, tests left vestibular apparatus
- Skew deviation testing (predicts central pathology):
 - Face patient
 - Patient to look at your nose
 - Alternately cover each eye
 - Normal: Eyes motionless
 - Abnormal: Refixation saccade after uncovered, (refixation upward,

ipsilateral medullary stroke, refixation downward, contralateral stroke)

- Dix–Hallpike test for posterior canal BPPV
- Supine Roll test for lateral canal BPPV
- Auscultation of the carotid and vertebral arteries for bruits
- Pulses and pressures in both arms
- Inspection of the ears:
 - Evaluation of hearing (Weber and Rinne tests)
 - Ocular assessment (pupils, fundi, visual acuity, nystagmus)
- Cardiac auscultation
- Full neurologic exam, common stroke findings:
 - Unilateral limb weakness
 - Gait ataxia
 - Unilateral limb ataxia and/or sensory deficit
 - Dysarthria

ESSENTIAL WORKUP

- Ask patient to describe the sensation without using the word “dizzy.”
- Determine whether the cause is a peripheral or a central process using patient’s clinical presentation (see above).

DIAGNOSIS TESTS & INTERPRETATION

Lab

Electrolytes, BUN, creatinine, glucose

Imaging

- EKG for any suspicion of cardiac etiology
- Head CT/MRI for evaluation of suspected tumor, or post-traumatic cause
- MRI/MRA for suspected vertebrobasilar insufficiency (CT poor sensitivity)

Diagnostic Procedures/Surgery

Audiology or electronystagmography often helpful in outpatient follow-up

DIFFERENTIAL DIAGNOSIS

More likely other cause when “dizziness” actually is lightheadedness or malaise:

- DM
- Hypothyroidism
- Drugs (e.g., alcohol, barbiturates, salicylates)
- Hyperventilation
- Cardiac (i.e., arrhythmia, MI, or other etiologies of syncope); peripheral vascular disease (i.e., HTN, orthostatic hypotension, vasovagal)
- Infection/sepsis



TREATMENT

PRE HOSPITAL

Treatment and medication per EMS protocol based on symptoms

INITIAL STABILIZATION/THERAPY

- IV access for dehydration/vomiting
- Monitor
- Trauma evaluations as indicated
- Finger-stick blood glucose

ED TREATMENT/PROCEDURES

- Based on accurate diagnosis:
 - Central etiologies require more aggressive workup than peripheral.
 - Neurosurgical intervention for cerebellar bleed
 - Symptomatic treatment for peripheral vertigo with appropriate follow-up
- Administer medication to control vertiginous symptoms and/or nausea:
 - Antihistamines
 - Benzodiazepines
 - Antiemetics
- Initiate IV antibiotics for acute bacterial labyrinthitis (rare).
- Repositioning maneuvers such as Epley and Semont for posterior BPPV. Roll or Lempert maneuver for lateral BPPV

MEDICATION

- Diazepam (Valium): 2.5–5 mg IV q8h or 2–10 mg PO q8h
- Dimenhydrinate (Dramamine): 25–50 mg IV, IM or PO q6h
- Diphenhydramine (Benadryl): 25–50 mg IV, IM, or PO q6h
- Lorazepam (Ativan): 1 mg IV, IM or 1–2 mg PO q4–6h
- Meclizine (Antivert): 25 mg PO q6h PRN
- Promethazine (Phenergan): 12.5 mg IV q6h or 25–50 mg IM, PO, or PR q6h



FOLLOW-UP

DISPOSITION

Admission Criteria

- Cerebellar infarct/hemorrhage
- Vertebrobasilar insufficiency
- Acute suppurative labyrinthitis
- Intractable nausea/vomiting
- Inability to ambulate

Discharge Criteria

Patient with peripheral etiology and stable

Issues for Referral

Otolaryngology follow-up for suspected acoustic neuroma or perilymphatic fistula

FOLLOW-UP RECOMMENDATIONS

- Primary care, neurology, or otolaryngology follow-up for all
- Epley and Semont maneuvers are extremely effective in treating BPPV.

PEARLS AND PITFALLS

- Isolated vertigo can be the sole symptom of stroke or bleed
- Central cause clues: Imbalance and/or ataxia out of proportion to vertigo
- Learn the specialized exam and repositioning techniques

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Dizziness
- Labyrinthitis



CODES

ICD9

- 386.10 Peripheral vertigo, unspecified
- 386.11 Benign paroxysmal positional vertigo
- 780.4 Dizziness and giddiness

ICD10

- H81.10 Benign paroxysmal vertigo, unspecified ear
- H81.399 Other peripheral vertigo, unspecified ear
- R42 Dizziness and giddiness

VIOLENCE, MANAGEMENT OF

Elizabeth R. Dunn • David S. Kroll



BASICS

DESCRIPTION

- EDs and waiting rooms are areas of high prevalence for violence
- Higher risk associated with busier EDs
- Patients with primary psychiatric complaints are likely to be boarding > 24 hr and may not be receiving psychiatric care
- Risk factors for violence in the ED:
 - Prior history of violence OR being a victim of violence
 - Patient arriving in police custody
 - Substance abuse history/intoxication
 - Poor impulse control
 - Male gender
 - Psychiatric illness (complex relationship to risk)
- No clear difference in risk associated with:
 - Ethnicity
 - Language
 - Education
 - Medical diagnosis

ETIOLOGY

- Primary psychiatric problem:
 - Most commonly psychosis or mania, but associated with many different diagnoses
- Acute primary medical problem:
 - Infection
 - Metabolic:
 - Hypoglycemia
 - Hypoxia
 - Hypothermia or hyperthermia
 - Toxicologic:
 - Alcohol intoxication or withdrawal
 - Illicit drug intoxication or withdrawal
 - Sedatives
 - Pain medications
 - Anticholinergics
 - Steroids
 - Neurologic:

- Seizure
- Stroke
- Head injury or bleed
- Brain lesion or mass
- Chronic primary medical problem:
 - Dementia
 - Intellectual disability
 - Traumatic brain injury
- Criminal behavior or psychopathy

DIAGNOSIS

SIGNS AND SYMPTOMS

- Early signs of impending violence risk (nonspecific):
 - Loud speech
 - Physical agitation or tension (pacing, clenching fists, darting eyes)
- Later signs of impending violence risk:
 - Abusive or provocative language
 - Behaving irrationally; unable to comply with reasonable limit setting
 - Invading personal space
 - Eliciting anger in staff

History

- Prior history:
 - Violent behavior
 - Self-injurious behavior
 - Medical and psychiatric histories
 - Substance use history
 - Legal or criminal history
- Current HPI:
 - Recent substance use
 - Potential head injury
 - Pain or discomfort from medical or psychiatric symptoms or environment
 - Plan or threat of violence
- Indicators of a higher likelihood of medical etiology:
 - Age >40 without a history of similar symptoms or behaviors
 - Concurrently emerging medical complaints
 - Comorbid medical conditions commonly associated with mental status changes:
 - Neurologic problems (including seizure disorders, CNS infections)
 - Chronic cognitive impairment
 - Vascular or cardiovascular disease

- Diabetes mellitus
- Chronic pain treated with opiates
- Inflammatory disorders treated with steroids
- Cancer
- HIV/AIDS
- Recent traumatic injury

Physical-Exam

- Exam signs suggesting a medical cause for the mental status change:
 - Abnormal vital signs
 - Focal neurologic findings
 - Seizure activity
 - Speech or gait deficits without evidence of alcohol or substance abuse

ESSENTIAL WORKUP

- Identify early warning signs
- Pay careful attention to findings during neurologic and mental status exams and note vital signs
- May be performed with the patient in restraints in an emergency

DIAGNOSIS TESTS & INTERPRETATION

- Follow clinical indicators for further testing, but if planning a psychiatric admission, labs and/or imaging may be required
- Basic labs and ECG may be useful in assessing and monitoring risks associated with chemical restraint use

Lab

- CBC, electrolytes, BUN, creatinine, and glucose if medical cause is suspected or if psychiatric admission or chemical restraint use is likely
- Consider LFTs, Ca, Mg, and Ph if chronically medically ill or pursuing delirium
- Drug screen if ingestion is likely

Imaging

CT head if bleed or stroke suspected

Diagnostic Procedures/Surgery

Obtain ECG if chemical restraint use is likely



TREATMENT

ALERT

- Medical workup is important, but in an emergency you may need to restrain

potentially violent patients 1st to reduce risk of harm to self or others

- Involve security or police as needed

PRE HOSPITAL

- Physically restrain violent patients and seek police assistance if necessary
- Keep weapons and other dangerous items (sharp objects, medications, cords, etc.) out of the patient's reach

INITIAL STABILIZATION/THERAPY

- Prevention:
 - _ Environmental:
 - Control access to ED: Secured doors, protected entrances, metal detectors, cameras
 - Visible security staff
 - Post visible rules stating clearly that weapons are not allowed
 - Exam room exits clear of obstruction
 - _ Procedural:
 - Identify high-risk patients at triage
 - Shorter ED wait times are helpful
 - Search/derobe patients after triage; if involuntary, ensure careful documentation of reasons in terms of risk to patient and providers
 - See to patients' comfort quickly
 - Alleviate pain
 - Online alerts for patients with past history of violence in ED
 - Clear ED protocols for managing violence and documenting interventions
 - Enlist family support when possible; if not, remove family to safe place
 - Train all clinical staff to recognize and manage potentially violent situations
- Approaching the potentially violent patient:
 - _ Do not go alone
 - _ Remove your own personal articles that could be used as weapons (neckties, jewelry, trauma shears, etc.)
 - _ Keep 2 arm's lengths between you and patient; open stance
 - _ Introduce yourself and try to address the patient's concerns as soon as possible
 - _ Maintain open exit for patient and staff
 - _ Leave immediately and initiate seclusion or restraint if there is an open threat of violence or imminent violence seems likely

ED TREATMENT/PROCEDURES

- Verbal de-escalation:

- Attempt to clarify and validate patient’s immediate concerns
- Calmly explain potential need for a restraint if de-escalation is not successful
- Offer patient choices when possible
- Seclusion:
 - If an appropriate room is available, this may obviate the need for restraint
- Physical restraint:
 - Follow your institutional protocol
 - Must document appropriate reason for restraint, attempts to verbally de-escalate, and plans for appropriate monitoring and reassessments
 - Whenever possible, treating physician should not be part of restraint team
 - Use leather restraints for combative patients; soft restraints for patients who are unlikely to be combative or try to elope
 - Supine position if patient needs to be examined; side position if aspiration risk is significant
 - If restraint in prone position is needed, ensure adequate airway is maintained
- Chemical restraint:
 - Offer voluntary PO or IM sedative medication prior to initiating involuntary restraint
 - Avoid PO medications for involuntary restraint due to bite risk
 - Choice of medication should depend on underlying cause; either a benzodiazepine or a neuroleptic or both may be appropriate:
 - If agitation results from delirium or other medical condition, 1st attempt to treat the underlying cause
 - Consider benzodiazepines for hyperadrenergic (including cocaine) state or if there is a contraindication to neuroleptics
 - Consider neuroleptics for most primary medical or psychiatric causes, sedative intoxication, or primary behavioral cause
 - Often used in combination
 - Contraindications to neuroleptics:
 - Knowledge of or suspicion for Parkinson disease, dementia with Lewy bodies or frontotemporal dementia
 - Neuroleptic malignant syndrome, dystonic reaction, or catatonia
 - Prolonged QT
 - Anticholinergic overdose
 - Potential adverse effects:
 - Dystonia: Treat with IM benztropine 1 mg or IM diphenhydramine 50 mg
 - QTc prolongation and/or torsades de pointes (rare)
 - Neuroleptic malignant syndrome (rare): Stop all antipsychotics; begin intensive monitoring and supportive care

MEDICATION

ALERT

- Patients who are elderly, have medical or neurologic illness, or have cognitive impairment are more vulnerable to adverse effects and may respond to lower doses (e.g., haloperidol 0.5 mg)
- If 1st dose of IM haloperidol is ineffective, may be repeated after 30–60 min.
- First line:
 - _ Haloperidol: 5–10 mg IV, IM, or PO
 - _ Lorazepam: 1–2 mg IV, IM, or PO
- Second line:
 - _ Droperidol: 2.5–5 mg IV or IM; watch QTc
 - _ Olanzapine: 5–10 mg IM or PO; if IM, do not give with IM/IV benzodiazepines due to risk of respiratory depression
 - _ Risperidone: 0.5–1 mg PO
 - _ Ziprasidone: 10 mg IM every 2 hr, not to exceed 40 mg IM per day



FOLLOW-UP

DISPOSITION

Admission Criteria

- Medical admission for medical conditions not temporary or reversible in the ED
- Medical admission if further medical workup needed for which ED setting is not optimal
- Psychiatric admission if patient has a treatable psychiatric illness appropriate for inpatient level of care
- Involuntary admission for safety may be necessary according to criteria defined by individual state laws

Discharge Criteria

- Underlying medical or psychiatric causes have been stabilized
- Appropriate follow-up is in place
- Access to weapons has been assessed
- If intoxication played a role in presentation, sober re-evaluation should occur prior to discharge
- Discharge to police custody may be appropriate if no psychiatric or medical issues remain
- If patient elopes, must consider imminent danger to self or others; notify police if risk is high or if safety evaluation not complete
- Duty to warn or protect 3rd parties from risk of harm: “Tarasoff” laws vary among states, so know yours

ADDITIONAL TREATMENT

Issues for Referral

- Psychiatric consultation in the ED can be helpful, especially if primary mental illness suspected
- Other consultation may be indicated based on the underlying etiology

FOLLOW-UP RECOMMENDATIONS

- Patients with psychiatric illness should follow-up with community mental health provider
- Patients who are using substances should be offered counseling and/or detox

PEARLS AND PITFALLS

- Do not assume that patients with violent behavior have only psychiatric problems
- Patients who have been restrained require appropriate monitoring, including regular nursing checks and VS, and labs/ECG if chemical restraints are used
- “Distracting staff” is annoying and may interfere with the care of other patients, but this is not an indication for restraints
- Document need for restraints and renewal of restraints per your hospital’s protocol

ADDITIONAL READING

- Coburn VA, Mycyk MB. Physical and chemical restraints. *Emerg Med Clin North Am.* 2009;27:655–667.
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- Rossi J, Swan MC, Isaacs ED. The violent or agitated patient. *Emerg Med Clin North Am.* 2010;28:235–256.
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- Zun LS. Pitfalls in the care of the psychiatric patient in the emergency department. *J Emerg Med.* 2012;43(5):829–835.



See Also (Topic, Algorithm, Electronic Media Element)

- Psychosis, Acute
- Delirium



CODES

ICD9

- 292.89 Other specified drug-induced mental disorders
- 312.9 Unspecified disturbance of conduct
- 312.30 Impulse control disorder, unspecified

ICD10

- F19.929 Oth psychoactive substance use, unsp with intoxication, unsp
- F63.9 Impulse disorder, unspecified
- R45.6 Violent behavior

VISUAL LOSS

Jason Hoppe

BASICS

DESCRIPTION

- Decrease in visual function (i.e., visual acuity, visual fields, blurry vision)
- Visual loss has many etiologies and can be caused by multiple body systems

ETIOLOGY

- Ophthalmologic:
 - Eyelid or tear film abnormality
 - Anterior segment (cornea, anterior chamber, iris, lens)
 - Posterior segment (vitreous, retina, optic nerve)
 - Posterior to the eye (optic nerve, chiasm, radiations)
- Traumatic:
 - Corneal abrasion
 - Hyphema
 - Lens dislocation
 - Ruptured globe
 - Commotio retinae
 - Retinal detachment
 - Retinal/vitreous hemorrhage
 - Retrobulbar hemorrhage
 - Intraocular foreign body
- Neurologic:
 - Cerebral (cerebrovascular accident [CVA]) or intracranial pathology (mass lesion)
 - Multiple sclerosis
 - Optic neuritis
 - Migraine
- Cardiovascular system:
 - Embolic
 - Thrombotic
 - Ischemic
 - Hypertensive events
- Immunologic system:
 - Uveitis
 - Giant cell arteritis
- Infection:
 - Orbital cellulitis/abscess

- Cavernous sinus thrombosis
- HIV optic neuropathy or cytomegalovirus (CMV) retinitis
- Endocrine:
 - Diabetic retinopathy
 - Thyroid disease may cause diplopia (muscle hypertrophy) or corneal erosions
- Toxic:
 - Methanol (acute severe loss, subacute optic atrophy)
 - Licorice (transient loss, self-limited)
 - Digitalis (flashing lights, color changes)
 - Amiodarone (rare cause of optic neuropathy)



DIAGNOSIS

- Categorize visual loss by the properties associated with the decrease in visual function
- Transient (< 24 hr):
 - Minutes:
 - Transient ischemic attack = amaurosis fugax (unilateral)
 - Vertebrobasilar artery insufficiency (bilateral)
 - Minutes to hours:
 - Migraine
 - Sudden BP changes
- Persistent (> 24 hr):
 - Painless: Sudden:
 - Retinal artery or vein occlusion
 - Vitreous hemorrhage
 - Retinal detachment
 - Optic neuritis
 - Giant cell arteritis
 - Cerebral infarct
 - Painless: Gradual (weeks to years):
 - Cataract
 - Presbyopia
 - Refraction errors
 - Open-angle glaucoma
 - Chronic retinal disease
 - Macular degeneration
 - Diabetic retinopathy
 - CMV retinopathy
 - CNS tumor
 - Painful:
 - Corneal abrasion, ulcer, burn, or foreign body

- Angle-closure glaucoma
- Optic neuritis
- Iritis/uveitis/endophthalmitis
- Keratoconus with hydrops
- Orbital cellulitis/abscess
- Monocular: Pathology anterior to optic chiasm
- Binocular: Pathology posterior to optic chiasm
- Associated with systemic neurologic symptoms of visual field defects:
 - CVA (especially posterior or occipital circulation)
 - Mass lesion (pituitary adenomas, aneurysm, meningioma, other tumors)
- Malingering/hysteria

SIGNS AND SYMPTOMS

History

- Decreased vision:
 - Loss of vision
 - Blurry vision
 - Double vision:
 - Horizontal or vertical
- History of trauma
- Use of corrective lenses:
 - Contacts
 - Glasses
- Prior eye surgery or problems
- Eye pain
- Conjunctival redness or discharge
- New floaters
- Flashing lights
- Pain with eye movement
- Key elements to determine:
 - Acute or gradual onset
 - Length of symptoms
 - Transient vision loss or permanent
 - Binocular or monocular
 - Degree of vision loss
 - Painful or painless
 - Other comorbidities

Physical-Exam

- Ophthalmologic:
 - Visual acuity
 - Pupil exam

- Afferent papillary defect
- Confrontational visual field exam
- Extraocular muscle function
- Slit-lamp exam
- Intraocular pressure (Tonometry)
- Fundoscopy:
 - Optic nerve swelling
 - Pale retina with a cherry-red spot
- Cardiovascular:
 - Murmurs
 - Carotid bruits
 - Temporal artery tenderness
- Neurologic exam:
 - Complete exam for other deficits
 - Optic chiasm and intracerebral lesions
 - Occipital and posterior circulation lesions
- General:
 - Signs of immune, endocrine, or toxic disorders

ESSENTIAL WORKUP

Thorough history and physical exam

DIAGNOSIS TESTS & INTERPRETATION

Lab

- May be obtained to determine extent of other comorbidities in association with vision loss (i.e., diabetes, cardiovascular disease)
- Erythrocyte sedimentation rate if giant cell arteritis is suspected

Imaging

- Tests should be directed toward the suspected etiology of visual loss
- Dilated fundus exam may be performed to assess for posterior segment disease
- Temporal artery biopsy may be obtained if giant cell arteritis is suspected
- Brain CT, MRI, MRA, and transcranial Doppler may be used to evaluate neurologic symptoms and vertebrobasilar artery
- Urgent cardiac and carotid US if a retinal artery occlusion is diagnosed
- Facial CT may be used to evaluate extent of traumatic injuries

DIFFERENTIAL DIAGNOSIS

- Trauma
- Neurologic lesion
- Infectious
- Cardiovascular

- Toxic/metabolic
- Autoimmune



TREATMENT

PRE HOSPITAL

- Chemical burns:
 - Begin copious irrigation with water or saline

ED TREATMENT/PROCEDURES

- Direct therapy toward cause of visual loss
- Ophthalmology consultation for visual loss with an uncertain diagnosis
- 3 conditions for which identification and treatment must begin within minutes:
 - Central retinal artery occlusion
 - Chemical burn
 - Acute angle-closure glaucoma

Central Retinal Artery Occlusion

- Clinical criteria:
 - Unilateral, painless, dramatic vision loss
 - Afferent pupillary defect
 - Pale fundus with a cherry-red spot (macula)
 - Counting fingers to light perception in 94% of patients
- Therapy:
 - Immediate ophthalmology consultation
 - Maneuvers and medications to lower intraocular pressure, allowing the embolus to move to the periphery:
 - Ocular massage: Direct pressure to eye for 5–15 sec then sudden release, repeat for 15 min
 - Acetazolamide: 500 mg IV or PO
 - Topical β -blocker
 - Anterior chamber paracentesis by an ophthalmologist
 - Referral for cardiac and carotid artery workup
 - Rule out giant cell arteritis

Chemical Burn

- Clinical criteria:
 - Alkali worse than acids
 - White eye (vessels have already sloughed) worse than red eye (vessels are intact)
 - Examples: Mace, cements, plasters, solvents
- Therapy:

- Topical anesthetic
- Copious irrigation of the eyes with LR or NS (nonsterile water is acceptable if others not available); minimum of 30 min
- Goal: Neutral pH at 5–10 min after ending irrigation
- Do not try to neutralize acids with alkalis or vice versa
- Evert lids and use moist cotton-tipped applicator to sweep furnaces for residual chemical precipitants
- Dilate with cycloplegic (atropine, cyclopentolate, tropicamide)
- Do not use phenylephrine; vasoconstricts already ischemic conjunctival blood vessels
- Erythromycin ointment q1–2h
- Artificial tears q1h
- Check intraocular pressure

Acute Angle-closure Glaucoma

- Signs and symptoms:
 - Unilateral, painful vision loss
 - Nausea, vomiting, headache
 - Cornea injected, edematous
 - Mid-dilated, sluggish/nonreactive pupil
 - Swollen, “steamy” lens
 - Cell, flare in a shallow anterior chamber
 - Increased intraocular pressure (> 20 mm Hg)
- Therapy:
 - Topical β -blocker
 - Topical prostaglandin analog
 - Acetazolamide
 - Topical α -2 agonist
 - Pilocarpine
 - Mannitol: If no decrease in IOP after 1 hr

MEDICATION

- Antibiotic drops:
 - Ciprofloxacin 0.3%: 1–2 gtt q1–6h
 - Gentamicin 0.3%: 1–2 gtt q4h
 - Ofloxacin 0.3%: 1–2 gtt q1–6h
 - Levofloxacin 0.5%: 1–2 gtt q2h
 - Polymyxin (Polytrim) 1 gtt q3–6h
 - Sulfacetamide 10%, 0.3%: 1–2 gtt q2–6h
 - Tobramycin 0.3%: 1–2 gtt q1–4h
 - Trifluridine 1%: 1 gtt q2–4h
- Antibiotic ointments:
 - Bacitracin 500 U/g 1/2 in ribbon q3–6h

- Ciprofloxacin 0.3%: 1/2 in ribbon q6–q8h
- Erythromycin 0.5%: 1/2 in ribbon q3–6h
- Gentamicin 0.3%: 1/2 in ribbon q3–4h
- Neosporin 1/2 in ribbon q3–4h
- Polysporin 1/2 in ribbon q3–4h
- Sulfacetamide 10%: 1/2 in ribbon q3–8h
- Tobramycin 0.3%: 1/2 in ribbon q3–4h
- Vidarabine 1/2 in ribbon 5 times per day
- Mydriatics and cycloplegics:
 - Atropine 1%, 2%: 1–2 gtt/day to QID
 - Cyclopentolate 0.5%, 1%, 2%: 1–2 gtt PRN
 - Homatropine 2%: 1–2 gtt BID–TID
 - Phenylephrine 0.12%, 2.5%, 10%: 1–2 gtt TID–QID
 - Tropicamide 0.5%, 1%: 1–2 gtt PRN dilation
- Corticosteroid–antibiotic combination drops (with ophthalmology consultation):
 - Prednisolone (Blephamide) 1–2 gtt q1–8h
 - Hydrocortisone/neomycin/bacitracin/polymyxin B (Cortisporin) 1–2 gtt q3–4h
 - Dexamethasone/neomycin/polymyxin B (Maxitrol) 1–2 gtt q1–8h
 - Prednisolone/gentamicin (Pred-G) 1–2 gtt q1–8h
 - Dexamethasone/tobramycin/chlorobutanol (TobraDex) 1–2 gtt q2–26h
- Glaucoma agents (always with ophthalmology consultation):
 - α -2 agonists:
 - Brimonidine 1% 1 gtt TID
 - Apraclonidine 1% 1 gtt TID
 - β -blocker:
 - Betaxolol 0.25%, 0.5%: 1–2 gtt BID
 - Carteolol 1%: 1 gtt BID
 - Levobunolol 0.25%, 0.5%: 1 gtt QD–BID
 - Carbonic anhydrase inhibitor:
 - Acetazolamide 500 mg PO/IV QD–QID
 - Miotic (parasympathomimetic):
 - Pilocarpine 0.25%, 0.5%, 1%, 2%, 3%, 4%, 6%, 8%, 10%: 1–2 gtt TID–QID
 - Osmotic agent:
 - Mannitol 1–2 g/kg IV over 45 min
 - Prostaglandin analog:
 - Latanoprost 0.005%: 1 gtt QD
- Only if mechanical closure is ruled out:
 - Timolol 0.25%, 0.5%: 1 gtt BID



DISPOSITION

Admission Criteria

- Ruptured globe
- Hyphema (depending on severity)
- Orbital cellulitis/abscess
- Cavernous sinus thrombosis
- Significant cardiac, carotid, or neurologic disease
- Unexplained, progressive vision loss

Discharge Criteria

If the diagnosis is certain and visual loss will not progress

FOLLOW-UP RECOMMENDATIONS

- Follow-up should be discussed with ophthalmology for emergent or urgent issues
- Referral for cardiac and carotid workup in embolic disease

PEARLS AND PITFALLS

- Document visual acuity for all eye complaints
- Topical anesthesia will aid in diagnosis as well as facilitating a proper eye exam
- Consider ocular issues and a detailed eye exam with headache complaints

ADDITIONAL READING

- Khare GD, Symons RC, Do DV. Common ophthalmic emergencies. *Int J Clin Pract.* 2008;62:1776–1784.
- Kunitomo DY, Kanitkar KD, Makar MS. *The Wills Eye Manual: Office and Emergency Room Diagnosis and Treatment of Eye Disease.* 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2004. Website: www.eyeatlas.com
- Mahmood AR, Narang AT. Diagnosis and management of the acute red eye. *Emerg Med Clin North Am.* 2008;26:35–55.
- Vortmann M, Schneider JI. Acute monocular visual loss. *Emerg Med Clin North Am.* 2008;26:73–96.



See Also (Topic, Algorithm, Electronic Media Element)

- Chalazion
- Conjunctivitis
- Corneal Abrasion
- Corneal Burn
- Corneal Foreign Body
- Dacryocystitis
- Giant Cell Arteritis

- Globe Rupture
- Hordeolum
- Hyphema
- Iritis
- Red Eye
- Optic Artery Occlusion
- Optic Neuritis
- Orbital Cellulitis
- Ultraviolet Keratitis
- Vitreous Hemorrhage



CODES

ICD9

- 368.8 Other specified visual disturbances
- 368.11 Sudden visual loss
- 369.9 Unspecified visual loss

ICD10

- H53.8 Other visual disturbances
- H53.139 Sudden visual loss, unspecified eye
- H54.7 Unspecified visual loss

VITREOUS HEMORRHAGE

Kevin F. Maskell, Jr. • Carl G. Skinner

BASICS

DESCRIPTION

Vitreous hemorrhage is a secondary diagnosis; identification of a specific cause is necessary for successful treatment:

- Retinal vessel tear due to vitreous separation
- Sudden tearing of vessels due to trauma
- Spontaneous bleeding due to neovascularization (e.g., diabetics)

ETIOLOGY

- Blunt or penetrating trauma
- Retinal break/tear/detachment
- Any proliferative retinopathy
- Diabetes mellitus
- Sickle cell disease
- Retinal vein occlusion
- Eales disease
- Senile macular degeneration
- Retinal angiomas
- Retinal telangiectasia
- Peripheral uveitis
- Subarachnoid or subdural hemorrhage:
 - Terson Syndrome
- Intraocular tumor

Pediatric Considerations

- Prematurity
- Congenital retinoschisis
- Pars planitis
- Child abuse:
 - Shaken-baby syndrome

DIAGNOSIS

SIGNS AND SYMPTOMS

- Sudden, painless unilateral loss or decrease in vision
- Appearance of dark spots (floaters), cobwebs, or haze in visual axis:
 - Above findings sometimes accompanied by flashing lights; floaters move

with head movements

- Blurred vision, decreased visual acuity
- Loss of red reflex
- Inability to visualize fundus
- Mild afferent papillary defect

History

- Ocular or systemic diseases
- Trauma

Physical-Exam

Fundoscopic exam:

- Absent red reflex
- No view of the fundus
- Acute:
 - RBCs in anterior vitreous
- Chronic:
 - Yellow appearance from hemoglobin breakdown

ESSENTIAL WORKUP

- History with special attention to pre-existing systemic disease and trauma
- Complete ocular exam including:
 - Slit lamp
 - Tonometry
 - Dilated fundoscopic exam

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC
- PT/PTT/INR if indicated
- Electrolytes, BUN, creatinine, glucose

Imaging

- B-scan US when no direct retinal view is possible to rule out retinal detachment or intraocular tumor
- Fluorescein angiography to define the cause
- CT scan/anteroposterior/lateral orbital films to rule out intraocular foreign body

Diagnostic Procedures/Surgery

If nontraumatic, scleral depression

DIFFERENTIAL DIAGNOSIS

- Vitreitis (leukocytes in the vitreous):
 - May include anterior or posterior uveitis
- Retinal detachment without hemorrhage
- Central retinal venous occlusion (CRVO)
- Central retinal artery occlusion (CRVA)



TREATMENT

PRE HOSPITAL

Protect the eye from trauma or pressure:

- Monitor BP

INITIAL STABILIZATION/THERAPY

- Bed rest with head of bed elevated
- No activity resembling Valsalva maneuver (lifting, stooping, or heavy exertion)
- Avoid NSAIDs and other anticlotting agents.

ED TREATMENT/PROCEDURES

- Urgent ophthalmologic consultation within 24–48 hr is needed with treatment based on the cause of the hemorrhage; an exam is carried out by the consultant:
 - Laser photocoagulation or cryotherapy for proliferative retinal vascular diseases
 - Repair of retinal detachments
- Surgical vitrectomy is needed for:
 - Blood that does not clear with time
 - VH from retinal detachment
 - Associated neovascularization
 - Hemolytic or ghost-cell glaucoma



FOLLOW-UP

DISPOSITION

Admission Criteria

Retinal break or detachment

Discharge Criteria

Retinal break or retinal detachment must be excluded as cause of hemorrhage.

FOLLOW-UP RECOMMENDATIONS

Re-evaluation daily for 2–3 days; if etiology is still unknown, B-scan US every 1–3 wk.

PEARLS AND PITFALLS

- Be sure to consider alternate diagnoses of CRVO or CRAO.
- Consider retinal detachment.
- Get history of trauma and use of blood thinners.
- Even minor bleeds require urgent ophthalmology consultation.

ADDITIONAL READING

- Dahl AA. Vitreous Hemorrhage in Emergency Medicine. *Medscape Reference*. February 2013.
- Gerstenblith AT, Rabinowitz MP. *The Wills Eye Manual: Office and Emergency Room Diagnosis and Treatment of Eye Disease*. 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2012.
- Hollands H, Johnson D, Brox AC, et al. Acute-onset floaters and flashes: Is this patient at risk for retinal detachment? *JAMA*. 2009;302(20):2243–2249.
- Leveque T. Approach to the patient with acute visual loss. In: DS Basow, ed. *UpToDate*. Waltham, MA: UpToDate; 2013.
- Lorente-Ramos RM, Armán JA, Muñoz-Hernández A, et al. US of the eye made easy: A comprehensive how-to review with ophthalmoscopic correlation. *Radiographics*. 2012;32(5):E175–E200.



See Also (Topic, Algorithm, Electronic Media Element)

- Central Retinal Artery Occlusion (CRVA)
- Central Retinal Venous Occlusion (CRVO)
- Retinal Detachment
- Visual Loss



CODES

ICD9

- 250.50 Diabetes with ophthalmic manifestations, type II or unspecified type, not stated as uncontrolled
- 362.16 Retinal neovascularization NOS
- 379.23 Vitreous hemorrhage

ICD10

- E13.39 Oth diabetes mellitus w oth diabetic ophthalmic complication
- H35.059 Retinal neovascularization, unspecified, unspecified eye
- H43.10 Vitreous hemorrhage, unspecified eye

VOLVULUS

Ronald E. Kim

BASICS

DESCRIPTION

- Axial twist of a portion of the GI tract around its mesentery causing partial or complete obstruction of the bowel
- Often associated with other GI abnormalities
- In pediatric setting, infants typically involved:
 - Abnormal embryonic development
- Can be precipitated by pathologic distention of the colon
- Blood supply may be compromised by venous congestion and eventual arterial inflow obstruction, leading to gangrene of the bowel and potential infarction

ETIOLOGY

- 3rd most common cause of colonic obstruction (10–15%) following tumor and diverticular disease
- Epidemiology:
 - 0–1 yo: 30%
 - 1–18 yo: 20%
 - Over 18 yo: 50%
- Often associated with other GI abnormalities
- Cecum (52%):
 - More common in young adults, < 50 yr old
 - Due to improper congenital fusion of the mesentery with the posterior parietal peritoneum, causing the cecum to be freely mobile in varying degrees
 - Associated with increased gas production (malabsorption and pseudo-obstruction)
 - Can be seen in pregnancy and after colonoscopy
- Sigmoid (43%):
 - More common in:
 - Elderly
 - Institutionalized
 - Chronic bowel motility disorders (Parkinson)
 - Psychiatric diseases (schizophrenia)
 - Due to redundant sigmoid colon with narrow mesenteric attachment
 - Associated with chronic constipation and concomitant laxative use
- Transverse colon and splenic flexure (5%)
- Gastric volvulus (rare) associated with diaphragmatic defects

Pediatric Considerations

- Midgut volvulus:
 - Due to congenital *malrotation* in which the midgut fails to rotate properly in utero as it enters the abdomen
 - Entire midgut from the descending duodenum to the transverse colon rotates around its mesenteric stalk, including the superior mesenteric artery
 - Common in neonates (80% < 1 mo old, often in 1st week; 6–20% > 1 yr old)
 - Males > females, 2:1
 - Sudden onset of bilious emesis (97%) with abdominal pain
 - May have previous episodes of feeding problems/bilious emesis
 - In children > 1 yr old, associated with failure to thrive, alleged intolerance to feedings, chronic intermittent vomiting, bloody diarrhea
 - Constipation
 - Mild distention, since obstruction higher in GI tract
 - May not appear toxic based on degree of ischemia



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Infants: Vomiting in 90%:
 - May be bilious
- Older children and adults: Variable and often insidious:
 - 80% with chronic symptoms; weeks to months to years
- Bowel obstruction secondary to volvulus:
 - Colicky, cramping abdominal pain (90%)
 - Abdominal distention (80%)
 - Obstipation (60%)
 - Nausea and vomiting (28%)
- Cecal volvulus:
 - Highly variable; intermittent episodes to sudden onset of pain and distention
- Sigmoid volvulus:
 - Vomiting uncommon
 - More insidious onset
 - Abdominal pain/distention, nausea, and constipation
- Gastric volvulus:
 - Triad of Borchardt: Severe epigastric distension, intractable retching, inability to pass nasogastric tube (30% of patients)

Physical-Exam

- Presence of gangrenous bowel:
 - Increased pain
 - Peritoneal signs: Guarding, rebound, and rigidity
 - Fever
 - Blood on digital rectal exam
 - Tachycardia and hypovolemia
- Cecal volvulus:
 - Distended abdomen
 - Often a palpable mass in the left upper quadrant/midabdomen

Pediatric Considerations

- Child will appear well with normal exam early in clinical course
- 70% present with chronic symptoms
- 40% of neonates with bilious vomiting will require a surgical intervention
- Hematochezia, abdominal distention or pain, and shock indicate ischemia/necrosis

ESSENTIAL WORKUP

- CBC, BMP, UA
- Plain abdominal radiograph
- Upper GI series (best initial exam for children)
- CT abdomen/pelvis with IV contrast (optimal for adults)
- Barium enema
- US

DIAGNOSIS TESTS & INTERPRETATION

Lab

- May give clues as to the presence of gangrenous bowel, but normal lab values do not exclude the diagnosis
- CBC:
 - Leukocytosis (WBC > 20,000) suggests strangulation with infection/peritonitis.
- Electrolytes, BUN, creatinine, glucose:
 - Anion gap acidosis due to lactic acidosis
 - Prerenal azotemia due to dehydration
- Urinalysis:
 - Elevated specific gravity and ketones

Imaging

- Plain abdominal radiograph:
 - Suggestive but often inconclusive
 - Diagnostic finding present in < 70% of cases

- Sigmoid volvulus: Inverted U-shaped loop of dilated colon arising from the pelvis
- Cecal volvulus—dilated and displaced:
 - Cecum in the left abdomen (kidney shaped), often with dilated loops of small bowel
- CT scan:
 - “Whirl” sign in cecal volvulus
 - May be useful in sigmoid volvulus to determine extent of obstruction
- Upper GI series (best for duodenum, but operator dependent):
 - Abrupt ending or corkscrew tapering of contrast seen (75%)
 - Subtle findings (25%)
- Barium enema:
 - “Bird’s beak” deformity at the site of torsion
 - Perform cautiously because of perforation risk
 - Beware of false positives with infants who normally have inadequately fixed cecums
- US (specific but not sensitive):
 - Abnormal position of the superior mesenteric vein (anterior or left of SMA)
 - “Whirlpool” sign of volvulus: Vessels twirled around the base of the mesentery
 - 3rd part of duodenum not in normal retromesenteric position (between mesenteric artery and aorta)

Pediatric Considerations

- Diagnosis of midgut volvulus:
 - Duodenum lies entirely to the right of the spine on plain films
 - “Double-bubble” sign on an upright film due to distended stomach and proximal duodenal loop
 - Established by upper GI swallow: Coiled spring/corkscrew appearance of jejunum in the right upper quadrant
 - Plain x-ray normal or equivocal in 20% of cases

ALERT

- Evaluate any child with signs/symptoms of obstruction (including bilious vomiting and abdominal pain) for malrotation, even if he or she appears nontoxic
- Delay in diagnosis > 1–2 hr results in gangrenous bowel, necessitating large resection and leading to permanent parenteral nutrition with its associated complications

Diagnostic Procedures/Surgery

- Laparoscopy:
 - Useful when diagnostic imaging equivocal
 - Can differentiate congenital malrotation from volvulus

DIFFERENTIAL DIAGNOSIS

- Obstruction due to colonic tumor or diverticulitis
- Small bowel obstruction
- Ileus
- Intussusception
- Appendicitis
- Pelvic inflammatory disease and salpingitis, especially for cecal volvuli
- Ovarian torsion
- Perforated viscus
- Cyclic vomiting syndrome

Pediatric Considerations

- Meconium ileus
- Hirschsprung disease
- Duodenal atresia
- Meckel diverticulum
- Necrotizing enterocolitis (especially premature infants)
- Intussusception
- Appendicitis
- Medical conditions:
 - Colic
 - Henoch–Schönlein purpura
 - Inborn errors of metabolism
 - Trauma
 - Gastroesophageal reflux
 - Pyelonephritis
 - Meningitis



TREATMENT

PRE HOSPITAL

- Establish IV assess
- NPO

INITIAL STABILIZATION/THERAPY

- ABCs
- Aggressive fluid resuscitation with 0.9% NS bolus of 20 mL/kg (peds) or 2 L bolus (adult)
- NGT

ED TREATMENT/PROCEDURES

- Obtain surgical and/or GI consultation

- NPO
- Correct hypovolemia and electrolyte abnormalities
- Preoperative broad-spectrum antibiotics if suspected sepsis or perforation

Definitive Therapy

Sigmoid Volvulus

- Nontoxic patient:
 - Reduce volvulus nonoperatively with sigmoidoscopy:
 - 80–95% successful
 - 60% recurrence (within hours to weeks)
 - Follow with elective sigmoid resection and primary anastomosis (< 3% recurrence)
- Toxic patient:
 - Emergent resection of sigmoid and any gangrenous bowel, with placement of end colostomy
- Endoscopic decompression with rectal tube placement:
 - Successful in 78% of patients with sigmoid volvulus; less effective for cecal volvulus
 - Recurrence is common
 - Elective surgical treatment after endoscopic detorsion

Cecal Volvulus

- Emergent operative reduction followed by colectomy and primary anastomosis (preferred), or cecopexy if the cecum is still viable (higher recurrence)

Pediatric Considerations

- Laparotomy within 1–2 hr to reduce risk for ischemia
- Surgical detorsion of bowel with resection of gangrenous bowel and a Ladd procedure is performed to prevent recurrent volvulus

MEDICATION

- Ampicillin sulbactam (Unasyn): 3 g (peds: 100–200 mg/kg/24 h) IV q6h
- Cefoxitin (Mefoxin): 2 g (peds: 80–160 mg/kg/24 h) IV q6h
- Ceftriaxone 1–2 g IV q12–24h (peds: 50–75 mg/kg/d q12–24h) AND metronidazole 500 mg IV q8h (peds: 30 mg/kg/24 h q6h)
- Piperacillin–tazobactam 3.375–4 g IV q4–6h (peds: 200–300 mg/kg/d of piperacillin component q6–8h)



FOLLOW-UP

DISPOSITION

Admission Criteria

Admit with a surgical consult all suspected of having a volvulus.

Discharge Criteria

None

Issues for Referral

- Surgical consultation necessary
- Atypical malrotation: Asymptomatic or symptoms of gastroesophageal reflux:
 - _ Close observation with repeat contrast study
 - _ Defer surgery

FOLLOW-UP RECOMMENDATIONS

Surgical follow-up postoperatively

PEARLS AND PITFALLS

- Consider volvulus in any child < 1 mo old presenting with vomiting:
 - _ Bilious vomiting is due to mechanical intestinal obstruction until proven otherwise
- Delayed diagnosis leads to increased morbidity, more often with adults than children:
 - _ 70% adults not diagnosed until > 6 mo from initial presentation; most present with chronic abdominal symptoms
 - _ If gangrene present, mortality = 25–80%
- Operative repair for all adult patients
- Upper GI contrast series is the best initial test for children
- CT abdomen/pelvis is preferable for adults

ADDITIONAL READING

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- Madiba TE, Thomson SR. The management of cecal volvulus. *Dis Colon Rectum.* 2002;45(2):264–267.
- Nehra D, Goldstein AM. Intestinal malrotation: Varied clinical presentation from infancy through adulthood. *Surgery.* 2011;149:386–393.



See Also (Topic, Algorithm, Electronic Media Element)

Bowel Obstruction



CODES

ICD9

- 537.89 Other specified disorders of stomach and duodenum
- 560.2 Volvulus
- 751.5 Other anomalies of intestine

ICD10

- K31.89 Other diseases of stomach and duodenum
- K56.2 Volvulus
- Q43.8 Other specified congenital malformations of intestine

VOMITING, ADULT

Scott G. Weiner



BASICS

DESCRIPTION

- 3 phases:
 - Nausea: Unpleasant sensation prior to vomiting
 - Retching: Rhythmic contractions of diaphragm, abdominal muscles, intercostals that bring gastric contents up the esophagus
 - Vomiting: Forceful retrograde expulsion of gastric contents through the mouth
- Vomiting center in medulla coordinates vomiting through vagus, phrenic, spinal nerves
- Irritated by impulses from the GI tract, pharynx, vestibular system, heart, genitalia, or via stimulation of chemoreceptor trigger zone (CTZ) in the area postrema of the brain by medications, toxins, or hormones in circulation
- CTZ response mediated by dopamine D₂, serotonin (5-HT₃), cholinergic, and histamine receptors:
 - Medications providing symptomatic treatment of vomiting antagonize these receptors

ETIOLOGY

- GI:
 - Appendicitis
 - Boerhaave syndrome
 - Bowel obstruction or ischemia
 - Cholecystitis, biliary colic
 - Gastric outlet obstruction, gastroparesis
 - Gastritis
 - Gastroenteritis (e.g., infectious)
 - GI bleeding
 - Hepatitis
 - Inflammatory bowel disease
 - Pancreatitis
 - Peptic ulcer disease, dyspepsia
 - Perforated viscus
 - Peritonitis
- Neurologic:
 - Elevated intracranial pressure (ICP)
 - Intracranial blood

- Labyrinthitis, vertigo
- Meningitis
- Migraine
- Stroke
- Tumor
- Endocrine:
 - Adrenal insufficiency
 - Diabetic ketoacidosis (DKA)
 - Hypoparathyroid, hyperparathyroid
 - Hypothyroid, hyperthyroid
 - Uremia
- Pregnancy:
 - Hyperemesis gravidarum
 - Nausea/vomiting of pregnancy
- Drug toxicity:
 - Acetaminophen
 - Aspirin
 - Digoxin
 - Theophylline
- Therapeutic medication use:
 - Antibiotics
 - Aspirin
 - Chemotherapy
 - Ibuprofen
- Drugs of abuse:
 - Narcotics/narcotic withdrawal
 - Alcohols
- Genitourinary:
 - Gonadal torsion
 - Nephrolithiasis
 - UTI/pyelonephritis
- Miscellaneous:
 - Carbon monoxide or organophosphate poisoning
 - Electrolyte disorders
 - Glaucoma
 - Motion sickness
 - Myocardial infarction/ischemia (MI)
 - Pain
 - Post-procedural (after anesthesia)
 - Self-induced (eating disorders)
 - Sepsis/shock

DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Symptom duration, frequency, severity:
 - Acute, recurrent, chronic, cyclic
- Characteristics of vomiting: Timing, description, content of vomitus
- Associated symptoms: Pain, fever, diarrhea, neurologic
- Past surgical or GI history
- Medication and drugs use
- Last menstrual period
- Complete past medical history

Physical-Exam

- Vital signs:
 - Fever: Appendicitis, gastroenteritis, cholecystitis, hepatitis, bowel perforation
 - Tachycardia: Dehydration
- Head, ears, eyes, nose, throat:
 - Abnormal anterior chamber: Glaucoma
 - Dry mucous membranes: Dehydration
 - Nystagmus: Labyrinthitis, stroke, tumor, intracranial hemorrhage
 - Papilledema: Elevated ICP
- Abdomen:
 - Blood in stool or emesis: Peptic ulcer, Mallory–Weiss tear
 - Decreased bowel sounds: Ileus
 - Distention, high-pitched bowel sounds, scars or hernias: Intestinal obstruction
 - Pain: Appendicitis, cholecystitis, pancreatitis, perforated viscus, ovarian torsion
 - Testicular pain: Torsion
- Neurologic:
 - Abnormal mental status, cerebellar test abnormalities, cranial nerve abnormalities: CNS pathology

ESSENTIAL WORKUP

The workup is aimed at determining the underlying cause of vomiting and excluding dangerous sequelae

DIAGNOSIS TESTS & NTERPRETATION

Lab

- **CBC:**
 - Elevated WBC: Infectious process (e.g., appendicitis, gastroenteritis)
 - Elevated hematocrit: Dehydration
 - Decreased hematocrit: GI bleed from ulcer
- **Electrolytes/renal function:**
 - Prolonged vomiting may cause hyponatremia, hypokalemia.
 - BUN/creatinine ratio > 20 may indicate dehydration.
 - Renal insult may occur from dehydration
- **Liver/pancreatic function tests:**
 - Amylase/lipase elevation: Pancreatitis
 - AST/ALT elevation: Hepatitis
 - Alkaline phosphatase elevation: Cholecystic etiology
- **Urine analysis:**
 - WBC, nitrites, leukocyte esterase, bacteria: UTI
 - Ketones: Dehydration, DKA
 - Pregnancy test in women of childbearing age
- **Toxicology screen/drug levels:**
 - For suspected drug toxicity or overdose

Imaging

- **Abdominal series (kidney, ureter, bladder/upright):**
 - Suspected bowel obstruction or perforated viscus
- **CT abdomen/pelvis:**
 - Suspected appendicitis, obstruction, nephrolithiasis
- **CT/MRI head:**
 - Suspected intracranial etiology
- **US:**
 - Suspected biliary disease, gonadal torsion, nephrolithiasis

Diagnostic Procedures/Surgery

- **EKG:**
 - Suspected MI
- **Endoscopy:**
 - Peptic ulcer disease leading to significant GI bleed



TREATMENT

PRE HOSPITAL

- Aimed at stabilizing patient until arrival in the ED, where the workup of underlying cause of vomiting can proceed
- Placement of IV, oxygen, cardiac monitor

- Begin administration of isotonic fluids in suspected dehydration
- Fingerstick glucose in mental status change
- Specific protocols may permit antiemetics for motion sickness or other etiologies of vomiting

INITIAL STABILIZATION/THERAPY

- Address ABCs
- Urgent fluid resuscitation if vomiting has led to hypovolemic shock
- Urgent antiemetic therapy for patient comfort
- Urgent analgesic therapy if indicated

ED TREATMENT/PROCEDURES

- 3 principles of ED treatment:
 - Correct fluid, electrolyte, and nutritional deficiencies as a result of vomiting
 - Identify and treat underlying cause
 - Suppress or eliminate symptoms.
- Antibiotics if indicated: UTI, appendicitis, bacterial gastroenteritis
- Medications:
 - Serotonin antagonists often 1st line treatment:
 - Ondansetron, dolasetron, granisetron
 - Useful in chemotherapy-induced nausea
 - Ondansetron available as an oral dissolving tablet for patients who cannot tolerate pills
 - Can cause QT prolongation
 - Dopamine D₂ antagonists also useful in most types of nausea:
 - Prochlorperazine, promethazine, metoclopramide, droperidol
 - Side effects (e.g., akathisia, dystonia) more common than in serotonin antagonists
 - Note black box warnings on use of droperidol (potential QT prolongation and/or torsades de pointes) and promethazine (tissue injury with IV administration)
 - Anticholinergic and antihistamine agents useful in labyrinthitis, positional vertigo, and motion sickness:
 - Meclizine, diphenhydramine, scopolamine
 - Benzodiazepines and glucocorticoids have mild antiemetic properties and can be used as adjuncts
- Consultation with other specialties (e.g., surgery, gynecology, gastroenterology) depending on underlying etiology

MEDICATION

- Diphenhydramine: 25–50 mg IM/IV/PO
- Dolasetron: 12.5 mg IV
- Droperidol: 0.625–1.25 mg IM/IV

- Granisetron: 1 mg IV or 2 mg PO
- Hydroxyzine: 25–100 mg IM
- Meclizine: 25–50 mg PO
- Metoclopramide: 10 mg IM/IV/PO
- Ondansetron: 4–8 mg IM/IV/PO
- Prochlorperazine: 5–10 mg IM/IV/PO or 25 mg PR
- Promethazine: 12.5–25 mg PO/PR/deep IM
- Scopolamine: 1.5 mg patch applied behind the ear 4 hr prior to travel

Geriatric Considerations

- Dopamine-antagonizing antiemetics have potential cardiac side effects:
 - The doses of these medications should be reduced in the elderly
- Serotonin antagonists are safer in this population:
 - Still consider using lower doses and obtaining an EKG to detect QT prolongation prior to administration

Pediatric Considerations

- Vomiting in children can result from a host of other diagnoses, e.g., structural/anatomical disorders, infections, and metabolic disorders:
 - Workup and treatment may therefore be different in children

Pregnancy Considerations

- Vomiting occurs in >25% of pregnancies
- Dopamine D₂ antagonists (e.g., promethazine, chlorpromazine, metoclopramide) or serotonin antagonists (e.g., ondansetron, granisetron) most commonly used

First Line

- Serotonin antagonists
- Dopamine D₂ antagonists

Second Line

- Anticholinergics
- Antihistamines
- Benzodiazepines
- Glucocorticoids



FOLLOW-UP

DISPOSITION

Admission Criteria

- Depends on underlying pathology
- Significant underlying disease or symptoms necessitating close observation or surgical procedure
- Uncontrolled emesis resulting in inability to tolerate food or liquids by mouth
- Severe dehydration requiring continued IV fluids
- Significant electrolyte disturbances
- Unknown etiology of vomiting with inadequate outpatient follow-up

Discharge Criteria

- Significant underlying pathology is excluded
- Patient is sufficiently hydrated
- Emesis is controlled
- Close follow-up is arranged (preferably within 24–36 hr)

FOLLOW-UP RECOMMENDATIONS

- All patients who are unable to tolerate fluids at home should return to the ED
- Patients in whom the etiology of vomiting is unknown or who had electrolyte disturbances should follow-up

PEARLS AND PITFALLS

- Vomiting is a symptom and not a diagnosis:
 - It is important to be familiar with the broad differential diagnoses and exclude dangerous etiologies
- Many antiemetics have notable side effects, ranging from dystonia to cardiac arrhythmias.
 - Know contraindications and treatment of adverse reactions before using these agents
- Oral dissolving tablets and suppositories useful to avoid IV and for home care

ADDITIONAL READING

- Longstreth GF. Approach to the adult with nausea and vomiting. Up to Date on-line text. www.uptodate.com. March 2012.
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See Also (Topic, Algorithm, Electronic Media Element)

Vomiting, Pediatric



CODES

ICD9

- 643.00 Mild hyperemesis gravidarum, unspecified as to episode of care or not applicable
- 787.01 Nausea with vomiting
- 787.03 Vomiting alone

ICD10

- O21.0 Mild hyperemesis gravidarum
- R11.10 Vomiting, unspecified
- R11.2 Nausea with vomiting, unspecified

VOMITING, CYCLIC

Rosaura Fernández



BASICS

DESCRIPTION

- A chronic, idiopathic disorder characterized by recurrent, discrete episodes of disabling nausea and vomiting separated by symptom-free intervals lasting a few days to months
- Adult population – average age of diagnosis is 31:
 - Average age of onset is 21
- Pediatric population – average age of diagnosis is 5
- General characteristics:
 - Phase 1: Interepisodic phase:
 - Symptom free
 - Phase 2: Prodrome:
 - Varying intensity of nausea and diaphoresis
 - Phase 3: Emetic phase:
 - Intense nausea/vomiting/retching/dry heaving up to 7 days
 - Phase 4: Recovery phase:
 - Improvement of nausea and tolerance of PO intake

EPIDEMIOLOGY

Incidence and Prevalence Estimates

- True incidence and prevalence in adult general population unknown due to limited data and research, increasing recognition in syndrome
- In pediatric population, cyclic vomiting syndrome affects 0.04–2% of population with estimated new cases 3/100,000 annually

ETIOLOGY

- Etiology unknown
- Pathophysiology is also unknown and is under research:
 - Limited research suggests multifactorial factors such as autonomic, central, and environmental to be involved



DIAGNOSIS

SIGNS AND SYMPTOMS

Commonly present to ED with unexplained onset of nausea/vomiting and abdominal pain.

History

- History of similar prior episodes
- No preceding trigger identified at times but typically when asked specifically may identify
- Will complain of abdominal pain, usually epigastric

Physical-Exam

May have benign physical exam or various findings based on degree of dehydration:

- Normal vital signs or abnormal vital signs demonstrating:
 - Tachycardia
 - Hypotension (including orthostatic hypotension)
 - Tachypnea
- Cool extremities and/or delayed (> 2 s) capillary refill indicating shock
- Varying degrees of consciousness:
 - Alert, lethargic, or obtunded
- Dry mucous membranes:
 - Sunken eyes
 - Dry/sticky or cracked mouth
- Poor skin turgor
- Oliguria or anuria

Pediatric Considerations

May present with above in addition to refusal to eat/drink, reduced or lack of tear production, sunken fontanel, reduced or absent urine output (reduced wet diapers)

ESSENTIAL WORKUP

Must rule out other potentially serious conditions (see Differential Diagnosis)

DIAGNOSIS TESTS & INTERPRETATION

- Perform necessary exam and lab or radiographic tests necessary to rule out other conditions with similar presenting signs and symptoms
- Cyclic vomiting has no specific diagnostic feature nor specific biochemical marker
- Extensive list of other diagnostic possibilities
- Diagnosis of adult cyclic vomiting is based on Rome III criteria:
 - Stereotypical episodes of vomiting regarding onset (acute) and duration (< 1 wk)
 - At least 3 episodes in the past year
 - Absence of nausea/vomiting between episodes

Lab

- CBC
- Electrolytes, BUN/Cr, glucose
- Liver enzyme, liver profile

- Lipase
- Lactate
- Urinalysis
- Pregnancy test
- Toxicology screen/drug levels:
 - Acetaminophen
 - Salicylic acid
 - Alcohols:
 - Ethanol, isopropanol, methanol, ethylene glycol
 - Digoxin

Imaging

Atypical severity or atypical episodes should raise suspicion of underlying disorder not due to cyclic vomiting:

- Tailor imaging to individual patient presentation

Diagnostic Procedures/Surgery

Outpatient gastric emptying study should be done to r/o gastroparesis or other gut motility disorders as cause of frequent emesis.

DIFFERENTIAL DIAGNOSIS

- Infectious:
 - Appendicitis
 - Pyelonephritis
 - Pneumonia
 - Cholecystitis
- Metabolic/endocrine:
 - Renal failure/uremia
 - Electrolyte disorder
 - Diabetic ketoacidosis
 - Thyroid disorder
 - Adrenal insufficiency
 - Pheochromocytoma
 - Pregnancy or hyperemesis gravidarum
- Renal:
 - Nephroureterolithiasis
 - UVJ obstruction/hydronephrosis
- GI:
 - Gastroparesis
 - Bowel obstruction
 - Peptic ulcer disease
 - Cholelithiasis
 - Pancreatitis

- Malrotation with volvulus
- Inflammatory bowel disease
- CNS:
 - Intracranial hemorrhage
 - Brain tumor
 - Hydrocephalus
 - CVA
- Cardiovascular:
 - Anginal equivalent
 - STEMI/NSTEMI
- Toxicology (examples):
 - Cannabinoid hyperemesis
 - Mushroom toxicity:
 - >100 species
 - Acute alcohol/toxic alcohol ingestion:
 - Ethanol, isopropanol, methanol, ethylene glycol
 - Alcohol withdrawal
 - Heroin withdrawal
 - Any acute/subacute ingestion; consider:
 - Acetaminophen
 - Salicylic acid
 - Digoxin
- Psychiatric:
 - Self induced
 - Bulimia
 - Anorexia
 - Anxiety

Pediatric Considerations

Munchausen by proxy



TREATMENT

PRE HOSPITAL

- Address airway/breathing/circulation
- Initiate IV, oxygen (if indicated), place on cardiac monitor
- Start IV fluids if presenting with vomiting and/or abnormal vital signs

INITIAL STABILIZATION/THERAPY

- Address airway/breathing/circulation
- Continue IV/O₂ (as indicated), cardiac monitor
- Address abnormal vital signs specifically hypotension and tachycardia:

- Adults: 500 to 1000 mL bolus 0.9% NS
- Pediatric: 20 mL/kg bolus 0.9% NS

ED TREATMENT/PROCEDURES

- Supportive care in acute phase
- Abort emetic phase of nausea/vomiting with antiemetics
- IV 0.9 normal saline:
 - Add dextrose after initial boluses
- Correct electrolyte abnormalities
- Treat pain with analgesics
- Provide light sedation for very symptomatic patients
- Administer gastric acid suppressants:
 - H₂ receptor antagonist
 - Proton pump inhibitors
- Consider antimigraine triptans

MEDICATION

Antiemetics

- Ondansetron 4–8 mg IV/PO/ODT q4–8h prn
- Metoclopramide 10 mg IV/IM q2–3h prn 4–8 mg IV/PO/ODT q4–8h prn
- Prochlorperazine 5–10 mg IV/PO/IM (peds: 0.1 mg/kg/dose PO/IM/PR) q6–8h prn
- Promethazine 12.5/25 mg PO/IM/PR q4–6h (IV use common but not approved) (peds: 0.25–1 mg/kg PO/IM/PR q4–6h prn if > 2 yr)

Pain/Sedation

- Ketorolac 15–30 mg IV
- Lorazepam 0.5–1 mg IV/IM/PO
- Morphine 0.1 mg/kg IV
- Sumatriptan 4–6 mg SC-repeat in 1 hr prn

Gastric Acid Suppressants

- Cimetidine (H₂-blocker): 800 mg PO at bedtime nightly (peds: 20–40 mg/kg/24 h)
- Famotidine 20 mg IV q12h
- Pantoprazole 40 mg IV q24h
- Ranitidine 50 mg IV/IM q8h



FOLLOW-UP

DISPOSITION

Admission Criteria

- Vital signs/lab or physical exam findings suggestive of moderate to severe dehydration
- Inability to tolerate PO fluids

Discharge Criteria

- Stable vital signs
- Cessation of vomiting and pain control
- Able to tolerate PO fluids and keep self hydrated

Issues for Referral

- GI consult for further outpatient workup when symptom free
- Consider additional referral to specialist managing this syndrome

FOLLOW-UP RECOMMENDATIONS

- Prophylaxis:
 - Identification and avoidance of triggers:
 - Emotional stress, poor sleep, fasting, illness, marijuana, specific foods (chocolate, cheeses, etc.)
 - Management of coexisting conditions:
 - Migraine headaches, psychiatric disorders, chronic narcotic, and marijuana use
 - Medications (outpatient-in active research):
 - Tricyclic antidepressants (amitriptyline)
 - Propranolol
 - Coenzyme Q-10
 - Antihistamines
 - Antianxiety medications

PEARLS AND PITFALLS

- Obtain good history about prior cyclic episodes and similarities to prior episodes
- Manage active coexisting conditions if applicable
- Exclude other disorders with similar presentations of nausea/vomiting/abdominal pain

ADDITIONAL READING

- Abell TL, Adams KA, Boles RG, et al. Cyclic vomiting syndrome in adults. *Neurogastroenterol Motil.* 2008;20:269–284.
- Fleisher DR, Gornowicz B, Adam K, et al. Cyclic Vomiting Syndrome in 41 adults: The illness, the patients, and problems of management. *BMC Med.* 2005;3:20.
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CODES

ICD9

- 346.20 Variants of migraine, not elsewhere classified, without mention of intractable migraine without mention of status migrainosus
- 346.21 Variants of migraine, not elsewhere classified, with intractable migraine, so stated, without mention of status migrainosus
- 536.2 Persistent vomiting

ICD10

- G43.A Cyclical vomiting
- G43.A0 Cyclical vomiting, not intractable
- G43.A1 Cyclical vomiting, intractable

VOMITING, PEDIATRIC

Christina M. Conrad



BASICS

DESCRIPTION

- Forceful, coordinated act of expelling gastric contents through the mouth; characterized by nausea, retching, and emesis; no gastric contents are expelled during retching.
- Emesis results from sustained contraction of abdominal muscles and diaphragm; at the same time, the pylorus and antrum contract.

ETIOLOGY

Mechanism:

- GI/mechanical: Gastroesophageal reflux (GER), meconium ileus, necrotizing enterocolitis, hypertrophic pyloric stenosis, intussusception, malrotation with midgut volvulus, Hirschsprung disease, congenital obstructions (atresias, stenoses, and webs), hernia, foreign body/bezoar, pancreatitis, appendicitis, paralytic ileus
- Metabolic/endocrine: Inborn errors of metabolism (amino acidurias, fatty acid oxidation disorders, urea cycle defects), uremia, diabetic ketoacidosis, congenital adrenal hyperplasia, kernicterus
- Neurologic: CNS bleeding (often due to trauma), tumor, hydrocephalus
- Infectious: Otitis media, UTI, pneumonia, sepsis, gastroenteritis, meningitis/encephalitis
- Feeding problems: Chalasias, improper technique (overfeeding, improper position), milk allergy
- Other: Toxicologic, nonaccidental trauma, pregnancy



DIAGNOSIS

SIGNS AND SYMPTOMS

- General:
 - Appearance variable depending on the underlying cause
 - Signs of dehydration, including tachycardia, tachypnea, pallor, decreased perfusion, and shock
 - Altered mental status may occur secondary to shock, hypoglycemia, or extra-abdominal conditions (sepsis, inborn error of metabolism, increased intracranial pressure, toxicologic poisoning).
- Vomiting characteristics:
 - Assess color, composition, onset, progression, and relationship to intake and position.

- Nonbilious emesis is caused by a lesion proximal to the pylorus.
- Bilious (green) emesis indicates obstruction below the duodenal ampulla of Vater; in infants, bilious emesis is associated with a more serious underlying condition (malrotation, volvulus, intussusception, bowel obstruction); may also be due to adynamic ileus or sepsis.
- Bloody emesis (hematemesis) involves a lesion proximal to the ligament of Treitz; bright red bloody emesis has little or no contact with gastric juices due to an active bleeding site at or above cardia.
- “Coffee-grounds” emesis results from reduction of heme by gastric secretions.
- Feculent odor suggests lower obstruction or peritonitis.
- Undigested food in emesis suggests an esophageal lesion or one at or above the cardia.
- GER: Begins shortly after birth, remains relatively constant, usually with normal weight gain.
- Hypertrophic pyloric stenosis: Begins insidiously at 2–6 wk of age and progresses, becoming increasingly forceful (projectile) after feedings.
- Obstruction and/or ischemic bowel (malrotation with midgut volvulus, intussusception, necrotizing enterocolitis): Sudden onset associated with rapid progression to appearing ill out of proportion to the duration of illness; abdomen distended and tender.
- Abdominal:
 - Distention suggests obstruction.
 - Peritoneal signs suggest inflammation and possible perforation.
- Complications:
 - Aspiration
 - Mallory–Weiss tear
 - Boerhaave syndrome

History

- Constitutional:
 - Fever
- Vomiting characteristics:
 - Timing, duration
 - Bilious?
 - Bloody?
- Associated symptoms:
 - Diarrhea
 - Anorexia
 - Abdominal pain
 - Dysuria
 - Inguinal swelling
- PMHx:

- History of similar
- Past surgical history

Physical-Exam

- General:
 - General appearance, vital signs, hydration status
- Cardiovascular:
 - Quality heart tones
 - Pulses, perfusion
- Abdominal:
 - Tenderness, distention, mass
 - Bowel sounds
- Genitourinary:
 - Scrotal swelling, tenderness, mass
- Rectal:
 - Presence of blood, mass, tenderness

ESSENTIAL WORKUP

Exclude life-threatening causes of vomiting.

DIAGNOSIS TESTS & NTERPRETATION

Lab

- As indicated by history and physical exam and consideration of differential:
 - Metabolic assessment (glucose, electrolytes)
 - Infection assessment (CBC, culture—urine)
 - Pregnancy tests for females of childbearing age

Imaging

- As indicated by differential considerations
- Abdominal radiographs (flat plate, upright, and decubitus) helpful for evaluation of obstruction or perforation
- Pelvic and abdominal US for evaluation of hypertrophic pyloric stenosis, intussusception, appendicitis as well as pelvic or scrotal pathology
- Abdominal CT scan helpful for evaluation of appendicitis, mass/tumor often requiring contrast

Diagnostic Procedures/Surgery

Nasogastric tube:

- Location, character, and severity of gastric bleeding

DIFFERENTIAL DIAGNOSIS

- Neonate/infant:

- GI/mechanical: GER, meconium ileus, necrotizing enterocolitis, hypertrophic pyloric stenosis, intussusception, malrotation with midgut volvulus, Hirschsprung disease, congenital obstructions (atresias, duplications, imperforate anus. stenoses, and webs), hernia, foreign body/bezoar, paralytic ileus
- Metabolic/endocrine: Inborn errors of metabolism (amino acidurias, fatty acid oxidation disorders, urea cycle defects), uremia, congenital adrenal hyperplasia, kernicterus
- Neurologic: CNS bleeding (often due to trauma), tumor, hydrocephalus
- Infectious: Otitis media, UTI, pneumonia, sepsis, pertussis, meningitis/encephalitis
- Feeding problems: Chhalasia, improper technique (overfeeding, improper position), milk allergy
- Other: Toxicologic, nonaccidental trauma
- Child/adolescent:
 - GI: Gastroenteritis, obstruction (hernia, adhesions, intussusception, foreign body, bezoar), pancreatitis, appendicitis, peptic ulcer, peritonitis, paralytic ileus, trauma (duodenal hematoma)
 - Metabolic/endocrine: Diabetic ketoacidosis, uremia, adrenal insufficiency
 - Infectious: Gastroenteritis, UTI, sinusitis, upper respiratory infection, sepsis, meningitis, encephalitis, pneumonia, hepatitis
 - Neurologic: CNS mass/tumor, CNS bleeding (often due to trauma), cerebral edema, concussion, migraine
 - Other: Toxicologic, (nonaccidental) trauma, pregnancy, bulimia



TREATMENT

PRE HOSPITAL

Not applicable

INITIAL STABILIZATION/THERAPY

- Fluid resuscitation with 0.9% NS IV; caution if concern about increased intracranial pressure.
- Determine bedside fingerstick glucose.

ED TREATMENT/PROCEDURES

- Continue fluid resuscitation and correction of electrolyte imbalance if present.
- Decompress stomach with nasogastric or orogastric tube if abdomen distended or vomiting persistent.
- Continue evaluation for underlying cause.
- Consider antiemetic medications.
- Surgical consultation if acute abdomen; antibiotics if peritonitis or other systemic

infection present

MEDICATION

Antiemetics may be helpful once the underlying cause of vomiting has been determined.

First Line

Ondansetron: 4–8 mg (peds: 0.1 mg/kg per dose) IV or PO q6h

Second Line

- Metoclopramide: 10 mg (peds: 0.1 mg/kg per dose) PO q6h
- Prochlorperazine: 2.5–5 mg (peds: 0.1 mg/kg per dose) IV, IM, or PR q6h
- Promethazine: 12.5–25 mg (peds: 0.25 mg/kg per dose) PO, PR, or IM q6h



FOLLOW-UP

DISPOSITION

Admission Criteria

- Unstable vital signs, including persistent tachycardia or other evidence of hypovolemia
- Serious etiologic condition or inability to exclude serious etiologic conditions
- Intractable vomiting or inability to take oral fluids
- Inadequate social situation or follow-up

Discharge Criteria

- Stable; able to tolerate oral fluids
- Benign etiology considered most likely and serious or potentially important etiologies excluded
- Parental understanding of instructions to advance clear liquids slowly and return for continued vomiting, abdominal distention, decreased urination, fever, lethargy, or unusual behavior

Issues for Referral

- Chronic or recurrent episodes of vomiting or abdominal pain:
 - Pediatric gastroenterology

FOLLOW-UP RECOMMENDATIONS

PCP in 1–2 days

PEARLS AND PITFALLS

- Determine presence or absence of bile or blood in emesis.

- Bilious vomiting in the neonate is an important anatomic abnormality such as malrotation until proven otherwise.
- Consider causes of vomiting other than just GI (see Differential Diagnosis).

ADDITIONAL READING

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- Hostetler MA. Gastrointestinal disorders. In: Marx JA, Hockerberger RS, Walls RM, et al., eds. *Emergency Medicine: Concepts and Clinical Practice.* 7th ed. St. Louis: Mosby; 2010:2168–2187.
- Pepper VK, Stanfill AB, Pearl RH. Diagnosis and management of pediatric appendicitis, intussusception and Meckel diverticulum. *Surg Clin North Am.* 2012;92(3):505–526.



CODES

ICD9

- 530.81 Esophageal reflux
- 787.03 Vomiting alone
- 787.04 Bilious emesis

ICD10

- K21.9 Gastro-esophageal reflux disease without esophagitis
- R11.10 Vomiting, unspecified
- R11.14 Bilious vomiting

VON WILLEBRAND DISEASE

Matthew A. Wheatley • Ryan A. Stroder



BASICS

DESCRIPTION

- Coagulopathy caused by deficiency or dysfunction of von Willebrand factor (vWF)
- vWF functions:
 - Mediates platelet–endothelial cell adhesion
 - Carrier protein for factor VIII
- Prevalence as high as 1–2% in the general population
- Genetics:
 - Most cases inherited—multiple genetic defects identified
 - Type 1—quantitative defect of vWF:
 - 70% of cases
 - Autosomal dominant
 - vWF deficiency results from decreased synthesis and increased clearance of protein.
 - Manifestation ranges from asymptomatic to moderate bleeding.
 - Type 2—qualitative defect of vWF:
 - 10–15% of cases
 - Divided into types 2A, 2B, 2M, 2N—all are autosomal dominant except 2N which is autosomal recessive.
 - Decrease in intermediate and high molecular-weight multimer
 - 2N—decreased binding to factor VIII
 - Leads to decreased levels of VIII and thus more serious coagulopathy
 - Type 3—absent or severe deficiency in amount of vWF:
 - Rare disease—1 per million cases
 - Autosomal recessive
 - Severe coagulopathy
 - vWD genetically associated with sickle cell disease, hemophilia A, factor XII deficiency, hereditary hemorrhagic telangiectasia, and thrombocytopenia

ETIOLOGY

- In addition to genetic causes, acquired forms exist.
- Multiple mechanisms:
 - vWF antibody production
 - Decreased synthesis
 - Proteolysis
 - Increased clearance from binding to tumor cells
- Seen in association with the following:

- Malignancy:
 - Wilms tumor
 - Multiple myeloma
 - Chronic lymphocytic leukemia
 - Non-Hodgkin lymphoma
 - Chronic myelogenous leukemia
 - Waldenstrom macroglobulinemia
 - Monoclonal gammopathy of uncertain significance
- Immunologic:
 - Systemic lupus erythematosus
 - Rheumatoid arthritis
- Medication induced:
 - Valproic acid
 - Ciprofloxacin
 - Hetastarch
 - Griseofulvin
- Miscellaneous:
 - Hypothyroidism
 - Uremia
 - Hemoglobinopathies
 - Cirrhosis
 - Congenital heart disease
 - Disseminated intravascular coagulation



DIAGNOSIS

SIGNS AND SYMPTOMS

- Symptoms vary depending on type of disease.
- Many type 1 and some type 2 are asymptomatic, severe type 2 and type 3 are symptomatic:
 - Easy bruising
 - Menorrhagia
 - Recurrent epistaxis
 - Gum bleeding
 - GI bleeding
 - Soft-tissue bleeds and hemarthroses
 - Prolonged or excessive procedural bleeding
 - Postoperative hemorrhage

History

- Most often diagnosed in pediatric and adolescent populations
- Family history

- Minor/moderate recurrent mucosal bleeding most common historical clue
- Heavy menses

Physical-Exam

- Most will have normal exam
- Multiple large bruises
- Deep-tissue hematomas, hemarthroses

Pregnancy Considerations

- Pregnancy causes increased vWF levels in patients with types 1 and 2 disease
- Pregnancy, labor, and delivery are usually uncomplicated
- vWF levels fall quickly after delivery:
 - Patients may suffer postpartum bleeding 10–28 days after delivery

Pediatric Considerations

Always consider nonaccidental trauma in an infant or child presenting with bruising or bleeding of unknown cause

ESSENTIAL WORKUP

- Screen and refer for testing if historical concerns or consistent physical findings
- For type 1 diagnosis, patient must have significant mucocutaneous bleeding, lab confirmation, and family history of type 1 disease

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CBC: Normal platelet count and morphology
- PT: Normal
- PTT:
 - Mildly prolonged in 50%
 - Due to low factor VIII levels or coexistent factor deficiency
- Measurement of vWF level and activity:
 - vWF ristocetin cofactor activity (vWF:RCo):
 - Uses platelet agglutination to determine vWF function
 - vWF antigen—tests for vWF level in serum using rabbit antibodies
- Bleeding time:
 - May be normal in type 1 (50%); prolonged in types 2 and 3
 - Not specific and hard to reproduce; has fallen out of favor for diagnosis

DIFFERENTIAL DIAGNOSIS

- Hemophilia A, B
- Platelet defects
- Use of antiplatelet drugs—NSAIDs

- Platelet-type pseudo vWD
- Bernard–Soulier syndrome



TREATMENT

PRE HOSPITAL

Direct pressure for control of hemorrhage

INITIAL STABILIZATION/THERAPY

Resuscitation with crystalloid and packed RBCs as needed

ED TREATMENT/PROCEDURES

- As with all significant bleeding, apply direct pressure to site of bleeding
- 3 treatment strategies:
 - _ Increase endogenous vWF
 - _ Replacement of vWF
 - _ Agents that generally promote hemostasis but do not alter levels of vWF
- Desmopressin acetate (DDAVP):
 - _ Promotes release of vWF from endothelial cells, increases factor VIII levels
 - _ Maximal levels obtained at 30–60 min, with duration of 6–8 hr
 - _ Effective for type 1; variable effectiveness for type 2; not indicated for type 3
 - _ Patients may use intranasal spray at home before menses or minor procedures
- vWF replacement therapy:
 - _ Humate-P factor VIII concentrate with vWF:
 - Treated to reduce virus transmission risks
 - Indicated for type 3 vWD and severe bleeding in all types
 - Doses, length of treatment depend on severity of bleeding
 - Cryoprecipitate is no longer a treatment of choice as it carries risk of virus transmission. If no other treatments are available and patient having life-threatening hemorrhage, it can be used
- Antifibrinolytic therapy:
 - _ Aminocaproic acid (Amicar) and tranexamic acid (Cyklokapron)
 - _ Block plasmin formation to prevent clot degradation
- Topical agents—applied directly to bleeding site:
 - _ Gelfoam or Surgicel soaked in thrombin
 - _ Micronized collagen
 - _ Fibrin sealant
- Avoid antiplatelet agents

- Minor bleeding (epistaxis, oropharyngeal, soft tissue):
 - IV or intranasal desmopressin
- Major bleeding (intracranial, retroperitoneal):
 - Replace vWF and factor VIII so activity level is at least 100 IU/dL

Second Line

- Minor bleeding:
 - vWF concentrate:
 - Given if desmopressin is ineffective
 - Should be given in consultation with a hematologist
 - Aminocaproic acid or tranexamic acid:
 - For mild mucocutaneous bleeding

MEDICATION

- Aminocaproic acid: 50–60 mg/kg PO/IV q4–q6h
- Cryoprecipitate: 10–12 U initial dose or 2–4 bags/10 kg
- Desmopressin (DDAVP):
 - 0.3 µg/kg IV, max. 20 µg
 - 0.3 µg/kg SQ, max. 20 µg
 - 300 µg (1 spray each nostril) intranasal
 - Peds: < 50 kg—150 µg (1 spray in each nostril) intranasal
- Antihemophilic factor/vWF complex, human (Humate-P): 20–40 U/kg IV
- Tranexamic acid: 20–25 mg/kg PO, IV q8h
- Fresh frozen plasma (FFP)—10–20 mL/kg IV



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients with significant bleeding requiring further IV medical management
- Observation after major trauma for types 2 and 3 vWD
- Consider transferring patients with major bleeding events to a center with round-the-clock lab capability, and a care team that includes a hematologist and a surgeon skilled in management of bleeding disorders

Discharge Criteria

- Control of hemorrhage
- Adequate follow-up and access to medical therapy

FOLLOW-UP RECOMMENDATIONS

Hematology:

- Severe, difficult-to-manage bleeding
- Prior to elective/semielective procedures
- Definitive workup of suspected cases

PEARLS AND PITFALLS

Patients may not know their type of hemophilia:

- Consider FFP for the patient with unknown type of hemophilia in the setting of trauma or bleeding

ADDITIONAL READING

- Mannucci P. Treatment of von Willebrand disease. *N Engl J Med*. 2004;351:683–694.
- Nichols WL, Hultin MB, James AH, et al. von Willebrand disease (vWD): Evidence-based diagnosis and management guidelines, the National Heart, Lung and Blood Institute (NHLBI) Expert Panel report (USA). *Haemophilia*. 2008;14:171–232.
- Pacheco L, Costantine M, Saade G, et al. von Willebrand disease and pregnancy: A practical approach for the diagnosis and treatment. *Am J Obstet Gynecol*. 2010;203(3):194–200.
- Robertson J, Lillicrap D, James P. Von Willebrand disease. *Pediatr Clin North Am*. 2008;55(2):377–392.
- *The Diagnosis, Evaluation and Management of von Willebrand Disease*. National Heart, Lung and Blood Institute. Available at <http://www.nhlbi.nih.gov/guidelines/vwd/> Accessed January 14, 2013.



See Also (Topic, Algorithm, Electronic Media Element)

Hemophilia



CODES

ICD9

286.4 Von Willebrand's disease

ICD10

D68.0 Von Willebrand's disease

WARFARIN COMPLICATIONS

Molly C. Boyd

BASICS

DESCRIPTION

- Most commonly prescribed oral anticoagulant
- Inhibits vitamin K metabolism required for activation of factors II, VII, IX, and X
- Blocks the coagulation cascade's extrinsic system and common pathway
- Commonly used for venous thromboembolism and prevention of embolism with prosthetic heart valves or atrial fibrillation
- Adjustments based on the international normalization ratio (INR)
 - Typical therapeutic range 2–3
 - 2.5–3.5 for mechanical valves and antiphospholipid syndromes
- Contraindications include any condition in which the risk of hemorrhage or adverse reaction outweighs clinical benefit
 - Prior hypersensitivity
 - Skin reactions
 - Recent surgeries
 - Active or potential GI, intracerebral, or genitourinary bleeding
 - Fall risk

ETIOLOGY

- Bleeding complications:
 - 15% of patients/yr
 - 4.9% major bleeding events
 - Up to 0.8% fatal, most commonly intracranial hemorrhage (ICH)
 - Bleeding risk is directly related to INR
 - Increases dramatically above 4
- Risk factors for nontherapeutic INR:
 - Age >75 yr
 - Hypertension, cerebrovascular disease, severe heart disease
 - Diabetes, renal insufficiency
 - Alcoholism or liver disease
 - Hypermetabolic states, fever
 - Hyperthyroidism
 - Cancer
 - Collagen vascular disease
 - Hereditary warfarin resistance
 - Cytochrome P450 polymorphism

Common Interactions

Increase INR	Decrease INR
Multiple antibiotics	Carbamazepine
NSAIDs	Barbituates
Amiodorone	Rifampin
Propranolol	Haloperidol
Prednisone	St. John wort
Cimetidine	High vitamin K foods
Grapefruit, garlic	
Ginko biloba	

Pregnancy Considerations

- Pregnancy class X
- Crosses the placenta causing spontaneous abortion and birth defects



DIAGNOSIS

SIGNS AND SYMPTOMS

- Presentation may be occult or dramatic:
 - High index of suspicion required to detect potentially life-threatening complications
- Subtherapeutic/low INR: Breakthrough thrombosis
- Therapeutic and suprathereapeutic: GI, CNS, retroperitoneal bleeding
- Skin necrosis and limb gangrene:
 - Classic lesions of warfarin skin necrosis and limb gangrene begin on the 3rd–8th day of therapy
 - Capillary thrombosis in subcutaneous fat (skin necrosis) and obstruction of venous circulation of the limb (limb gangrene)
 - Often associated with protein C deficiency
 - Eschar in center differentiates lesions from ecchymosis
- Intentional overdose
 - May be asymptomatic
 - Superwarfarin (rat poison) can result in prolonged bleeding risk (months)
 - Follow serial INR
 - Do not start vitamin K empirically, may mask late development of INR elevation
 - Consider activated charcoal

ESSENTIAL WORKUP

- Thorough history:
 - Many chief complaints are complicated by anticoagulation.
 - Reason for anticoagulation, recent dose changes, compliance, recent INR testing, other prescriptions, over the counter, and alternative medicines
 - Subtle changes in mental status, recent “minor falls,” or bleeding
- Check for vital sign abnormalities:
 - Early hemorrhagic shock
 - Hypertension and bradycardia may be secondary to Cushing response in ICH.
 - Cardiac meds often mask important changes in vital signs.
- Examine carefully for:
 - Pallor, contusions, abrasions, ecchymosis, palpable pulses in affected extremity and skin lesions
 - Check stool for blood.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- PT/PTT/INR:
 - Significant bleeding may occur even in INR therapeutic range.
 - PTT also elevated with toxicity
 - Elevations will be delayed in overdose
- CBC:
 - Initial HCT inaccurate measure of acute rapid bleeding
 - Platelets:
 - Aspirin and ADP inhibitors/Plavix result in normal platelet levels but qualitative deficits.
- Electrolytes, BUN, creatinine, LFTs, and glucose:
 - Elevated BUN may indicate blood in GI tract.
 - Coingestants if intentional ingestion
- Type and cross-match

Imaging

- Low threshold for CT imaging to detect occult but life-threatening bleeding:
- Head CT:
 - Minor mechanisms of blunt head trauma without loss of consciousness
 - Detect ICH prior to symptom onset
- Abdominal CT:
 - Blunt abdominal trauma without significant tenderness
 - Retroperitoneal hemorrhage
 - Solid organ or visceral injury

DIFFERENTIAL DIAGNOSIS

- All causes of bleeding:
 - GI, retroperitoneal, CNS, and traumatic
- Skin lesions—hemorrhagic skin disorders:
 - Hemostatic deficits such as platelet disorders
 - Vascular purpuras including glucocorticoid use, vitamin C deficiency, purpura fulminans, disseminated intravascular coagulation, Henoch–Schönlein purpura, protein C deficiency



TREATMENT

PRE HOSPITAL

- ABCs
- Treat hypotension with 2 large-bore IV lines and 0.9% NS infusion.
- Cardiac and pulse oximetry monitoring

INITIAL STABILIZATION/THERAPY

- Establish central IV access for hypotension not responsive to initial fluid bolus:
 - Compressible sites only
- Replace lost blood as soon as possible
 - Initiate with O-negative blood until type-specific blood available.
 - 10 mL/kg bolus in children

ED TREATMENT/PROCEDURES

- Specific management depends on the INR, presence of bleeding, reason for anticoagulation, and reliability of patient:
 - INR < 5 without bleeding:
 - Lower or omit next dose.
 - Recheck INR in 24 hr.
 - INR ≥ 5 –<9 without bleeding:
 - Omit next 1 or 2 doses or omit 1 dose and give 1–2.5 mg PO vitamin K.
 - If at increased risk for bleeding or pre-op, then administer vitamin K 1–5 mg PO, INR will be lowered in 24 hr.
 - Recheck INR in 24 hr.
 - INR ≥ 9 without significant bleeding:
 - Hold warfarin and give vitamin K 2.5–5 mg PO; INR will be substantially lowered in 24–48 hr
 - INR > 20 with minor bleeding or life-threatening bleeding regardless of INR:
 - Hold Warfarin
 - Vitamin K 10 mg by slow IV infusion
 - Fresh frozen plasma (FFP) or prothrombin complex concentrate (PCC)

- depending on volume status and availability
 - PCC shows faster INR reversal and hemostasis; however, no clear benefit has been shown in patient outcome
 - PCC preferred in cases of ICH, volume overload, or massive bleeding.
- In the setting of controlled bleeding, maintain the INR at the lower level of therapeutic efficacy:
 - 1.5–2 for atrial fibrillation
 - 2–2.5 with mechanical heart valves
- Starting reversal agents before transferring patient leads to better outcomes

MEDICATION

- Vitamin K1 phytonadione:
 - Side effects:
 - Anaphylaxis with IV >> IM or PO
 - SC absorption unpredictable
 - IM administration may result in hematoma formation.
 - Breakthrough thromboembolism with complete correction, prolonged risk if high dose vitamin K
 - 10 mg IV infusion over 10–30 min is recommended for life-threatening active bleeding with effects beginning in 1–2 hr
- FFP:
 - Traditionally 3–4 U of FFP (1 L) are given to control continued bleeding in the short term without excessive risk of thromboembolism.
 - Additional units may be necessary
 - Follow serial INR closely
 - Patient response is variable and may not correlate with correction of the INR.
 - Side effects:
 - Fluid overload
 - Virus transmission-rare
 - Transfusion-related acute lung injury (TRALI) – rare
- PCC:
 - Long shelf life and easy reconstitution into a highly concentrated volume (20 mL vs. 1 L of FFP per dose)
 - Rapid reversal without volume overload
 - Side effects:
 - Thrombosis
 - Less virus transmission than FFP
 - Multiple studies show more rapid reversal of INR, reduction bleeding compared to FFP
 - Relationship to patient outcome has not been demonstrated
 - **4-factor PCC** (Kcentra) is a fractionation product of FFP containing equal amounts of factors II, VII, IX, and X:

- FDA approved in 2013, not widely available
- For patients with an INR of 2–3.9, administer 25 U/kg, 4–5.9, 35 U/kg, and >6, 50 U/kg
- **3-factor PCC** (Bebulin-VH, Profilnide-SD) contains very little factor VIIa:
 - Some use in combination with VIIa or VIIa alone depending on availability
 - 50 U/kg PCC and 1–2 mg FVIIa has been suggested
 - Consider FFP supplementation of FVIIa unavailable
 - More widely available in US
 - Warfarin reversal is off label use



FOLLOW-UP

DISPOSITION

Admission Criteria

- Active GI, retroperitoneal, or CNS bleeding
- Anticoagulated trauma patient with evidence of active bleeding requires:
 - Reversal of anticoagulation and blood replacement
 - Early surgical consultation for operative intervention
 - Transport to a level 1–trauma center after initial stabilization for definitive care.
- Skin necrosis and limb gangrene requires admission for anticoagulation with alternative agents in consultation with a hematologist.
- Subtherapeutic patient may require adequate anticoagulation with inpatient heparin or low molecular weight heparin to prevent a breakthrough thromboembolism:
 - Outpatient Lovenox therapy followed by increased warfarin with close follow-up prevents unnecessary hospitalization

Discharge Criteria

- Asymptomatic reliable patient with a supratherapeutic INR after consideration of:
 - Indication for anticoagulation, reason for supratherapeutic level, underlying comorbidities, overall risk of bleeding, fall risk, social situation, reliability, and availability of follow-up
- Asymptomatic anticoagulated patient with minor trauma, therapeutic INR, stable hemoglobin, normal imaging studies, and reliable caretakers, can be discharged with close follow-up.

Issues for Referral

- Patient should follow up with primary care physician or specialist within 24–48 hr of discharge for INR check and further warfarin adjustments.

- Psychiatric referral for intentional overdose

FOLLOW-UP RECOMMENDATIONS

Educate patient on monitoring for signs and symptoms of excessive bleeding and/or new thrombotic event.

PEARLS AND PITFALLS

- Maintain a low threshold for imaging trauma patients on warfarin
- No vitamin K for an INR <5 without bleeding
- Vitamin K1 IV may result in fatal anaphylaxis:
 - Use only in patients with INR >20 with minor bleeding, or patients with life-threatening bleeding
 - Administer PO for everyone else.
- For rapid reversal, FFP is still considered a 1st-line agent
- 4-factor PCC, or 3-factor PCC/FVIIa should be used in patients with ICH, volume overload, or massive bleed

ADDITIONAL READING

- Ansell J, Hirsh J, Hylek E, et al. Pharmacology and management of the vitamin K antagonists: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines (8th Edition). *Chest*. 2008;133:160S–198S.
- Denas G, Marzot F, Offelli P, et al. Effectiveness and safety of a management protocol to correct over-anticoagulation with oral vitamin K: A retrospective study of 1,043 cases. *J Thromb Thrombolysis*. 2009;27(3):340–347.
- Garcia, DA, Crowther MA. Reversal of warfarin: Case-based practice recommendations. *Circulation*. 2012;125:2944–2947.
- Sarode R, Matevosyan K, Bhagat R, et al. Rapid warfarin reversal: A 3-factor prothrombin complex concentrate and recombinant factor VIIa cocktail for intracerebral hemorrhage. *J Neurosurg*. 2012;116:491–497.



CODES

ICD9

- 286.59 Other hemorrhagic disorder due to intrinsic circulating anticoagulants, antibodies, or inhibitors
- V58.61 Long-term (current) use of anticoagulants

ICD10

- D68.318 Oth hemorrhagic disord d/t intrns circ anticoag,antib,inhib
- T45.515A Adverse effect of anticoagulants, initial encounter

- Z79.01 Long term (current) use of anticoagulants

WARTS

Gary M. Vilke



BASICS

DESCRIPTION

- Warts are caused by the human papillomaviruses (HPV)
- Causes cellular proliferation and vascular growth
- Lesions are typically verrucous and hyperkeratotic
- Lesions resolve spontaneously in most cases:
 - 1/3 within 6 mo
 - 2/3 within 2 yr
 - 90% within 5 yr
 - Likely due to cell-mediated immune response
- Cutaneous warts:
 - Verrucae vulgaris (common warts):
 - Dorsum of hands
 - Sides of fingers
 - Adjacent to nails
 - Usually asymptomatic
 - Verrucae plantaris (plantar warts):
 - Weight-bearing parts of sole: Heels, metatarsal heads
 - Often symptomatic and painful
 - More common in adolescents and young adults
 - Flat (juvenile) warts:
 - Primarily on light-exposed areas
 - Head, face, neck, legs, dorsum of hands
 - Small in size
 - Range from a few to hundreds
- Anogenital warts:
 - Known as condyloma acuminata or venereal warts
 - Most are asymptomatic and may go unrecognized
 - HPV types 6 and 11 account for 90% of anogenital warts
- HPV types 16 and 18 account for 70% of cervical cancers

ETIOLOGY

- HPV is host-specific to humans
 - Cause infection of epithelial tissues and mucous membranes
 - Infects the basal layer of skin or mucosa
- There are > 100 types of HPV that variably infect different body sites
- HPV transmission is:

- Direct: Skin to skin
- Indirect: Contaminated surface to skin
- Autoinoculation: Scratching, sucking (especially in young children)
- Incubation period can range from weeks to >1 yr

Pediatric Considerations

- 10–20% of children will have warts
- Peak incidence between 12 and 16 yr
- May produce laryngeal papillomatosis in infants from viral exposure at birth
- Must consider sexual abuse in children with anogenital warts



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Complete sexual history
- Prior history of warts and treatment
- HIV status
- Cutaneous warts:
 - Common warts:
 - Usually asymptomatic unless on a pressure point
 - May present with bleeding secondary to minor trauma
 - Plantar warts:
 - Often painful with weight bearing
 - Flat (or juvenile) warts:
 - On light-exposed areas of skin
 - May spread with shaving face, neck, legs
- Anogenital warts:
 - In men, usually on glans penis, shaft, scrotum, or anus
 - In women, found on labia, vagina, cervix, or anus
 - May extend into urethra, bladder, or rectum:
 - Dysuria
 - Pain, itching, and/or bleeding with bowel movements
 - May have symptoms involving mouth or throat if oral sexual contact

Physical-Exam

- Cutaneous warts:
 - Common warts:
 - Hard, rough, raised, dome-shaped lesions
 - Obscure normal skin markings
 - Hypervascular and may bleed with minor trauma

- Plantar warts:
 - Soles of the feet
 - Obscure normal skin markings
 - Hypervascular and may bleed with gentle scraping
- Flat (or juvenile warts):
 - Flesh colored
 - Flat top and smooth
 - Small: Range from pinpoint to size of pencil eraser
- Anogenital warts:
 - Pedunculated growths often with cauliflower-like appearance
 - Lesions are soft and usually present in multiples
 - Flesh colored to slightly pigmented or red

ESSENTIAL WORKUP

Diagnosis made by characteristic appearance of lesions

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Pregnancy test for females
- Biopsy and viral typing not recommended for typical lesions
- If difficult to see, add acetic acid to suspected area, which will cause infected areas to whiten and become more visible
- Screen for other sexually transmitted diseases

Diagnostic Procedures/Surgery

Biopsy indicated if failing therapy, patient immunocompromised, or warts are pigmented, indurated, fixed, or ulcerated

DIFFERENTIAL DIAGNOSIS

- Cutaneous warts:
 - Common wart
 - Callus; will not bleed
 - Plantar wart:
 - Callus, corn, bunion
 - Flat (or juvenile) wart:
 - Moles, skin tag, lichen planus
- Anogenital wart:
 - Condyloma latum (secondary syphilis)
 - Herpes simplex
 - Prominent glands around head of penis
 - Benign or malignant neoplasm
 - Molluscum contagiosum



TREATMENT

INITIAL STABILIZATION/THERAPY

None required

ED TREATMENT/PROCEDURES

- Cutaneous warts:
 - Occlusion with duct tape:
 - Least invasive
 - Maintain on wart for 6 days
 - Gentle debridement with pumice stone or nail file on day 7
 - Good for young children
 - May also enhance other topical treatments
 - Salicylic acid:
 - Inexpensive, mild side effects
 - OTC is 17% salicylic acid
 - Prescription strength has up to 70% salicylic acid
 - Soak wart in warm water for 10–20 min
 - Apply salicylic acid overnight
 - Gently debride in morning
 - Patches are also available
 - Resolution may take weeks to months
 - May be more effective combining with cryotherapy
- Anogenital warts:
 - May use imiquimod, podofilox, podophyllin, trichloroacetic acid (TCA), bichloroacetic acid (BCA), or alternative therapies listed below
 - Nonintervention may be best course in children, as treatment has not been well studied
- Alternative treatments:
 - Cryotherapy with liquid nitrogen or dry ice
 - OTC cryotherapy kits
 - Electrocautery
 - Laser therapy
 - Surgical excision
 - Interferon for use by subspecialists
- Provide appropriate referral

MEDICATION

- Topical medications (patient applied):
 - Imiquimod 5% cream:
 - Apply 3 times/wk for up to 16 wk
 - Cream may weaken diaphragms and condoms

- Podofilox 0.5% gel or solution:
 - Apply BID for 3 days, then rest 4 days; may repeat for 4 cycles
 - Do not use on perianal, rectal, urethral, or vaginal lesions
- Salicylic acid:
 - Wash off 6–10 hr later
 - May be repeated weekly
- Topical medications (provider administered):
 - Podophyllin 10–25% in benzoin:
 - Weekly topical application:
 - Protect surrounding normal tissue with petroleum jelly
 - Wash off 1–4 hr later
 - Do not use in pregnancy: Highly toxic and teratogenic
 - Do not use on cervix, vagina, or anal canal as may cause dysplastic changes
 - TCA or BCA 80–90%
 - Apply weekly for 6–10 wk
 - Cryotherapy with liquid nitrogen or cyroprobe
 - May be repeated every 1–2 wk
- Vaccine:
 - Gardasil: Targets HPV types 6, 11, 16, 18:
 - Recommended for girls > 9 yr
 - 3-shot series over 6 mo
 - For the prevention of cervical cancer, vulvar and vaginal cancer, genital warts, and other low-grade cervical lesions
 - Cervarix: Targets HPV types 16, 18:
 - 3 shots over 6 mo
 - Universal vaccination may provide significant reduction of cervical cancer in developing countries without well-established screening
 - Both vaccines are 96% effective
 - There are still controversies surrounding routine use and acceptance



FOLLOW-UP

DISPOSITION

Admission Criteria

Disseminated cases in immunocompromised patients may require admission

Discharge Criteria

Most patients can be treated as outpatients

Issues for Referral

- All medication-based therapies require follow-up and subsequent dosing. Should not initiate treatment unless follow-up can be secured
- For treatment failures, referral to PMD or dermatology should be made for alternative treatment options
- Refer sexually active teenage girls to pediatrician or primary care for HPV vaccination

FOLLOW-UP RECOMMENDATIONS

- Pain, burning, redness, or other changes in symptoms require prompt re-evaluation
- Arrange follow-up with appropriate provider: Pediatrician, gynecologist, dermatologist, primary care physician

PEARLS AND PITFALLS

- Pregnancy test must be done before initiation of medical therapy
- HPV vaccine does not protect from all forms of HPV, just those most commonly associated with cervical cancer
- Consider sexual assault in children with anogenital warts

ADDITIONAL READING

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See Also (Topic, Algorithm, Electronic Media Element)

- Herpes, Genital
- HIV/AIDS
- Molluscum Contagiosum



CODES

ICD9

- 078.10 Viral warts, unspecified
- 078.12 Plantar wart
- 078.19 Other specified viral warts

ICD10

- B07.0 Plantar wart
- B07.8 Other viral warts
- B07.9 Viral wart, unspecified

WEAKNESS

Kathryn A. Volz • Jason C. Imperato

BASICS

DESCRIPTION

- Defined as a decrease in physical strength or energy
- Often multifactorial
- Distinguish neuromuscular disorder vs. non-neuromuscular disorder
- Categories of neuromuscular disorders:
 - Upper motor neuron (UMN) lesions:
 - Deep tendon reflexes (DTR) increased
 - Plantar reflexes upgoing
 - Increased muscle tone
 - Muscle atrophy absent
 - Lower motor neuron (LMN) lesions:
 - DTRs decreased to absent
 - Plantar reflexes absent or normal
 - Decreased muscle tone
 - Muscle atrophy present
 - Fasciculations
 - Neuromuscular junction (NMJ) lesions:
 - DTRs normal
 - Plantar reflexes normal or absent
 - Decreased muscle tone
- Categories of non-neuromuscular disorders:
 - Infectious
 - Endocrine
 - Metabolic
 - Cardiac
 - Rheumatologic
 - Toxic
 - Psychiatric

ETIOLOGY

- Neuromuscular disorders:
 - UMN lesions:
 - Multiple sclerosis
 - Amyotrophic lateral sclerosis (mixed)
 - Transverse myelitis
 - Poliomyelitis

- LMN lesions:
 - Guillain–Barré syndrome
 - Toxic neuropathies
 - Impingement syndromes
 - Diphtheria
 - Porphyria
 - Seafood toxins
- NMJ lesions/others:
 - Myasthenia gravis
 - Lambert–Eaton syndrome
 - Botulism
 - Periodic paralysis
 - Tick paralysis
- Non-neuromuscular disorders:
 - Dehydration
 - Anemia
 - Electrolyte imbalances
 - Malignancy
 - Cerebrovascular accident
 - Head or neck trauma
 - Myocardial ischemia
 - Infection/sepsis:
 - UTI
 - Pneumonia
 - Meningitis
 - Mononucleosis
 - HIV
 - Arboviruses
 - Endocrine abnormalities:
 - Hypothyroidism
 - Adrenal crisis
 - Periodic paralyzes
 - Rheumatologic disorders:
 - Systemic lupus erythematosus
 - Polymyalgia rheumatica
 - Toxins:
 - Medications
 - Environmental
 - Carbon monoxide poisoning
 - Cocaine
 - Alcohol

SIGNS AND SYMPTOMS

- Altered physical strength:
 - Assessment of strength:
 - 1: No contraction
 - 2: Active movement with gravity eliminated
 - 3: Active movement against gravity
 - 4: Active movement against gravity and resistance
 - 5: Normal power
 - Change in muscle tone:
 - Flaccidity
 - Spasticity
 - Rigidity
 - Abnormal DTRs
 - Abnormal plantar reflexes
 - Muscle atrophy:
 - Difference of > 1 cm in the leg and thigh and > 0.5 cm in the forearm and arm
- Systemic findings:
 - Weakness
 - Fatigue
 - Dizziness
 - Paresis
 - Paresthesias
 - Hoarse voice
 - Dysphagia
 - Visual changes
 - Confusion
 - Associated symptoms:
 - Fever
 - Chest pain
 - Dyspnea
 - Cough
 - Weight loss
 - Rash
 - Dysuria
 - Upper respiratory infection symptoms

ESSENTIAL WORKUP

- Review of medications
- Clinical suspicion gathered through history and physical exam guides further testing:

- Generalized vs. focal
- Acute vs. chronic
- Proximal vs. distal
- Ascending vs. descending
- Symmetric vs. asymmetric
- Improved vs. worsened with activity

DIAGNOSIS TESTS & INTERPRETATION

Diagnostic testing should be broad unless history and physical exam identify the cause of weakness.

Lab

- Serum glucose
- CBC
- Electrolytes
- BUN/creatinine
- Toxin screen
- Urinalysis
- Thyroid function tests (rule out hypothyroidism)
- ESR (rule out rheumatologic cause)
- Carboxyhemoglobin (rule out CO poisoning)
- Troponin/CK-MB (rule out cardiac ischemia)
- Digoxin level (rule out digoxin toxicity)

Imaging

- EKG (rule out acute coronary syndrome [ACS]/arrhythmia)
- CXR (rule out pneumonia)
- CT/MRI head (rule out intracranial pathology)

Diagnostic Procedures/Surgery

- Bedside spirometry:
 - Forced vital capacity, negative inspiratory force, peak expiratory flow rate
 - May identify those with impending ventilatory failure
- Lumbar puncture:
 - In suspected Guillain-Barré syndrome:
 - Albumin-cytologic dissociation in CSF (protein > 400, WBC < 10) is virtually diagnostic.
- Tensilon test:
 - Distinguishes myasthenic crisis from cholinergic crisis in myasthenia gravis

DIFFERENTIAL DIAGNOSIS

- Physiologic causes of weakness:
 - Simple fatigue:

- Excessive physical activity
- Inadequate rest
- Excessive or inadequate diet
- Pregnancy
- Psychiatric causes of weakness:
 - Anxiety/depression
 - Dependent personality
 - Hypochondriasis
 - Chronic fatigue syndrome
 - Fibromyalgia
 - Malingering



TREATMENT

Treatment is geared to the underlying cause of weakness.

PRE HOSPITAL

- Supplemental oxygen
- IV access
- Finger-stick glucose determination
- Consider endotracheal intubation in patients with severe respiratory distress.

INITIAL STABILIZATION/THERAPY

- Supplemental oxygen
- IV access
- Endotracheal intubation for impending ventilatory failure

ED TREATMENT/PROCEDURES

- Neurology consult if needed
- When the diagnosis is determined, specific therapies can be applied:
 - TPA for CVAs meeting criteria
 - Plasma exchange and/or IV immunoglobulin (IVIG) for Guillain–Barré syndrome
 - Hydrocortisone for adrenal insufficiency
 - Potassium supplementation for hypokalemia
 - Dextrose for hypoglycemia
 - Antibiotics for infectious etiologies
 - Specific antidotes for botulism and diphtheria
 - Digibind for digoxin toxicity



FOLLOW-UP

DISPOSITION

Admission Criteria

- All patients with new-onset neuromuscular disorders should be admitted for definitive diagnosis.
- Any evidence of impending ventilatory or circulatory compromise warrants ICU admission.

Discharge Criteria

- Resolution of symptoms
- Stable vital signs
- Definitive diagnosis and correction of abnormality

FOLLOW-UP RECOMMENDATIONS

- Discharged patients with non-neurologic etiologies should have follow-up with their PCP.
- Discharged patients with neurologic etiologies should have urgent neurology follow-up.

PEARLS AND PITFALLS

- Identify early and aggressively treat patients at risk for respiratory compromise due to Guillain–Barré, botulism, myasthenia gravis.
- Identify elderly patients with ACS or infection presenting as weakness.
- Consider endocrine causes of weakness, including adrenal insufficiency and hypothyroidism.

ADDITIONAL READING

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- Losman E. Weakness. In: Marx J, ed. *Rosen's Emergency Medicine: Concepts and Clinical Practice*. 7th ed. St. Louis, MO: Mosby; 2009:87–92.
- LoVecchio F, Jacobson S. Approach to generalized weakness and peripheral neuromuscular disease. *Emerg Med Clin North Am*. 1997;15(3):605–623.



CODES

ICD9

- 728.2 Muscular wasting and disuse atrophy, not elsewhere classified
- 728.87 Muscle weakness (generalized)
- 780.79 Other malaise and fatigue

ICD10

- M62.50 Muscle wasting and atrophy, NEC, unsp site
- M62.81 Muscle weakness (generalized)
- R53.1 Weakness

WEST NILE VIRUS

Roger M. Barkin

BASICS

DESCRIPTION

Infectious agent is an arbovirus, an RNA member of the *Flaviviridae* family.

ETIOLOGY

- Vector-borne virus
- Transmitted by infected mosquitoes in late summer/early fall
- Wild birds are primary reservoir hosts; humans are infected by cross-feeding mosquitoes.
- Introduced to Western Hemisphere in 1999; became more widespread owing to vector of *Culex* mosquito and is now endemic in North America
- Infection after blood transfusion and solid-organ transplant can occur.
- There are case reports of occupational exposure and infection of lab workers via percutaneous inoculation.
- Following recovery, immunity is considered lifelong. Reoccurrence is rare
- The 2011 outbreak had a mortality rate of 4–5%. Cases were reported in 48 states.

Pregnancy Considerations

Infection via transplacental transmission and breast-feeding has been reported.

DIAGNOSIS

SIGNS AND SYMPTOMS

- Variable severity of illness:
 - 80% asymptomatic
 - 20% mild symptoms, flu-like illness
 - ~1/150 with CNS involvement (encephalitis, meningitis)
- Incubation period is usually 2–6 days but can be up to 14 days in average patient and up to 21 days in immunocompromised patient.
- Symptoms have a sudden onset and last < 1 wk with mild infection.
- Mortality rate in severe cases is estimated at 7%.
- Severity of illness is related to degree of CNS invasion by virus. Risk is enhanced with increased age and immunosuppression
- Immunocompromised patients have prolonged viremia, delayed development of antibody, and increased likelihood of severe disease.
- Persistent symptoms of fatigue, memory impairment, weakness, and headache have been reported to last for 1–2 mo

Geriatric Considerations

- Patients >60 yr, if infected, are at higher risk for developing more severe disease and neurologic consequences.
- Advanced age is the most important risk factor for death.

History

- General:
 - _ Fever
 - _ Malaise
 - _ Anorexia
 - _ Headache
 - _ Acute phase resolves within several days but fatigue and weakness may persist for weeks
- Neurologic:
 - _ Altered mental status (change in level of consciousness, confusion, agitation, irritability)
 - _ Severe, diffuse muscle weakness; may be asymmetric and involve the face
 - _ Flaccid paralysis, which may resemble poliomyelitis-like syndrome, associated with anterior horn cell injury. Cranial nerve and bulbar abnormalities have been reported
 - _ May resemble Guillain–Barré syndrome
 - _ Seizures
 - _ Encephalitis more commonly reported in adults and meningitis in children
- GI:
 - _ Nausea, vomiting, diarrhea, anorexia
 - _ Abdominal pain
- Musculoskeletal:
 - _ Myalgia
 - _ Arthralgia
 - _ Back pain
- Respiratory:
 - _ Cough
 - _ Sore throat
- Ophthalmologic:
 - _ Photophobia
 - _ Eye pain

Physical-Exam

- General:
 - _ Temperature >38°C (>100°F)
 - _ Transient maculopapular rash
 - _ Rhabdomyolysis

- Neurologic:
 - Altered mental status
 - Hyporeflexia, areflexia
 - Ataxia
 - Extrapyrarnidal signs
 - Cranial nerve palsies, paresis
 - Myoclonus
 - Profound motor weakness
 - Flaccid paralysis
- GI:
 - Hepatosplenomegaly, hepatitis, pancreatitis
- Musculoskeletal:
 - Nuchal rigidity
- Hematologic:
 - Lymphadenopathy
- Dermatologic:
 - Rash (maculopapular or morbilliform on neck, trunk, extremities) usually lasting < 1 wk
- Cardiovascular:
 - Myocarditis (rare)
- Ophthalmologic:
 - Optic neuritis
 - Vitritis
 - Chorioretinitis

ESSENTIAL WORKUP

- Most sensitive screening test is serologic testing of CSF and serum for IgM antibody-capture enzyme-linked immunosorbent assay (MAC-ELISA) and culture.
- Centers for Disease Control and Prevention (970-221-6400)
- Can be detected during 1st 4 days of illness, nearly all tests are positive by day 7–8; may remain positive up to 1 yr after infection
- Procedures for submitting samples vary by state.
- Refer to local public health department for guidelines.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- CSF:
 - Pleocytosis with lymphocyte predominance
 - Elevated protein
 - Normal glucose
- CBC:
 - WBCs may be mildly elevated (50%) or normal.

- Leukopenia may be present (15%).
- Anemia can occur.
- Chemistry:
 - Hyponatremia sometimes seen:
 - Cause uncertain, possibly syndrome of inappropriate antidiuretic hormone (SIADH) when CNS involvement exists
 - Pancreatitis (rare)
 - Fulminant hepatitis (rare)

Imaging

- CT head usually normal
- MRI can be useful to identify CNS inflammation:
 - 1/3 of patients show abnormality.
 - Imaging findings generally nonspecific but may include enhancement of leptomeninges and/or periventricular white matter or can mimic demyelinating process.

Diagnostic Procedures/Surgery

- Lumbar puncture
- MAC-ELISA may be used on serum and CSF samples

DIFFERENTIAL DIAGNOSIS

- Other causes of meningitis:
 - Bacterial
 - Viral
 - Tuberculous
 - Fungal
- Other causes of viral encephalitis:
 - Other arboviruses, especially St. Louis encephalitis virus
 - Enterovirus, particularly in patients ≤ 16 yr of age
 - Herpes simplex virus (HSV)
 - Cytomegalovirus (CMV)
 - Epstein–Barr virus (EBV)
 - Mumps virus
 - Varicella zoster virus
 - Rabies virus
- Intracranial abscess
- CNS vasculitis
- Nonspecific viral syndrome
- Gastroenteritis



TREATMENT

INITIAL STABILIZATION/THERAPY

- ABCs
- Seizure precautions

ED TREATMENT/PROCEDURES

- Supportive care
- IV fluids for signs of dehydration
- For signs of meningitis, administer antibiotics pending results of CSF.
- Consider acyclovir if index of suspicion for the only treatable cause of viral encephalitis, HSV, is high.
- Administer antipyretics and pain medications.
- No known effective antiviral therapy or vaccine
- No controlled studies proving effectiveness of interferon α -2b, ribavirin, corticosteroids, anticonvulsants, or osmotic agents



FOLLOW-UP

DISPOSITION

Admission Criteria

- Neurologic symptoms
- Dehydration
- Concerning risk factors (advanced age, immunocompromise)

Discharge Criteria

- No signs of CNS involvement (encephalitis, meningitis)
- Able to tolerate oral solutions

FOLLOW-UP RECOMMENDATIONS

Neurologist to monitor for potential ongoing residual.

PEARLS AND PITFALLS

Consider HSV in differential, since HSV is treatable.

ADDITIONAL READING

- Centers for Disease Control and Prevention (CDC): Interim guidelines for the evaluation of infants born to mothers infected with West Nile Virus during pregnancy. *MMWR Morb Mortal Wkly Rep.* 2004;53:154–157.
- Hayes EB, O’Leary DR. West Nile virus infection: A pediatric perspective. *Pediatrics.* 2004;113:1375–1381.
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See Also (Topic, Algorithm, Electronic Media Element)

Meningitis; Encephalitis, HSV



CODES

ICD9

- 066.40 West Nile Fever, unspecified
- 066.41 West Nile Fever with encephalitis
- 066.42 West Nile Fever with other neurologic manifestation

ICD10

- A92.30 West Nile virus infection, unspecified
- A92.31 West Nile virus infection with encephalitis
- A92.32 West Nile virus infection with oth neurologic manifestation

WHEEZING

Stephen K. Epstein

BASICS

DESCRIPTION

- Result of turbulent airflow:
 - High-pitched sound with dominant frequency at 400 Hz:
 - Gas flowing through constricted airways analogous to a vibrating reed
 - Resonant vibration of the bronchial walls when airflow velocity reaches critical values
- Caused by airway narrowing between 2–5 mm:
 - Wheezing is very low pitched with airway diameters of 5 mm.
 - Airways of <2 mm are unable to transmit sound because the energy is lost as friction heat.
- Airway narrowing is caused by a combination of ≥ 1 of the following:
 - Constriction (as with reactive airway disease)
 - Peribronchial interstitial edema
 - Inflammation
 - Obstruction

ETIOLOGY

- Pulmonary (small airway):
 - Asthma
 - Acute respiratory distress syndrome
 - Anaphylaxis
 - Aspiration pneumonia:
 - Wheezing occurs early in the disease due to intense bronchospasm following the event.
 - Byssinosis:
 - Occupational lung disease of textile workers exposed to cotton dust
 - Drugs:
 - Can precipitate angioedema or allergic reaction
 - ACE inhibitors
 - β -blockers
 - Aspirin and NSAIDs
 - Forced exhalation in normal patients
 - Hyperventilation
 - Chronic obstructive pulmonary disease
 - Chronic cor pulmonale

- Chemical pneumonitis
- Carcinoid tumors
- Paroxysmal nocturnal dyspnea
- Pulmonary edema
- Pulmonary embolism:
 - Rarely associated with wheezing
 - Focal
- Pneumonia
- Sleep apnea
- Pulmonary (large airway):
 - Vocal cord dysfunction (paralysis, paradoxical movement)
 - Foreign body
 - Epiglottitis:
 - Wheezing associated with stridor in 10% of cases
 - Diphtheria
 - Smoke inhalation
 - Bronchial tumor
 - Tracheal tumor

Pediatric Considerations

- Viral bronchiolitis in patients < 3 yr of age
- Asthma
- Infection:
 - Croup
 - Rhinovirus
- Foreign-body aspiration
- Congenital abnormalities:
 - Tracheomalacia
 - Tracheal stenosis
- Cystic fibrosis
- CHF



DIAGNOSIS

SIGNS AND SYMPTOMS

- A whistling sound made while breathing:
 - Diffuse:
 - As with reactive airway disease or pulmonary edema
 - Focal:
 - As with pneumonia or pulmonary embolism
- Dyspnea
- Respiratory distress

- Chest pain
- Cough
- Sputum production:
 - Frothy (pulmonary edema)
- Stridor
- Fever
- Cyanosis
- Tachypnea
- Tachycardia

History

- Current URI:
 - Rhinoviruses implicated in reactive airways
- Recent exercise:
 - Exercise-induced asthma, vocal cord dysfunction

Physical-Exam

- Mental status:
 - Lethargy, confusion, and fatigue in the setting of respiratory distress are the primary reasons for airway management.
- Presence of muscle retractions
- Lung auscultation

ESSENTIAL WORKUP

- Pulse oximetry:
 - Useful for assessing severity, but not for predicting hospital admission
- Peak flow:
 - Useful in assessing need for hospitalization
- CXR

DIAGNOSIS TESTS & INTERPRETATION

Lab

- ABG:
 - Sometimes used to determine whether patient is fatiguing by noting falling oxygenation, rising CO₂, and acidosis
 - Clinical assessment is a more reliable indicator of the need for airway management.
- WBC:
 - Elevated WBC does not distinguish infection from other disorders, as stress causes demargination.
 - WBC is also elevated in noninfected patients taking steroids.

- A normal WBC does not rule out an underlying pneumonia.

Imaging

- Peak expiratory flow (PEF):
 - To assess function of small airways
 - Use to determine severity and track the progress of therapy in patients with reactive airway disease.
- CXR:
 - Assess for diagnosis of pulmonary conditions:
 - Pneumonia
 - Foreign-body aspiration
 - Assess for pulmonary edema.
- EKG:
 - Useful when patient is at risk for cardiac ischemia
 - Indicated in all cases in which wheezing is caused by pulmonary edema
- Soft-tissue neck:
 - Used to assess for foreign body or obstructing mass

Diagnostic Procedures/Surgery

Laryngoscopy/bronchoscopy:

- Indicated when obstruction is thought to be causal
- Used to retrieve an inhaled foreign body or diagnose an underlying tumor

DIFFERENTIAL DIAGNOSIS

See Etiologies.



TREATMENT

PRE HOSPITAL

- Supplemental oxygen
- Initiate pulse oximetry and cardiac monitoring.
- Initiate therapy for underlying condition when indicated:
 - Asthma
 - Pulmonary edema
- Intubate for respiratory failure or anticipated respiratory failure.

INITIAL STABILIZATION/THERAPY

- ABCs
- Intubation for impending airway failure:
 - Prepare for possible foreign body in airway.
 - Anticipate difficult airway.

ED TREATMENT/PROCEDURES

- Correct hypoxemia: Supplemental oxygen
- Initial assessment of severity:
 - PEF >40%: Mild–moderate
 - PEF <40%: Severe
- Treat the underlying condition.
- Rapid reversal of airflow obstruction:
 - Bronchodilators:
 - Reversibility following the use of short-acting β -agonists such as albuterol or terbutaline suggests reactive airway disease.
 - Anticholinergics: Ipratropium bromide:
 - Add to β -agonist therapy for severe disease
- Reduce likelihood of relapse:
 - Trial of steroids indicated if wheezing is caused by bronchospasm or noninfectious inflammation.
- Adjunctive agents:
 - Heliox:
 - Less dense than air or oxygen alone
 - Decreases work of breathing
 - More efficacious in large-airway disease
 - Not as effective for small-airway disease
 - Magnesium sulfate:
 - Evidence for benefit only in moderate to severe asthmatics
 - Ketamine:
 - For intubation of the asthmatic patient

MEDICATION

First Line

- Albuterol: 2.5–5 mg in 2.5 mL NS q20min inhaled \times 3 doses (peds: 0.15 mg/kg/dose q20min \times 3 doses; min. dose 2.5 mg)
- Levalbuterol: 0.63 mg q8h (peds: 6–12 yr 0.31 mg q8h; >12 yr 0.63 mg q8h) via nebulizer
- Prednisone: 40–80 mg PO (peds: 1 mg/kg/d in 2 div. doses; max. 60 mg/d)
- Prednisolone: Peds 1–2 mg/kg/d in 2 div. doses PO; ipratropium insert in peds dose (peds: >12 yr 0.25–0.5 mg)
- Racemic epinephrine: Peds 0.25–0.5 mL nebulized for croup

Second Line

- Ipratropium bromide: 0.5 mg q20min \times 3 doses (peds: 0.25–0.5 mg q20min \times 3 doses); may mix with albuterol
- Methylprednisolone: 40–80 mg IV (peds: 1–2 mg/kg/d IV or PO in 2 div. doses, max. 60 mg/d) for patients who cannot tolerate PO

- Terbutaline: 0.25 mg SC q0.5h for 2 doses (peds: 0.01 mg/kg up to 0.3 mg SC):
 - No proven advantage over aerosol therapy
- Magnesium sulfate: 0.1 mL/kg of 50% solution IV over 20 min, then 0.06 mg/kg/h



FOLLOW-UP

DISPOSITION

Admission Criteria

- Hypoxia
- Persistent or worsening wheezing
- Underlying condition requires hospital admission

Discharge Criteria

- Improvement or resolution of wheezing
- PEF > 70% predicted
- Adequate oxygenation

Issues for Referral

Asthma:

- Referral should be made for a written asthma action plan.

FOLLOW-UP RECOMMENDATIONS

The patient should be instructed to return to the ED with shortness of breath, fever, hemoptysis, or chest pain.

PEARLS AND PITFALLS

Be prepared to manage the airway if administering an anxiolytic.

ADDITIONAL READING

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- Fernandes RM, Bialy LM, Vandermeer B, et al. Glucocorticoids for acute viral bronchiolitis in infants and young children. *Cochrane Database Syst Rev*. 2013;6:CD004878.
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See Also (Topic, Algorithm, Electronic Media Element)

- [Asthma, Adult](#)
- [Asthma, Pediatric](#)



CODES

ICD9

- [493.90 Asthma, unspecified type, without mention of status asthmaticus](#)
- [519.11 Acute bronchospasm](#)
- [786.07 Wheezing](#)

ICD10

- [J45.909 Unspecified asthma, uncomplicated](#)
- [J98.01 Acute bronchospasm](#)
- [R06.2 Wheezing](#)

WITHDRAWAL, ALCOHOL

Trevonne M. Thompson



BASICS

DESCRIPTION

- Alcohol withdrawal is the most common withdrawal syndrome encountered in the emergency department
- Neuroexcitation is the hallmark of alcohol withdrawal
- Alcohol withdrawal may be life threatening.
- More severe symptoms and signs are seen in patients with prior episodes of withdrawal, a process called kindling
- Alcoholism is not uncommon among older adults.
- Age-related increase in alcohol sensitivity
- Alcohol-related problems may be misdiagnosed as normal consequences of aging.

ETIOLOGY

- Chronic alcohol use downregulates GABA (inhibitory) receptors, upregulates NMDA (excitatory) receptors.
- Abstinence or reduction in use leads to increased adrenergic activity because of these receptor adaptations
- 4 components to alcohol withdrawal:
 - _ Early withdrawal
 - _ Withdrawal seizures
 - _ Alcoholic hallucinosis
 - _ Delirium tremens (DTs)
- DTs occur in 5% of patients experiencing alcohol withdrawal
- DTs have a 5–15% mortality rate



DIAGNOSIS

SIGNS AND SYMPTOMS

- Early withdrawal:
 - _ Occurs: 6–8 hr after the last drink
 - _ Duration: 1–2 days
 - Tremulousness
 - Anxiety
 - Palpitations
 - Nausea
 - Anorexia
- Withdrawal seizures:

- Occurs: 6–48 hr after the last drink
- Duration: 2–3 days
 - Generalized seizures, generally brief
- Alcoholic hallucinosis:
 - Occurs: 12–48 hr after the last drink
 - Duration: 1–2 days
 - Visual hallucinations (most common)
 - Tactile hallucinations
 - Auditory hallucinations
 - Sensorium typically otherwise clear
- DTs:
 - Occurs 48–96 hr after the last drink
 - Can last up to 5 days
 - Not necessarily preceded by hallucinosis or seizures:
 - Tachycardia
 - HTN
 - Diaphoresis
 - Delirium
 - Agitation
 - Sensorium typically not clear

History

- Obtain substance abuse history:
 - Time of last substance use
 - History of previous withdrawal and how severe

Physical-Exam

A thorough physical exam is necessary

ESSENTIAL WORKUP

Thorough history and physical exam with attention to the vital signs

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Electrolytes, BUN, creatinine, glucose, magnesium
- CBC
- Alcohol level
- Urine drug screening rarely alters management
- Urinalysis
- Blood/urine culture:
 - For suspected infection

Imaging

- Not necessary if early withdrawal is clearly the presenting issue
- CT head:
 - _ For altered mental status or if the clinical situation is not straightforward
- CXR:
 - _ If secondary infection (e.g., aspiration pneumonia) is suspected.

Diagnostic Procedures/Surgery

ECG when clinically warranted

DIFFERENTIAL DIAGNOSIS

- Benzodiazepine withdrawal
- Barbiturate withdrawal
- Intracerebral hemorrhage
- CNS infection
- Epilepsy
- Hypoglycemia
- Hyperthyroidism
- Sepsis
- Drug intoxication
- Psychosis
- Electrolyte disorder



TREATMENT

PRE HOSPITAL

- Assess vital signs
- Assess capillary glucose

INITIAL STABILIZATION/THERAPY

- Attention to the ABCs
- Obtain IV access
- IV fluid administration
- Cardiopulmonary monitoring

ED TREATMENT/PROCEDURES

- Aggressive supportive care
- Benzodiazepines:
 - _ The standard therapy
 - _ No single benzodiazepine is more effective than another
 - _ High doses are often required to control symptoms and signs
- Barbiturates may be used as an alternate or adjunct to benzodiazepines.

- Propofol may also be used in severe cases.

MEDICATION

- Diazepam: 5–20 mg PO for mild symptoms and signs; 5–10 mg IV; repeat for severe symptoms and signs
- Lorazepam: 2 mg PO, repeat q2–4h as needed for mild symptoms and signs; 2 mg IV in repeated doses as necessary for severe symptoms and signs
- Phenobarbital: 30–60 mg PO for mild symptoms and signs; 15–20 mg/kg slow intravenous administration for severe symptoms or status epilepticus
- Propofol: Start with 25–75 µg/kg/min, then titrate as necessary



FOLLOW-UP

DISPOSITION

Admission Criteria

- Moderate-to-severe symptoms
- Persistent symptoms despite treatment
- DTs or impending DTs
- Comorbid medical illness

Discharge Criteria

Mild symptoms and signs responsive to therapy

FOLLOW-UP RECOMMENDATIONS

Referral to detox program or facility

PEARLS AND PITFALLS

- Misdiagnosis of medical disease as withdrawal syndrome
- Misunderstanding the relationship between withdrawal syndromes and comorbid medical illness
- Administer sufficient quantities of benzodiazepines to control symptoms.

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See Also (Topic, Algorithm, Electronic Media Element)

Withdrawal, Drug



CODES

ICD9

- 291.0 Alcohol withdrawal delirium
- 291.3 Alcohol-induced psychotic disorder with hallucinations
- 291.81 Alcohol withdrawal

ICD10

- F10.231 Alcohol dependence with withdrawal delirium
- F10.239 Alcohol dependence with withdrawal, unspecified
- F10.951 Alcohol use, unsp w alcoh-induce psych disorder w hallucin

WITHDRAWAL, DRUG

Trevonne M. Thompson



BASICS

DESCRIPTION

- Neuroexcitation is the hallmark of benzodiazepine, barbiturate, and opiate withdrawal
- Benzodiazepine and barbiturate withdrawal can be life threatening
- Opiate withdrawal can be extremely uncomfortable but is not typically life threatening
- Cocaine and amphetamine withdrawal are similarly not life threatening

ETIOLOGY

- Chronic exposure to certain drugs cause adaptive changes in the CNS
- Withdrawal syndromes occur when the constant presence of drug is removed or reduced and the adaptive changes persist
- Tolerance occurs when increasing amounts of drug are required to achieve a given response
- Withdrawal and tolerance are distinct entities



DIAGNOSIS

SIGNS AND SYMPTOMS

- Benzodiazepines and barbiturates:
 - Anxiety
 - Agitation
 - Irritability
 - Tremor
 - Sleep disturbance
 - Tachycardia
 - Hypertension
 - Hyperthermia
 - Autonomic instability
 - Seizures
- Opiates:
 - Restlessness
 - Irritability
 - Drug craving
 - Yawning
 - Piloerection

- Mydriasis
- Nausea
- Vomiting
- Diarrhea
- Abdominal pain
- Tachycardia
- HTN
- Cocaine:
 - Depressed mood
 - Fatigue
 - Vivid dreams
 - Sleep disturbance
 - Psychomotor retardation or agitation
- Amphetamines:
 - Fatigue
 - Irritability
 - Sleep disturbance
 - Anxiety

History

- Obtain substance abuse history
 - Time of last substance use
 - History of previous withdrawal

Physical-Exam

A thorough physical exam is necessary

ESSENTIAL WORKUP

Thorough history and physical exam with attention to the vital signs

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Electrolytes, BUN, creatinine, glucose
- CBC
- Urine drug screening rarely alters management

Imaging

If the clinical situation is not straightforward for withdrawal, CNS or plain radiograph imaging may be indicated depending on the clinical presentation.

DIFFERENTIAL DIAGNOSIS

- Ethanol withdrawal

- Intracerebral hemorrhage
- CNS infection
- Encephalopathy
- Hypoglycemia
- Hyperthyroidism
- Sepsis
- Psychosis
- Electrolyte disorder
- Drug intoxication



TREATMENT

PRE HOSPITAL

- Assess vital signs
- Assess capillary glucose

INITIAL STABILIZATION/THERAPY

- Attention to ABCs
- Obtain IV access
- IV fluid administration
- Cardiopulmonary monitoring

ED TREATMENT/PROCEDURES

- Benzodiazepine and barbiturate withdrawal:
 - Aggressive supportive care
 - Begin long-acting agent of the same class causing the withdrawal
- Opiate withdrawal:
 - Supportive care
 - Antiemetics for nausea and vomiting
 - Clonidine to reduce severity of signs and symptoms
 - Opiate therapy if withdrawal is complicating other disease states
- Cocaine and amphetamine withdrawal:
 - Supportive care

MEDICATION

- Clonidine: 0.1–0.3 mg PO q4–6h
- Diazepam: 5–20 mg PO for mild signs and symptoms; 5–10 mg IV in repeated doses as necessary for severe symptoms and signs
- Lorazepam: 1–2 mg PO for mild symptoms and signs; 2 mg IV in repeated doses as necessary for severe symptoms and signs
- Phenobarbital: 30–60 mg PO for mild symptoms and signs; 15–20 mg/kg slow IV administration for severe symptoms or status epilepticus.
- Ondansetron: 4–8 mg PO/IV



FOLLOW-UP

DISPOSITION

Admission Criteria

- Moderate-to-severe withdrawal symptoms
- Persistent withdrawal symptoms
- Psychosis with withdrawal
- Autonomic instability
- Concomitant medical condition that may complicate withdrawal
- Suicidal ideation or otherwise psychiatrically unstable

Discharge Criteria

- Mild symptoms responsive to therapy
- Psychiatrically stable

FOLLOW-UP RECOMMENDATIONS

Referral to detox program or facility

PEARLS AND PITFALLS

- Misdiagnosis of medical disease as withdrawal syndrome
- Misunderstanding the relationship between withdrawal syndromes and comorbid medical illness
- Important to administer sufficient quantities of benzodiazepines for patient in benzodiazepine withdrawal states.

ADDITIONAL READING

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CODES

ICD9

292.0 Drug withdrawal

ICD10

- F11.23 Opioid dependence with withdrawal
- F15.23 Other stimulant dependence with withdrawal
- F19.239 Oth psychoactive substance dependence with withdrawal, unsp

WOLFF-PARKINSON-WHITE (WPW) SYNDROME

James J. Rifino



BASICS

DESCRIPTION

- Syndrome resulting from the presence of an abnormal (accessory) pathway that bypasses the AV node (Kent bundles) between the atria and ventricles
- Wolff-Parkinson-White (WPW) pattern on the ECG is defined by a short PR interval and a Δ -wave reflecting early conduction (pre-excitation):
 - Accessory pathways occur in 0.1–0.3% of the population.
- WPW syndrome requires ECG evidence of the accessory pathway and related tachycardia.
- Accessory pathways:
 - Small bands of tissue that failed to separate during development:
 - Left lateral (free wall) accessory pathway: Most common
 - The posteroseptal region of the AV groove: 2nd most common location
 - Right free wall
 - Anteroseptal
- Conduction in WPW may be antegrade, retrograde, or both.
- Orthodromic re-entrant tachycardia is the most common (70%):
 - Impulse travels antegrade from the atria down the AV node to the ventricle and then retrograde up the accessory pathway.
 - This re-entrant tachycardia is a narrow complex rhythm unless a bundle branch block or intraventricular conduction delay is present.
- Antidromic is less common (30%):
 - Impulse travels antegrade down the accessory pathway and retrograde through the AV node resulting in a wide quasi-random signal (QRS) complex.
- Sudden death occurs in 1 per 1,000 patient-years in persons with known ventricular pre-excitation.

ETIOLOGY

- Idiopathic:
 - Unknown mechanism in most cases, with familial predisposition
- Rarely inherited as an autosomal dominant trait
- Associated in rare cases with a familial hypertrophic cardiomyopathy



DIAGNOSIS

SIGNS AND SYMPTOMS

History

- Asymptomatic
- Palpitations:
 - Fast or irregular
- Chest pain
- Dyspnea
- Dizziness
- Diaphoresis
- Syncope
- Sudden death (rare)

Physical-Exam

- Tachycardia:
 - Rapid and regular:
 - Supraventricular tachycardia
 - Atrial flutter
 - Irregular:
 - Atrial fibrillation
- Signs of instability:
 - Chest pain
 - Hypotension
 - Change in mental status
 - Rales
 - Cyanosis

ESSENTIAL WORKUP

- WPW syndrome should be considered the underlying etiology in all cases of tachydysrhythmia.
- The diagnosis should be based on the characteristic ECG findings.

DIAGNOSIS TESTS & INTERPRETATION

Lab

- Cardiac enzymes only if signs of ischemia
- Consider electrolytes and thyroid disease

Diagnostic Procedures/Surgery

- EKG
- Pre-excitation:
 - Short PR interval, < 0.12 sec
 - Δ -wave: Small slurred upstroke at the beginning of the QRS

- Prolonged QRS, >0.10 sec with variable morphology linked to specific accessory pathway
- Left lateral pathway:
 - Positive Δ -waves
 - Q-waves with negative to isoelectric deflections in V1 and in the inferior leads:
 - May suggest a former high lateral MI and right axis deviation
- Posteroseptal accessory pathway:
 - Negative deflecting Δ -waves
 - QRS complexes in the inferior leads:
 - Often mistaken for prior inferior MI
- Tachydysrhythmias:
 - Orthodromic atrioventricular re-entrant tachycardia (OAVRT):
 - The pathway that conducts the impulse to the ventricle is the AV node/His–Purkinje system
 - Narrow QRS complex tachycardia
 - However, this may be associated with a wide QRS complex in the presence of a pre-existing or rate-related functional bundle branch block.
 - P-wave following the QRS
 - Rate between 150–250 bpm
 - The Δ -wave seen during sinus rhythm is lost since antegrade conduction is not via the accessory pathway
 - Antidromic AVRT:
 - Regular
 - Wide QRS complex
 - The antegrade limb is usually the accessory pathway.
 - Atrial fibrillation:
 - Irregular
 - Wide complex with variable QRS morphologies

DIFFERENTIAL DIAGNOSIS

- Pre-excitation:
 - Inferior MI
- Narrow complex supraventricular tachycardias without an accessory pathway:
 - AV nodal re-entry tachycardia (AVNRT)
- Wide complex tachycardia:
 - Atrial fibrillation with intraventricular conduction delay
 - Ventricular tachycardia



TREATMENT

PRE HOSPITAL

- Supplemental oxygen and monitor
- Vagal maneuvers (Valsalva), carotid massage, and ice water on the face
- Synchronized cardioversion for:
 - Signs of instability (hypotension, AMS, etc.)
 - Atrial fibrillation with WPW; wide complex tachycardia
- Pre-hospital use of adenosine:
 - Stable patients: No emergent conversion.
 - Unstable patients: Need cardioversion, not adenosine.

INITIAL STABILIZATION/THERAPY

- Unstable patients:
 - Synchronized cardioversion (start with 100 J)
 - Increase incrementally until sinus rhythm is restored (200 J then 360 J).
- Stable patients with wide complex tachycardia:
 - Amiodarone
 - Procainamide
 - **DO NOT USE:** Lidocaine, calcium channel blockers, β -blockers, and Digoxin in patients with wide complex tachycardia and suspected WPW.

ED TREATMENT/PROCEDURES

- Stable patients:
 - Vagal maneuvers: Valsalva and carotid massage:
 - Right carotid artery massage for no more than 10 sec
 - Auscultate the artery 1st for a bruit that would contraindicate this procedure.
 - Fluid replacement and Trendelenburg if the patient has mild hypotension
 - Pharmacologic conversion if carotid massage fails
- Orthodromic (usually narrow complex) AVRT:
 - Adenosine or verapamil
- Antidromic (usually wide complex) AVRT:
 - Procainamide is the drug of choice
 - Although verapamil and β -blockers can be used when the diagnosis is certain, their administration can be dangerous in ventricular tachycardia and WPW with atrial fibrillation, which can be hard to distinguish from this dysrhythmia.
- Irregular wide complex tachycardia:
 - WPW syndrome with atrial fibrillation
 - Amiodarone or procainamide.

Pediatric Considerations

- Children may develop ventricular rates up to 320 bpm that are poorly tolerated.
- Cardiovert unstable children with 0.5–2 J/kg.

- Vagal maneuvers and adenosine are safe in stable children.

MEDICATION

- Adenosine: 6 mg rapid IV bolus over 1–2 sec; if ineffective, repeat with 12 mg (peds: 0.1 mg/kg rapid IV push, repeat with 0.2 mg/kg)
- Amiodarone: 150 mg IV over 10 min, 360 mg over the next 6 hr
- Magnesium: 2 g IV bolus
- Procainamide: 6–13 mg/kg IV at 0.2–0.5 mg/kg/min until either arrhythmia controlled, QRS widens 50%, or hypotension, then 2–6 mg/min, max. of 1,000 mg

First Line

- Amiodarone for wide complex tachycardias
- Adenosine for narrow complex tachycardias

Second Line

- Procainamide for wide complex tachycardias
- IV Procainamide, IV Verapamil 5–10 mg, IV Diltiazem 10–20 mg, or Esmolol can be considered as 2nd-line agents for patients with WPW presenting with regular narrow complex tachycardias.



FOLLOW-UP

DISPOSITION

Admission Criteria

- Signs of instability and/or history of syncope
- Failure of outpatient therapy for continuous pharmacologic control or ablation

Discharge Criteria

- Most patients will be stable and can be discharged once converted to sinus rhythm
- Follow-up should be arranged with a cardiologist

Issues for Referral

Electrophysiology studies to assess for radiofrequency ablation or surgery may be performed on outpatient basis.

FOLLOW-UP RECOMMENDATIONS

The patient should be instructed to return to the ED with any symptoms suggestive of a tachydysrhythmia:

- Palpitations
- Dizziness
- Chest pain

- Feeling faint or actual syncope

PEARLS AND PITFALLS

Never use calcium channel blockers, β -blockers, or digoxin in patients with pre-excitation with atrial fibrillation or wide complex tachycardia:

- These medications prolong the refractory period of the AV node, increasing the rate of transmission through the accessory pathway, and may result in fatal ventricular dysrhythmias.
- If symptoms > 48 hr, anticoagulation must be addressed prior to cardioversion as 1–3% of patients will have embolic event. Transesophageal echo should be considered to rule out left atrial thrombus.

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See Also (Topic, Algorithm, Electronic Media Element)



CODES

ICD9

426.7 Anomalous atrioventricular excitation

ICD10

I45.6 Pre-excitation syndrome

WOUND BALLISTICS

Brian K. Snyder

BASICS

DESCRIPTION

The physical forces that determine the wounding potential of gunshot and other penetrating wounds

ETIOLOGY

- Wounding potential of bullet is determined by mass and velocity.
- The type and severity of a wound is determined by:
 - Wounding potential
 - Construction and shape of the bullet
 - Orientation of the bullet upon striking body
 - Deformity or fragmentation
 - What tissues the bullet traverse
- Traditional distinction between low and high muzzle **velocity** does not differentiate kind and severity of wounding:
 - A civilian hunting rifle or a large-caliber handgun with a hollow-point bullet may produce a more severe wound than a bullet with a full metal jacket from a “high-velocity” military rifle.
- Bullets wound by 2 main mechanisms—**crush** and **stretch**:
 - Sonic pressure wave that precedes bullet has no role in wounding.
 - Bullet crushes the tissue it directly passes through, forming a *permanent cavity*.
 - *Stretch* is produced by radial energy transferred from bullet as it slows down in tissue, forming a *temporary cavity*.
 - A bullet is stabilized in flight by *spin* transmitted from rifling in the barrel.
 - Spin minimizes *yaw*, which is the angle between the long axis of the bullet and its flight vector.
 - Without spin, a bullet would yaw to its most stable flight configuration, which is base and center of mass forward:
 - Not aerodynamically efficient
- As bullet enters tissue, spin of bullet is reduced and bullet will yaw.
- When yaw is 90°, a bullet crushes maximal amount of tissue, slows down the most, and maximal stretch injury occurs.
- Bullets designed to deform in tissue (soft point, hollow point) will expand on impact:
 - Increases amount of crush injury
 - Moves bullet center of mass forward

- Jacketed bullets prevent lead stripping in the barrel, which occurs at high muzzle velocities:
 - Jacketed bullets do not deform but may fragment.
 - Fragmentation increases surface area and crush injury.
- Bullets striking bone often fragment and may cause bone fragments to become secondary projectiles.
- Severity of wound also depends upon *tissue composition and thickness*:
 - Minimally elastic tissues, near-water-density tissue (brain, liver), fluid-filled (heart, bowel) and dense organs (bone) may be injured by the temporary cavity.
 - More elastic tissue, such as lung and skeletal muscle, may absorb the energy from temporary cavity formation and sustain minimal damage.
 - Extremities are often not thick enough for the bullet to fully yaw:
 - Temporary cavity formation is minimal.
 - Most damage is caused by direct crush injury of the bullet, its fragments, or secondary projectiles.
- Short-range shotgun blasts can produce severe wounds with compromise of the blood supply:
 - In short-range shotgun injuries, pellets may be greatly scattered in tissue secondary to the pellets striking each other.
- Stab wounds with knives and other sharp instruments are low-energy wounds with tissue injury from direct weapon contact.



DIAGNOSIS

SIGNS AND SYMPTOMS

- Severe underlying tissue damage and life-threatening injury may occur with even small entrance wounds.
- A knowledge of how different kinds of weapons and bullets wound, the trajectory of the bullet through the body, and the effect on different body tissues will allow the physician to carefully evaluate gunshot and stab wounds and their potential morbidity and mortality.

History

- Field personnel can provide information about weapon type and size, distance, and angle between the weapon and victim:
- This information may not be available or may be inaccurate.

Physical-Exam

- Evaluate for entrance and exit wounds:
- May estimate trajectory and potential for tissue damage
- Exit wounds are often stellate and larger than entranced wounds unless energy is

dissipated at skin surface by special bullet type (hollow point, etc.).

- With high-velocity projectiles, exit wound may be much more extensive than entrance wound.
- Because of the elasticity of the skin, bullet can often be palpated subcutaneously.
- It is not always possible to differentiate entrance from exit wounds; clinicians do this poorly, so wounds should be described fully and classification as entrance or exit wounds should be avoided.

ESSENTIAL WORKUP

- ABCs must be stabilized prior to any workup.
- Account for all injury tracts.
- Place markers at wound sites.
- Examine all areas of the body for wounds (remember the perineum, axillae, and scalp).

DIAGNOSIS TESTS & INTERPRETATION

Lab

Initial lab tests are not especially helpful in diagnosis but may be helpful in guiding resuscitation.

Imaging

- Anteroposterior and lateral radiographs help localize bullet:
 - With placement of markers at wound sites, wound trajectory can be estimated
 - Fragments, fractures, pneumothoraces, or hemothoraces can be identified.
- US:
 - A positive FAST scan is highly predictive of a therapeutic laparotomy. A negative FAST scan does not exclude significant intra-abdominal injury.
 - US for pericardial effusion in potential mediastinal injuries
- CT scanning:
 - Identify location of projectile.
 - Location and amount of tissue damage (especially to the head and brain)
 - Abdominal CT is increasingly used in the evaluation of stable patients with penetrating back/flank or abdominal trauma.
 - In penetrating trauma of the thorax, an initial negative CT scan of the thorax obviates the usual practice of repeated chest radiographs.
- Angiography may be necessary if patient has potential vascular injury and surgical exploration is not otherwise warranted.

Diagnostic Procedures/Surgery

- Local wound exploration with clear delineation of the base of the wound tract that does not penetrate deep structures may be sufficient to evaluate stab wounds.

- Abdominal wounds that encroach the posterior fascia require further evaluation, either diagnostic peritoneal lavage or surgical exploration.
- Extent of tissue injury often apparent only on surgical exploration.

DIFFERENTIAL DIAGNOSIS

Organs at risk of damage can be inferred from weapon type, distance, locations of entrance and exits wounds, or projectiles on imaging.

- Tissues surrounding the projectile tract are also at risk of injury (i.e., from temporary cavity).
- Projectiles may fragment and create multiple injury tracts.



TREATMENT

PRE HOSPITAL

- Gunshot and stab wounds to chest with unstable vital signs warrant a needle thoracostomy in the side of the chest with the entrance wound:
 - _ Relieves tension pneumothorax
 - _ If no improvement, a needle thoracostomy should be placed in the contralateral hemithorax.
- Impaled objects or projectiles should not be removed:
 - _ Immobilize with tape and gauze and transport.
- Clothing should be preserved if possible:
 - _ Clothing should be cut around holes made by the projectiles to preserve evidence.
- Patient should be transported to the closest trauma center.
- Hypotensive patient may be taken directly to the OR.

INITIAL STABILIZATION/THERAPY

Stabilize airway, breathing, and circulation. Secure adequate IV access.

ED TREATMENT/PROCEDURES

- Impaled objects should be removed only in the OR.
- In the ED, estimate tissue injury based on the above principles.
- Wound care includes appropriate exploration, irrigation, and debridement of devitalized tissue.
- All bullets are contaminated with bacteria and are **not** sterilized by being fired:
 - _ All nongrazing bullet wounds warrant empiric antibiotics.
- Early trauma, orthopedic, and vascular surgery consultation is necessary.

MEDICATION

Prophylactic antistaphylococcal antibiotics should be prescribed for several days:

- Cefazolin 1 g IV q6h
- Cephalexin 500 mg PO q6–8h

- For penicillin-allergic patients or patients at risk for methicillin-resistant *Staphylococcus aureus* then vancomycin 1 g IV q12h, clindamycin 300 mg IV/PO q6h, or sulfamethoxazole/trimethoprim DS 1 tablet BID can be prescribed
- Intra-abdominal wounds require broader coverage (many regimens available) such as cefotetan 1 g IV q12h, piperacillin/tazobactam 3.75 mg IV q6h, or the combination of ciprofloxacin 500 mg IV q12h with metronidazole 500 mg IV q8h



FOLLOW-UP

DISPOSITION

Admission Criteria

- Patients with neurovascular compromise and extensive tissue damage must be admitted for appropriate surgical intervention.
- Patients with nontrivial injury to the head, neck, torso, or abdomen should be admitted.
- Patients with injury from high-velocity projectiles or gunshot wounds should be admitted to a monitored setting for observation of neurovascular status.

Discharge Criteria

Patients with minor penetrating extremity trauma or stabbing victims found not to have significant injury may be discharged with appropriate follow-up.

Issues for Referral

Emergent consultation of appropriate surgical specialists should be obtained for patients with potential injuries to vascular or nervous structures.

FOLLOW-UP RECOMMENDATIONS

Patients not admitted to the hospital should have scheduled follow-up with a trauma surgeon or an appropriate surgical specialist (e.g., orthopedist for extremity trauma).

PEARLS AND PITFALLS

- Do not underestimate the extent of underlying tissue damage or injury to critical structures given the size or location of entrance or exit wounds.
- Account for all projectiles and all injury tracts.
- Gunshot and stab wounds are usually reportable to local law enforcement.

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CODES

ICD9

879.8 Open wound(s) (multiple) of unspecified site(s), without mention of complication

ICD10

T14.8 Other injury of unspecified body region

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