URINARY DISEASES OF EQUINES

EQUINE MEDICINE PART - I

MODULE No. – 13 Course Code – Vetm4146

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Function of kidney in horses

- Blood arrives in the kidneys via the renal artery--a branch of the aorta--and is processed in two layers of kidney tissue(Medula and Cortex).
- Within the tissue, more than a million microscopic units called nephrons handle the purification duties.
- Only about 25 to 30 % of the nephrons actively process fluid full-time.
- The remainder stay as stand by.
- In case of an increase in blood flow, which may be triggered by illness, excitement or cold temperatures the remainder nephrons start working.
- Additional nephrons also may be called into service when a horse consumes more water than usual.
- The kidney is the organ which detoxifies the body.
- If the kidney is ill, harmful substances stay, and other organs get damaged.
- **Indeed horses do not often have an inflammation of the kidney.**
- Each nephron begins at a tuft (Bunch) of capillaries on one of the tiny branches of the renal artery, Known as the glomerulus.
- Glomerulus is surrounded by a cup-shaped funnel, called Bowman's capsule, that receives the watery part of the blood for processing.
- Molecules of protein, fats and blood cells that are too large to fit through the filters of Bowman's capsule are rejected and remain in the circulation.

Function of kidney in horses

- The fluid that passes through the capsule moves into a long winding (Curly) tube called the loop of Henle.
- Here, appropriate amounts of salt, potassium, sulfate, phosphate, glucose and amino acids are extracted and returned to the blood to meet the requirement and deficits.
- Water that is needed by the circulatory system is absorbed at the end of the nephron.
- The waste substances, such as creatinine, urea, uric acid, pigments and excess salts and water are sent to the bladder to be excreted out.
- Almost 99 % of the filtered blood is reabsorbed.
- Remaining blood constituents are unwanted and excreted out in the urine.
- Any waste that is too large to fit through the kidneys' tubes is rerouted to the liver or the gut wall, where it is processed for excretion with solid feces.
- **The entire blood volume in horses (on average, about 9 gallons or 35lits) passes through the kidneys more than 60 times in 24 hours.**
- As the kidneys carry out their basic filtration duties they also monitor and regulate fluid volume and composition.
- Kidney promote red blood cell production, modulate blood pressure and control the blood's pH (acidity).

Function of kidney in horses

- Most important function of the kidney is detoxification of the blood.
- **Every day nearly 6600 liters blood flow through kidney of the horse.**
- Kidney filters the blood and cleans it from degradation products of several metabolic processes.
- Especially the final degradation product of the protein metabolism, so called as urea, gets separated from the blood and excreted out.
- The urea accumulates inside the kidney (renal pelvis) before it leaves the body through the ureter and the bladder.
- Thus kidney detoxifies the body from harmful and dangerous substances.
- Additionally it produces two hormones**(Erythropoitin and Renin**).
- Reabsorption of water is by anti diuretic hormone (ADH).
- *It is* released by t he posterior pituitary gland.
- Kidney secretes two important endocrine substances:
- Erythropoietin – Help in RBCs synthesis.
- Renin: Regulates aldosterone secretion by adrenal cortex.
- In poor blood supply to kidney, Juxtaglomerular stimulate RAAS(Renin Angiotensin Aldosterone System) to produce Renin which convert Angiotenogen to Angiotensin-I.
- ACE inhibitors convert Angiotensin-I to Angiotensin –II.
- Angiotensin –II is a vasoconstrictor and cause ARF.

Drug Selection for Equine Bacterial Pathogens

Gram-Positive:

- *Rhodococcus* equi--- Erythromycin +/- rifampin
- *Streptococcus* spp --- Penicillin G, ampicillin, ceftiofur
- *Staphylococcus aureus* ----Trimethoprim-sulfonamide

Gram-Negative:

- *Escherichia coli* ---- Gentamicin, amikacin(With caution).
- *Klebsiella pneumoniae* ---- Gentamicin, amikacin
- *Enterobacter* spp. ----- Gentamicin, amikacin
- *Pseudomonas aeruginosa* ----- Gentamicin, amikacin, ticarcillin
- *Pasteurella* spp. ----- Ampicillin, ceftiofur, trimethoprim + sulfonamide
- *Actinobacillus* spp.- Ampicillin, penicillin,trimethoprim +sulfonamides

Renal failure in Horses

- According to Schott, acute **renal failure** is most often **caused** by a loss of blood volume due to colic, diarrhea, hemorrhage or severe dehydration in horses.
- Ingested toxins and antibiotics administered to a dehydrated **horse** also may cause **Renal failure**.
- Long use of Aminoglycosides(Nephrotoxic) damage kidney.

It is of Two Type :

1. Acute Renal Failure (Reversible)
2. Chronic Renal Failure(Irreversible)

Renal failure in Horses

- Horses eat too much proteins which causes increased quantity of urea.
- Kidney needs to decompose overdose of urea and becomes overloaded.
- Additionally a wrong dose of calcium and vitamin D can cause Renal failure.
- An overdose of Ca and vit D causes lumpy deposits between kidney cells and causes kidney damage.
- Less water, colic or heart problems cause a disturbed circulation in kidney.
- Oxygen supply gets interrupted, that causes an immense damage of cells.
- Any damage of the tissue cells is not curable. **Chronic kidney disease is an irreversible condition.**
- **Acute kidney disease is a reversible condition.**
- **Chronic kidney disease is an irreversible condition.**

Acute Renal Failure

- Primary renal disease remains uncommon in equid.
- Acute kidney injury (AKI) with other diseases cause mortality in equides.
- 2,000 horses presented to Michigan State University's Veterinary Medical Center each year suffering from ARF or CRF.
- Approximately **4% of horses** have increased amounts of waste products in their blood due to kidney insufficiency.
- Azotemia [excess nitrogen-type wastes in the bloodstream] develop.
- With a serum creatinine concentration [Cr] > 2.5 mg/dL [220 µmol/L]), and 1% horses have more severe azotemia with Cr levels exceeding 5.0 mg/dL (440 µmol/L).
- **1mg/L = 88 µmol/L**

Causes of Acute Renal Failure in Horses

Three Types Prerenal Failure are :

1. Pre Renal failure
2. Renal failure
3. Post renal Failure

1. **Prerenal Failure:**

- Functional decrease in glomerular filtration rate associated with renal hypoperfusion.
- Hypotension and/or hypovolemia associated with the following:
  1. Gastrointestinal fluid losses (colic, enterocolitis)
  2. Acute blood loss
  3. Exercise-associated sweat losses
  4. Sepsis/endotoxemia
  5. Volume redistribution (severe hypoalbuminemia; pleural or peritoneal effusion)
  6. Disseminated intravascular coagulation(DIC)

Causes of Acute Renal Failure in Horses

2. **Renal Failure**:

- Acute tubular necrosis secondary to the following:
- Profound or persistent renal hypoperfusion lead to ischemic necrosis (continuum from prerenal failure).
- Especially in horses receiving nephrotoxic agents in the face of inadequate fluid replacement.

Nephrotoxins for Horses:

1. Antimicrobial agents (aminoglycosides,tetracyclines)
2. Heavy metals (mercury, arsenic, gold, lead)
3. Endogenous substances (myoglobin, hemoglobin)
4. Miscellaneous (NSAIDs, vitamin D and vitamin K3)

- Interstitial nephritis or glomerulonephritis secondary to bacterial infections *(Leptospirosis pomona, Actinobacillus equuli* in neonates.
- Nephrolithiasis / ureterolithiasis.

3. Post renal Failure: Urinary bladder rupture (uroperitoneum) in neonates (rarely in postpartum mares).

Chronic kidney disease (CKD) or CRF

- Chronic kidney disease (CKD) develop as a consequence of incomplete treatment of AKD leading to tissue degeneration.
- Fibrosis (>90% of cases) or immune-mediated renal disease(<10% of cases).
- Rare cases associated with anomalies development, such as:
    1. Renal aplasia (one or both fetal kidneys failing to develop).
    2. Hypoplasia (abnormally small kidneys).
    3. Dysplasia (kidney malformation)
    4. Polycystic kidney disease (an inherited disorder in which clusters of cysts develop in the kidneys).
- Presenting complaints for CKD are vague and include decreased performance or loss of function of kidney.

Chronic kidney disease (CKD) or CRF

- By nature CKD is a progressive disease but varies widely between horses.
- Supportive treatment is focused on maintaining body condition through nutrition (pasture is the ideal diet)
- Avoiding use of nephrotoxic medications.
- In horses with immune-mediated renal disease and proteinuria, the treatment with corticosteroids and angiotensin-converting enzyme(ACE) inhibitors could also be helpful.
- Supplementation with omega-3 fatty acids slows progression of CKD.
- Pasture is an ideal source of omega-3 fatty acids.
- Long-term prognosis for CKD is poor.
- Horses can do well for months to several years.

Immune-mediated renal diseases

- Glomerulonephritis is a renal disease in which immune-mediated glomerular damage is the initiating factor.
- The hallmark of glomerulonephritis is increased permeability of the glomerular barrier.
- Proliferative glomerulonephritis is characterized by proliferation of the mesangial cells with an influx of inflammatory cells(IL-I ,IL-II).
- Membranous glomerulonephritis is characterized by accumulation of matrix and thickening of the glomerular basement membrane (GBM) and capillary wall.
- **Mesangial cells** are the glomerular capillary equivalent to the smooth muscle cell and in that capacity respond to injury in a similar fashion.

Chronic kidney disease (CKD) or CRF

- Lower urinary tract disorders— Like bladder stones (cystolithiasis), urinary incontinence, and blood in the urine (hematuria)—are actually more common than upper tract disease.
- Considering that horses normally excrete large amounts of calcium carbonate and oxalate crystals in urine.
- It is somewhat surprising that urinary stones (urolithiasis) are less common in horses than dogs and cattle.
- Cystolithiasis in equids have a recurrence rate approaching 50%.
- Suggesting a genetic predisposition in affected horses.

Chronic kidney disease (CKD) or CRF

- Mares with bladder muscle dysfunction due to foaling or trauma
- Neurological disease, develop progressive bladder enlargement and overflow incontinence.
- Affected horses can also accumulate a large concretion of urine crystals into a mass that can be confused with a cystolith i.e Urolith in bladder (Sabulous urolithiasis).
- Sabulous urolithiasis can be treated by dissolution through bladder lavage.
- Affected horses should not be subjected to an unnecessary cystotomy surgery.

Causes Of ARF and CRF

- Dehydration
- Heat stroke
- Natural toxins : When the cells are disabled, the loop of Henle cannot distinguish what to absorb or what to excrete ultimately wastes remain in the body.
- Aminoglycoside antibiotics, nonsteroidal anti-inflammatory drugs (NSAIDs), vitamin D and vitamin K are toxic in high doses.
- Massive blood loss.
- Shock : depresses blood flow to the kidneys.
- Colic
- **Bacterial infection,** which can damage the kidney cells.
- The resultant inflammation may affect the tubules to the point where plasma water can no longer pass through.
- **Chronic urinary passage obstruction(Uroliths).**

Symptoms for an inflammation of the kidney

- Fever
- Restlessness
- Stick walk
- Physical weakness/ tiredness
- Lack of appetite
- Loss of weight
- Swelling of certain parts of the body
- High blood pressure

Diagnosis of a kidney disease

- Blood screening test like creatinine, urea and certain electrolytes.
- Beside blood screening test , ultrasonic examination.
- Ultrasonic examination shows the shape of the organ might help to see external changes (caused by a tumor or kidney stones).
- X-rays .
- The range of a normal creatinine level is less than 1.8 to 2.0 mg/dL.
- Severe kidney dysfunction serum cr level go upto 15 or 20 mg/dL.
- BUN : Normal - 20- 30 mg%
- Anemia, due to the destruction of red blood cells.
- Normally a horse Drink 50ml/kg/day
- A Horse Urinate 20ml/kg/day

Water Deprivation Test

- This test is performed in horses that are not azotemic or dehydrated.
- Initially horse is weighed and its bladder emptied with a urinary catheter.
- Water is withheld for 8 hrs.
- Urine specific gravity is measured at baseline and then every 6 to 12 hours.
- Water deprivation is stoped once horse becomes clinically dehydrated.
- In addition to urine specific gravity, urine osmolality can be measured to assess urine concentration.
- Urine osmolality should normally be three to four times of serum osmolality, which is 900 to 1200 mOsm/kg.
- **Horse**: Normal Urine Specific gravity(USGP) = G 1.025–1.060.
- Water deprivation increases urine osmolality as well as USPG in ARF, CRF .

Urine PH - Normal is 7.5-9.5
Specific Gravity – Normal is 1.020-1.060

Carry out water deprivation test for renal failure.
Pigmenturia: urine is normally straw colored.
Myoglobinuria: Dark coloured urine
Haemoglobin/blood (Hemoglobunuria)=Coffee coloured
Hematuria = (Red urine)

- Discolored urine can be caused by contamination with red blood cells, hemoglobin, myoglobin, oxidizing agents normally found in urine, and plant-derived pigments.

TREATMENT

- I/V Fluid therapy to maintain dehydration and flushing toxic materials
- Blood transfusion or Plasma Transfusion if severe anemia.
- Diuretics as per condition.
- Mostly Loop diuretics (Lasix) are recommended.
- No diuretics in urolithiasis.
- Vitamin B complex and anabolic steroids may help boost cell production and prevent muscles from wasting away.

Horse Shoe Kidney

- It occurs during fetal development as the **kidneys** move into their abnormal position.
- With **horseshoe kidney**, as the **kidneys** of the fetus rise from the pelvic area, they become attached ("fuse") together at the lower end or base.
- By fusing, they form into a U shape, **like** a **horseshoe**.
- Hydronephrosis.
- Enlargement of the kidneys that is usually the result of a **urinary** tract obstruction.
- Wilm's tumor - an embryonic (newly-formed) tumor of the kidneys that usually occurs during early age of **renal** cancer.

PHIMOSIS IN HORSES

Phimosis refers to the inability of the **horse** to protrude its penis from the prepuce because of a congenital or acquired stricture of preputial orifice or preputial ring.

- Trauma or chronic infection may cause inflammation or edema of prepuce (posthitis Penis: balanoposthitis) → stenosis of preputial orifice.
- **Horses** castrated when young may fail to protrude penis when urinating → inflammation → scarring → constriction of preputial orifice → **phimosis**.
- **Cause**: stenosis of the preputial orifice, eg congenital stricture, trauma, edema, inflammation.
- **Signs**: inability to protrude penis from the sheath, inflammation of the prepuce, excessive smegma accumulation.
- **Diagnosis**: signs, biopsy, microbiology, serology and parasitology.
- **Treatment**: depends on cause. surgical.
- **Prognosis**: guarded to fair - depends on cause.

Paraphimosis in Horses

Paraphimosis is the inability of the horse to retract its penis into the preputial cavity.

- It most commonly occurs in stallions as a result of breeding trauma, but geldings can also be affected.
- Penile prolapse occurs initially, which then results in excessive edema and swelling of the penis and prepuce.
- The condition is probably more common in the horse than any other domestic animal with the possible exception of the dog.
- **The causes of paraphimosis can be either inflammatory or non-inflammatory.**
- It may occur in horses debilitated by severe infections, febrile diseases.
- Senility in stallions may be responsible for the condition.
- Paralysis of retractor muscles following injuries of the posterior spinal cord.
- Tumor formation either of benign or malignant type may cause the penis to become too large to be withdrawn into the sheath.
- The most common cause is injury to the penis or preputial folds.
- Such injuries may be caused by false copulation in which the penis strikes the pelvis of the female, striking with a whip, or kicking.
- **The most common method of treatment in the many cases is amputation of penis.**

NEOPLASIA IN URINARY TRACT

- Primary neoplasms of the equine urinary tract are uncommon.
- Published cases document mainly malignant epithelial tumors of the kidney and urinary bladder.
- Renal carcinoma (or renal adenocarcinoma) is the most common form of primary upper urinary tract neoplasia in horses.
- Renal carcinoma can be locally invasive and/or metastasize to various organs.
- Therefore, the prognosis is generally poor.

CYSTITIS IN HORSES

Bacterial cystitis in horses is usually a complication of followings:

- Urolithiasis
- Bladder neoplasia
- Bladder paralysis
- Anatomical defect of the bladder such as:
- A persistent urachal remnant.

Clinical Findings:

- Dysuria may be manifested by pollakiuria, stranguria, hematuria or pyuria.
- Scalding(Burn) and accumulation of urine crystals may be observed on perineum of affected mares or on the front of the hind limbs of male.
- These findings should not be confused with normal estrus in a mare.
- Physical and rectal examinations and collection of a urine sample for urinalysis and quantitative bacterial culture.
- Although the bladder is usually felt to be normal during rectal palpation.
- Endoscopic examination of the bladder may be helpful in assessing mucosal damage caused by cystitis.
- **Blood at end of urination is typical sign of cystitis.**

Diagnosis

- Normal equine urine is rich in mucus and crystalloid material.
- Gross examination of urine may be unrewarding.
- However, sediment examination may reveal increased numbers of white blood cells (> 10 leukocytes per high power field).
- Presence of bacteria (>20 organisms per high-power field) in some, but not in all cases of cystitis.
- Quantitative culture results exceeding 10,000 organisms/ml in a urine sample collected by midstream catch.
- For best results, urine sediment should be evaluated within 30 minutes of collection.
- Samples for culture should be cooled during transport, because bacterial numbers may increase in samples left at room temperature.
- Organisms that may be recovered on culture include *Escherichia coli* and species of *Proteus, Klebsiella, Enterobacter, Corynebacterium, Streptococcus, Staphylococcus,* and *Pseudomonas.*
- Isolation of more than one organism is not uncommon.

Treatment

- Successful treatment of bacterial cystitis requires correction of predisposing problems such as urolithiasis.
- Selection of an antibiotic is ideally based on the results of sensitivity testing of isolated organisms.
- Initial course of treatment should not be less than 1 week.
- A trimethoprim/sulfonamide combination is given.
- Ampicillin, penicillin and an aminoglycoside, or ceftiofur are recommended.
- If signs return after treatment is discontinued, a urine culture should be repeated and longer-term treatment be instituted.

Treatment

- Trimethoprim@5mg/kg and sulfonamide@25mg/kg with combinations of penicillins@2000 IU/kg are excreted through the kidneys and are concentrated in urine.
- Although sensitivity testing may indicate resistance, these agents may have effective antimicrobial activity against the causative agents because of the high concentrations achieved in urine.
- Sulfamethoxazole is largely metabolized to inactive products prior to urinary excretion.
- Sulfadiazine is excreted largely unchanged in urine and is effective.
- Addition of 50 g to 75 g salt to the diet or warm water during cold weather increase water intake and urine production.
- This has a benefit in cases of bacterial cystitis to flush out bacterial load.

Treatment

- Urine acidification is necessary for treatment of cystitis in horses.
- Use of ammonium chloride at a dose of 520 mg/kg per day orally, or
- Ammonium sulfate at 175 mg/kg per day orally is successful in reducing urine pH to below 6.0.
- Between a **pH** range of 5.0-6.5, urine has a **antibacterial** activity.
- At these doses, the oral medications are difficult and had to be administered by dose syringe.
- Addition of grain to diet is another simple way to decrease urine pH.
- Horse urine pH typically remains higher than 7.0.

UROLITHIASIS ,URETEROLITHIASIS AND NEPHROLITHIASIS

- Nephrolithiasis **is stone in Kidney.**
- Ureterolithiasis is stone in ureter.
- Nephroliths may develop around a nidus associated with a variety of renal diseases, which include:
- Pyelonephritis, renal tubular or papillary necrosis, and neoplasia.
- It has been speculated that race horses are at greater risk for this disease because of the common use of NSAIDs.
- Horses with nephrolithiasis or ureterolithiasis often remain asymptomatic until bilateral obstructive disease results in acute or chronic renal failure and renal pain.

Nidus formation:

- A nidus favors the deposition of crystals around itself.
- A nidus may be a group of desquamated epithelial cells or necrotic tissue.
- It may be formed as a result of occasional infection in the urinary tract.
- Vitamin A deficiency- It causes desquamation of epithelial cells around which crystals deposit.
- Nonspecific signs of uremia such as poor performance, inappetence, lethargy, and weight loss are observed more commonly than signs of obstructive disease, including colic, stranguria and hematuria.
- Rectal palpation may reveal an enlarged kidney or ureter and in some instances the calculus can be palpated in the enlarged ureter.

UROLITHIASIS, Diagnosis (USG)

- The diagnosis is made based on rectal and **transcutaneous** ultrasonographic examination findings.
- Calculi are detected as **hyperechoic** structures with a strong anechoic shadow on ultrasonographic examination.
- Small stones may be missed on ultrasonographic examination.
- Other findings such as dilation of the renal pelvis, proximal ureters, or hydronephrosis, indicate upper urinary tract obstruction.
- Infection often accompanies urolithiasis, a quantitative urine culture should be performed in all cases.
- **Ultrasound** is a very good tool to direct the diagnostic pathway.

Some Ultrasound terms:

- **Hyperechoic** – more **echogenic** (brighter) than normal.
- **Hypoechoic** – less **echogenic** (darker) than normal.
- **Hypoechoic** literally **means** that it **does** not bounce back sound waves very well (**does** not echo the sound).
- Anechoic: Deaden sound. It means that the cyst absorbs the sound waves of the ultrasound and does not bounce them back.

UROLITHIASIS

- Most horses are in chronic renal failure at the time of diagnosis of upper urinary tract lithiasis.
- Treatment includes nephrectomy, nephrotomy or ureterolithectomy.
- Surgical treatment does not seem to improve.
- Few cases are managable to medical treatment.
- Less invasive treatments have been attempted in more recent years.
- A distal ureteral calculus was removed using a basket stone dislodger through a vestibule urethral approach in a mare.
- Electrohydraulic lithotripsy through a ureteroscope has been used successfully in a horse with a single unilateral ureterolith.
- A more recent means of upper urinary tract stone removal is Extracorporeal shock wave lithotripsy (ESWL).
- Extracorporeal shock wave lithotripsy(ESWL) is not useful for horses.
- It is useful for human beings.

PYELONEPHRITIS

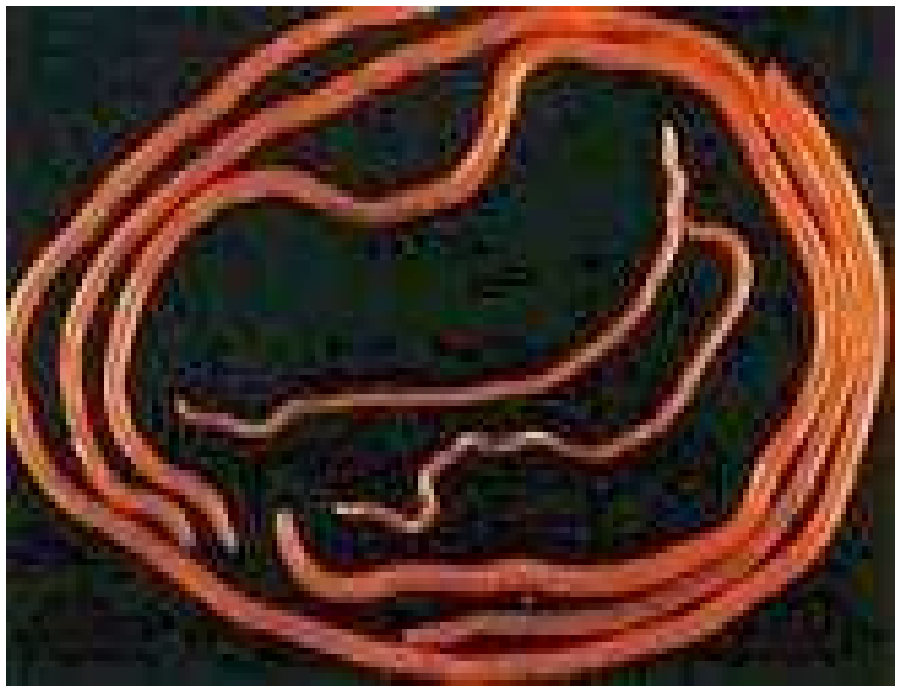
Definition: Pyelonephritis is a bacterial inflammation of renal parenchyma and renal pelvis.

- Pyelonephritis develops most commonly from ascending infection through the lower urinary tract.
- It is characterized clinically by pyuria, dysuria or stranguria(Difficult dribbling), suppurative nephritis, cystitis and ureteritis.
- Pyelonephritis is usually caused by a bacterial infection that climbed into the bladder and then later into the kidneys (Pelvis).

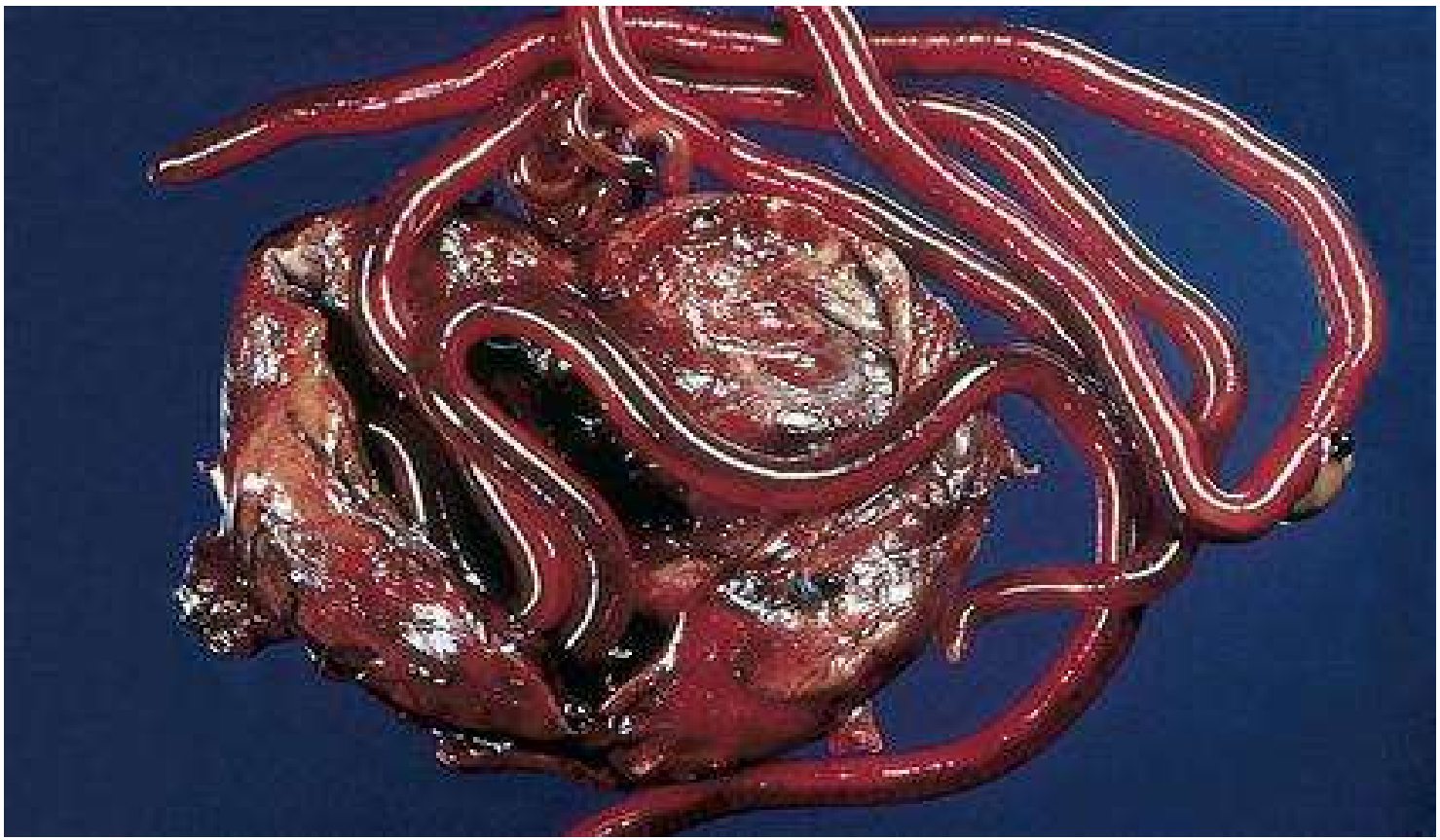
Etiology

- In most cases it results following bacterial infection of the lower urinary tract.
- Hematogenous spread of bacteria causing pyelonephritis.
- Septicemia of cattle caused by *Pseudomonas aeruginosa*.
- E.coli infection most common in man and dogs.
- Specific pyelonephrit is caused by *Corynebacterium renale* in cattle.

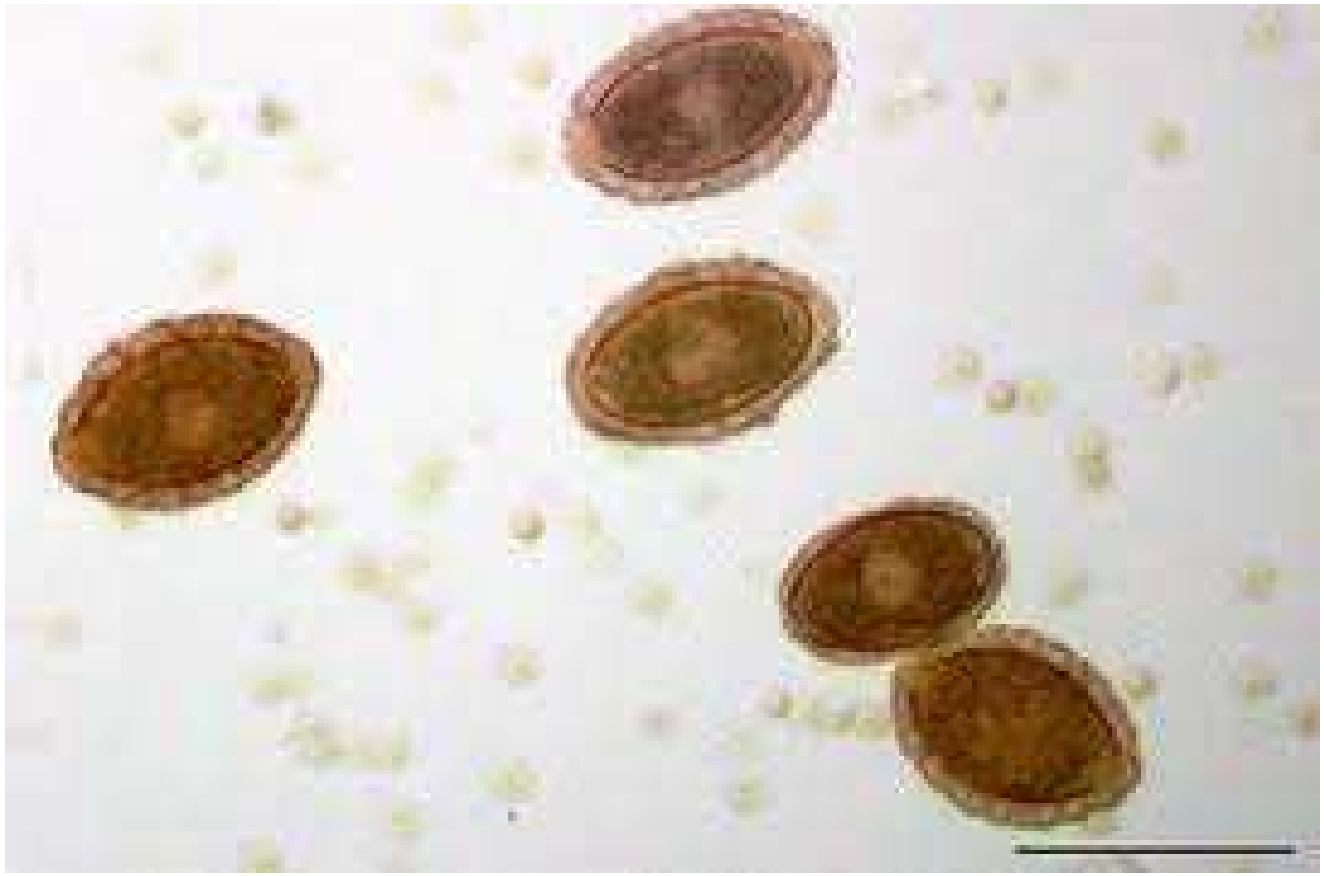
Kidney Worms (Adult)



Dioctophyma renale frequently infects the right kidney of dogs and ingests the entire parenchyma, leaving only the capsule of the kidney.



Dioctophyma renale ova in affected animal urine sediment



Transmission

- Transmission is by direct contact (such as tail switching).
- Service by infected or contaminated stalion.
- Poor urinary catheterization techniques.
- Poor management
- Urine deposit in ditches in mare houses.

Pathogenesis

- Pyelonephritis results from an ascending UTI.
- Urinary obstruction (urolithiasis, prostatic hyperplasia) will lead to urinary stasis.
- This predisposes the animal to bacterial cystitis.
- Once an infection is established in bladder, it may ascend up the ureters via **vesico ureteral reflux.**
- Once the renal pelvis is infected, bacteria gain access to the parenchyma via the collecting ducts.
- This is how pyelonephritis develop.
- **Vesicoureteral reflux** (**VUR**) is the backward flow of urine from the bladder into the kidneys.
- Normally, urine flows from the kidneys through the ureters to the bladder.
- The muscles of the bladder and ureters, along with the pressure of urine in the bladder, prevent urine from flowing backward through the ureters.

Pathogenesis

- In severe cases of reflux, bacteria present in urine, flows up into the **proximal and distal** tubules via the collecting ducts.
- This carry bacteria up to the urinary space of **glomeruli.**
- If the infection is from bacteria it may reach to tubules through glomerulus reflux.
- **This glomerulus reflux is called intra renal reflux.**

Development of pyelonephritis depend up on:

1. Common presence of infection in urinary tract.
2. Stagnation of urine.
3. Reflux of urine from the bladder to ureter then to kidney.

Urine stasis can occur as a result of:

- Blocking of the ureters by inflammatory swelling or debris.
- Pressure from the uterus in pregnant mare.
- Obstructive urolithiasis.

Pathogenesis

- Infection ascends ureters and invades renal pelvis.
- Extends to involve the medulla and cortex of kidney.
- Signs of toxemia and fever, usually accompany extensive bilateral infection.
- Uremia may develop if the lesions are sufficiently extensive.
- Pyelonephritis is always accompanied by hematuria.
- It is due to Inflammatory lesions of ureters and bladder.

Clinical findings

- First sign passage of blood stained urine in some cases.
- The first sign may be an attack of colic in horses.
- Swishing of tail, kicking at abdomen and treading (walking in Specific way) of the feet.
- Straining to urinate.
- Such attacks are caused by obstruction of the ureter or renal calyx by pus or tissue debris.
- Pyelonephritis (along with toxic nephrosis) is one of clinically recognized renal disorders of horses.

Treatment

- On the basis of culture and drug sensitivity test.
- If the **nephrotic syndrome** is manifested, it should be treated with diuretics and through dietary salt restriction.
- Fluid therapy.

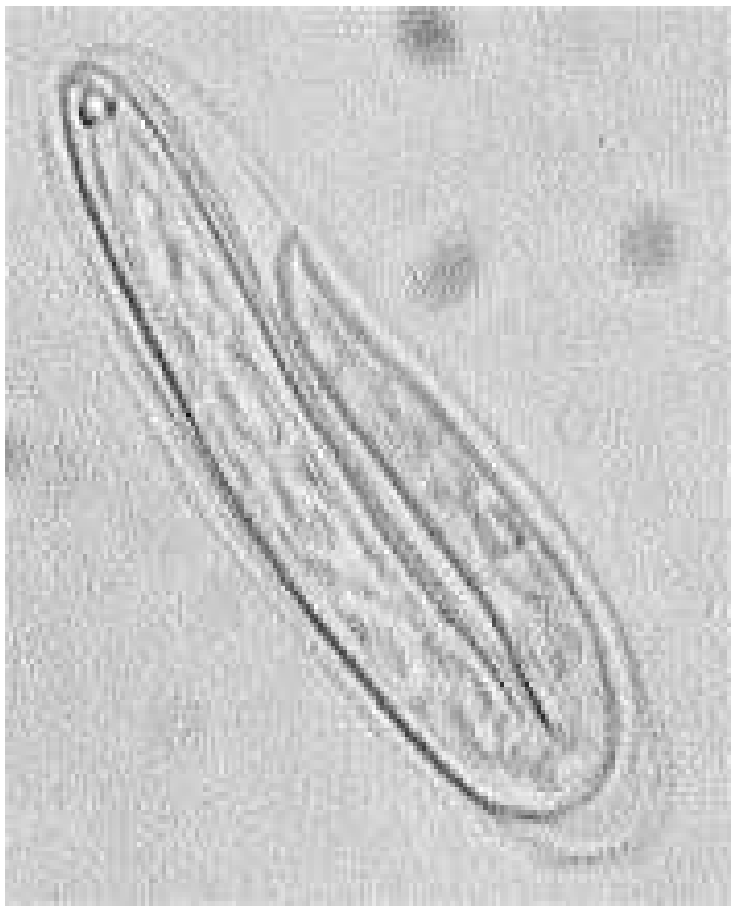
CUTANEOUS HABRONEMIASIS (SUMMER SORE) IN HORSES

- "**Summer Sores**" or "Fly **Sores**" is a seasonal skin disease in **horses** referred as Cutaneous Habronemiasis.
- It is caused by infection of the skin by the larvae of the large-mouth stomach worm Habronema (and Draschia).
- **Summer sores in horses** can occur any time of year.
- During warm months they tend to become more prevalent.
- This is because biting insects are at their peak of breeding.
- Insect leave larvae of stomach worms in bite wounds that they carry.

SUMMER SORE IN HORSES

- Summer sores are a massive inflammatory reaction to the larvae of the equine stomach worm , Habronema muscae.
- Worms deposited onto wounds and muco cutaneous junctions including the sheath, eyes and corners of the mouth.
- This reaction then forms granulomas: This is called “summer sore”.
- Adult stomach worms shed larvae into the environment through the manure of infected horses.
- These larvae are then ingested by maggots and develop within these maggots as adult flies.
- Habronema reside in mouth parts of adult flies and they deposit on the wounds or mucocutaneous junctions of horses causing an infection.

Stomach worm(Habronema muscae)



SUMMER SORE IN HORSES

- Horses can ingest these larvae by consuming dead flies in feed or water.
- The larvae continue development in the stomach of the horse and begin laying eggs within eight weeks but cause very little clinical signs in the horse.
- Usually, the horses shed the eggs in their manure to contaminate pasture.
- Some horses are shedders, but even a wormed horse can get summer sores.
- This is because the larvae that cause the massive inflammatory reaction are actually already dead.
- It is the body's immune response to the dead larvae that causes the intense itchiness and summer sore formation.
- The intensity of these wounds is very intensive.

SUMMER SORE IN HORSES**(Control)**

- Removal of the dead larvae from the sore.
- Usually this is aggressive and has to be repeated.
- The area needs to be covered at all times if possible and a medicated ointment applied daily on the body of horses.
- Cryotherapy has also proven successful in some cases.
- This involves the freezing of summer sores with liquid nitrogen.
- Success by using **immune mediating products."**
- An oral deworming product is often administered to the entire stable of horses when a summer sore is seen.
- Deworm all horses if carrying the adult Habronema worms in the stomach.
- Fecal worm egg counts do not detect Habronema larvae.

SUMMER SORE IN HORSES(Control)

- Use of solution containing **Cyromazine** in stable for fly control.
- **Cyromazine** is a triazine insect growth regulator used as an insecticide and an acaricide.
- **Cyromazine** works by affecting nervous system of the immature larval stages of insects.
- In veterinary medicine, **cyromazine** is used as an ectoparasiticide.
- This prevents the larvae from developing exoskeletons so they cannot become adults.
- It does not get absorbed by the horse but passes through their digestive tract to be passed in their manure.

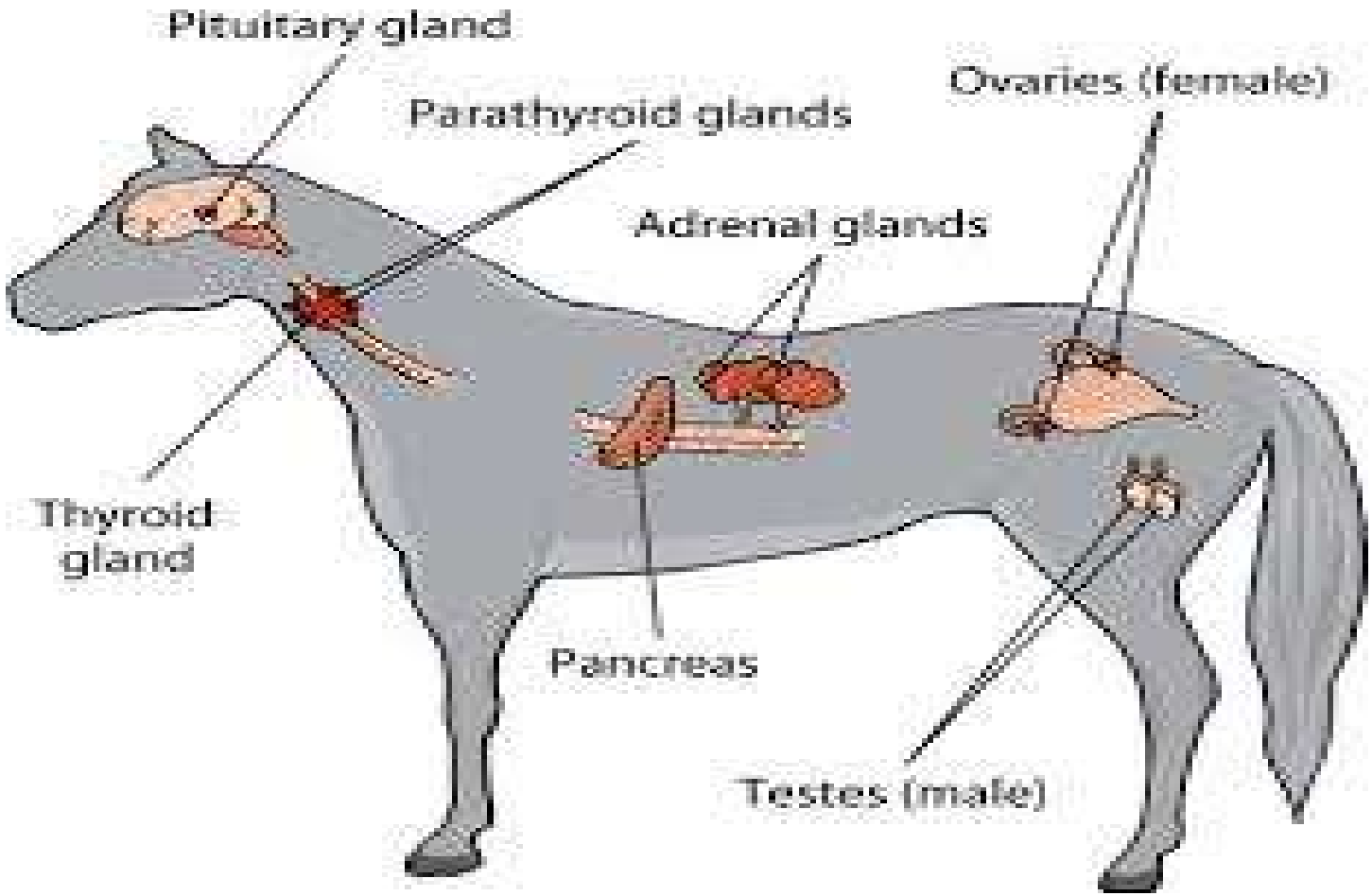
SUMMER SORE IN HORSES(Control)

- The maggots then ingest the cyromazine while feeding on the manure and the immature fly will die before spreading Habronema larvae.
- This significantly reduces the fly population in just 4 to 6 weeks.
- Removing manure as often as possible from the stall and keeping it away from the stable
- Open wounds should also be covered as soon as they appear.

DISORDES OF ENDOCRINE SYSTEM

- Endocrine system consists of a number of glands.
- These glands produce and secrete hormones which control the body's metabolism, growth, sexual development and function.
- When the hormones leave the glands they enter the blood stream and are transported to organs and tissues in every part of the body.

Endocrine Glands of Horses



Adrenal glands (suprarenal glands)

- Located at the top of the kidneys.
- Adrenal glands secrete catecholamines.
- Adrenal glands are divided into 2 regions,right gland is triangular while the left one is semilunar in shape.
- These glands secrete corticosteroids and catecholamines, such as norepinephrine and adrenaline (epinephrine)
- These hormones are released due to stress.
- **Adrenaline** (epinephrine) is the neurotransmitter of the adrenal gland that is secreted in moments of crisis.
- It stimulates the heart to beat faster and work harder.
- The adrenal glands also produce androgens, male sex hormones that promote the development of male characteristics.
- **Testosterone** is the major androgen.
- These glands produce aldosterone which affects kidney function.
- The adrenal glands also produce androgens, male sex hormones that promote the development of male characteristics.

Hypothalamus:

It is located just above the brain stem, below thalamus.

- This gland activates and controls involuntary body functions, **appetite, sleep**, temperature, as well as the circadian cycles(24hrs body physiology).
- The hypothalamus links the nervous system to the endocrine system via the hypophysis (pituitary gland).
- Parathyroid Glands and Pineal gland:This is a small endocrine glands located in neck.
- They produce parathyroid hormone, which regulates calcium and phosphorous in the blood, blood clotting, and neuromuscular excitation.

Pineal body (Pineal gland):

- A small endocrine gland located in the brain.
- It secretes melatonin, and is probably involved in controlling the body's sleep patterns.

Thyroid gland:

An endocrine gland located just below the Adam's apple in the neck in man and neck in animals.

- It produces hormones that play a key role in regulating blood pressure, body temperature, heart rate, metabolism, and how the body reacts to other hormones.
- The thyroid gland uses iodine to manufacture hormones. The two main secreted hormones are thyroxine (T4) and triiodythyronine (T3).
- The thyroid gland also produces calcitonin, regulating calcium metabolism.
- Disorders of thyroid gland function in horses are uncommon.
- Not well documented, and in most cases incompletely understood.
- Hypothyroidism accounts for most cases described in horses.

Thymus gland:

- An endocrine gland located beneath the breast bone (sternum).
- T lymphocytes, types of immune cells, mature and multiply in the thymus gland early in life.
- After puberty the gland shrinks.
- The thymus gland plays a role in the body's immune system.

Pituitary Gland:

- An endocrine gland located just off the hypothalamus at the base of the brain (a protrusion off the hypothalamus).
- It is Known as the main endocrine master gland.
- It secretes hormones that regulate the functions of other glands.
- It maintain growth and several body functions.

Anterior Pituitary Gland:

Anterior Pituitary Gland secretes hormones that affect:

1. sexual development
2. thyroid function
3. body growth
4. skin pigmentation
5. adrenocortical function.

NB : If the anterior pituitary is underactive, it can lead to dwarfism in animals in early age.

Posterior Pituitary gland:

SWEAT GLAND

Sweat glands: They are simple tubular glands found in almost every part of the skin.

- About 2-4 million sweat glands distributed all over animal bodies).

Each gland consists of two parts:

1- A secretory portion: This lies deep in the dermis, where the tubule is twisted into a fairly compact tangle.

2- A duct portion: Passing outwards through the overlying dermis and the epidermis

SWEAT GLAND

There are two types of sweat glands :

1- The Eccrine sweat gland

2- The *Apocrine* sweat glands

1. **The Eccrine sweat gland** are located over entire body surface, except for lips,nipples and part of external genitals.

- Eccrine sweat gland are innervated by sympathetic nerves.
- Tiny ducts of the eccrine glands pass through the dermis and epidermis, open up and empty directly on to the skin.

They are active since birth producing an odorless, clear fluid which is sweat and is a mixture of water and salts .

2. The *Apocrine* sweat glands :

- ***Apocrine* sweat glands** are limited in their location to axillary, pubic, and perianal region , and armpits (less than eccrine glands).
- They are larger in size compared to the eccrine glands.
- They become active with the onset of puberty.
- They are associated with hair follicles and open up at the hair follicles.
- They produce a thick fluid, which in contact with bacteria on the skin's surface, produces a characteristic potent "body odor".

SWEAT GLAND

In Animals Non-primate mammals have eccrine sweat glands only on the palms and soles.

- Apocrine glands cover the rest of the body.
- Apocrine glands are not as effective as humans' in temperature regulation.
- Horses are exception in which it regulate temperature.
- Dogs and cats have apocrine glands that are specialized in both structure and function located at the eyelids (Moll's glands), **ears** (ceruminous glands).

Sweating control and its stimulation:

Sweating is controlled by a center in hypothalamus where thermosensitive neurons are located .

- Sweat glands are stimulated in response to:

  1-high temperature .

  2-exercise.

  3- hormones.

  4- emotional stress (emotionally induced) Normal

- Sweating is restricted to palms ,soles,armpits and forehead
- While temperature induced sweating causes sweating throughout the body.

SWEAT GLAND

Composition of sweat:

Eccrine sweat : It is composed mainly of water and 0.2 – 1% solutes :

- solutes are: sodium, potassium lactate, urea, ammonia, serine, ornithine, citrulline, aspartic acid, heavy metals, organic compounds, and proteolytic enzymes.
- Eccrine sweat secretions are isotonic which travels through the eccrine duct.
- Here NaCl and bicarbonate are actively reabsorbed (only about 25% of the sodium can be reabsorbed) .
- Apocrine sweat: It consists mainly of sialomucin (Its viscosity) and a large amounts of protein and steroids with unpleasant odours.

Mechanism of sweat secretion

Eccrine sweat:

- When the sweat glands are stimulated, the secretory portion of the sweat gland secrets a fluid called the primary secretion.
- This is absorbed by the gland cells from the interstitial fluid.
- Then it is produced and secreted into the gland lumen by active secretory activity of the epithelial cells lining , the coiled portion of the sweat gland.
- As the **precursor** flows through the duct portion, it is modified by the reabsorbtion of Na^+ and Cl^-.

Factors affecting the composition of sweat

- Animal sweat is highly variable, both between individuals and within an indivisuals.

Low sweat rate (Resting, At cool temperature):

- When the sweat glands are stimulated, only slightly, the **precursor** fluid passes through the duct very slowly
- More Na^+ and Cl^- are absorbed which reduces the osmotic pressure of the fluid leading to water reabsorption and concentrating sweat constituents.
- Thus at low sweating rates the sweat produced is low in volume and is more concentrated in its constituents, such as urea, lactic acid and K^+.
- But Less concentrated in Na^+ and Cl^- .

SWEAT GLAND

High sweat rate is due to work, exercise, and hot temperature.

- When the sweat glands are strongly stimulated large amounts of primary secretion is formed.
- It will flow too rapidly through the sweat duct reducing the chance of Na^+ and Cl^- reabsorption.
- Water reabsorption is also reduced leading eventually the production of a large volume but less concentrated sweat.

Eccrine glands:

It has three primary functions:

1. Thermoregulation: sweat cools the surface of the skin and reduces body temperature.
2. Excretion: Eccrine sweat gland secretion can also provide a significant excretory route for water and electrolytes.
3. Protection: Eccrine sweat gland secretion aids in preserving the skin's acid balance.

- It helps protect the skin from colonization from bacteria and other pathogenic organisms.

Appocrine glands:

- Appocrine glands have little physiological importance.
- Their importance is derived more from the negative effects of their product, from which unpleasant odours arise.

Sweating Abnormalities

1. Hyperhidrosis
2. Hypohidrosis
3. Anhidrosis

1. Hyperhidrosis:

- overactive thyroid gland (hyperthyroidism)
- Diabetes
- Certain medications.

2. Hypohidrosis: is a partial loss of sweating.

3. Anhidrosis : is a complete lack of sweating.

This can occur for a number of reasons:

- Some skin disorders
- Burns to skin that damage the sweat glands
- Underactive thyroid (hypothyroidism)
- Dehydration

ANHYDROSIS

- Equine anhidrosis has been recognized as a clinical condition of horses since 1920s.
- This condition is characterized by the inability of the horse to sweat effectively in response to appropriate Exercise.
- The disease is predominant in hot, humid climates.
- Any horse can be affected of any coat color, age, sex and breed.
- Increased frequency of anhidrosis in horses in training.
- Decreased frequency in adolescent(Immature) horses.
- Altered sweat gland function.

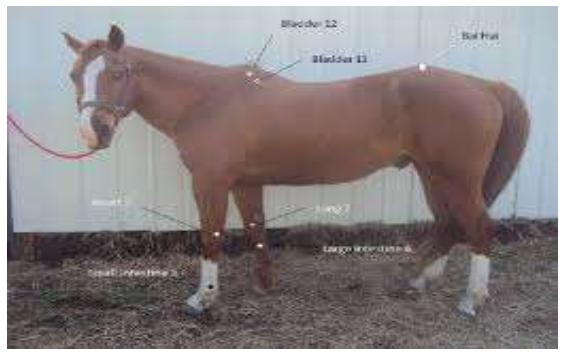
Pathophysiology of anhidrosis:

- Potential down-regulation or desensitization of the sweat gland B2-receptors may be responsible for anhydrosis.

CLINICAL SIGNS

- Tachypnea is probably the first indication.
- Overt exercise intolerance.
- Inappropriate tachypnea at the outset of anhidrosis.
- Horses with severe anhidrosis may be tachypneic at rest.
- Horses after exercise:
- Respiratory rates is high – 120/ minute.
- A normal horse should cool down within 30 minutes.
- If temperature remains elevated for longer than 30 minutes, it is suspected for anhidrosis. Anhydrosis is mis diagnosed as Respiratory diseases.
- In some horses ability to sweat may be lost completely.
- In partial anhidrosis sweating in some body areas.
- Under the mane, in the inguinal, axillary, and the perineal regions.
- Chronic cases of anhidrosis reveal a dry, flaky skin with areas of alopecia.
- Alopecia around the face and along the neck.
- latter anorexic, lethargic and no water intake.
- **Thyroidectomized (Removal)** horses sweat normally.

ANHYDROSIS




Typical areas of sweat patches after exercise
neck
buttock
throat
flank

DIAGNOSIS

- Definitive diagnosis is on intradermal testing to evaluate the sweating response.
- Intradermal testing: At sites on the neck below the mane.
- Salbutamol sulfate or terbutaline sulfate are injected intradermally in dilutions of 10- 3 w/v to 10-8 w/v.
- It is compared with a negative control injection of physiologic saline.
- Results are evaluated 20 to 30 minutes later.
- Sweating will occur at all sites except the control site in normal horses.
- Horses with severe anhidrosis may not sweat at any of the dilutions.
- Whereas horses with partial anhidrosis may respond at the 10- 4 w/v and 10- 6 w/v sites

TREATMENT

- At present no effective treatments exist.
- Management and environmental control are most appropriate way to resolve anhidrosis.
- Physical activities should be restricted.
- Well ventilated faned room
- No concentrates feed.
- Potassium salts (60 g of KCl -oraly) daily. Methyldopa has been used by some practitioners with reported success.
- An initial dosage of 3000 mg every 24 hours is used.
- Increased to 4000 mg every 24 hours if no positive response occurred in 3 to 4 days.
- This problem of anhydrosis may be life long in horses in humid climate.

Cushing’s Disease

The most common hormonal disorders in horses affect the adrenal glands and produces diseases are:

1. Addison's disease.

- It is also called as **Hypoadrenocorticism.**
- It is caused by a deficiency of adrenal gland hormones.
- It is seen occasionally in horses.

2. Cushing's disease (Hyperadrenocorticism) common in horses.

3. Hypothyroidism and goiter

4. Diabetes mellitus(Pancrea).

Cushing's Disease

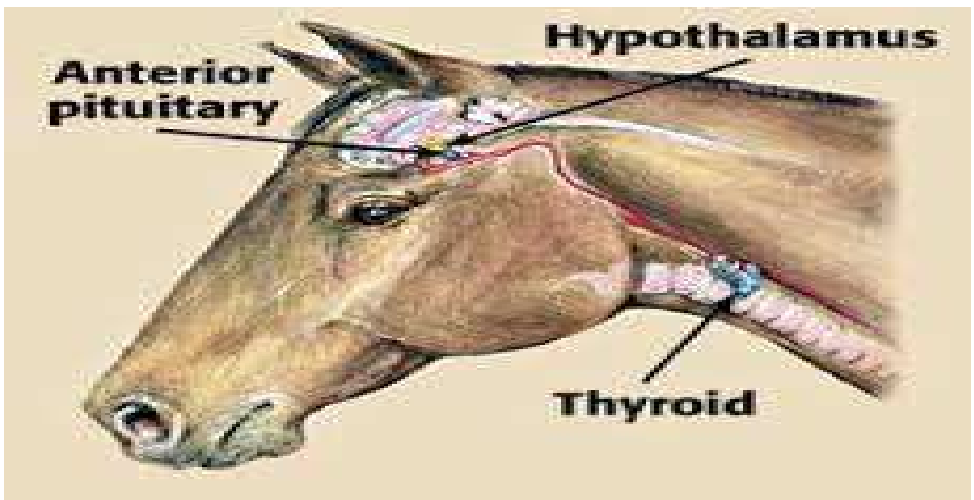
- Equine Cushing's disease occurs when a tumor called a pituitary adenoma develops in the *pituitary gland*.
- Hyperplasia or adenoma formation in the *pars intermedia* of pituitary gland is Cushing's disease.
- As this tumor slowly grows, it sends inappropriate signals to the rest of the body to secrete excessive hormones.
- It is now called as Pituitary Pars Intermedia Dysfunction (PPID).

Cushing’s Disease

- In humans, Cushing's disease is most commonly attributed to a corticotroph adenoma in the ***pars distalis* of the pituitary gland.**
- Parkinson's disease iis called in man.
- It is not called equine Cushing's disease.
- All breeds and types are affected.
- Ponies are at greatest risk.
- Cushing’s is usually seen in horses and ponies over the age of 15 years.
- Often in their 20s or 30s of life.
- Mares and geldings are equally affected.

Etiology

- The cause of Cushing's disease in horses is due to a tumor found in the pituitary gland.
- This tumor affects the pars intermedia - the small middle region of the pituitary gland.
- Sometimes *equine* Cushing's disease is also called as pars intermedia dysfunction (PID).



Pathogenesis

- The tumors caused varying degrees of compression of the *pars distalis* and occasionally infiltrated-or even ablated-the neurohypophysis.
- Dorsal expansion of the tumor through the diaphragma sella can lead to compression of hypothalamus and optic chiasm.
- Resulting in blindness and other neurologic deficits. Equine Cushing's disease occurs when a tumor called a pituitary adenoma develops in the *pituitary gland*.
- As this tumor slowly grows, it sends inappropriate signals to the rest of the body to secrete excessive hormones.
- Primarily a stress hormone called cortisol.
- Too much cortisol can affect the body negatively in many different ways.
- This disease in horses is similar to Cushing's disease in humans and in dogs.

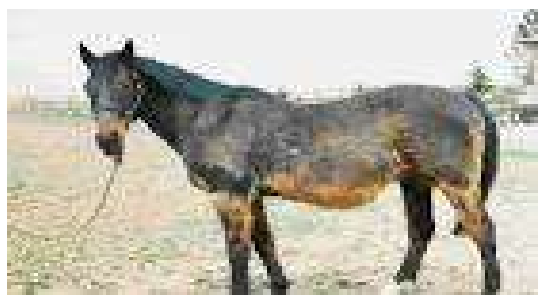
Pathogenesis

- It is caused by progressive nerve degeneration in an area of the brain called hypothalamus.
- (2^{nd}) thought--This cause reduction in a neurotrasmitter substance called dopamine.
- Dopamine is important in controlling the secretions of pars intermedia of pituitary glands.
- Dopamine is also responsible for controlling the secretion of ACTH and cortisol.
- If pars intermedia is not exposed to enough dopamine, which come from the hypothalamus.
- Then abnormal production of high levels of pituitary hormones (cortisol) will be produced.
- This result in Equine Cushing's Disease.
- For this reason, Equine Cushing's Disease is now a days termed as Equine Pituitary Pars Intermedia Dysfunction (PPID).
- There is suppression of immune system.

Clinical Findings

- Signs are usually slow to develop, but are progressive.
- Laminitis (inflammation within the structure of the *hoof*)
- Weight loss
- Ulcers in mouth
- Excessive thirst (Frequent trips to water trough)
- Excessive Urination (due to excessive drinking)
- Hirsutism (long, thick coat without shedding)
- Abnormal shedding
- Changes in body shape (e.g., development of large fat deposits along the *mane).*
- Muscle wasting, and pot-bellyed.
- Prone to infection (cuts and scrapes to take longer to heal).

Cushings Diseases



Clinical Findings

- "Pot belly" due to weakening and stretching of the abdominal muscles.
- Fat may be deposited along the crest of the neck, above the tail and above and behind the eyes.
- Sweating is a common sign, particularly in areas where the coat is long.
- Affected horses may drink and urinate more than usual.
- Wavy haircoat that fails to shed according to normal seasonal patterns.
- Lethargy and poor athletic performance.
- Chronic recurrent laminitis.
- Muscle wasting, especially along the topline.
- **A long, curly coat** that fails to shed normally is the classical clinical sign of **Cushing's Disease in horses**.

Diagnosis

- It is a disorder of the pituitary gland that results in hormonal imbalances.
- Endocrinologic tests.
- In affected horses the pars intermedia produces excessive amounts of pro-opiomelanocortin (POMC) and ACTH.
- Adrenocorticotropic hormone (ACTH).
- POMC and ACTH level goes higher than normal.
- Delayed shedding of a long, uneven coat, as well as other clinical signs help in diagnosis.

Clinical Pathology

Dexamethasone suppression and ACTH test.

- Pre and post treatment blood samples for cortisol estimation.
- Dexamethasone @ (40 micro g/kg, 1M) is given.
- Dexamethasone suppresses secretion of cortisol from the adrenal gland.
- After 24 hrs cortisol is estimated which is lower than pre val
- In diseased horses, no effect on cortisol level(Remain high).
- Measurement of plasma ACTH concentration.
- Pituitary gland in affected horses often secretes excessive amounts of ACTH into the bloodstream as compared to normal horses.

Treatment

- Cushing’s disease cannot be cured.
- Effective treatment can improve quality of life.
- Pergolide is given with good result.
- Pergolides are dopamine receptor agonist (activates dopamine receptors and secretion).
- Parkinson's disease in human is also incurable.
- Bromocriptine (replaces dopamine production)
- Trilostane (inhibits cortisol production).
- Vetoryl capsules 120mg @5 capsules daily reduces cortisol production.
- Cyproheptadine (Inhibits serotonin production or serotonin antagonist) - Practin @ 5 tab bid
- Improvement in a horse’s demeanour and attitude may be within 1-2 weeks.
- Improvement hair coat can take up to 6 months

Treatment

Medications used to treat **Cushing's disease** is focus on:

- (1) Reducing the amount of ACTH and other POMC derivatives secreted by the pituitary (e.g. cyproheptadine and pergolides).
- (2) suppression of cortisol synthesis and release by the adrenal glands (e.g. Trilostane).
- The drug of choice is currently pergolide mesylate (Permax®), which is administered daily by the oral route.
- Permax @ 0.002 mg/kg (approximately 1 mg for a 500 kg horse) once daily is recommended.
- This dose may be gradually increased if clinical improvement fails to occur after one to two months.

Diabetes Mellitus

- **Diabetes mellitus (DM),** commonly referred to as diabetes, is a group of metabolic diseases in which there are high blood sugar.
- Diabetes is due to either the pancreas not producing enough insulin.
- Or the cells of the body not responding properly to insulin produced at cellular level.

There are three types of diabetes mellitus:

- Type 1 DM results from the pancreas's(Islets) failure to produce enough insulin.
- This form was previously referred to as "insulin-dependent diabetes mellitus" (IDDM) or "juvenile diabetes". The cause is unknown
- Type 2 DM begins with insulin resistance, a condition in which cells fail to respond or utilise insulin properly.
- Also called "non insulin-dependent diabetes mellitus"(NIDDM) or "adult-onset diabetes".
- The primary cause is excessive body weight and not enough exercise.
- **Gestational diabetes: Mainly and only during pregnancy period.**

Diabetes Mellitus

- Diabetes mellitus (often called simply diabetes) is a chronic disorder of carbohydrate metabolism caused by either a deficiency of insulin or a resistance to insulin utilisation.
- Primary diabetes mellitus is caused by deficiency of insulin and is rare in horses.
- Secondary diabetes mellitus caused by resistance to insulin utilization by cells and is more common in horses.
- It develop in horses with Cushing's disease.

Pathogenesis(Diabetes Melitus)

- When food is ingested and passes through the intestine during digestion , sugar is absorbed from the food.
- This sugar is transported to the cells for metabolic function and energy in the intestine and are converted into simple sugar (glucose and sucrose).
- This glucose is absorbed into the blood stream for delivary to all the body cells and tissues for energy.
- At this stage insulin is require for transfer of blood glucose to the different body cells and tissues.
- If there is no sufficient insulin liberated by island of Langerhans (Beta cells), glucose level in blood increases leading to hyperglycemia(Diabetes mellitus).

Pathogenesis(Diabetes Melitus)

- When cells and tissues do not get sugar for energy , cells become starved for energy either due to poor insulin or due to low carbohydrate intake or over work.
- Sugar is needed for energy.
- This metabolic starvation causes the body fat to breakdown to sugar for energy.
- The liver convert fat into sugar then to energy.
- This breakdown of fat and tissues causes loss of body wt and fatty liver syndrome.
- Glucose provide much energy if it is absorbed by cells and tissues.
- Insulin attaches to receptors on the surface of cells and open pores in the cell wall that allows glucose to leave the blood stream and enter into the cells interior.
- Without an adequate amount of insulin to open the door of cells , glucose is unable to enter into the cells.
- Thus glucose accumulate in blood stream nad causes diabetes mellitus.

Treatment

- A diagnosis of diabetes mellitus is based on finding high levels of sugar in the blood and urine after a period of fasting.
- Treatment with insulin cannot reverse the insulin resistance seen in secondary diabetes mellitus.
- An initial dose of Insulin @ 0.002 mg/kg (approximately 1 mg for a 500 kg **horse**)/ OD
- Metformin is now a days a choice of drug @ 5mg/kg body wt in horses.

Hypothyroidism

- In hypothyroidism, decreased levels of thyroid hormones result in a slower metabolic rate.
- Adult horses rarely develop hypothyroidism.
- Hypothyroidism slow metabolism.
- Results in lethargy, unwillingness or inability to exercise.
- Weight gain without an increase in appetite.
- Goiter in foals mostly develop.
- When TSH is low, his thyroid gland won't produce enough thyroid hormones.
- Hypothalamus is not releasing enough thyroid releasing hormone(TRH).
- TRH stimulates production of TSH by thyroid gland.
- This cause low thyroid hormones production.
- Thus hypothyroidism occur.

Hypothyroidism

- The thyroid function is mainly of the production and storage of thyroid hormones.
- Iodine is essential for the manufacture of the two main thyroid hormones, known as T3 and T4.
- It is regulated by another hormone — known as "thyroid stimulating hormone" or TSH.
- TSH is produced by the pituitary gland nearby.
- When things go wrong they can be classified as:
- Primary, secondary or tertiary failure or **thyroid disease**.
- There is an **inverse** relationship between thyroid hormones (T3 and T4) and TSH.
- Thyroxine (T4) and Triiodythyronine (T3).
- If T4 and T3 are **high**, TSH goes down.
- If T3 and T4 are low, TSH goes up.
- When patients see their TSH being low, they think that their thyroid hormones are low.

Hypothyroidism

Primary thyroid disease:

- Inadequate thyroid hormone production, caused by:
- Usually due to a deficiency in iodine — is rare in horses.
- Too much or, less commonly, too little dietary iodine.
- Certain chemicals in forage, for example, **nitrates** — known as "goitrogenic agents" — can block hormone production.
- Tumours
- Infection — "Thyroiditis"

Secondary thyroid disease:

- Inadequate production of TSH due to a tumour in the pituitary gland.

Tertiary thyroid disease:

- Inability to use the thyroid hormones within the body, due to certain other "non-thyroidal" factors:
- Medicines such as steroids and excessively high-energy or high-protein diets.
- increased zinc or copper in diet.
- Regular starvation.
- Foals appear to be especially vulnerable to thyroid disease.
- They can even become affected while still in the womb if their dam's diet is deficient in iodine.

Symptoms of Hypothyroidism

- Dullness and lethargy
- Slow heart rate
- Thickening of tissues,especially around face, lower limb
- Poor performance and/or exercise intolerance.
- Obesity, despite a variable or even reduced appetite.
- Muscle problems, including tying up.
- Hypothermia (tendency to feel much cold).
- Infertility in stallions and mares.
- Dry, scaling skin and a dull coat.
- Delayed shedding of coat or patchy hair loss.

Signs of Hypothyroidism in foals

- Weak at birth and often not full-term
- Poor sucking and righting reflexes
- Lack of coordination
- Hypothermia
- Long coat
- Respiratory problems
- Stunted growth
- Developmental bone and muscle problems, such as undershot jaw and bent legs
- Early death

Goiter

- A lack of iodine reduces the ability of the thyroid to make thyroid hormone.
- As thyroid hormone levels drop, the pituitary gland secretes more thyroid-stimulating hormone.
- Over action of thyroid glands
- This results in the thyroid gland enlargement in an effort to make more thyroid hormones.
- Enlarge thyroid gland is goiter.

Diagnosis:

- Low level of thyroid hormones.
- Thyroxin, T3 and T4 are mostly involved.
- In fact, confirm a lower than normal concentration of the two major thyroid hormones in horses—
- Triiodothyronine (T3).
- Thyroxine (T4).

Treatment

- Thyroxin 0.1mg - @ 5 tab daily for 3months
- Feed supplementation with Vit E and Selenium.
- Feeding the correct amount of iodine to pregnant and foals.
- Iodised salt shold be recommended to foals particularly.

Hyperlipemia

- Hyperlipemia syndrome is a metabolic disease of ponies, miniature horses and donkeys.
- Hyperlipemia rarely occurs in horses but more severe in donkeys.
- Hyperlipemia refers to the condition-disease.
- Hyperlipidemia refers to the increase in serum triglyceride level (hypertriglyceridemia).
- Hyperlipidemia can cause organ failure (liver failure, renal failure, multiorgan dysfunction) and death.

Etiology:

- Any condition that results in a negative energy balance or is associated with anorexia can cause hyperlipemia.
- Anorexia, illness, neoplasia, stress(Over work load), pregnancy, lactation and parasitism can cause hyperlipemia.
- Typically these are obese animals with a recent history of stress, weight loss, parasites, sepsis, gestation/lactation, colic, endotoxemia, azotemia and Cushing's disease.

Etiology

- If body system start utilizing fat as a source of energy in place of carbohydrate- Hyperlipidemia.
- A decrease energy intake → increased blood triglycerides and deposition of fat in the liver and other organs.
- The excessive deposition of fat may lead to fatty liver → organ dysfunction (liver, renal, intestine).
- Thus hyperlipidemia develop.
- Breed and obesity are predisposing factors.

Clinical Sign

- Depression, anorexia, weakness, diarrhea, ataxia, seizures and other signs of liver or multi-organ failure may be present.
- Dullness, depression.
- Anorexia, weakness.
- Fetid breath.
- Muscle **fasciculations**, ataxia, head-pressing, severe depression, coma and death.
- Laminitis
- Occasionally jaundice and mucosal hemorrhages.

Hyperlipemia

- Diagnosis : history, clinical findings, blood biochemistry, transabdominal ultrasonography, post-mortem examination.
- Treatment : Supportive therapy, including fluid therapy, parenteral glucose administration (with or without insulin), partial parenteral nutrition, treatment of concurrent disease.
- Prognosis : Guarded; depending on how severe the increase in triglycerides is and how rapidly the condition is recognized and treated, the mortality can be up to 70%. Glucose therapy.

POLYCYTHEMIA

- Definition: Polycythemia, or erythrocytosis, is an increase in red blood cell mass.
- Polycythemia, or hemoconcentration is common in horses.
- Plasma volume decreased.
- There is dehydration and endotoxemia.
- It is an abnormally increased concentration of Hb.
- Splenic contraction may cause transient polycythemia.
- Cancer also cause Polycythemia.

POLYCYTHEMIA

Polycythemia is of two types:

1. Primary absolute polycythemia: It indicates an increased red cell mass in the absence of plasma volume change.

- When RBCs increases without concurrent increase in erythropoietin concentrations.

2. Secondary absolute polycythemia: It reflects a bone marrow response to increased erythropoietin production that results in polycythemia.

Etiology

- Bone marrow disorder in which the body produces an excessive amount RBCs.
- Blood (RBCs) runs through veins and arteries.
- When too many red blood cells exist in blood, they begin to form clots in the blood vessels.
- Clots formed and cells grouped together.
- Clots block blood from reaching the heart or brain, causing serious damage to vital organs.

Symptoms

- It is usually associated with a gene mutation.
- Mutations are changes or damage in the body's gene DNA.
- DNA is responsible for all physical characteristics including eye color.
- Fatigue on exercise of horses.
- Difficult breathing due to low oxygen tension (viscosity of blood increased).
- Tingling in the fore and hind feet.
- Heart attack and deep vein thrombosis.

Symptoms

- lethargy, weight loss, and mucosal hyperemia.
- Increased blood viscosity impairs oxygen delivery to tissues.
- Packed cell volume(PCV)exceeds 60%.(N=30%)
- At this point, additional clinical signs-such as:
- Abnormal mentation, epistaxis(Common in horses), tachycardia and tachypnea.

Diagnosis and Treatment

- Persistently increased PCV and Hb concentration.
- High TEC and bone marrow assessment.
- It is chronic and genetic diseases and is not cured.
- **Hydroxyurea**: @ 35mg/kg/day.
- **Hydroxyurea** suppresses the production of RBCs.
- This reduces the risk for clots, but increases the risk of leukemia.
- Interferon alpha 2b : It prevent clots but is expensive @ 1.5 microg/kg once weekly.

Haemorrhage and Haemorrhagic Shock

- Hemorrhagic shock: It occurs when there is a reduction in circulating blood volume due to rapid blood loss (Hemorrhages).
- Acute hemorrhage with loss of 35% or more of total blood volume due to traumatic injury or rupture of large vessels.(35% in Whole Blood).
- Equivalent to an acute blood loss of 2.8% of total body weight.
- Blood volume is 8% of total body weight.

Haemorrhage and Haemorrhagic Shock

- Severe blood loss cause low blood supply to the heart-low blood to the periphery.
- Ultimately hypovolemia.
- This will lead to clinical signs of severe hemorrhagic shock.
- 10% of total blood volume (equivalent to 0.8% of body weight) produces minimal clinical changes.

PATHOGENESIS

Hypovolemic shock:

- When cardiac output falls as a result of decreased venous return, the carotid and aortic baroreceptors stimulate the sympathetic nerves and adrenal medulla to release catecholamines.
- **Catecholamines is a vasoconstrictor.**
- This result in vasoconstriction in vessels with alpha adrenergic receptors.
- A**drenergic receptors** are a class of G protein-coupled **receptors** that are targets of the catecholamines
- Especially norepinephrine (noradrenaline) and epinephrine (adrenaline)= Increasesd emotions and more energy.

PATHOGENESIS

- Vasoconstriction leads to decreased renal perfusion.
- This activates the Renin-Angiotensin -Aldosterone system(RAAS).
- Juxtaglomerulus nephron activate and renin liberates.
- RAAS cause decrease in renal perfusion which can result in renal ischemia.
- Renin convert Angiotensinogen to angiotensin-I
- ACE convert angiotensin I to angiotensin-II.
- Angiotensin-II is highly **vasoconstrictor.**

Diagnosis and Treatment

- Symptoms of dehydration and Anaemia
- Pinch test (8%)
- Capillary refil time(Less than 1-2 seconds)
- Blood transfusion @ 10 ml/kg body wt
- Major and minor cross matching.
- Plasma expander , Haemacil can be given if blood transfusion is not possible.

Immune-Mediated Hemolytic Anemia (IMHA)

- Immune-mediated hemolytic anemia results from cross-reacting antibodies to RBCs.
- It induces enhanced red blood cell destruction.
- Autoimmune (primary IMHA) hemolysis results from loss of self-tolerance.
- Most commonly, hemolysis results from adherence of cross-reacting antibodies to erythrocyte surface antigens (secondary IMHA).
- **Antibodies**, also known as immunoglobulins (Ig) are a form of protein.

Immune-Mediated Hemolytic Anemia (IMHA)

- The presence of these molecules on red blood cells surface causes intravascular destruction by complement activation (IgM-mediated AB).
- Or, extravascular removal by macrophages.
- Any infectious agent- like equine infectious anemia, *Babesia* , exogenous substances such as penicillin and phenylbutazone may cause alterations in epitopes of the erythrocyte membrane.
- This change in epitopes of RBCs cause lysis of rbc.

Immune-Mediated Hemolytic Anemia (IMHA)

- For example, a change in the red blood cell membrane (Epitopes) may form a novel antigen that evokes an immune response.
- Drugs, neoplasia, or infection may induce changes in red cell antigens.
- Infectious agents(Bacteria) that express similar antigens as host red blood cell antigens, there will be IMHA .
- This result in pathogen-induced immune-mediated hemolysis.
- It is termed as ***molecular mimicry.***

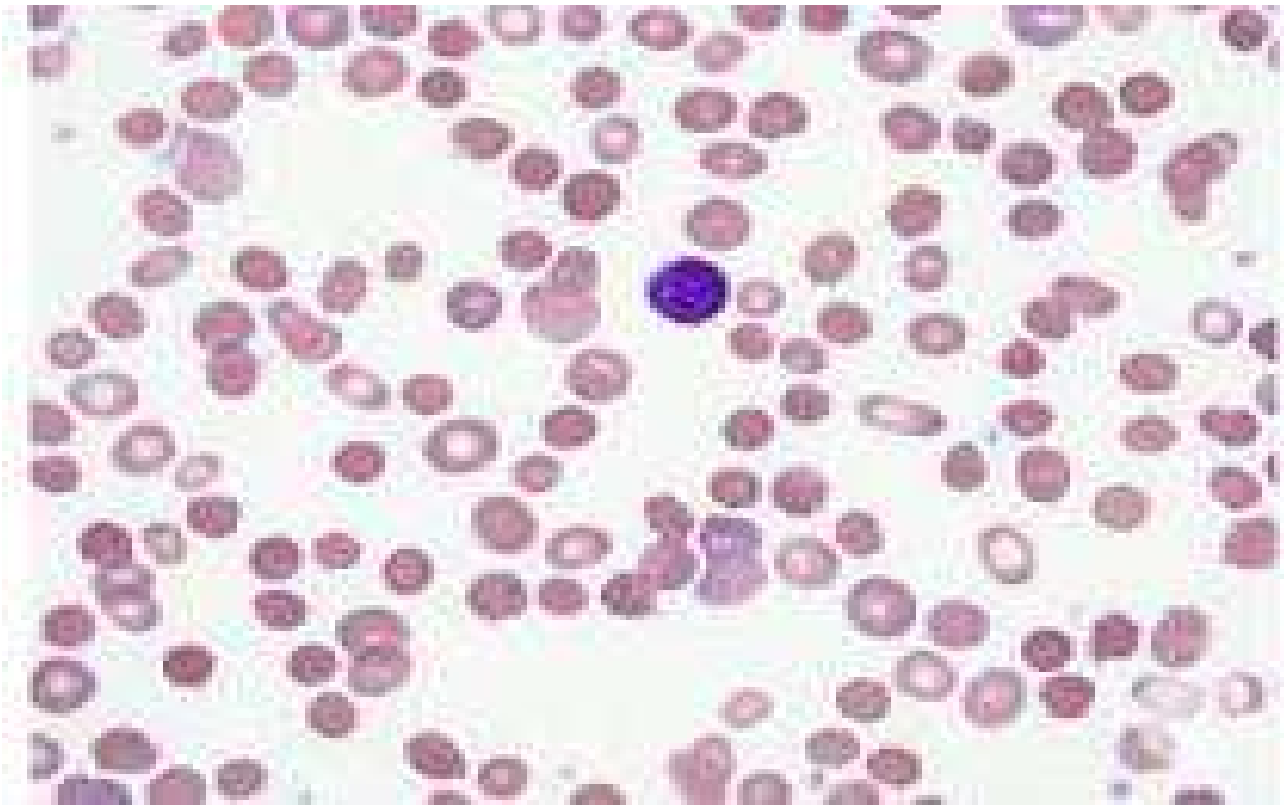
Immune-Mediated Hemolytic Anemia

- It occurs when antibodies directed against its own red blood cells (RBCs) and cause lysis of RBCs.
- This cause Immune-Mediated Hemolysis.
- This cause low plasma concentration and IMHA.
- Lifetime of RBCs reduced from the normal 100–120 days to few days in serious cases.

Clinical Findings

- Horses with IMHA most commonly present with signs of extravascular hemolysis.
- Intravascular hemolysis is possible, especially when IgM antibodies or complement is involved.
- **Spherocytes (Sphere shaped)** may be present on cytology of peripheral blood smears.
- Spherocytosis occur in auto-hemolytic anemia (AIHA).
- Antibody fight germs, but sometimes antibodies make a mistake and target own body's healthy red blood cells instead and cause AIHA.

RBCs are sphere-shaped rather than bi-concave disk shaped(Spherocytosis).



Diagnosis

- Auto agglutination will suggest surface-bound antibody.
- Dilution of the sample with saline (1: 1).
- If erythrocytes still agglutinate after dilution,it is positive for surface-bound antibody.
- Coombs' test: The End point of the Coombs' test is agglutination.
- Direct immunofluorescence assay that uses class-specific antibodies to equine IgM, IgG, and IgA.

Coombs' test

- Antibodies are a part of immune system.
- They fight germs, but sometimes antibodies make a mistake and target own body's healthy cells instead.
- The **Coombs test** check blood for antibodies that attack own red blood cells.
- Destruction of own RBCs by its own Antibody is tested through Coobs Test.

Treatment

- Therapy will be determined based on the level of anemia.
- In severe cases, whole blood transfusion may be indicated.
- Immunosuppressive therapy.
- Glucocorticoid reduces the function of macrophages.
- Dexamethasone @ 0.05 to 0.2 mg/kg IV-24hly

IRON DEFIDIENCY ANAEMIA

- Vitamin B12 and folic acid are important co factors in erythrocyte maturation.
- In horses, gastrointestinal bacteria synthesize vitamin B12- No need for supplementation.
- Iron-deficiency anemia in horses is *almost* invariably the result of chronic blood loss.
- Iron-deficiency anemia is diagnosed on the basis of hypoferritinemia, hypoferremia, increased total iron binding capacity,

IRON DEFIDIENCY ANAEMIA

- In horses, normal serum ferritin has been reported as 152 ng/ml.
- Normal iron concentration is 120 microgm/dl
- Normal total iron binding capacity -388mic/dl
- Iron deficiency anemia is not very common.
- It occur mostly in foals feed on only milk.
- Milk is the poorest source of iron.
- Iron suplementation with ferrous sulphate.

ANEMIA OF CHRONIC DISEASE (ACD)

There are three mechanisms of ACD :

1. Shortened erythrocyte life span
2. Insufficient bone marrow response to demand for red blood cells.
3. Decreased release of iron from the reticuloendothelial system.

Etiology and Pathogenesis

- Accelerated red cell destruction
- Intravascular response to inflammation.
- Increased RBCs damage while passing through micro circulation.
- Damaged and subsequent removal by R.E system.
- Normally, the bone marrow respond with increase in RBCs.
- Erythropoietin(CRF) is primarily responsible for regulation of erythropoiesis in the bone marrow.
- Cytokines(ILK-1,TNF) produced in response to inflammation, infection and neoplasia.

Diagnosis, Clinical Signs, and Treatment

- Non regenerative, normochromic, and normocytic anemia develop.
- Serum iron and total iron-binding –decreased.

  Anemia of chronic Disease(ACD) are:

- Pleuropneumonia,internal abscessation,CRF
- Neoplasia, and equine infectious anemia.
- Hypoferremic horses with ACD have normal iron stores and do not require iron supplementation.
- Symptomtic treatment as per causative factors.

GENERALISED BONE MARROW SUPPRESSION OR FAILURE

- Aplastic anemia results from congenital or acquired developmental failure of hematopoietic progenitor cells(Stem cell) in the bone marrow.
- Bacterial,viral , chronic renal or hepatic failure. and irradiation therapy.
- Drug administration but mostly are idiopathic.
- Endogenous **erythropoietin** is produced by the liver and is activated in the kidney.

Clinical Signs, Diagnosis, and Treatment

- Bone marrow assessment.
- Bone marrow myeloid and erythroid ratios is 6.7 and 3.2 (normal 0.5-1.5).
- This indicate severe nonregenerative anemia.
- Loss of neutrophils(Leukemia) and platelets.
- Fever, localized infection and thrombo cytopenic hemorrhages.
- Steroids stimulate erythropoiesis by increasing erythropoietin production.
- No treatment for myeloid aplastic anemia(Leukemia).

IMMUNE MEDIATED THROMBOCYTOPENIA

- Thrombocytopenia is low platelets production

There are three major mechanisms:

(1) Reduced platelet production.

(2) Abnormal platelet distribution

(3) Increased consumption or decreased platelet survival.

IMMUNE MEDIATED THROMBOCYTOPENIA

- Thrombocytopenia causes altered hemostasis due to insufficient phospholipid substrate for coagulating proteins.
- The platelet activate clotting factors and maintains vascular integrity.
- Severe thrombocytopenia causes prolonged bleeding times.
- It will not affect clotting times or plasma fibrinogen concentration

Disseminated Intravascular Coagulation(DIC)

- DIC is most common hemostatic dysfunction in horses.
- DIC is an acquired process.
- DIC is not a primary disease.
- It occurs in conjunction with diseases that generate excessive pro coagulant activity in the blood.
- There is widespread fibrin deposition in micro circulation, thrombosis and ischemic damage.
- Hemorrhagic diathesis occurs as a result of consumption of pro coagulants or hyperactivity of fibrinolysis.

Etiology and Pathogenesis of DIC

- In normal coagulation, thrombin activates the conversion of plasma soluble fibrinogen to the insoluble fibrin, which forms a clot.
- Simultaneously, the fibrinolytic system is activated to prevent tissue ischemia that may occur from persistent fibrin clots through Plasmin
- Plasmin is the fibrinolytic protein.
- It is primarily responsible for reducing fibrin clot formation and providing a mechanism for clot removal .

Etiology and Pathogenesis of DIC

- Antithrombin III and protein C also minimize clot formation by inhibiting the actions of thrombin.
- In DIC, antithrombin III and protein C become depleted.
- This results in excessive, unchecked thrombin and clot formation.
- At this crucial point plasmin(fibrinolytic protein) is activated to prevent clot formation.

Etiology and Pathogenesis of DIC

Diseases associated with DIC are:

- Strangulating obstruction, colitis and enteritis.
- Sepsis,renal diseases, hemolytic anemia, and neoplasia.
- 96% horses that develop DIC have colic.
- Endotoxin is a prominent cause of ischemic or inflammatory disease of the equine GIT.

Etiology and Pathogenesis of DIC

Endotoxin can initiate DIC by several mechanisms:

(1) Direct damage to the endothelium.

(2) Induction of cytokine synthesis by phagocytes.

(3) Stimulation of thromboxane A2 synthesis by platelets which promotes irreversible platelet aggregation.

(4) Inhibition of fibrinolysis by increasing production of plasminogen activator inhibitor.

Clinical Signs of DIC

- Oppositbto humans, frank hemorrhage with DIC is rare.
- Petechial or ecchymotic hemorrhages of the mucous membranes or sclerae.
- Epistaxis and melena can occur.
- Colic, laminitis, renal failure, pulmonary and cerebral diseases.
- Peripheral veins are susceptible to spontaneous thrombosis.
- Increased thrombus formation after catheterization or simple venipuncture.
- *Diagnosis:* A single test cannot confirm DIC.
- Clinical signs.
- Thrombocytopenia.
- Fibrinogen concentration may be increased.
- Reduced antithrombin III activity (<80% is normal)

Treatment and Prognosis

- Therapy for DIC is difficult and controversial.
- Identification and treatment of the underlying disease process is important.
- Intravenous fluid therapy is necessary to maintain tissue perfusion and prevent shock.
- Antimicrobials are indicated if sepsis exist.
- If a strangulating intestinal obstruction is present immediate surgery needed.
- Endotoxemia – control by specific antitoxins.

Treatment and Prognosis

- Flunixin meglumine @0.25 mg/kg I/V q8h will mitigate the detrimental effect.
- **Corticosteroids are contraindicated.**
- Corticosteroids potentiate the vasoconstrictive effect of catecholamines.
- Corticosteroids reduces the activity of phagocytic system.
- Fresh plasma therapy (15-30 ml/kg-I/V) may act as "fuel the fire." and is fast effective.

Treatment and Prognosis

- Heparin @20 to 100 U/kg q8-12hly with fresh plasma.
- It minimize clot formation by potentiating the anticoagulative effects of antithrombin III.
- Heparin therapy must be used, with adequate antithrombin III.
- Heparin cause thrombocytopenia and low PCV
- In general, the prognosis is guarded to poor.

Purpura Haemorrhagica

Definition: Purpura hemorrhagica is a noncontagious, type III immune-mediated vasculitis of horses, characterized by S/C edema of the head, ventral abdomen and limbs and by petechial hemorrhages of the mucous membranes.

- It is caused by bleeding from capillaries which results in red spots on the skin and mm.
- It is more common in younger animals.

Etiology

- Most of the clinical signs of the disease result from deposition of antigen-antibody complexes in the small blood vessels of the skin.
- Purpura hemorrhagica occurr after vaccination,drug administration idiopathically.
- There seem to be two prerequisites for the development of purpura hemorrhagica:

  1. A large amount of antigenic material and an exaggerated immune response.

2. Bacterial and viral organisms, particularly those that cause formation of purulent or necrotic foci.

Etiology

- Purpura hemorrhagica can also rarely be seen after infection with:
- *Streptococcus equi* subsp. *equi*
- *Rhodococcus equi*
- *Corynebacterium pseudotuberculosis*
  (causative agent of pigeon fever)
- Equine influenza virus
- Equine herpes virus type 1
- Even without any apparent infection

Pathophysiology

- A recent history of strangles (infection with *Streptococcus equi* subsp. *equi*).
- Vaccination (I/M or intranasal) for strangles.
- It is thought to be caused by an auto-immune reaction.
- Antibodies against the *S. equi* M- or R-protein cross-react with proteins on endothelial cells.
- This results in vasculitis.
- Leading to subsequent severe peripheral edema in the legs and ventral abdomen.
- Petechiation or ecchymoses over the mm.

Pathophysiology

- Purpura hemorrhagica secondary to strangles reportedly occurs in 1% to 17% of cases
- Clinical signs typically appear 1 to 2 weeks after recovery from a strangles episode.
- There seem to be two prerequisites for the development of purpura hemorrhagica:

1. Large amount of antigenic material
2. Exaggerated immune response.

Pathophysiology

- Draining abscesses is the source of this antigenic material.
- Purpura hemorrhagica is a sequela to a variety of bacterial and viral organisms associated with formation of purulent foci.
- Antigen within these purulent foci is M-like protein.
- M-like protein is the major streptococcal antigen in cases of purpura hemorrhagica caused by strangles infection.

Clinical Signs

- S/C edema of the limbs and ventral abdomen.
- Hemorrhages on mucous membranes.
- Anorexia, fever, elevated heart and respiratory rate.
- Reluctance to move.
- Drainage from lymph nodes.
- Exudation of serum from the skin . Rarely DIC
- Colic,epistaxis due to submucosal hemorrhage in nasal.
- Hematuria associated with glomerulonephritis.
- Tachycardia occurs frequently and is probably the result of decreasing circulating fluid volume.

Clinical Signs

- Mostly in young horses but in geriatric also.
- In mild cases, edema may be more localized to the legs.
- Severe edema of the head often leads to respiratory stridor due to occlusion of the upper airway.
- Dyspnea due to swelling around upper or lower respiratory tract or, pulmonary edema.
- Petechial or ecchymotic hemorrhages of the mucosal, nasal, and conjunctival membranes
- Concurrent signs of strangles, such : lymphadenopathy, draining abscesses, coughing, and nasal discharge.
- Pyrexia may or may not be present

Clinical Pathology

- CBC and serum clinical chemistry tests are nonspecific.
- Hematologic changes include moderate anemia and leukocytosis with neutrophilia and a mild left shift.
- In general, horses with purpura hemorrhagica have normal clotting profiles and platelet counts.
- Thrombocytopenia is extremely rare and absent.
- Hyperproteinemia, and hyperglobulinemia.
- Elevated muscle enzymes: Including
- Aspartate aminotransferase(AST) and creatine kinase(CK).
- This is in association with *S. equi*-induced myopathy.

Diagnosis

- Necrotizing vasculitis.
- Edematous blood vessel walls
- Dermal and subcutaneous hemorrhage, inflammation, and thrombi may be visible.
- An agar gel immunodiffusion test (Coggins test).
- ELISA for carriers.
- PPH mostlyn diagnosed ,based on clinical signs and biopsy results.

Differential Diagnosis

- Other causes of vasculitis should be ruled out.
- Equine granulocytic Babesiosis
- Equine infectious anaemia.
- Equine viral arteritis (EVA).
- Equine herpesvirus infection
- Anaplasmosis (*Anaplasma phagocytophilum* infection; formerly Ehrlichia equi infection).
- Immune-mediated thrombocytopenia
- All can produce vasculitic syndromes, but affected animals are usually thrombocytopenic.

Treatment

Treatment of PPH is of three fold:

1. Removal of the antigenic stimulus (if identified)
2. Reduction of the immune response
3. Supportive care.

- Creatinine and blood urea nitrogen levels as well as urine specific gravity should be carefully monitored for evidence of dehydration and azotemia.
- Treat with appropriate amounts of I/V isotonic fluids.
- Hypoalbuminemic animals with edema may benefit from judicious use of colloids, such as plasma or hetastarch (10 ml/kg).

Treatment

- Potassium or sodium penicillin at an initial dosage of 22,000 to 44,000 IU/kg IV q6h is recommended
- I/M injections is avoided because inflamed muscle tissue may be further aggravated.
- Thrombophlebitis is a complication of purpura hemorrhagica
- Careful attention to aseptic placement of intravenous catheters.
- Switching to oral antibiotics such as trimethoprim sulfadiazine (15 to 30 mg/kg PO q6h) to avoid complications of long-term catheter.
- Continue Antibiotic throughout treatment of disease.

Treatment

- Suppressing the immune response by using glucocorticoids(Dexamethasone).
- Dexamethasone (0.05 to 0.2 mg/kg IV q24h initially).
- As clinical signs improve, the dose of dexamethasone should be slowly tappered over a 2- to 3-week period.
- If the final dose of dexamethasone is 0.04 mg/kg, switch to prednisolone at 0.4 mg/kg).

Treatment

- While high doses of steroids may risk laminitis, low doses are associated with refractory cases.
- Antibiotics should be given for residual nidus of *S. equi*.
- (NSAIDs), such as phenylbutazone or flunixin, may be useful to reduce fever and relieve pain.
- Additionally, wrapping the legs may reduce edema and skin sloughing.
- Supportive care with oral or IV fluids may be given

PPH Management

- Supportive therapy includes hydrotherapy.
- Thick bedding, counter-pressure leg wraps.
- Protective dressings for weeping skin lesions.
- Some clinicians advocate the use of furosemide (0.3 mg/kg IV q6-8h) to decrease edema, particularly pulmonary edema.
- However, in animals that are already azotemic, furosemide use may cause further renal complication.
- Light exercise for horses that are not laminitic or too sick to move can dissipate edema.
- Oxygen therapy is beneficial in horses with P/ edema.

POLYCYTHEMIA

- Definition: Polycythemia, or erythrocytosis, is an increase in red blood cell mass.
- Polycythemia, or hemoconcentration is common in horses.
- Plasma volume decreased.
- There is dehydration and endotoxemia.
- It is an abnormally increased concentration of Hb.
- Splenic contraction may cause transient polycythemia.
- Cancer also cause Polycythemia.

POLYCYTHEMIA

Polycythemia is of two types:

1. Primary absolute polycythemia: It indicates an increased red cell mass in the absence of plasma volume change.

- When RBCs increases without concurrent increase in erythropoietin concentrations.

2. Secondary absolute polycythemia: It reflects a bone marrow response to increased erythropoietin production that results in polycythemia.

Etiology

- Bone marrow disorder in which the body produces an excessive amount RBCs.
- Blood (RBCs) runs through veins and arteries.
- When too many red blood cells exist in blood, they begin to form clots in the blood vessels.
- Clots formed and cells grouped together.
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- Packed cell volume(PCV)exceeds 60%.(N=30%)
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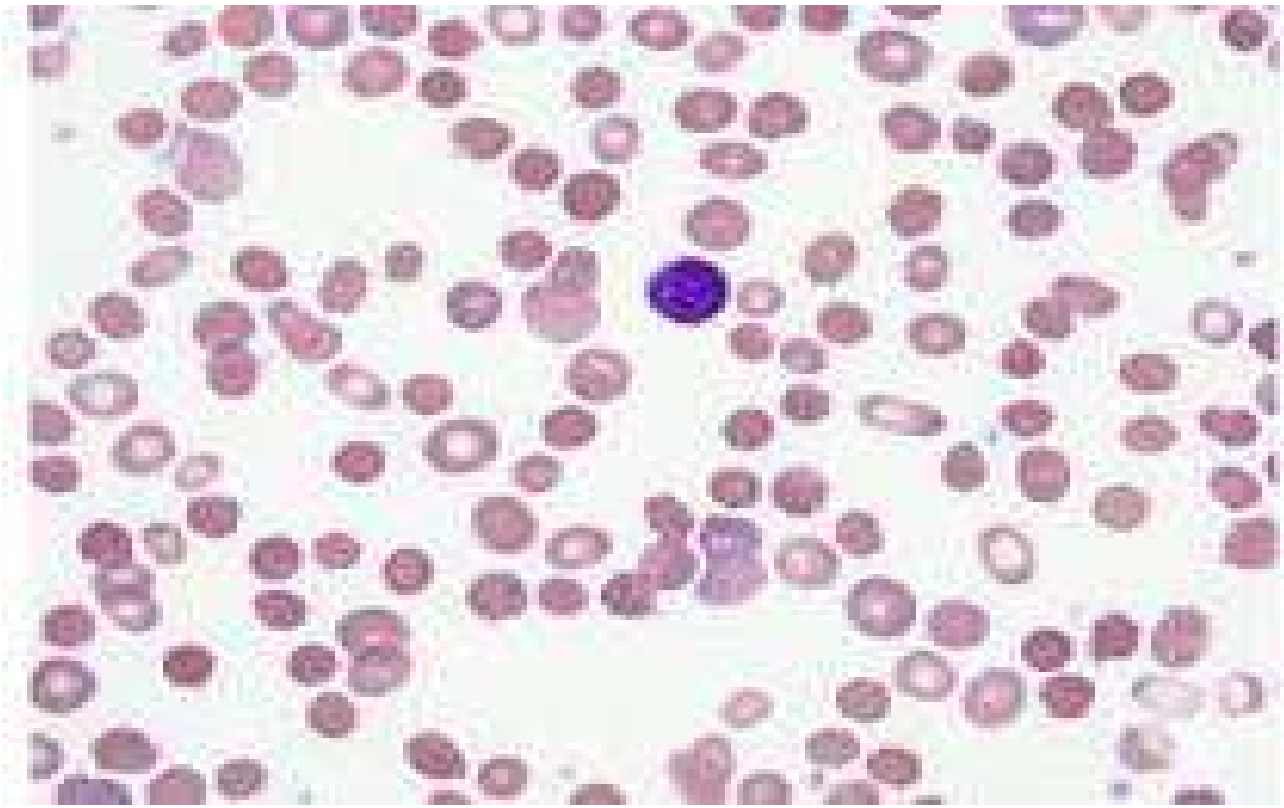

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  Anemia of chronic Disease(ACD) are:

- Pleuropneumonia,internal abscessation,CRF
- Neoplasia, and equine infectious anemia.
- Hypoferremic horses with ACD have normal iron stores and do not require iron supplementation.
- Symptomtic treatment as per causative factors.

GENERALISED BONE MARROW SUPPRESSION OR FAILURE

- Aplastic anemia results from congenital or acquired developmental failure of hematopoietic progenitor cells(Stem cell) in the bone marrow.
- Bacterial,viral , chronic renal or hepatic failure. and irradiation therapy.
- Drug administration but mostly are idiopathic.
- Endogenous **erythropoietin** is produced by the liver and is activated in the kidney.

Clinical Signs, Diagnosis, and Treatment

- Bone marrow assessment.
- Bone marrow myeloid and erythroid ratios is 6.7 and 3.2 (normal 0.5-1.5).
- This indicate severe nonregenerative anemia.
- Loss of neutrophils(Leukemia) and platelets.
- Fever, localized infection and thrombo cytopenic hemorrhages.
- Steroids stimulate erythropoiesis by increasing erythropoietin production.
- No treatment for myeloid aplastic anemia(Leukemia).

IMMUNE MEDIATED THROMBOCYTOPENIA

- Thrombocytopenia is low platelets production

There are three major mechanisms:

(1) Reduced platelet production.

(2) Abnormal platelet distribution

(3) Increased consumption or decreased platelet survival.

IMMUNE MEDIATED THROMBOCYTOPENIA

- Thrombocytopenia causes altered hemostasis due to insufficient phospholipid substrate for coagulating proteins.
- The platelet activate clotting factors and maintains vascular integrity.
- Severe thrombocytopenia causes prolonged bleeding times.
- It will not affect clotting times or plasma fibrinogen concentration

Disseminated Intravascular Coagulation(DIC)

- DIC is most common hemostatic dysfunction in horses.
- DIC is an acquired process.
- DIC is not a primary disease.
- It occurs in conjunction with diseases that generate excessive pro coagulant activity in the blood.
- There is widespread fibrin deposition in micro circulation, thrombosis and ischemic damage.
- Hemorrhagic diathesis occurs as a result of consumption of pro coagulants or hyperactivity of fibrinolysis.

Etiology and Pathogenesis of DIC

- In normal coagulation, thrombin activates the conversion of plasma soluble fibrinogen to the insoluble fibrin, which forms a clot.
- Simultaneously, the fibrinolytic system is activated to prevent tissue ischemia that may occur from persistent fibrin clots through Plasmin
- Plasmin is the fibrinolytic protein.
- It is primarily responsible for reducing fibrin clot formation and providing a mechanism for clot removal .

Etiology and Pathogenesis of DIC

- Antithrombin III and protein C also minimize clot formation by inhibiting the actions of thrombin.
- In DIC, antithrombin III and protein C become depleted.
- This results in excessive, unchecked thrombin and clot formation.
- At this crucial point plasmin(fibrinolytic protein) is activated to prevent clot formation.

Etiology and Pathogenesis of DIC

Diseases associated with DIC are:

- Strangulating obstruction, colitis and enteritis.
- Sepsis,renal diseases, hemolytic anemia, and neoplasia.
- 96% horses that develop DIC have colic.
- Endotoxin is a prominent cause of ischemic or inflammatory disease of the equine GIT.

Etiology and Pathogenesis of DIC

Endotoxin can initiate DIC by several mechanisms:

(1) Direct damage to the endothelium.

(2) Induction of cytokine synthesis by phagocytes.

(3) Stimulation of thromboxane A2 synthesis by platelets which promotes irreversible platelet aggregation.

(4) Inhibition of fibrinolysis by increasing production of plasminogen activator inhibitor.

Clinical Signs of DIC

- Oppositbto humans, frank hemorrhage with DIC is rare.
- Petechial or ecchymotic hemorrhages of the mucous membranes or sclerae.
- Epistaxis and melena can occur.
- Colic, laminitis, renal failure, pulmonary and cerebral diseases.
- Peripheral veins are susceptible to spontaneous thrombosis.
- Increased thrombus formation after catheterization or simple venipuncture.
- *Diagnosis:* A single test cannot confirm DIC.
- Clinical signs.
- Thrombocytopenia.
- Fibrinogen concentration may be increased.
- Reduced antithrombin III activity (<80% is normal)

Treatment and Prognosis

- Therapy for DIC is difficult and controversial.
- Identification and treatment of the underlying disease process is important.
- Intravenous fluid therapy is necessary to maintain tissue perfusion and prevent shock.
- Antimicrobials are indicated if sepsis exist.
- If a strangulating intestinal obstruction is present immediate surgery needed.
- Endotoxemia – control by specific antitoxins.

Treatment and Prognosis

- Flunixin meglumine @0.25 mg/kg I/V q8h will mitigate the detrimental effect.
- **Corticosteroids are contraindicated.**
- Corticosteroids potentiate the vasoconstrictive effect of catecholamines.
- Corticosteroids reduces the activity of phagocytic system.
- Fresh plasma therapy (15-30 ml/kg-I/V) may act as "fuel the fire." and is fast effective.

Treatment and Prognosis

- Heparin @20 to 100 U/kg q8-12hly with fresh plasma.
- It minimize clot formation by potentiating the anticoagulative effects of antithrombin III.
- Heparin therapy must be used, with adequate antithrombin III.
- Heparin cause thrombocytopenia and low PCV
- In general, the prognosis is guarded to poor.

Purpura Haemorrhagica

Definition: Purpura hemorrhagica is a noncontagious, type III immune-mediated vasculitis of horses, characterized by S/C edema of the head, ventral abdomen and limbs and by petechial hemorrhages of the mucous membranes.

- It is caused by bleeding from capillaries which results in red spots on the skin and mm.
- It is more common in younger animals.

Etiology

- Most of the clinical signs of the disease result from deposition of antigen-antibody complexes in the small blood vessels of the skin.
- Purpura hemorrhagica occurr after vaccination,drug administration idiopathically.
- There seem to be two prerequisites for the development of purpura hemorrhagica:

  1. A large amount of antigenic material and an exaggerated immune response.

2. Bacterial and viral organisms, particularly those that cause formation of purulent or necrotic foci.

Etiology

- Purpura hemorrhagica can also rarely be seen after infection with:
- *Streptococcus equi* subsp. *equi*
- *Rhodococcus equi*
- *Corynebacterium pseudotuberculosis*
  (causative agent of pigeon fever)
- Equine influenza virus
- Equine herpes virus type 1
- Even without any apparent infection

Pathophysiology

- A recent history of strangles (infection with *Streptococcus equi* subsp. *equi*).
- Vaccination (I/M or intranasal) for strangles.
- It is thought to be caused by an auto-immune reaction.
- Antibodies against the *S. equi* M- or R-protein cross-react with proteins on endothelial cells.
- This results in vasculitis.
- Leading to subsequent severe peripheral edema in the legs and ventral abdomen.
- Petechiation or ecchymoses over the mm.

Pathophysiology

- Purpura hemorrhagica secondary to strangles reportedly occurs in 1% to 17% of cases
- Clinical signs typically appear 1 to 2 weeks after recovery from a strangles episode.
- There seem to be two prerequisites for the development of purpura hemorrhagica:

1. Large amount of antigenic material
2. Exaggerated immune response.

Pathophysiology

- Draining abscesses is the source of this antigenic material.
- Purpura hemorrhagica is a sequela to a variety of bacterial and viral organisms associated with formation of purulent foci.
- Antigen within these purulent foci is M-like protein.
- M-like protein is the major streptococcal antigen in cases of purpura hemorrhagica caused by strangles infection.

Clinical Signs

- S/C edema of the limbs and ventral abdomen.
- Hemorrhages on mucous membranes.
- Anorexia, fever, elevated heart and respiratory rate.
- Reluctance to move.
- Drainage from lymph nodes.
- Exudation of serum from the skin . Rarely DIC
- Colic,epistaxis due to submucosal hemorrhage in nasal.
- Hematuria associated with glomerulonephritis.
- Tachycardia occurs frequently and is probably the result of decreasing circulating fluid volume.

Clinical Signs

- Mostly in young horses but in geriatric also.
- In mild cases, edema may be more localized to the legs.
- Severe edema of the head often leads to respiratory stridor due to occlusion of the upper airway.
- Dyspnea due to swelling around upper or lower respiratory tract or, pulmonary edema.
- Petechial or ecchymotic hemorrhages of the mucosal, nasal, and conjunctival membranes
- Concurrent signs of strangles, such : lymphadenopathy, draining abscesses, coughing, and nasal discharge.
- Pyrexia may or may not be present

Clinical Pathology

- CBC and serum clinical chemistry tests are nonspecific.
- Hematologic changes include moderate anemia and leukocytosis with neutrophilia and a mild left shift.
- In general, horses with purpura hemorrhagica have normal clotting profiles and platelet counts.
- Thrombocytopenia is extremely rare and absent.
- Hyperproteinemia, and hyperglobulinemia.
- Elevated muscle enzymes: Including
- Aspartate aminotransferase(AST) and creatine kinase(CK).
- This is in association with *S. equi*-induced myopathy.

Diagnosis

- Necrotizing vasculitis.
- Edematous blood vessel walls
- Dermal and subcutaneous hemorrhage, inflammation, and thrombi may be visible.
- An agar gel immunodiffusion test (Coggins test).
- ELISA for carriers.
- PPH mostlyn diagnosed ,based on clinical signs and biopsy results.

Differential Diagnosis

- Other causes of vasculitis should be ruled out.
- Equine granulocytic Babesiosis
- Equine infectious anaemia.
- Equine viral arteritis (EVA).
- Equine herpesvirus infection
- Anaplasmosis (*Anaplasma phagocytophilum* infection; formerly Ehrlichia equi infection).
- Immune-mediated thrombocytopenia
- All can produce vasculitic syndromes, but affected animals are usually thrombocytopenic.

Treatment

Treatment of PPH is of three fold:

1. Removal of the antigenic stimulus (if identified)
2. Reduction of the immune response
3. Supportive care.

- Creatinine and blood urea nitrogen levels as well as urine specific gravity should be carefully monitored for evidence of dehydration and azotemia.
- Treat with appropriate amounts of I/V isotonic fluids.
- Hypoalbuminemic animals with edema may benefit from judicious use of colloids, such as plasma or hetastarch (10 ml/kg).

Treatment

- Potassium or sodium penicillin at an initial dosage of 22,000 to 44,000 IU/kg IV q6h is recommended
- I/M injections is avoided because inflamed muscle tissue may be further aggravated.
- Thrombophlebitis is a complication of purpura hemorrhagica
- Careful attention to aseptic placement of intravenous catheters.
- Switching to oral antibiotics such as trimethoprim sulfadiazine (15 to 30 mg/kg PO q6h) to avoid complications of long-term catheter.
- Continue Antibiotic throughout treatment of disease.

Treatment

- Suppressing the immune response by using glucocorticoids(Dexamethasone).
- Dexamethasone (0.05 to 0.2 mg/kg IV q24h initially).
- As clinical signs improve, the dose of dexamethasone should be slowly tappered over a 2- to 3-week period.
- If the final dose of dexamethasone is 0.04 mg/kg, switch to prednisolone at 0.4 mg/kg).

Treatment

- While high doses of steroids may risk laminitis, low doses are associated with refractory cases.
- Antibiotics should be given for residual nidus of *S. equi*.
- (NSAIDs), such as phenylbutazone or flunixin, may be useful to reduce fever and relieve pain.
- Additionally, wrapping the legs may reduce edema and skin sloughing.
- Supportive care with oral or IV fluids may be given

PPH Management

- Supportive therapy includes hydrotherapy.
- Thick bedding, counter-pressure leg wraps.
- Protective dressings for weeping skin lesions.
- Some clinicians advocate the use of furosemide (0.3 mg/kg IV q6-8h) to decrease edema, particularly pulmonary edema.
- However, in animals that are already azotemic, furosemide use may cause further renal complication.
- Light exercise for horses that are not laminitic or too sick to move can dissipate edema.
- Oxygen therapy is beneficial in horses with P/ edema.

DISEASES OF EYE OF EQUINES

- The eye is a paired organ and is the organ of vision.
- The eye is made up of various components.
- Eye receive light stimuli from the environment.
- Eye deliver this stimuli through optic nerve to the brain in the form of an electrical signal and then image and thus horses see.
- Vision involves all components of the eye.
- Eye is contained within the bony orbit of the head.
- Bony orbit is a cavity of lacrimal bone and maxilla.
- The bony orbit is located laterally in herbivores, but is set forward in hunting animals e.g. carnivores.

Layers of the retina
(From vitreous humour to Choroid)

1. Inner limiting membrane
2. Optic nerve fibres and axons of ganglion cells
3. Ganglion cell bodies
4. Inner plexiform layer - synapse of bipolar cells with ganglion cells
5. Inner nuclear layer – nuclei of bipolar cells
6. Outer plexiform layer - synapses of photoreceptors with bipolar cells
7. Outer nuclear layer – photoreceptor cell nuclei
8. Outer limiting membrane
9. Photoreceptor layer – rods and cones

Glossary (Definition) of Eye Terms

- Anterior chamber - The space in the eye bounded in front by the cornea and behind by the iris.
- It is filled with aqueous humor.
- Anterior uvea - The front portion of the uvea, made up of the iris and ciliary body.
- Anterior uveitis - Inflammation of the iris and ciliary body.
- Aqueous humor - The clear, watery fluid which fills the eye.
- Blepharitis - Inflammation of the eyelids.

Glossary (Definition) of Eye Terms

- Blepharospasm - Spasm of muscles that control eyelids.
- Cataract - An opacity of the lens or its capsule, or both.
- Choroid - The part of the vascular layer of the eye (uvea) located at the back of the eye.
- It helps to nourish the retina. It is the posterior uvea.
- Ciliary body - The part of the vascular layer of the eye (uvea) located between the iris and the choroid.
- Conjunctiva - The mucous membrane lining the back of the eyelids and the front of the eye, except for the cornea.
- Conjunctivitis - Inflammation of the conjunctiva.

Glossary (Definition) of Eye Terms

- Cornea - The transparent portion of the outer layer of the eye, which allows light to enter the eye.
- It attaches to the sclera.
- Corneal ulceration - An open lesion or sore on the surface of the cornea.
- Enucleation - Removal of the eye.
- Epiphora - The abnormal flow of tears over the face.
- Fibrous - Composed of cells that form tough, connective tissue.
- Fluorescein dye - A special dye that is applied to the cornea that will not penetrate intact cornea or conjunctiva.
- But will adhere to and outline areas of ulceration of cornea.

Glossary (Definition) of Eye Terms

- Glaucoma - Increased ocular pressure resulting from altered flow of aqueous humor.
- Globe - The eyeball.
- Intraocular - Within the eyeball.
- Iris - The colored, circular portion of the eye located behind the cornea. It is part of the uvea.
- Keratoplasty -Corneal grafting used as repair technique.
- Keratectomy - Removal of part of the superficial layers of the cornea.
- Keratitis - Inflammation of the cornea.

Glossary (Definition) of Eye Terms

- Lacrimal apparatus - The structures responsible for tear production.
- Lens - A transparent structure suspended in the front portion of the eye. It refracts light that passes through the cornea on its way to the retina.
- Lens luxation - Displacement of the lens.
- Miosis - Contraction of the pupil so that the pupil becomes small.
- Miotic - A medication that causes the pupil to become small.
- Mucoid - This is a general term for types of secretions that are made up of mucous materials.

Glossary (Definition) of Eye Terms

- Mucopurulent - A secretion or exudate that is made up of pus and mucous.
- Mydriatic - A medication that causes the pupil to become dialated.
- Mydriasis - A dilated pupil.
- Nasolacrimal - The system of the eye responsible for proper drainage of tears out of the eye and into the inside of the nose.
- Nictitans (nictitating membrane) - The third eyelid.
- Ophthalmoscope - A device for studying the interior of the eyeball.
- Optic nerve - The nerve that originates from the retina and carries light signals to the brain.

Glossary (Definition) of Eye Terms

- Posterior uvea - The portion of the uvea made up of the choroid.
- Posterior uveitis - Inflammation of the choroid.
- Pupil - The circular opening in the center of the iris through which light passes into the eye.
- Primary problem of eye - The first, or foremost. Used in this case to describe problems that occur first and may be followed by other, secondary problems.
- Proptosis - Prolapse of the eye.
- Retina - The innermost coat of the eye, formed of cells sensitive to light.
- Retinal detachment - Separation of the retina from the underlying tissue, the choroid. It cause blindness.

Glossary (Definition) of Eye Terms

- Sclera - This is a layer of dense connective tissue that supports the eye. It is the white portion of the eye that connects to the cornea.
- Tonometry - The measurement of intraocular pressure.
- Topical - Medication that is applied locally on an external portion of the eye.
- Uvea - The portion of the eye which contains many blood vessels.
- Uvea is comprised of the iris, ciliary body, and choroid. It is called the vascular tunic.
- Uveitis - Inflammation of the uvea.

Functions of Eye

- To function properly, eyes must be kept moist.
- Tears are the source of this needed moisture.
- Tears are produced by 2 types of glands.

1. Lacrimal glands produce the watery portion of tears.

- They are located at the top outer edge of each eye.

2. Mucus glands: It is in the conjunctiva produce mucus that mixes with the watery portion of tear.

Functions of Eye

- This creates a more protective tear that is slower to evaporate.
- Nasolacrimal ducts allow tears to drain from each eye into the nose.
- Each of these ducts has openings at the edge of the upper and lower eyelids near the nose.

Examination (Test) of the Eye

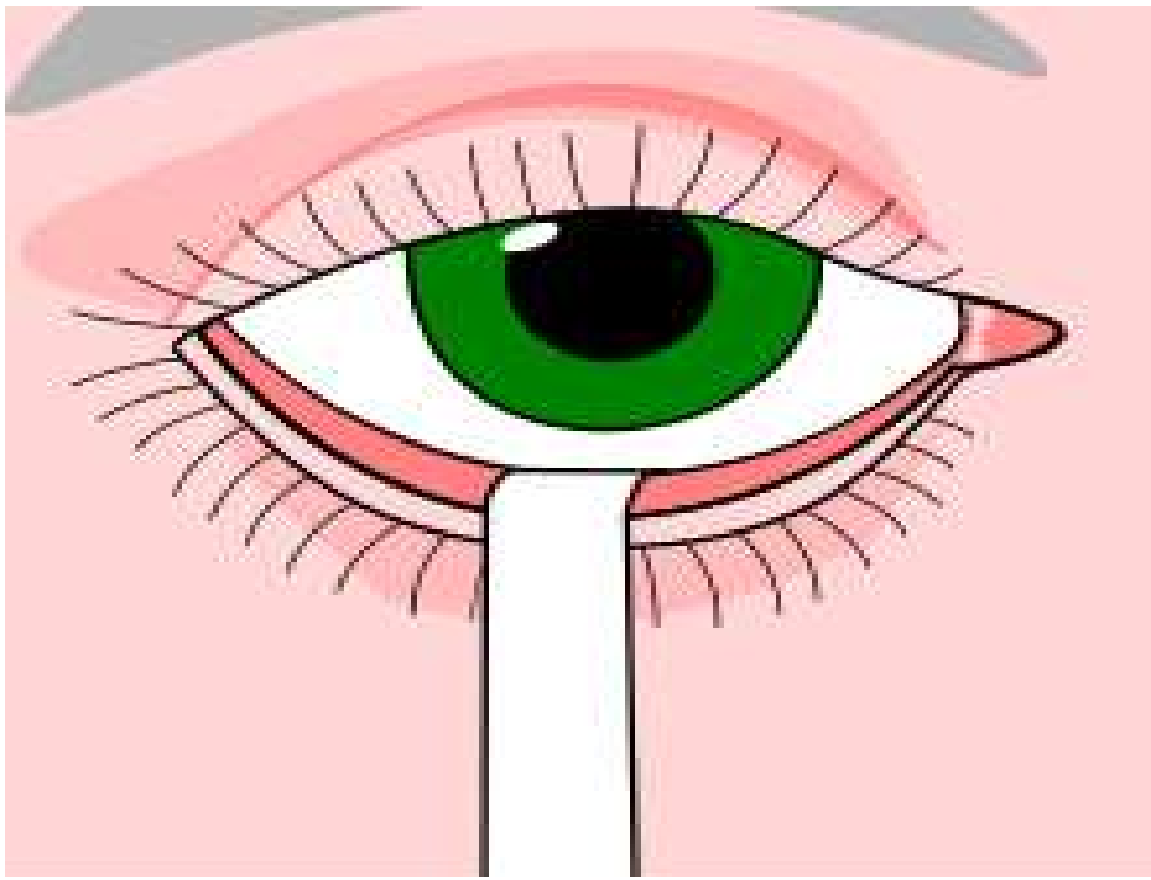
- Examine the shape and outline of the eyes.
- To see there are no obvious abnormalities.
- Using light and magnification in a darkened room.
- The reflexes of the pupils and the front part of the eye will be examined.
- A test, called the Schirmer tear test, may be performed to ensure that the eyes are producing enough tears to keep the eyes moist.

Schirmer's test(Dry Eye Test)

- To determines, enough tears produce or not
- Placing a small strip of filter paper inside the lower eyelid (inferior fornix).
- The eyes are closed for 5 minutes.
  - The paper is then removed and the amount of moisture is measured.

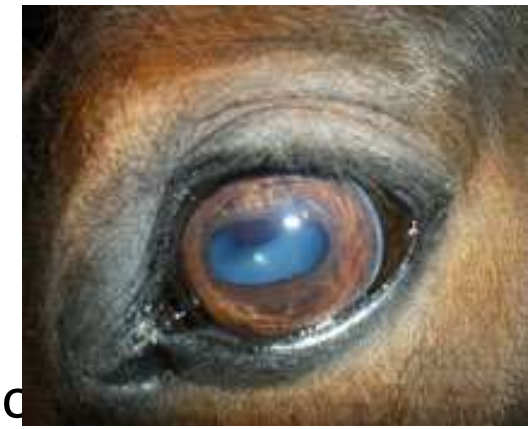
1. **Normal which is ≥15 mm wetting of the paper after 5 minutes.**
2. **Mild which is 9 -14 mm wetting of the paper after 5 minutes.**
3. **Moderate which is 4-8 mm wetting of the paper after 5 minutes.**
4. **Severe which is <4 mm wetting of the paper after 5 minutes**

Schirmer's test, placing the strip in the lower eyelid pouch



Ophthalmological examination

Vision
Lids
Tear film, discharge
Conjunctiva
Cornea
Sclera
Anterior chamber and aquec
Iris constricted/dilated Pupillary light reflex
Lens
Vitreous
Retina



Opthalmoscope

ENTROPIAN

- Eyelid Folding Inwards in **Horses** is called **Entropion**.
- **Entropion** is a condition of the eye that is seen in neonatal foals.
- In **Entropion** eyelids fold inward and press against their cornea.
- This creates a problem because the inward folding causes the eyelashes to rub against the cornea, resulting in **corneal ulcerations.**
- It is a **hereditary,** when a foal is born with his eyelid turning inward.
- This causes hairs on the surface of eyelid to rub against the eyeball.
- Leading to irritation of conjunctiva and cornea(Conjunctivitis and Corneal ulceration).

ENTROPIAN

- The condition may also occur after injury, infection or the exposure to certain irritants in a foal or full grown horse.
- Entropion will cause excess tearing and the horse will feel uncomfortable.
- Very long eyelashes may cause scarring and sores on the cornea of a horse.
- Condition must be treated quickly.
- Rubbing from the eyelash and eyelid can lead to infection in the eye.
- Entropion is a significant cause of corneal ulceration.
- Causing excess tearing and discomfort in the foal or horse.
- Entropion is a condition where the eyelid turns inward, causing the animal's eyelashes to rub against his eyeball.

Causes of Entropion in Horses

- Entropion is very often a hereditary condition.
- The foal is born with his eyelid turning in, causing the eyelashes to rub against his eyeball.
- Much less frequently the condition can be the result of horse having an injury to the eyelid or due to chronic eye irritation or spasms.
- The condition can also occur due to either an eyelid laceration.
- Being poorly stitched, with the scarring causing the eyelids to roll in.

Symptoms of Entropion in Horses

- Eyelid will roll in against his eye
- More tearing than normal.
- Irritation and redness will lead to discomfort in foal or horse.
- Horse may be dehydrated or uninterested in eating
- Forced, involuntary closing of eyelids may be noticed.`
- May display conjunctivitis.
- Unusual intolerance of light.
- Corneal ulcers.

ENTROPIAN

Diagnosis of Entropion in Horses

- Entropion is typically easy to diagnose.
- The horse will avoid secondary complications with a good prognosis.
- The eye examination may include an intraocular pressure test.
- Schirmer tear test.
- Fluorescein stain test.
- If the horse is experiencing pain in the eye, sedation may be needed to perform the diagnostic evaluation.

Treatment of Entropion in Horses

- Sutures will be placed carefully (a small amount of skin around the eye is stitched together) in an effort to roll the lid away from the eye of the foal.
- Decrease the pain for injecting medication into the lid near the area where it is turning in or using anesthetics to block the nerves in the eyelid.
- In most foals, this will allow the condition to self-correct.
- Surgical correction will be required and results are typically very good.
- Another option is to inject fluid into the eyelid which will lead to it rolling out.

ECTROPIAN

- Ectropion is an out-turning of the eyelid.
- This results in increased exposure of the cornea and conjunctival surfaces that may result in corneal desiccation and inadequate distribution of tears.
- Ectropion is most commonly associated with scar formation secondary to trauma or previous surgery.
- Ectropion, or eversion of the eyelid affects the lower eyelid only.
- Clinically significant ectropion is much less common than entropion.
- Cicatricial ectropion: It is an alternate form of ectropion due to contraction of scar tissue from previous injuries or surgical procedures such as overcorrection of entropion.
- Cicatricial ectropion is most common in horses and dogs.
- Regardless of cause, ectropion may result in severe secondary corneo-conjunctival lesions.
- If ectropion is severe enough and is left untreated.

Treatment of Ectropion

- Ectropion requires surgical correction when it causes conjunctivitis, keratitis, or exfoliative blepharitis due to epiphora or when it exacerbates keratoconjunctivitis **sicca.**
- Many horses tolerate slight ectropion with no ill effects and correction is required much less frequently than for entropion.
- A variety of techniques has been described for the correction of ectropion.
- Patients with more complex ectropion and combined entropion-ectropion should be referred to an equine ophthalmologist.

KERATITIS IN HORSES

- Keratitis is the inflammation of Cornea
- Keratitis can be a serious disease, threatening horse's vision.
- It usually only affects one eye, but there have been cases where both eyes are affected, leading to total blindness.
- Immune-mediated keratitis, IMMK, or commonly called just keratitis, is a non-infectious eye disease.
- It is quite common in horses and is recognized by a chronic corneal opacity that does not present an ulceration or uveitis.

Symptoms of Keratitis in Horses

- Daily examinations of horse should be done, including looking at eyes.
- Inflammation of the eyelids
- Congestion of the blood vessels in the conjunctiva
- Swelling of the conjunctiva and **Squinting**
- Redness of the conjunctiva
- Watery eye with Corneal edema and Ocular discharge
- Excessive amount of blood vessels within the cornea
- Softening of the cornea
- Drying or ulceration of the cornea Proliferation of blood vessels within the cornea.
- Aversion to light Pupil constriction

Symptoms of Keratitis in Horses

There are four types of keratitis in horses:

1. Epithelial Keratitis: There is no stromal edema but within the superficial opacity there will be irregular clumps or lines of thickened epithelium.

- When a light is shined on the cornea, the cornea will look rough and jagged instead of smooth.
- It generally occurs where the upper and lower eyelids meet.
- Epithelial keratitis is the most superficial type with discomfort.

2. Chronic Superficial Stromal Keratitis:

- There is prominent subepithelial arborizing vascularity from the limbus.
- The limbus are the blood vessels that look like tree branches.
- There is usually an increased blood flow within the eye.
- Yellow-white tint to the cornea when light is shined into the cornea.

Symptoms of Keratitis in Horses

3. Chronic Deep Stromal Keratitis:

- The blood vessels within the eye have ruptured.
- This causes plasma to be released into the stroma, giving the cornea a green-yellow tint.
- Significant pain when the blood vessels rupture.

4. Endotheliitis:

- The innermost layer of cells within the cornea, or the endothelium, is affected.
- In **Endotheliitis, eye** stops moving water from cornea and it starts to turn color.
- This is the most dangerous and damaging type of keratitis.
- Endotheliitis can progress into glaucoma.
- This is very painful for horses.

Diagnosis of Keratitis in Horses

- Ocular examination to diagnose and assess the severity of the keratitis.
- Horses need to be properly restrained and given a light sedative.
- Eye is stained to see any defects in the cornea can be seen (**Fluorescein Test**).
- Fluorescein Test- It is orange in the tube but become green when goes to eyes in the case of corneal ulcer.
- Any abrasions to the cornea can be very hard to see without stains.
- Any defects in the outer layer of the cornea allow the stain to diffuse into the middle layer.
- The lesions will appear bright green.

Treatment of Keratitis in Horses

- Most horses that have been diagnosed with keratitis need to managed disease medically to avoid blindness in the affected eye.
- Surgery should be a last resort.
- Horse may be stubborn and refuse to accept their medications.
- Subpalpebral lavage where a flexible tube is inserted into the upper and lower eyelids and then stitched into place.
- The medication is administered through the tube.
- Antibiotic, anti-fungal, anti-inflammatory, atropine and anti-protein medications may be prescribed.

Keratoconjunctivitis in Horses

- Infectious keratoconjunctivitis is an acute, contagious disease characterized in earlier stages by photophobia, conjunctival hyperemia, epiphora, and edema.
- Later stages by ulceration and opacity.
- Perforation may result from the ulceration.
- In less severe cases, corneal healing associated with fibrosis and neovascularization occurs in 3–4 days.
- Keratitis or keratoconjunctivitis can result from the migration and death of *Onchocercia cervicalis* microfilaria in the corneal and conjunctival tissues.
- An immune response to dead microfilaria likely initiates disease.
- Onset may be preceded by administration of ivermectin.

Equine glaucoma

- In Glaucoma the drainage of the aqueous humor is blocked.
- In Glaucoma intraocular pressure is increased due to icreased accumulation of aqueous humor.
- **Equine glaucoma** is most often a result of intraocular inflammation from equine recurrent uveitis.
- Similar to **glaucoma** in humans, the disease in **horses** is sometimes painful and eventually results in vision loss.

Glaucoma in a Horse

Causes of Glaucoma in Horses

- Aqueous humor is a normal substance found in the eye, which supports the eye health, nourishing the different structures.
- This is produced by the ciliary body processes of the eye and travels through the eye and is drained through the **iridocorneal and uveoscleral outflow pathways.**
- In many cases of glaucoma the drainage of the aqueous humor is blocked.

Glaucoma can be due to:

- **Primary causes** – Due to alterations to the internal structure of the eye, leading to abnormal iridocorneal or uveoscleral outflow pathway.
- **Secondary causes** – Due to blocked outflow pathways caused by trauma such as perforation of the cornea, inflammation, neoplasia of the eye or repeated cases of uveitis.

Symptoms of Glaucoma in Horses

- Glaucoma a difficult disease for owners to spot, especially in early stages.
- Often, the only sign of this condition is slight pupil dilation.
- Other symptoms may be seen and vary depending on severity of disease.

These Symptoms may include:

- Corneal opacity with Red or painful eye.
- Eye closure or **squinting.**
- Reduced vision or blindness and excessive production of tears.
- Pupil constriction.
- Corpora nigra atrophy.
- Sequestered pupil. Retinal detachment in severe case.
- Enlarged or Buphthalmic eyes

Diagnosis of Glaucoma in Horses

- If horse is showing signs of glaucoma a quick eye examination, using a special light to look into the eye.
- This will allow to visualise the fluid in the eye, and may allow to see blockages, build up or trauma.
- Perform a test to measure the intraocular pressure (IOP).- Tonometry
- This test is performed using a tonometer, a small handheld instrument.
- This procedure is performed by applying a topical anesthetic to horse's eye to prevent blinking during the examination.
- Nerve block to the **auriculopalpebral nerve** to further aid in examination.
- The IOP is then recorded.
- Normal range is considered **20-28 mmHg**.
- If horse's IOP is above 32 mmHg a diagnosis of glaucoma may be made.

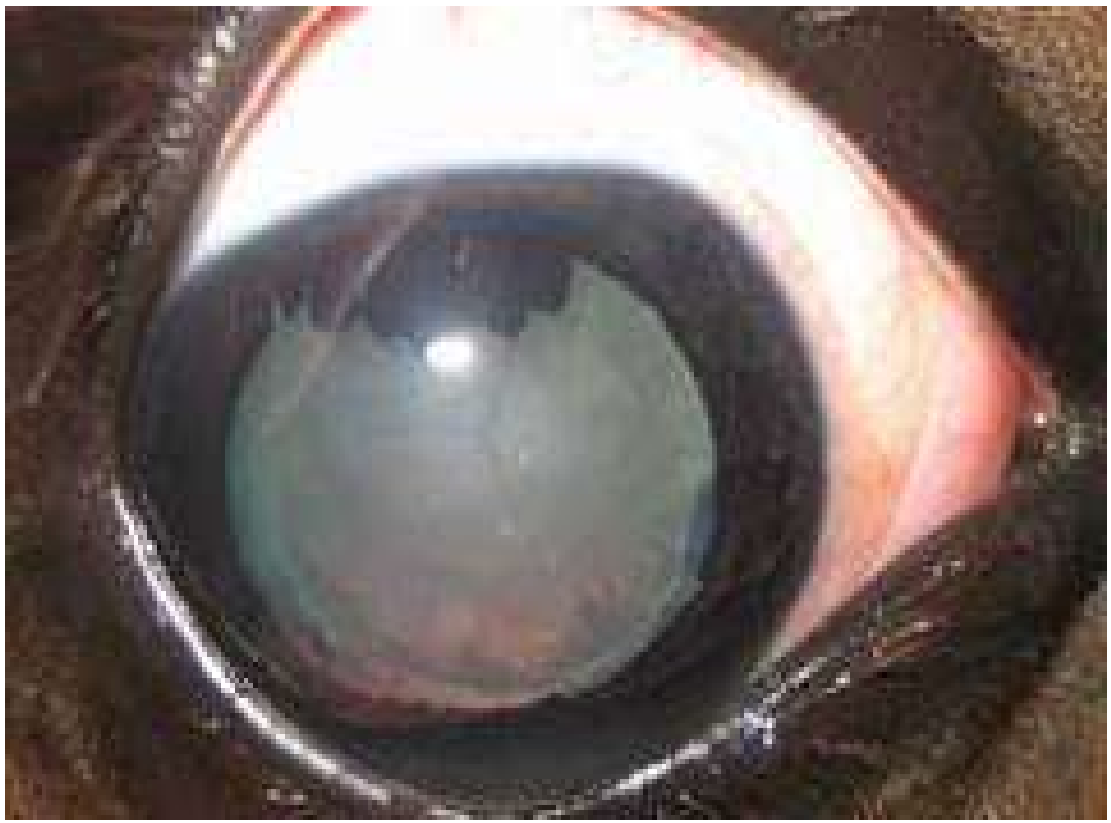
Treatment of Glaucoma in Horses

- First action taken is to relief pain of horse and attempt to reduce IOP.
- Topical medications such as corticosteroids may be given that can be placed in horse's eye to reduce inflammation.
- Oral NSAIDs may also be given to reduce inflammation and pain relief.
- Beta-blockers and carbonic anhydrase inhibitors may be given separately or in combination to reduce the IOP.

CATARACT IN HORSES

- A **cataract** may appear as cloudiness or haze in **horse's** eye(Lense) .
- The majority of **cataracts in horses** occur in adult animals.
- They most commonly develop secondary to diseases that cause intraocular inflammation, such as **Equine** Recurrent Uveitis (ERU or moon blindness).
- Cataracts are deep inside the eye.
- So it is hard to see the milky white opaqueness.
- Some people believe when outer eye is white and cloudy that is a cataract.

Cataract ina Horse



Causes of Cataracts in Horses

- Congenital cataracts are genetic and affect mostly Thoroughbred, Morgan and Belgian horses.
- Senile cataracts are thought to be caused by repeated bouts of eye inflammation and affects older horses eyes.
- Nutritional deficiency.
- Exposed to certain toxins can cause cataracts in some cases .
- Trauma or injury to the eye can cause cataracts.

Symptoms of Cataracts in Horses

- Cloudiness or white opacity of the pupil.
- Walking and dashing to objects and things
- Shying back for no reason.
- Jumpiness nil
- Inability to walk straight.

Cataracts are separated into three categories:

1. Incipient (or early) cataracts only affect a small part of the lens and usually does not cause a reduction in sight.

2. Immature cataracts are more serious and create a deterioration in sight that progresses with age.

3. Mature cataracts involve whole lens and cause blindness if not treated.

Treatment of Cataracts in Horses

- The only treatment for cataracts in horses is surgical removal of the cataracts.
- Cataract removal is a common surgery in horses and only takes a short time.
- The ophthalmologist will need to do a vision examination to make sure the horse is a good for surgery.
- It is not usually successful in horses with inflammation, infection, or other problems.
- Treat If horse has a inflammation or any problem before surgery.
- For inflammation, corticosteroids.
- Antibiotics for an infection.

Management of horses with eye disease

- Dark stable
- Eye protection

Helps maintain pupil dilation

Essential if horse has had atropine eye drops to prevent sunlight causing pain

- Clean and protect skin against discharges

Enucleation

- Removal of a painful non seeing eye is called **Enucleation.**
- This will greatly improve the welfare of a horse with a blind but painful eye.
- General or local anaesthetic (nerve blocks)
- Retrobulbar, auriculopalpebral or frontal nerve block.
- It is a major surgical intervention.

Parasitic Skin Diseases

1. **Rainrot (rain scald) ---- Dermatophilosis**
2. **Ringworm (fungal dermatitis)**
3. **Warts (papillomas)**
4. **Allopecia**
5. **Hives**
6. **Urticaria**
7. **Primary seborrhea (dandruff)**
8. **Mange**
9. **Lice (Pediculosis)**
10. **Seborrhea (Dandruff)**
11. **Sweet Itch**
12. **Onchocerciasis**

RAIN ROT

- Rain rot, or rain scald, is a bacterial skin infection caused by the spores of Dermatophilus congolensis.
- This bacterium can live on the skin of a horse and not cause any problems until the skin is compromised by prolonged periods of dampness and humidity.
- Biting insects can spread the bacterium from horse-to-horse.
- This infection is common in cows, sheep and goats and can also be found in humans.
- Dermatophilus can spread to humans, so don’t ride bareback on a horse with rain rot.

Etiology

- The causative organism, *Dermatophilus congolensis,* can reside on the skin without causing trouble.
- It multiplies rapidly in a moist environment.
- *D congolensis* is a gram-positive, non-acid-fast, facultative anaerobic.
- If the bacteria find a break in the skin, whether a small wound or insect bite, an active infection can develop.
- Anything that compromises a horse's immunity---advanced age, malnutrition, illness---can make him more susceptible to infection.
- Heavy winter coat, which tends to trap moisture against the skin.

Transmission

- The tropical bont tick is a three-host tick.
- Each life stage (larva, nymph and adult) feedon a blood meal on a particular host before dropping off and molting .
- The tropical bont tick *(Amblyomma variegatum* Fabricius) has its association with dermatophilosis.
- The tropical bont tick is originated in Africa.
- Factors such as prolonged wetting by rain, high humidity.
- Ticks and lice are major predisposing factors.

Clinical Findings

- Rain rot will start with bumps and matted coat on a horse's back, rump, head or neck – areas where insects bite.
- These will turn into scaly flakes of skin, or scabs that are itchy.
  - In long haired horses
    - There are large plaques of matted hairs and crusts which cover a moist gray to pink indurated surface.
  - In short haired horses;
    - There are multifocal nodules covered with crusts or scales in size of 1 to 2.5 mm
    - The lesion present on muzzle, around eye, distal extremities and along the back
  - In racing horses;
    - Lesion may present at pastern or fetlock as numerous scabs adherent to base of hair follicles with excessive exudation fissures and cracks resulting in lamness.

Treatment

- Chlorhexidine applied for five to 10 minutes, will aid in drying out the area and clearing up scabs.
- Dermatophilosis can be transmitted to people.
- Affected animals should be handled with gloves.
- Penstrip in recommended doses should be given.
- Anti-microbial shampoos and disinfectant rinses are recommended.
- Horse's coat will probably need to be treated daily for at least a week.

RINGWORM IN HORSES

Etiology:

- Ringworm is a skin infection caused by a dermatophyte (skin ‘loving’) fungus.
- Ringworm in horses include the Microsporum
- This can infect not only horses but other animal species, including humans.
- Skin lesions start as small raised spots with hair loss.
- These spread from these spots and usually become scurfy or a thick dry crumbly scab .
- Sometimes the lesions are sore and sometimes itchy.

Pathogenesis

- The respiratory efflux of carbon dioxide from the skin attracts the motile zoospores to susceptible areas on the skin surface.
- Zoospores germinate to produce hyphae, which penetrate into the living epidermis and subsequently spread in all directions from the initial focus.
- In wet scabs moisture enhances the proliferation and release of zoospores from hyphae.
- The high carbon dioxide concentration produced by the dense population of zoospores accelerates their escape to the skin surface.
- This result in to lesions of dermatophilosis.

Pathogenesis

- *Trichophyton mentagrophytes* requires approximately 6 hours to adhere firmly.
- Spore germination begins after 4 hours.
- Adherence occurs after 6 hours and germination of the conidia and branching after 16 hours.
- Other report showed ,12 hours is for adherence and 16 hours for germination.
- 72 hours for invasion of the stratum corneum.

Pathogenesis

- Once dermatophytes adhered to keratinized tissue, they release enzymes (keratinases, metalloproteases, and serine proteases).
- It also produce lipases and ceramides.
- The production of these enzymes is induced by the substrate on which they develop.
- Dermatophytes possess specific adhesins for some carbohydrates present on the skin surface.
- Why it is ring(It is Aerobic) ?

Clinical Findings

- The lesions can look very similar to rain rot.
- Statistically the bacterial infection is much more common than the fungal infection."
- A typical ringworm lesion is a round, whitish crust. Multiple lesions may coalesces each other.
- Ringworm is seen in young horses, the immune compromised and the geriatric horses, not in your average, healthy, well-maintained horses.
- Rounded hairless patches with crusty and scabby skin.
- The affected areas may be sore or itchy, but they often cause no discomfort, and the horse may appear otherwise healthy.

Diagnosis

- microscopic examination of scrapings and hairs from the lesions.
- Fungal culture in S.agar media
- Wood's lamp examination and histology of tissues.
- Some dermatophytes fluoresce when they are stimulated by the wavelengths of ultraviolet (UV) light in a Wood's lamp.
- Dermatophytes can often be detected by microscopic examination of infected hairs and skin or nail scrapings.
- Hyphae rounding up into arthroconidia (spores) are diagnostic.
- Samples are usually cleared with 10% potassium hydroxide (KOH).
- Fungal cultures, which identify the species of dermatophyte, can be useful in diagnosis.

Treatment

- First clean the area with a antifungal antiseptic, Chlorhexidine.
- Then dry thoroughly before applying an antifungal ointment or medication.
- Whole-body lime sulfur dips (1:16).
- 0.2% Enilconazole rinses.
- 2% miconazole rinse
- Repeat the treatments until the infection is resolved.
- Exposure to sunshine will also kill the fungi.
- Microsized formulation of griseofulvin is used in canides @3–5 mg/kg daily po or in divided doses.

Treatment

- Itraconazole @5–10 mg/kg/day orally.
- Pulse therapy: 5–10 mg/kg/day for 28 days then on an alternate-week regimen that is 1 week on and 1 week off
- ketoconazole @5–10 mg/kg/day orall- 2 wks.
- Terbinafine @30–40 mg/kg/day orally - 2 wks.
- Fluconazole @5–10 mg/kg/day po – 2 wks.
- Fluconazole is least effective among all drugs.

Warts (Papillomas)

Warts is a raised gray or pink cauliflower-like growths that are not much bigger than peas.

- They may appear singly or in clusters.
- Most commonly on the muzzle or around eye.
- Occasionally on ears, genitals and lower legs.
- The growths do not appear to cause any pain or discomfort.
- Wart is of two types- Pedunculated and diffused(Flat).
- Pedunculated is mostly curable.

Etiology: Warts are caused by equine papillomavirus.

- The virus can survive on skin, equipment and structures for weeks.
- Younger horses, under 3 years of age, are more susceptible.
- Warts can appear in adults.

Treatment

- The warts are harmless. No treatment
- Unless warts inhibit horse from eating, blinking or moving or they are interfering tack.
- Left alone warts typically shrink and disappear in young foals after four months.
- Persist for a year or more when they appear in older horses.
- Warts can be removed by excised with laser or cryosurgery, but leave scars.
- Autogenous vaccine made from the wart itself has been used and may speed resolution.

SWEET ITCH (an allergic skin reaction)

- Sweet itch is an allergic skin reaction caused by Culicoides midge bites.
- Symptoms appear during the warm months between April and September, when midges and other insects are biting.
- **Symptoms:** The skin becomes swollen and extremely itchy, prompting the horse to rub and scratch the affected areas vigorously, seeking relief.
- This can cause bald patches, broken skin and sometimes bleeding.
- These patches are found in the areas where the midges bite, such as the mane and tail and occasionally on the belly.
- **Treatment:** Oral supplements and topical treatments containing nicotinamide (vitamin B3) can help reduce itching by reducing the production of histamine (compound that causes allergic reaction) in skin.
- Corticosteroid prednisolone is given to ease the itching.

ALOPECIA

- Alopecia is baldness.
- It isn’t a disease or infection, but a symptom of many disorders in horse.
- It affects the hair follicle and can make the hair fall out.
- It can be caused by bacterial or fungal infections, parasites, allergies, hormonal changes lymphoma or other auto-immune diseases.
- “The way to know if the alopecia is caused by trauma or something else is to take a piece of hair (that is still remaining in the area) and look at the tip.
- If the tip is broken it is self-inflicted (trauma) and it can be some sort of allergy or disease that is making them itch.
- If the tip of the hair is not broken, it may be hormonal or systemic illness,”

HIVES (URTICARIA)

- **HIVES is** Known as urticaria in the veterinary Medicine.
- Hives are bumps on the skin that appear due to an overactive immune reaction to environmental triggers.
- There can be a million reasons for hives.
- Some horses can get hives just from physical exercise or from too hot or cold."

Symptoms:

- Bumps appear quickly in localized areas, most commonly on the back, belly and neck.
- It disappear as quickly as they appeared.
- Many horses are not itchy with hives and this is a difference from other species.
- **Treatment:** Antihistamines – both topical and oral – can ease itching.
- Hives resolve themselves quickly.

URTICARIA

- **It** is a common condition in horse compared with other domestic species caused by a variety of stimuli e.g. drugs, food, inhaled substances.
- Infectious organisms, bites/stings, contact allergy, stress, cold, heat etc.
- The characteristic signs are wheals up to a few centimetres in diameter which usually are circular.
- They develop rapidly over the chest, neck and face.
- Associated itch is not uncommon and serum leakage through the skin occasionally occurs.
- Treatment involves identification of stimuli (can be very difficult).
- In acute casess steroid with antihistamin is effective.

Seborrhea (Dandruff)

- Seborrhea can be dry or oily.
- *It is of two form– Dry and* Oily seborrhea
- In the dry form, small flakes of skin appear routinely, usually at the base of the mane and tail.
- Sand-like flecks may appear on the girth area or anywhere sweat accumulates.
- Oily seborrhea causes large, waxy crusts, often on the elbows, hocks or lower legs.
- When peeled off, these may leave hairless patches up to several inches in diameter.
- Dandruff sometimes causes fetid odor, but no itchy or pain in horses.

Etiology :

- Heredity cases of primary seborrhea.
- It is more common in Arabians and Thoroughbreds.
- Dandruff is likely to be a lifelong issue.
- Primary seborrhea looks just like secondary seborrhea.
- This can also be either oily or dry.
- Secondary seborrhea is due to liver or intestinal diseases.
- Onset of Secondary seborrhea is gradual.

Treatment

- Primary seborrhea is not curable, but it can be managed.
- Antidandruff shampoos are available that can dissolve flakes and loosen oily scales.
- It's best to select shampoo formulated for use on horses.
- Human products can be too harsh.
- Gentle scrubbing with a soft bristled brush.
- 2ndary seborrhea cured when causing disease is treated.

Prevention :

- Regular grooming of horses stimulates circulation and natural oil production that help keep the skin healthy and clean.
- Feeding vegetable oils, especially omega-3 fatty acids.
- Supplements that contain biotin may also help promote healthier skin.
- Equine nutritionist should be consulted.

MANGE IN HORSES

- Mange is a parasitic infection caused by several species of tiny mites that can barely be seen by the naked eye.
- Most common form in horses is chorioptic mange.
- It is caused by mite *Chorioptes equi.*
- This typically affects lower legs of horses.
- Rarely horses develop psoroptic mange *(Psoroptes equi).*
- Psoroptic mange produces lesions under mane and tail, under the jaw and in the groin and armpits.
- Demodectic mange *(Demodex equi)produces lesions* over the face, neck and shoulders.

Symptoms

- Small, round bumps at first, soon followed by bald spots, with scaly, thickened skin.
- Usually lesions on the lower legs of draft horses.
- Any horse can be affected.
- In more serious cases the skin may be rubbed raw and show signs of secondary infections.
- Mange causes itching.
- Horses rub, stamp and bite at their legs.
- Rarely mange appear on other parts of body.
- Permanent thickening and scarring of the skin .
- Thick skin impede the movement of pastern joints.

Diagnosis

- Each sample was shifted to clean petri dish and 10% KOH was used to release the mites from scabs & crusts .
- It is warmed at 38ºC for about two minutes.
- Examined microscopically for the presence of various stages of mites i.e. egg, larva, nymph and adult.
- Skin scrapings (negative) were shifted to separate test tubes containing 10 ml of 10% KOH.
- Reheated for five minutes centrifuged for 3 minutes at 2000 rpm and the supernatant was discarded.
- 5ml water added to the sediment and centrifuged again for 3 minutes at 2000 rpm. After discarding the supernatant the pellets were collected and examined microscopically for the presence of different stages of mites

Sarcoptic Mange

- Mites usually spend their entire life on Horses.
- The female mite burrows into the skin and lays eggs several times as she continues burrowing.
- These tunnels can actually reach the length of several centimeters. There is itching
- After she deposits the eggs, the female mite dies.
- In 3-8 days, eggs hatch into *larvae* which have 6 legs.
- The larvae mature into nymphs which have 8 legs.
- Nymph then molts into an adult while still in the burrow.
- The adults mate, and the process continues.
- The entire life cycle requires 2-3 weeks.

Treatment

- Administering oral ivermectin or moxidectin may also be helpful in some cases.
- Treatments may need to be repeated three or four times at 12- to 14-day intervals.
- Products that contain lime-sulfur solutions, organophosphates or permethrins.
- These products can be applied by spraying, sponging or dipping the leg.

Lice (Pediculosis)

- Lice infestations tend to be more severe during the winter months but can occur at any time of year.

Two types of these parasitic insects infect horses:

- Chewing lice *(Damalinia equi)* feed on sloughed-off dead skin cells.
- These are more likely to affect the neck, flanks and the base of the tail.
- Biting or sucking lice *(Haematopinus asini)* feed on blood.
- This species prefers longer hair and may be found under the forelock and mane, the tail and on the pasterns of long-feathered horses.
- Both types cause skin irritation and itching.

Lice (Pediculosis)

Symptoms:

- Hair loss from rubbing, usually appearing first on the shoulders and neck, as well as on the head and the base of the mane and tail.
- Affected areas are intensely itchy and may also have abrasions and scabs from rubbing and possibly secondary infections.
- Flattened insects up to two to four millimeters long may be visible if the hair is parted and the skin examined in good light.
- Pale, translucent eggs may be attached to nearby hairs.
- The horse is also likely to be generally unthrifty and in poor health.

Treatment

- Permethrin sprays and wipe-ons.
- Insecticidal powders and shampoos---are effective against lice.
- Diazinon Spray
- Oral ivermectin may be effective, but only against the biting lice.
- Insecticides will not kill eggs.
- Treatment needs to be repeated in two weeks to eliminate newly hatched adults.

Sarcoids

- ***Sarcoids*** are the most common skin tumors of the horse.
- Thought to be viral in cause.
- Sarcoids frequently occur in areas subject to trauma and may spread to other areas on the same horse or to other horses through biting, rubbing, fomites (inanimate objects such as tack, grooming tools, et cetera) or insects.
- *Treatment:* Surgical removal, topical therapies.

Fibroblastic sarcoid

Nutritional Skin Diseases

- Nutritional deficiencies are rare in horses.
- Zinc are important causes of generalised skin diseases.
- There are reports of vitamin A deficiency.
- Severe nutritional deprivation may lower immunity.
- Selenium deficiencies cause secondary skin infections.
- The major part of the sign relates to the secondary infection.

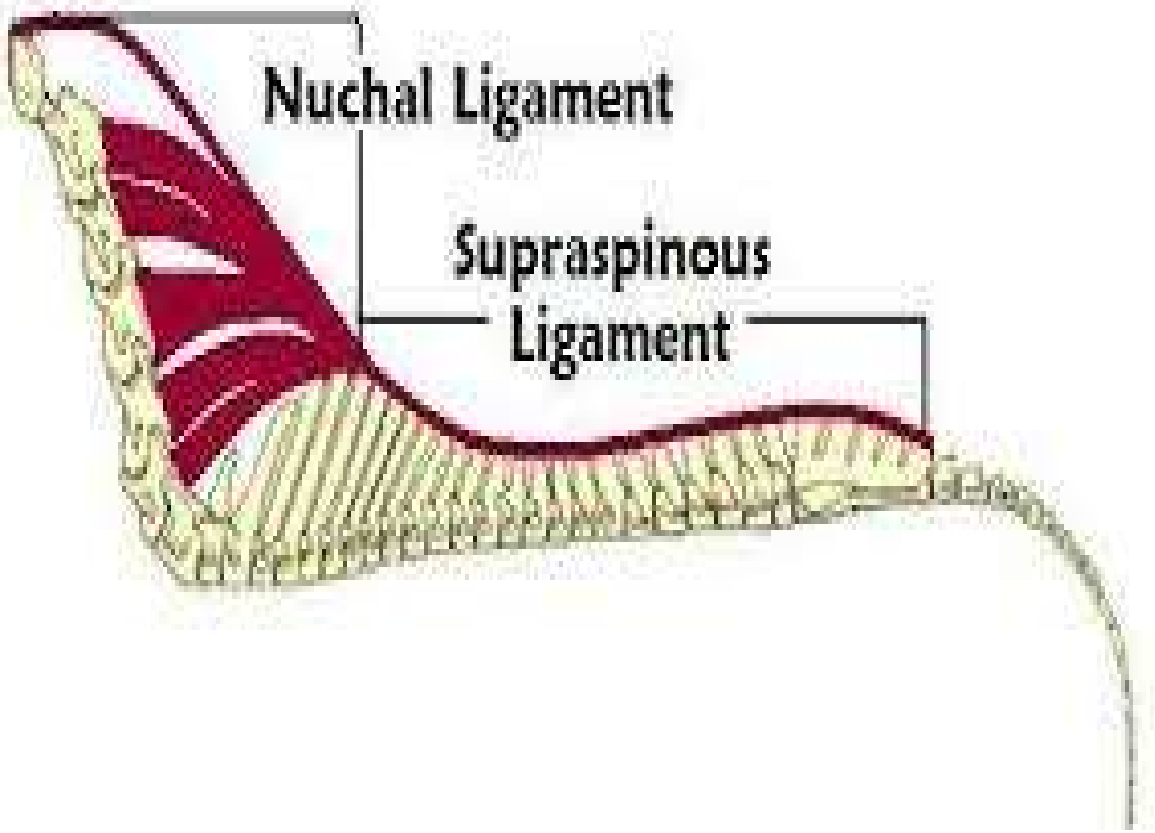
Protozoal Skin Disease

- Protozoal skin Disease is a rare cause of skin infection but it should not be entirely forgotten.
- *Besnoitia* sp. does cause some skin lesions in donkeys in some parts of the world including South Africa.

Onchocerciasis

- Onchocerciasis is an infestation by a roundworm in the Onchocerca family that lives in the nuchal ligament.
- Its larvae travel to the skin and cause severe itching.
- It is referred to as neck threadworms.
- Worms are around 2.5 to almost 12 inches long in adult.
- Generally live in ligament that runs down the nape of neck.
- Occasionally residing in other ligaments as well.
- When the adult worms release their larvae into the system, they travel to the skin where they cause small, itchy bumps.
- On rare occasions they may also travel to the eye, causing swelling and pain.

Nuchal Ligament



Etiology

- There are several parasitic worms in the onchocerca family, although not all of them infest horses.
- Onchocerca raillieti affects horses in Africa .
- Onchocerca cervicalis and onchocerca reticulata affect horses worldwide.
- Adult worms reproduce by releasing immature microfilaria.
- These are spread by biting insects such black flies.
- Onchocercosis (Microfilairiasis / Onchocerca *cervicalis*).
- Oxuyris *equi* is a cause of tail and peri-anal rubbing.

Symptoms

- The symptoms of onchocerciasis don’t appear until the larvae, microfilariae, migrate to skin.
- Once this journey has taken place, the larvae form small, itchy bumps.
- The horse generally rubs against fence posts or other hard surfaces, causing ulcerated sores, crustiness and hair loss.
- In some cases, the worms will travel to the eye instead. When this occurs, inflammation of the eye and discoloration can occur.

Diagnosis of Onchocerciasis in Horses

- A skin biopsy may be necessary to determine the exact cause of dermatitis.
- *Onchocerca* spp. microfilaria can be commonly found in the skin, even in asymptomatic horses, so their presence alone without inflammation may not confirm actual disease.
- Occasionally, radiographs of the neck can show mineralizations in the nuchal ligament.
- Adult worms can cause some inflammation and mineralization.

Treatment

- There is no effective treatment for eliminating the adult worms from the ligaments.
- Adult worms live there for up to a decade or longer with no negative symptoms.
- Dewormers such as ivermectin and moxidectin are given orally.
- It is quite successful in killing the microfilariae(Larvae).
- This eventually relief from itching and swelling.

Treatment

- The presence of microfilaria can cause intense itching and resultant skin trauma and swelling.
- Dead microfilaria are often more itchy than live ones.
- Horses may show intence signs after they have been dewormed with an effective dewormer.

EQUINE MEDICINE Part - II

MODULE No. – 13

Course Code – Vetm4146

Prepared by Dr. Abid Ali Bhat

Diseases of Alimentary Tract

Oral cavity and Soft palate

Dental disease

SIGNS OF DENTAL DISEASE

- ✓ Signs of dental disease are diverse and may present in many ways.
- ✓ It needs a thorough oral examination with a full mouth speculum.
- ✓ The oral cavity should be **inspected visually**, and each **tooth palpated** during the examination.

Signs of dental problems include:

- abnormal eating behavior (head tilt, quidding, dropping grain)
- excessive salivation
- discharge or fetid odor from mouth
- refuses to eat, eats slowly, or eats hay but not grain
- long (greater than 0.6 cm) hay particles in feces
- poor body condition
- dorsal displacement of the soft palate
- swelling or bumps on the maxilla or mandible

- purulent nasal discharge
- resists bridling or rears when bridled
- head tilts while ridden or lunged
- sticks tongue out of the mouth or over the hit

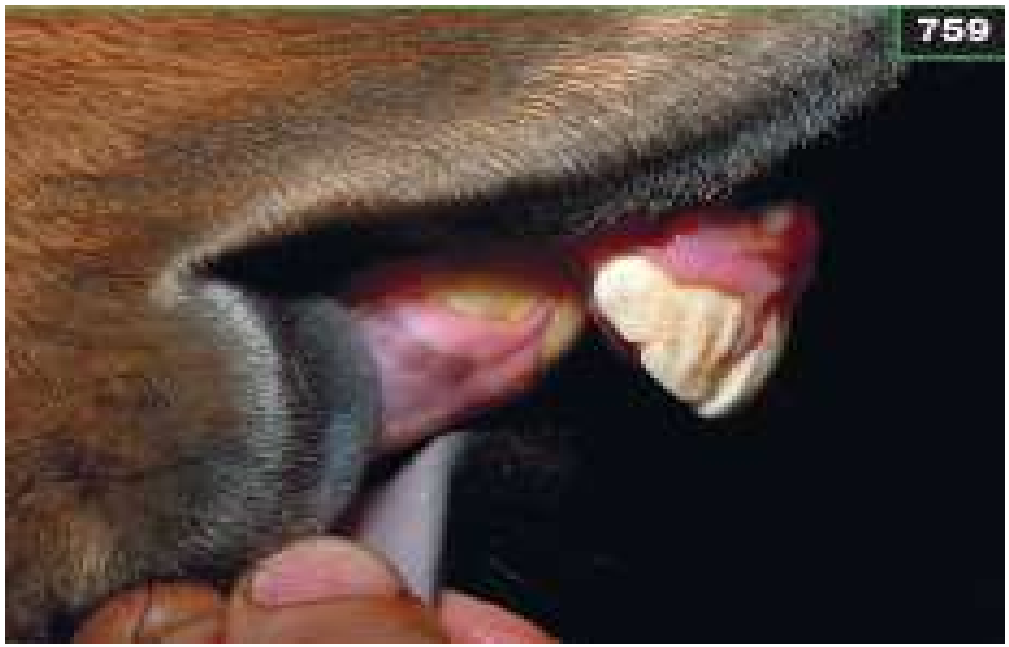
Management

As per condition: Periodontitis-antibiotics; malocclusion-tooth removal; sharp teeth: rasping; etc

DEVELOPMENTAL DISORDERS

Mandibular and maxillary **brachygnathia**

- It is the most common developmental oral abnormality when a mandible is shorter than the maxilla it is also called as **'parrot mouth‘ (Long-maxilla).**
- If the mandible is longer than the premaxilla (shortened premaxilla), the condition is called **'sow mouth**’ (**Prognathism-long mandible**)
- Both abnormalities are thought to be **inherited**.
- Sow mouth is less common than parrot mouth and is usually seen in **small breeds**, particularly miniature horses.
- **Foals** may be normal at birth, but develop these disorders by the time they are **2-6 months old**.
- **Parrot mouth** has also been classified as an **'overbite‘** or **'overjet'** deformity. An 'overjet' is where the maxilla protrudes further than the mandible, but the incisor arcades are maintaining their usual anatomic positions.



Brachygnathia

Treatment

Treatment for parrot or sow mouth is more successful if started while the horse is less than 6 months of age (**Surgical)**

Dental tumors

- ✓**Odontomas**: are **tumors** with histologic presence of both dentine and enamel, are rare in horses Or Odontomas are odontogenic benign tumors composed of dental tissue
- ✓Odontomas originate from **dental epithelium** and four types have been identified in the horse.
- ✓Odontogenic tumors generally **do not metastasize**, but they are **invasive** and successful removal depends on location and extent of bony, sinus, and soft tissue involvement.
- ✓Diagnosis is based on radiographic and histologic examination.
- ✓Treatment: is surgical excision

Dentigerous cysts

- Dentigerous cysts are **congenital abnormalities** due to an **embryologic disorder** leading to anatomically inappropriate dental tissue, often close to the pinna.

OR

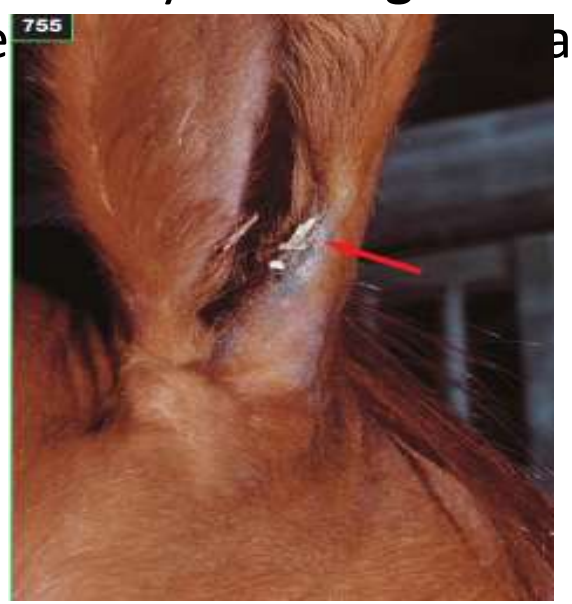
- Dentigerous cysts, also known as **ear teeth** or aural fistulae; are odontogenic cysts frequently containing stratified squamous or goblet cell epithelium or **fluid-filled sac** that develops in the jaw bone and soft tissue.

OR

- A cystic structure, typically present at the base of the ear, containing either **dental remnants** or **complete teeth.**
- They are commonly found at the **base of the ear**, other locations include the mandible, maxilla, and maxillary sinus.
- These cysts may have a seromucous or purulent discharge. Careful excision usually results in complete resolution.
- Radiographs are needed to differentiate between tumors, dentigerous cysts, and fluid cysts.

Pathophysiology

- The presence of ectopic dental tissue containing rudimentary enamel and other dental elements.
- The condition can present at any age and is often coincidental with the age of eruption of the teeth (usually less than 3 years).
- The most common site is at the base of the pinna.
- The cyst has a **stratified squamous epithelium** and **goblet cells** that secrete a **seromucinous fluid**, which commonly **discharges** through a **duct onto the skin**, often halfway up the le a.



The tract discharging from the base of the pinna in this horse (arrow) is typical in cases of dentigerous cyst.

Cysts

- Fluid filled cysts occasionally occur in the mandible, maxilla, and paranasal sinuses. They produce a variable degree of facial deformity and present as a smooth, firm, **non-painful**, gradually enlarging swelling.
- Aspiration of a pale yellow clear to turbid fluid coupled with **a radiolucent center** is indicative of a cyst.
- Surgical removal is the treatment of choice

Polyodontia

- **Supernumerary** teeth are considered congenital because they arise from abnormal differentiation of tooth germinal tissue.
- The condition is only recognized after tooth eruption. **Incisors** are the teeth most often affected.
- Removal is seldom indicated.

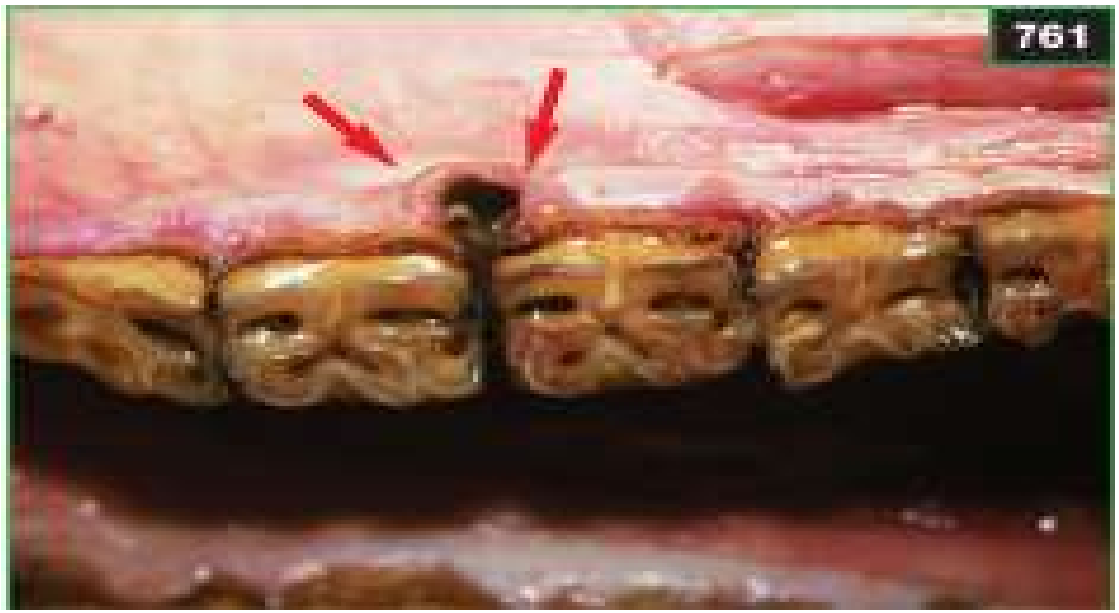
Periodontal disease

Periodontitis is

- inflammation of the gingiva
- resorption of alveolar bone
- loss of gingival attachment
- destruction of the periodontal ligament
- tooth loosening.

Periodontal disease is often **secondary to malocclusions**.

- Animals exhibiting **dysmasesis**: quidding, dropping grain, head tilt, and excessive salivation should be examined closely for early periodontal disease.
- Treatment for early periodontitis includes correction of any malocclusion, and routine (every 6 months) dental maintenance.
- Treatment of advanced periodontitis consists of **correcting any malocclusions** and evaluation of the diseased tooth for **extraction**.



Severe periodontal pocketing (arrows) resulting from diastema and periodontal food impaction.

Oral Tumors

- Oral tumors in horses are **rare**.
- The most common soft tissue tumor of the horse's oral cavity is **squamous cell carcinoma**.
- These tumors can involve any region of the mouth, occur in older horses, and produce a characteristic fetid smell.
- Treatment is **not rewarding** in cases of SCC.

Cleft Palate

The hard and soft palate function to

- **prevent feed contamination** of the nasal cavity and nasopharynx while eating
- **maintain an appropriate size** and **stability** to the nasal cavity and nasopharynx so that upper airway impedance is minimized during exercise.
- The **hard palate** separates the nasal cavity from the oral cavity.

- The **soft palate** separates the **nasopharynx** from the **oropharynx**.

✓Congenital cleft palate in horses is an **uncommon** deformity

✓**Most** defects affect the **caudal aspect of the soft palate**, and more rarely extend to the hard palate.

✓In addition **midline clefts** are more common than lateral defects. This disease **leads to nasal regurgitation of milk** and, later on, feed material, predisposing a horse to tracheal aspiration and **aspiration pneumonia**.

✓Affected animals therefore often have recurrent lower airway infection and stunted growth.

- The condition is discovered in most cases in the first few weeks of life because of the obvious clinical signs. The **appearance of milk at the nostrils** and coughing after nursing are distressful for both the foal and for its carers.

CLINICAL SIGNS

- milk, water, or food exuding from both nostrils
- coughing while nursing or eating
- un thriftiness
- stunted growth
- purulent nasal discharge
- fever
- depression
- chronic pneumonia.

Treatment: is achieved through surgical repair, but the anesthetic episode is complicated by the status of the lower airway.

Esophageal Disease

ESOPHAGEAL OBSTRUCTION

Esophageal obstruction or **choke** is the most common disorder of the esophagus and occurs as a consequence of **physical obstruction of the esophagus** with feed material or foreign bodies.

Etiology

- **Primary** choke is most often caused by **impaction** with common feeds that are of poor quality
- **Ingestion of foreign bodies**: carrots, apples, corncobs, shavings, or medicinal boluses can lead to esophageal obstruction.
- Other predisposing factors are **eating too early after sedation** or general anesthesia, consumption of pelleted or cubed feed, and inadequate water intake.
- **Secondary** esophageal obstructions are caused by anatomic abnormalities

- ✓**Intraluminal**: Esophageal strictures, diverticula, inflammation, ulcerated mucosa (from a previous obstruction), congenital disorders (megaesophagus, stenosis, cysts, and vascular ring abnormalities), and neoplasia.
- ✓**Extraluminal**: causes include mediastinal or cervical masses, such as tumors or abscesses.

Clinical Signs

Clinical signs of esophageal obstruction include **nasal discharge** of feed material and saliva, hypersalivation, coughing, and **frequent attempts to swallow**

Diagnosis

- Because esophageal obstruction can be considered an emergency, diagnosis is initially made on the basis of **clinical signs** of nasal discharge containing feed material and saliva, hypersalivation, coughing, and palpation of the larynx and pharynx.
- Palpation of the left cervical area may reveal a mass associated with the impaction.

- The veterinarian's **inability to pass a nasogastric tube** or **endoscope** is consistent with a complete obstruction.
- Careful passage of a **nasogastric tube** is a valuable method of confirming the location of an obstruction
- **Endoscopic examination** can help in determination of the nature and material of the obstruction
- **Radiographs** of the cervical portion

Management

- Most horses can tolerate obstruction for 24 hours or more without significant esophageal damage.
- However, **potential for dehydration and electrolyte disturbances**, aspiration pneumonia, and esophageal ulceration make emergency treatment essential.
- **Treatment** of choke initially consists of **sedating the horse** and providing supportive care because, in many cases, the obstruction will resolve spontaneously if the horse is rehydrated by intravenous fluid administration and the esophageal muscles are relaxed. **Hydraulic flush** is attempted several times.

- **Sedation:** with xylazine (0.25 to 0.5 mg/kg, IV) or detomidine (0.01 to 0.02 mg/kg, IV), in combination with acepromazine, (0.05 mg/kg, IV) or butorphanol (0.01 to 0.02 mg/kg, IV), will cause relaxation of the esophageal muscles and promote a **lower head carriage**, which assists drainage of feed and saliva from the nostril and prevents aspiration of feed material into the trachea.
- **Oxytocin** (0.11 to 0.22 IU/kg, IV) can be given for obstructions in the cervical portion of the esophagus to **relax striated** esophageal muscles.
- If sedation alone does not result in clearance of the obstruction: nasogastric tube is passed and gentle **irrigation with warm water** will soften most feed impactions and may be relieved.
- Surgical relief of the obstruction through esophagotomy is rarely indicated and should be reserved for cases in which repeated medical treatment fails to resolve the obstruction

ESOPHAGEAL STRICTURES

Strictures are circumferential areas of **narrowing** or **tightening** of the esophagus, which **lead to a reduction in the lumen** and can cause difficulties in swallowing

Etiology

- Strictures can be **congenital** or **acquired**. Factors associated with development of strictures are oral administration of corrosive medicinal agents, traumatic injury of the neck, previous esophageal obstruction, gastroesophageal reflux disease, esophagitis, dysfunctional lower esophageal sphincter, motility disorders, and hiatal hernia
- These processes may damage the mucosa and possibly the more external layers of the esophagus in a circumferential fashion, and while the area heals, a scar forms, causing the tissue to pull and tighten and impairing adequate swallowing.

Clinical Signs

- Signs are similar to those caused by esophageal obstruction because strictures result in a partial obstruction and accumulation of feed material in the lumen.

Diagnosis

Diagnosis can be difficult just based on clinical signs. Esophageal webs can be easily observed through endoscopy, whereas double-contrast radiography with barium or other substances may be necessary to confirm mural strictures.

Management

- Temporary strictures can cause **recurrent obstruction** for several weeks after the initial obstruction episode and usually resolve with medical therapy
- Nonsteroidal antiinflammatory drugs, coating agents (sucralfate, 22 mg/ kg, orally, every 6 to 8 hours), antimicrobials, and lidocaine drenches (relax smooth muscles)
- Several surgical procedures, including resection and anastomosis, and temporary esophagostomy.

ESOPHAGEAL DIVERTICULA

Etiology

- Diverticula are **pouchlike dilations** in the esophagus and may be congenital or acquired.

The two types of acquired diverticula are traction and pulsion diverticula.

- **Traction diverticula** arise from a **wound or inflammation** in periesophageal tissue that results in formation of **fibrous adhesions** that **retract** the esophageal wall and **create dilation** in the lumen.
- **Pulsion diverticula** result from **increased intraluminal pressure** or **deep esophageal inflammation** that creates a defect in the muscular layer of the esophagus. This results in **herniation** of the mucosa and submucosa into the muscularis.

- Diverticula **remain filled with food** after the rest of the esophagus empties, leading to further dilation.

Clinical Signs

- Traction diverticula are usually clinically silent, whereas pulsion diverticula, because of disruption of the muscular layers, tend to fill with feed, ultimately leading to **esophageal obstruction**.
- large diverticula lead to postprandial dyspnea secondary to **upper airway obstruction** or **aspiration** of feed material, and **regurgitation**

Diagnosis

- Diverticula should be suspected when a horse has **recurrent episodes of esophageal obstruction** or if **swelling** is observed in the cervical area, over the esophagus.
- Nasogastric intubation should be done very **cautiously** to avoid perforating the diverticulum. Endoscopic examination often reveals flattening of the lumen retained feed material.

Management

- **gruel or soft feeding** and **elevating the feed** trough so that the horse's neck is upright during eating can decrease the likelihood of obstruction.
- In severe cases, surgical correction can be undertaken.

MEGAESOPHAGUS

Megaesophagus is a persistent, diffuse, and often extensive **dilation of the esophagus**. It is associated with several diseases and can lead to retention of feed material.

Etiology

- Esophageal obstruction-induced megaesophagus is usually caused by persistent esophageal obstruction
- Esophageal motility–related megaesophagus is less common, and causes include equine grass sickness, botulism, poisoning by lead, thallium etc.

Clinical Signs

- Clinical signs include lethargy, ptyalism, dysphagia, **regurgitation**, **nasal discharge**, palpable swelling in the esophagus, and complicating **aspiration pneumonia**.

Diagnosis

- Clinical signs may be suggestive of megaesophagus, but a definitive diagnosis can be made on endoscopic examination and contrast radiography.

Management

- Treatment should be focused on correcting the underlying cause of megaesophagus. A diet of **slurries** or **pellets** will improve the transit of food. Additionally, the horse should be fed from an **elevated** trough to promote esophageal transit.
- If reflux esophagitis is the underlying cause, administration of **metoclopramide** (0.02 to 0.1 mg/kg, SC, every 4 to 12 hours)

DYSPHAGIA

Dysphagia is as difficulty in swallowing or inability to swallow

DIAGNOSIS OF DYSPHAGIA

Clinical signs

The signs of dysphagia include

- an unwillingness to eat
- slow eating
- messy feeding
- rejection of semi-masticated food onto the ground (quidding)
- productive coughing
- nasal reflux of saliva, ingesta, and fluids.

✓Horses that are unable to eat and swallow food **lose weight** rapidly, and this process is accelerated if the horse develops secondary **inhalation pneumonia** which is not an uncommon sequel to dysphagia.

- **Case history** recording the circumstances and rate of onset of dysphagia
- Whenever a horse shows return of ingesta from its mouth, the site of the lesion causing the dysfunction must lie in the oral cavity or oropharynx
- Nasal reflux of ingesta points to an abnormality of the pharyngeal or esophageal phase of deglutition.

Physical examination

- Thoracic auscultation should check for signs of inhalation pneumonia.
- Local lymphadenopathies and distension of the esophagus to the left side of the trachea are abnormalities that might be found during palpation of the throat area.

Nasogastric intubation

- This procedure determine whether pharyngeal swallow reflexes are still present, or whether the upper alimentary tract is physically obstructed.

Oral examination and Pharyngial examination

One should look for evidence of

- absence of teeth or dental malalignment
- enamel pointing of the cheek teeth
- fractures of the dental crowns
- periodontitis
- soft tissue lesions of the buccal cleft and palate
- oral foreign bodies
- lesions of the tongue.
- Oral tumors and Intralumenal pharyngeal neoplasia

Endoscopy

Endoscopy per nasum is necessary to confirm whether pharyngeal paralysis is present.

The usual findings of pharyngeal paralysis include:

- a mixture of saliva and ingesta on the walls of the nasopharynx
- poor nasopharyngeal constrictor activity during deglutition

Treatment

As per etiology

Medical: anti-inflammatory and analgesics drugs can be used to reduce symptoms.

Equine Gastric Ulcer Syndrome (EGUS)

EGUS describes **ulceration** in the terminal part of the **esophagus**, nonglandular and glandular portions of the **stomach**, and the proximal part of the **duodenum**.

- Equine gastric ulcer syndrome is complicated and has many causes.
- Prevalence of 25% to 51% and 60% to 90% has been reported in **foals** and **adult horses**, respectively.
- The prevalence of gastric ulcers is highest in younger performance age horses (<10 years of age).
- **Thoroughbreds** and **Standardbreds** in active training and racing are more affected.

PATHOGENESIS

✓ An **imbalance** between mucosal **aggressive factors** (i.e., hydrochloric acid, pepsin, bile acids, and organic acids) and mucosal **protective factors** (bicarbonate, mucus) is the likely cause of EGUS.

- ✓The mucus-bicarbonate layer covers the surface of the glandular mucosa.
- ✓**Prostaglandin E2** enhances mucosal blood flow, promotes secretion of this layer, and increases production of mucus and bicarbonate.
- ✓In addition, prostaglandins maintain the integrity of the nonglandular and glandular mucosa by stimulation of production of surface-active protective phospholipids, enhancement of mucosal repair, and prevention of cell swelling by stimulation of transepithelial sodium transport.
- ✓**Stress** resulting from various factors may lead to the **release** of excess endogenous **corticosteroids**, which can **inhibit prostaglandin synthesis**.
- ✓This decrease in prostaglandins may lead to **breakdown** of mucosal protective factors and can promote development of ulcers

RISK FACTORS

- **Stress and Exercise**

Exercise and concurrent illness lead to increased prevalence of gastric ulcers.

- **Drugs**

Treatment with nonsteroidal antiinflammatory drugs (NSAIDs) is associated with gastric ulcers in horses. Nonsteroidal antiinflammatory drugs inhibit cyclooxygenase, which in turn inhibits prostaglandin E2 production, thereby resulting in increased acid secretion, decreased mucosal blood flow, and disruption of the mucus–bicarbonate barrier.

- **Diet**

Feed deprivation is associated with ulceration of the nonglandular mucosa, likely as a result of repeated exposure of the mucosa to an acidic pH.

Clinical Syndromes

Four separate clinical syndromes have been recognized in foals: **silent** (subclinical) ulcers; **active** (clinical) ulcers; **perforating ulcers** with diffuse peritonitis; and **pyloric strictures** from resolving ulcers, which may result in gastric outflow obstruction.

Clinical signs

- Result when gastric ulcers become larger and more diffuse and coalesce.
- **Diarrhea** and **poor appetite** are the most frequent
- Clinical signs in foals: **Poor growth**, rough haircoat, **potbellied** appearance, **bruxism**, dorsal recumbency, **excessive salivation**, interrupted suckling, and colic also may be observed.
- Foals with **large ulcers** show signs of **severe colic**, tend to roll and lie in dorsal recumbency, and may be sensitive to abdominal palpation

DIAGNOSIS

Diagnosis of gastric ulcer disease is made on the basis of clinical signs, endoscopic examination, and response to treatment

TREATMENT

- Multiple types of medications are available to treat gastric ulcers in horses. These include antacids, H2 receptor antagonists, sucralfate, prostaglandin analogues, and omeprazole
- Giving antacids **alone** is **ineffective** for treatment of gastric ulcers.
- Types of antacids include aluminum hydroxide, magnesium hydroxide (0.5 mL/kg) and calcium carbonate.
- Ranitidine (6.6 mg/kg, PO, every 8 hours) and cimetidine are two drugs of this type used in the horse

Gastric Impaction

❖Gastric impaction in the horse is relatively **rare**, with an estimated prevalence of less than 1% of colic cases.

❖Gastric impactions are characterized as **primary** or **secondary**.

- **Primary** gastric impactions are caused by **functional** or **anatomic gastric defects**, including decreased gastric emptying, acid secretion, or pyloric stricture.
- **Secondary** gastric impactions are caused by **poor mastication**, **dehydration**, **hepatic disease**, or any gastrointestinal disturbance that causes generalized **ileus**. Gastric impactions often occur following consumption of substances that expand upon contact with water, including hay, persimmon fruit, bran, mesquite beans, and beet pulp or straw.

CLINICAL SIGNS

- Clinical signs of gastric impaction range from **inappetence to acute colic**. Pain is typically mild, but can be severe.
- **Pyrexia**, **dysphagia**, **fluid nasal discharge**, **decreased fecal output**, lethargy, weight loss, and **hypersalivation** have also been reported

DIAGNOSIS

- ✓Diagnosis of gastric impactions can be challenging because of the nonspecific clinical signs, but a history of ingestion of expansive substances should increase the index of suspicion.
- ✓Use of endoscopy and transcutaneous Ultrasonography
- ✓Horses with gastric impactions may have hematologic evidence of systemic inflammation (leukocytosis, leukopenia, or hyperfibrinogenemia (probably due to bacterial translocation or peritonitis).
- ✓Palpation per rectum may reveal medial displacement of the spleen in conjunction with gastric impaction.
- ✓exploratory celiotomy

TREATMENT

- Medical management is the preferred treatment for gastric impactions.
- The most important component of medical treatment is enteral administration of fluids. Frequent administration of low volumes of **enteral fluids** (about 2 L/hour) may help to hydrate and soften the impaction.
- **Intravenous fluids** are typically less efficacious than oral fluids at hydrating gastrointestinal impactions
- **Laxatives**, including dioctyl sodium succinate (10 to 50 mg/kg given in 2 to 4 L water), magnesium sulfate (0.5 to 1 g/kg in 2 to 4 L water every 24 hours), or mineral oil (2 to 4 L, every 12 to 24 hours).
- Horses with concurrent gastric ulcers may benefit from gastric ulcer treatment. **Sucralfate** (20 to 40 mg/kg, PO, every 6 hours) may be used in conjunction with acid suppressant therapy.
- In Non-responsive cases: **exploratory celiotomy** is performed , to remove impacted mass.

Hepatic Diseases in the Horse

CLINICAL SIGNS

- Signs of hepatic dysfunction are often **nonspecific** and depend on the severity and duration of hepatic disease.
- Common clinical signs include **depression**, **anorexia**, **colic**, and **weight loss**. Signs more specific for liver disease include icterus, photosensitization, and hepatic encephalopathy.
- Hepatic encephalopathy (HE) may manifest in multiple ways, including depression, aggression, yawning, circling or walking compulsively, or head pressing

DIAGNOSIS

Biochemical Tests

- Standard biochemical indices of hepatocellular disease include sorbitol dehydrogenase (SDH), aspartate aminotransferase (AST), and lactate dehydrogenase (LDH).
- **SDH** is the **most specific indicator** of acute hepatocellular damage in the **horse**.

- Standard biochemical indicators of hepatobiliary disease include γ-glutamyl transferase (GGT) and alkaline phosphatase (ALP).
- GGT is a specific indicator of biliary epithelial damage and is the **most sensitive** indicator of hepatic disease in horses.
- ALP is released from multiple sites, including liver, bone, and intestine, so elevations are not specific for liver disease.
- Serum bile acid concentrations may be consistently high with either hepatocellular or hepatobiliary disease.
- Serum or plasma bilirubin concentration can be high in horses with any type of liver disease.
- Blood ammonia levels may also be evaluated as an indicator of hepatic function.
- Hepatic Ultrasonography is a valuable diagnostic tool.
- Hepatic Biopsy: Liver biopsy is often required for a definitive diagnosis in horses with evidence of hepatic disease.

Acute Liver Disease

Acute Liver Disease is defined by a rapid decline in hepatic function characterized by **jaundice**, **coagulopathy**, and **hepatic encephalopathy** with no evidence of prior liver disease.

Theiler's Disease

Theiler's disease (acute hepatitis necrosis, serum-associated hepatitis, serum sickness) has been reported in horses.

- Theiler's disease is considered to be one of the **most common causes** of acute hepatic failure and disease typically occurs in individual horses.
- Horses with Theiler's disease have a **history** of receiving an equine-origin **biologic antiserum** in the 4 to 10 weeks before the onset of signs.
- Biologic products that have been implicated include vaccines, antisera, tetanus antitoxin, and plasma.
- Horses present with an acute onset of signs of hepatic failure, including **anorexia**, **icterus**, and **signs of HE**.
- There is no specific treatment aside from general supportive care. The prognosis is poor to grave.

Bacterial Hepatitis

- **Tyzzer's disease**, caused by ***Clostridium piliforme***, is the most common cause of bacterial hepatitis in foals.
- It is confined to foals between 7 and 42 days of age, which are often found acutely dead or with nonspecific signs of **enteritis** and **hepatitis**.
- ***C piliforme*** is found in the **feces** of healthy adult horses as well as in the **environment**.
- Foals are infected by **ingesting contaminated soil** or **feces**.
- The organism causes **acute multifocal hepatitis** and **enteritis** and can be identified on histologic examination of the liver.
- Other organisms or Isolates are usually enteric organisms, such as *Salmonella* spp, *Escherichia coli*, *Klebsiella* spp, and *Acinetobacter* spp. Treatment includes supportive care and long-term antimicrobial therapy.

Viral Hepatitis

- Viral hepatitis is most common in foals that were infected with ***equine herpesvirus 1*** while *in utero*.
- If the foals survive to birth, they are generally weak and have profound hepatic necrosis and pneumonia.
- **Intranuclear acidophilic inclusions** are found in hepatocytes and biliary epithelium. The prognosis is generally grave.

Parasitic Hepatitis

- Parasitic hepatitis may arise **secondary to parasitic infestation**.
- Hepatic damage is generally **focal** as a result of **parasite migration**, although generalized disease with hepatic insufficiency can occur rarely.
- Parasites that have been implicated include *Parascaris equorum*, *Strongylus edentatus*, *Strongylus equinus*, and *Strongylus vulgaris*. ***Hydatid cysts*** have been found in the livers of horses infected with *Echinococcus granulosa*.

Toxic Hepatopathy

- Numerous chemicals, **drugs**, **mycotoxins**, and **plant toxins** are hepatotoxic but rarely cause acute hepatic failure in horses.
- Some substances are directly hepatotoxic, whereas others **require biotransformation by the liver** to become toxic metabolites.
- Clinical signs and clinicopathologic testing rarely differentiate between toxins, so **diagnosis** is often based on ***history of exposure***, documentation of the ***toxin in the blood*** or ***liver***, and possible *pathognomonic* ***histologic*** *findings*.

Chronic Liver Disease

Progressive destruction of the liver parenchyma over a period greater than 6 months leading to fibrosis and cirrhosis.

- Horses with chronic liver disease may still be presented with acute onset of clinical signs. However, **histologic evidence** of **fibrosis** develops only with chronic injury.

Chronic Megalocytic Hepatopathy

- Chronic megalocytic hepatopathy occurs throughout the world and is the **most common cause** of chronic liver failure in horses.
- It is caused by the ingestion of **pyrrolizidine alkaloid** (PA)-containing plants.
- Horses are relatively sensitive to PA intoxication, and the effects of PA are cumulative, so chronic, **low-level exposure** is the most common cause of toxicosis.
- Clinical signs usually develop 4 weeks to 12 months after the consumption of PA.

- Horses often present with **photosensitization** or **signs of HE**.
- Less conspicuous signs of anorexia, weight loss, mild **icterus**, and exercise intolerance may also be seen.
- **Diagnosis** is confirmed by the **histologic findings** of megalocytosis, biliary hyperplasia, and fibrosis.
- Treatment consists of supportive care.

Chronic Active Hepatitis

- Chronic active hepatitis (CAH) is an idiopathic, **chronic, progressive hepatopathy** characterized histopathologically by biliary hyperplasia, periportal or biliary inflammation, and associated hepatocellular damage.
- Clinical signs often are **intermittent**, and include depression, anorexia, weight loss, colic, icterus, and fever.

Hepatic Neoplasia

- Primary hepatic neoplasia is **rare** in horses. **Cholangiocarcinoma** is the most common primary hepatic neoplasm, particularly in older horses.
- Hepatocellular carcinoma and hepatoblastoma have been reported in several young horses (<4 years of age).
- Hepatic neoplasia is more likely to **arise after metastasis** of some other primary tumor.
- Hepatic **ultrasonography** may indicate the presence of a discrete mass

TREATMENT

- Treatment of liver disease is generally focused on removing the instigating cause (especially in pyrrolizidine alkaloid toxicosis) and supportive care.
- More specific treatment recommendations can be prescribed on the basis of culture and histologic evaluation.
- The liver has the **ability to regenerate over time**, so many treatments aim to ease the workload of the liver or prevent fibrosis.

- ✓IV Fluids
- ✓Pentoxifylline (antifibrotic)
- ✓Silymarin (hepatoprotective)
- ✓S-adenosylmethionine (antioxidant)
- ✓Corticosteroids (antifibrotic)
- ✓vitamins A, D, E, and K (antioxidant)

Acute Colitis in Horses (Acute Diarrhea)

- Acute colitis is a general term referring to inflammation of the cecum (**typhlitis**), colon (**colitis**), or both (**typhlocolitis**), with subsequent rapid onset of diarrhea in the adult horse.
- Acute colitis is a common cause of rapid debilitation and death in horses.
- Colitis-associated diarrhea is sporadic in occurrence and is characterized by **intraluminal sequestration** of fluid, moderate to severe colic, and **profuse watery diarrhea**, with resultant *endotoxemia*, *leukopenia*, and *hypovolemia*.
- The condition can affect adult horses of all ages but usually affects horses 2 to 10 years of age. Disease onset is sudden, and progression is rapid.

PATHOPHYSIOLOGY AND CLINICAL SIGNS

- **Diarrhea** associated with acute colitis is a result of ***abnormal fluid and ion transport*** by cecal and colonic mucosa, with fluid loss resulting from a combination of both **malabsorption** and **hypersecretory** processes.

- Horses have **sudden**, **massive fluid loss** and severe electrolyte imbalances that can result in death in hours.
- This may be partly due to large population of **gram-negative endotoxin**-bearing bacteria that reside in the large intestine and the markedly high mucosal **prostaglandin** concentrations, manifested by a marked **chloride** secretory response.
- Another reason for the distinctive clinical signs is the intense inflammation that results from **activation** of resident intestinal mucosal and submucosal **phagocytic granulocytes** by intestinal bacterial products after mucosal barrier disruption.
- Typical hematologic findings include **hypovolemia**, dehydration, **metabolic acidemia**, **electrolyte derangements**, **leukopenia** with a left shift, toxic neutrophils, lymphopenia, and azotemia.

Etiology: Treatment with drugs such as nonsteroidal antiinflammatory drugs (NSAIDs), antimicrobials, or anthelmintics; changes in diet; inadequate deworming history; or a stressful event, *Clostridium difficile* ands *Salmonella* spp and *Neorickettsia risticii*.

Clinical features: include depression, inappetence, fever, tachycardia, dry mucous membranes, skin tenting, prolonged capillary refill time, colic, and watery, often fetid, diarrhea.

TREATMENT

Fl**uid Therapy**: Intravenous polyionic fluids should be administered at a volume based on total fluid deficit calculated from the clinical assessment of dehydration (e.g., for 8% or moderate dehydration, the calculated volume deficit would be 0.08 × 450 kg body weight = 36 L).

Colloid Replacement (Hetastarch-6%; to 10 mL/kg.)

Decreased colloid oncotic pressure leads to decreased effective circulating fluid volume and tissue edema.

Antiendotoxin Therapy: hyperimmune serum, Low-dose administration of flunixin meglumine and antimicrobials

Colic

Factors associated with increased risk of colic

SIGNALMENT

Age, sex, and breed have been associated with increased risk of colic.

AGE: Some forms of colic appear to be more prevalent in **younger** animals (e.g. intussusception in younger horses) while strangulating lipomas, for example, are more common in **older** horses

Risk of colic, surgery and poor prognosis for survival is **higher in older** horses.

Sex: Some forms of colic are **gender-specific** (e.g. uterine torsion or scrotal herniation).

Breed: **Arabian breed** found to be associated with increased risk of colic

Fecaliths and **impactions** of the small colon: more prevalent in younger **miniature horses**

Standardbreds: appear to be at increased risk of **scrotal hernias**.

MEDICAL HISTORY

Previous colic-History of previous colic has been repeatedly identified as a risk factor for colic.

FARM MANAGEMENT FACTORS

- Management practices are of particular importance
- **Management practices** have been **associated with increased risk** of colic
- Factors associated with colic in **one area** may not be relevant in other areas (e.g. **sand colic**).

Dietary factors can predispose to colic

- **Type** (e.g. corn) or **amount** (i.e. increased risk with increased amount) of concentrate fed is considered a risk factor, whereas **change in diet**, particularly a change in the type, quality, or batch of hay/forage fed may also increase risk for colic.
- Diet is widely regarded as an important risk factor for colic, dietary practices may be modified to decrease the risk.
- Constant **access to water** is important to prevent colic, and also the quality and palatability of the water is also important.

Pathophysiology of intestinal obstruction

PATHOPHYSIOLOGY OF INTESTINAL DISTENTION

- Intestine **proximal to an obstruction** becomes **distended** with secretions, gas, fluid, and digesta, and the bowel wall and mesentery become stretched resulting in abdominal pain.
- **Veins** in the small intestinal wall are **compressed** as lumenal pressure increases, and capillary hydrostatic pressure and capillary filtration rate increase.
- If **capillary filtration** into the interstitium overwhelms fluid removal through lymph flow, then **tissue edema** and a **net secretion of fluid into the intestine develops.**

PATHOPHYSIOLOGY OF INTESTINAL ISCHEMIA

- **Ischemia** results in loss of the epithelial layer with progression to complete **loss of the villus architecture**, with severe mucosal hemorrhage and loss of the lamina propria (**because anoxic injury**).
- In the equine colon, unlike the small intestine, complete ischemia causes cellular necrosis and detachment of small clusters of surface epithelial cells.

ENDOTOXEMIA

- Due to ischemia or inflammation **destroys the integrity** of the intestinal epithelial barrier, the **lipopolysaccharide** component of the outer wall of **enteric gram negative** microorganisms **gains access** to the circulation.
- **Circulating and tissue-fixed mononuclear phagocytes** release the cytokines, lipid-derived mediators, Interleukins, tumor necrosis factor (TNF a)' and coagulation/ fibrinolytic factors are responsible for **acute-phase response and fever**.

MOTILITY DISTURBANCES IN INTESTINAL OBSTRUCTION

- Non-strangulating occlusion of pony jejunum causes loss of gastric contractile activity in the distended stomach and immediate continuous spiking activity in intestine proximal to the obstruction.
- Occlusion of blood supply to the pony ileum decreases motility in the ischemic bowel, increases motility in the more proximal segment
- **Ileus** is a common postoperative complication of intestinal surgery in horses.

CLINICAL SIGNS OF COLIC

The horse affected by colic due to gastrointestinal pain may behave in a variety of ways depending on severity of pain.

- no pain
- mild pain
- moderate pain
- severe pain
- depression

The horse with **mild pain** may demonstrate one or more of the following signs

- occasional pawing
- turning the head to the flank
- stretching out
- lying down for longer than usual
- quivering of the upper lip inappetence

With **moderate pain** the following may be seen

- restlessness
- pawing
- cramping with attempting to lie down
- crouching
- kicking at the abdomen
- lying down
- rolling
- turning the head to the flank
- dog-sitting position

The horse in **severe pain** will show one or more of the following

- sweating
- violent rolling

- The **stage of depression** may be seen **after a severe bout of colic** as advanced intestinal necrosis and endotoxemia produce a state of indolence.
- Alternatively, depression may be seen as an **early sign** of other diseases that produce colic, especially inflammatory diseases such as **colitis** and **peritonitis**.
- **Strangulating obstructive diseases** usually cause **more severe pain** than simple obstructions and late in the course of these conditions depression takes over as the predominant sign.
- **Severe pain** that is **continuous** may be more likely in cases of **severe tympany** or in **strangulating diseases** where there is bowel wall stretching or tension on the mesentery.

Characteristic clinical signs suggesting the presence of a particular disease

- a **dog-sitting position** is seen in horses with **gastric distention**
- a **stretched-out** (‘trestle table') position is seen in horses with small intestinal **intussusceptions** and **sand impactions**
- **foals** that **roll onto their backs** and lie in dorsal recumbency for long periods may be affected by **gastric ulceration**.

Physical examination of a horse with colic

HISTORY

The initial history should include

- signalment
- duration of clinical signs
- severity and frequency of pain
- the time when the horse was last observed to be normal

- An accurate history can also help determine if a horse's colic is **acute**, **chronic**, or **recurrent**.
- **Nutritional history** can help determine if feed materials or feeding practices could predispose to colic (e.g. poor quality hay may predispose to impaction; grain overload predisposes to colic and laminitis).
- Acute changes in water intake from defects in automatic watering systems or freezing temperatures can lead to obstructive colic (impaction can occur secondary to decreased water intake)
- **Parasite control program**: date of last deworming, and agent used can be especially important for younger horses.
- **Manure production**, volume, and character should be determined.

CLINICAL EXAMINATION

This can provide information regarding

- the type and severity of pain
- the animal's general condition
- signs of colic

Rectal temperature

Increases in body temperature can occur after anxiety, excitement, or exertion.

- Temperatures greater than 39.5°C may suggest an inflammatory or primary infectious process, such as colitis, proximal enteritis, peritonitis, or pleuritis.
- Body temperature elevation can also occur early after stomach or intestinal rupture, leading to septic peritonitis.
- Decreased temperature (**hypothermia**) , in addition to **tachycardia**, is indicative of the development of circulatory compromise and potential **shock**.

Respiratory rate

- The respiratory rate of a horse with colic will usually be elevated because of pain or metabolic acidosis.
- Dyspnea or shallow breathing can result from pressure applied to the diaphragm by severe gastric or intestinal distention.

Heart rate/Pulse Rate

- Palpation of a peripheral **pulse** can offer a reflection of **cardiovascular function** and **tissue perfusion**.
- The absence of a palpable pulse may indicate cardiovascular compromise .

The normal equine heart rate is 24-40 bpm.

- Elevations of heart rate in horses with colic are usually the result of anxiety, **pain**, and **hypovolemia**.
- **Heart rate elevation** is a **good indicator** of the **severity of pain** and indirectly, the original intestinal disorder.

Mucous membranes

- The character and color of mucous membranes can **reflect** the **circulatory status** of the patient.
- Normal mucous membranes are **moist** and **pink**. Physiological capillary refill time is usually 1 .5 seconds or less.
- When peripheral vascular circulation is **impaired** capillary refill time is **prolonged**, this is considered severe when increased to 4 seconds or more
- **Dry** mucous membranes can indicate **systemic dehydration**.
- **Pale** mucous membranes can occur with **shock** from hypovolemia or pain.
- **Dark** mucous membranes or a toxic line are usually associated with **septic** or **endotoxic shock**, following resorption of bacterial endotoxins from intestinal compromise or enteritis.

Abdominal auscultation

- Intestinal motility can be evaluated subjectively by auscultation of the abdomen using a stethoscope.
- The frequency, duration, intensity, and location of intestinal sounds should be noted.
- Normally, organized intermittent peristaltic sounds can be heard.
- **Auscultation** should be performed on both the **right** and **left** flanks as well as the **ventral** abdominal wall, or over all four quadrants, dorsal/ventral and left/right.
- **Colonic** and **small intestinal** sounds can best be heard at the **left flank**, whereas **cecal sounds** can be heard at the **right flank**.
- The presence of sounds associated with sand in the large colon are best detected on auscultation of the ventral abdominal wall.
- **Excessive** frequency of sounds or intestinal hyperactivity is associated with conditions such as **enteritis** or **spasmodic colic**.
- The **absence** of intestinal sounds over a prolonged period of time may indicate **ileus** or **obstructive disease (complete obstruction)**

RECTAL EXAMINATION

- Rectal examination may be the most revealing component of the physical examination of a horse with colic.
- Any **distension** in the part of intestine should be **located**.

ABDOMINOCENTESIS

- Abdominocentesis can provide useful information when other examination techniques **fail** to reveal a clear diagnosis, or when further determination is required of the severity of the lesion.
- **Site**: right of midline, should be selected to avoid the spleen and stomach.
- **Normal** peritoneal fluid is **clear** or **straw colored**, with a protein concentration up to 2.5 g/dl (25 g/l) and total white blood cell count (WBC) of less than 5000 cells/dl
- The presence of **food particles** or **bacteria** in the peritoneal fluid can indicate loss of bowel integrity and a poor prognosis.

ULTRASOUND EXAMINATION: helpful in diagnosis

CLINICAL PATHOLOGY

- with severe or changing cases WBC, packed cell volume (PCV) and total plasma proteins (TPP) are often helpful.
- The **PCV** and **TPP** are useful for assessment of the degree of **dehydration**
- The higher the PCV, the greater the rate of mortality, with values greater than 65 per cent associated with a poor prognosis.
- Normal total protein levels range between 5.5 and 7.5 g/dl (55-75 g/l) .
- **Plasma protein** in a colic patient is usually **increased** as a result of **dehydration**.
- **Severe leukopenia** (< 3000 cells/µL) can indicate **gram-negative sepsis** or **endotoxemia** as a result of salmonellosis or severe acute peritonitis from intestinal rupture.

Medical therapies for colic

The aims of medical therapy in equine colic are to

- relieve pain
- restore normal propulsive motility of the gut
- correct and maintain hydration and electrolyte or acid-base balance
- treat endotoxemia
- treat bacterial or parasitic infections (if present) .

<u>These include</u>

- analgesics to control visceral pain
- agents to soften and facilitate the passage of ingesta (laxatives)
- fluids and electrolytes to improve cardiovascular function during endotoxic and hypovolemic shock
- anti-endotoxin therapy
- anti-inflammatory drugs to reduce the adverse effects of endotoxin

Medical therapies for colic

Analgesic therapy

Analgesics and their relative efficacy in controlling abdominal pain		
Drugs	**Dosage**	**Efficacy**
Dipyrone	10 mg/kg	poor to moderate
Phenylbutazone	2.2-4.4 mg/kg	poor to moderate
Flunixin meglumine	**0.25-1.1 mg/kg**	**good to excellent**
Ketoprofen	1.1-2.2 mg/kg	good
Xylazine hydrochloride	0.2-1.1 mg/kg	**excellent**
Detomidine hydrochloride	10-40µg/kg	**excellent**
Romifidine hydrochloride	40-80 µg/kg	**excellent**
Morphine sulfate	0.3-0.66 mg/kg*	good
Butorphanol tartrate	0.05-0.075 mg/kg**	good
Pentazocine	0.3-0.6 mg/kg	poor to moderate

*Use only with xylazine or another alpha2 adrenoceptor agonist to avoid CNS excitement

**Doses in the upper range may cause ataxia

Spasmolytics

Atropine: Atropine is not recommended for use in horses (longer effects)

Hyoscine: Hyoscine has a shorter muscarinic cholinergic blocking

effect compared to atropine and is effective in relaxing the bowel wall.

- In cases of spasmodic colic, the use of a spasmolytic, such as N-butylscopolammonium bromide1 (single injection of 0.3 mg/kg body weight or 1.5 mL/100 kg).

Laxatives

Mineral oil: is the most frequently used laxative in equine practice. It is a surface lubricant and is administered at a dosage of 5-10 ml/kg once or twice a day by nasogastric tube.

Psyllium hydrophilic mucilloid: is a bulk-forming laxative which causes the fluid and ion content of feces to increase by absorbing water. A dose of **1 g/kg** can be administered per os up to four times a day.

Osmotic laxatives

Magnesium sulfate (Epsom salt) and **sodium chloride** (table salt) can be used as osmotic laxatives in horses.

- Undiluted osmotic laxatives will cause enteritis by osmotic damage to the mucosal cells, so each dose of **0.5-1.0 gm/kg** should be diluted in **4 liters of warm water** and given orally.

Dioctyl sodium succinate (DSS)

- DSS is a surface-active agent with wetting and emulsifying properties. It reduces surface tension and allows water and fat to penetrate the ingesta. A dose of **10-20 mg/kg** can be administered as a **5%** solution orally.

Fluid therapy

- Fluid, electrolyte, and acid-base imbalances commonly occur in equine gastrointestinal diseases.
- Intravenous administration of polyionic-balanced electrolyte solutions (e.g. Hartmann's solution) will help **to maintain the intravascular fluid volume** and **aid tissue perfusion**. Normal saline (0.9% sodium chloride) should not be given for longer periods.

Anti-endotoxin therapy

- Purified endotoxin-specific IgG containing antibodies against lipopolysaccharide extracts of a variety of gram-negative bacteria is available

Drugs that alter intestinal motility

Neostigmine methyl sulfate: Neostigmine is an acetyl-cholinesterase inhibitor that directly stimulates intestinal contractions.

- Doses of 0.0044 mg/kg (2 mg for an average sized adult horse) can be administered subcutaneously or intravenously.

Metoclopramide: is a non-specific dopaminergic antagonist that also augments the release of acetylcholine.

- It is a potent gastrointestinal stimulant when given at a dosage of 0.25 mg/kg i.v. but it can produce severe CNS side effects.
- **Domperidone**: @ 0.2 mg/kg i.v.
- **Cisapride**: administered rectally@ 0.2 mg/kg, has been found effective.

Surgery

- Those cases which have severe obstructive colic and does not respond to treatment require surgery. In cases of strangulating colic, surgery is needed.

Peritonitis

- It is the inflammation of the peritoneum.
- peritoneum is a single layer of squamous mesothelial cells (over alveolar tissue containing blood vessels) and is divided into a parietal and visceral peritoneum.
- The parietal peritoneum lines the diaphragm, abdominal walls, and pelvic cavity.
- A small volume of peritoneal fluid lubricates the surface of the visceral and parietal peritoneum.
- Together the peritoneum and fluid are responsible for preventing adhesion formation.

PATHOPHYSIOLOGY

- Peritonitis in the horse may have an infectious (bacterial, viral, fungal, or parasitic) or noninfectious (traumatic, chemical, or neoplastic) cause.
- It is classified as primary or secondary (defined by cause); peracute, acute, or chronic (defined by onset and duration); diffuse or localized (defined by region); and septic or nonseptic (defined by the presence or absence of bacteria).

- Acute, diffuse, septic peritonitis secondary to surgical manipulation or perforation of the gastrointestinal tract is the most common manifestation of peritonitis in the horse.
- Sepsis usually involves a mixed bacterial population, whether from gastrointestinal origin or from environmental contamination following trauma
- Enterobacteriaceae, obligate anaerobic bacteria, and gram-positive organisms are commonly found.
- Anaerobic bacteria are reported to be present in at least 20% to 40% of cases of peritonitis.

Phases of peritonitis

***Contamination phase*:** lasts **3 to 6 hours** and is characterized by **increased vascular permeability** and influx of protein-rich fluid and white cells into the peritoneal cavity, resulting in the release of mediators of inflammation.

Diffuse acute peritonitis: lasts up to 5 days and reflects the spread of bacteria throughout the peritoneal cavity.

- The inflammatory response grows with fluid accumulation and buildup of fibrin and inflammatory products, resulting in ***ileus*** mediated by the sympathetic nervous system.

- However, if bacteria overwhelm the immune system, bacteremia and endotoxemia develop, resulting in hypovolemia and hypoproteinemia and ultimately adhesions and abscess formation.
- The ***acute localizing phase*** develops 4 to 10 days after the initial insult. Fibrin aggregates attempt to localize the infection.

CLINICAL SIGNS

- Clinical signs are often nonspecific, irrespective of cause, and may include fever, signs of depression, inappetence, tachycardia, dehydration, reduced gastrointestinal motility, signs of abdominal pain, diarrhea, and weight loss.
- Horses with ***peracute peritonitis*** may be found dead or showing signs of severe endotoxemia, which leads rapidly to circulatory shock and death

- Typical findings include severe depression, sweating, muscle fasciculations, tachycardia, rapid shallow breathing, cold extremities, and purple or dark red mucous membranes with prolonged capillary refill times.
- Fever is often not a feature because of the peracute nature of the disease.

Acute peritonitis has a slower onset with the gradual spread of bacteria within the abdomen.

- Horses may have a history of showing signs of intermittent abdominal pain, and may show signs of depression, inappetence, fever, dehydration, tachycardia, tachypnea, congested mucous membranes with delayed refill time, and ileus or diarrhea.

Chronic peritonitis may be associated with low-grade and nonspecific signs including intermittent or persistent fever, signs of depression and inappetence, progressive weight loss, dehydration, intermittent mild abdominal pain, **reduced fecal output**, **decreased intestinal motility**, intermittent diarrhea, and ventral edema.

DIAGNOSIS

(Peracute peritonitis): usually have a **markedly high hematocrit** and **serum hypoproteinemia** associated with acute hypovolemia, fluid shifts, and sequestration of protein in the abdomen.

Severe leukopenia with **neutropenia** and **degenerative left shift** with severe toxic changes in the neutrophils is common.

Prerenal azotemia and electrolyte imbalances.

(Acute peritonitis): often have a high hematocrit and a serum hypoproteinemia (low albumin).

Leukopenia and neutropenia may be seen initially, followed by **leukocytosis** and neutrophilia with a degenerative left shift.

(Chronic peritonitis): high hematocrit, **leukocytosis** and neutrophilia with, or without, a left shift.

Abdominocentesis

- Fluid is usually abundant, cloudy, and turbid or may be thick and purulent.
- Total protein values greater than 2 to 2.5 mg/dL (20 to 25 g/L) suggest

- Total nucleated cell count is usually markedly high in acute peritonitis (100,000 to 800,000 cells/µL) whereas in chronic peritonitis, TNCC is typically lower (20,000 to 40,000 cells/µL)

TREATMENT

- Treatment aims are to identify and treat the underlying cause; eliminate infection; reduce inflammation and relieve pain; address hypovolemia, hypoproteinemia, and any electrolyte abnormalities; treat endotoxemia; and provide nutritional support.

Medical therapy: for stabilization of animal

- *Isotonic balanced electrolyte solutions* should be administered to replace the fluid deficit and meet ongoing losses.
- *Potassium and calcium* can be added to the fluids as required. Horses will require colloids, preferably plasma, when the plasma protein concentration falls below 4 g/dL (40 g/L).
- *Hyperimmune plasma products*: for negative effects of bactremia and endotoxemia.
- *Antimicrobial therapy*: Na-penicillin, Procaine penicillin, Ceftiofur, Enrofloxacin (better: broad spectrum).

Anthelmintic treatment: may be required if there is suspicion of verminous arteritis secondary to migration of ***Strongylus vulgaris*** larvae or ***larval cyathostomes***.

- **Fenbendazole** (**10 to 15 mg/kg**, PO, for 5 days, or **50 mg/kg**, PO, for 3 days) and **ivermectin** (0.2 mg/kg, PO) may be suitable anthelmintics.

Surgical treatment: reserved for horses in which surgical intervention is clearly indicated.

Diseases and Disorders of Respiratory tract

Guttural Pouch Tympany

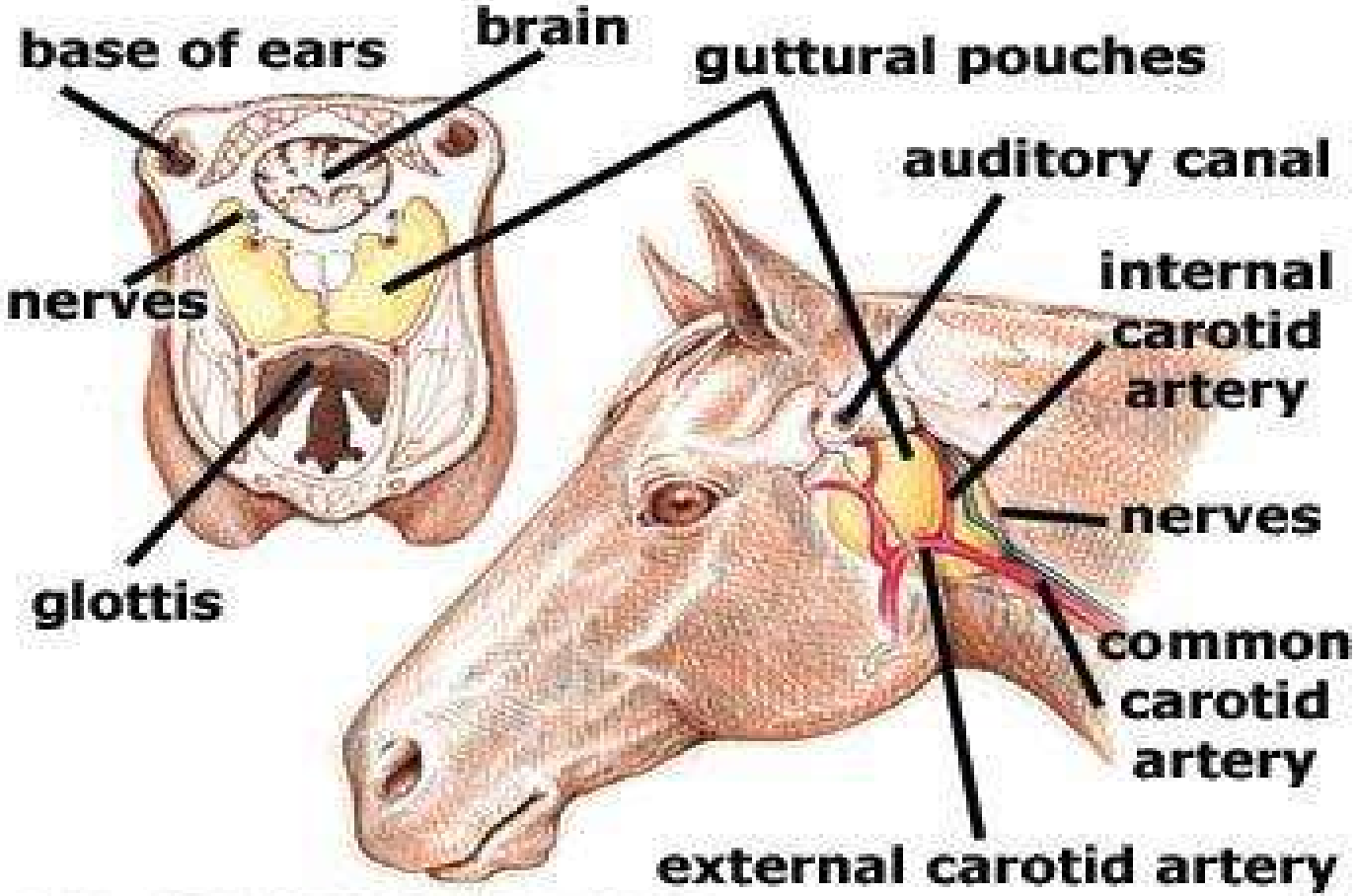
Anatomy

✓ Guttural pouches are **diverticula** of the Eustachian tubes, connecting the middle ear to the pharynx.

✓ Opening of the guttural pouch into the pharynx is called the **nasopharyngeal ostium**.

GTP: Guttural pouch tympany is seen in horses ranging from **birth to 1 yr** of age.

- Tympany is a unilateral or bilateral **distention of the guttural pouches** with **air**, with or without some fluid accumulation
- Possible causes include a **mucosal flap** (or plica salpingopharyngea) acting as a one-way valve that traps air and fluid in the pouch, **inflammation** from an upper airway infection, **persistent coughing**, and **muscle dysfunction**.
- A **genetic** basis of disease has been identified in **Arabian** and **German warmblood** breeds.
- The condition is **acquired** due to inflammation of the upper respiratory tract.



Clinical signs

- The affected guttural pouch is **distended with air** and forms a characteristic **non-painful swelling** in the parotid region.
- Breathing may become **stertorous** in severely affected animals.
- Dyspnea, dysphagia, secondary empyema, and inhalation pneumonia are rare **complications** of this disease.

Diagnosis: is based on **clinical signs** and **radiographic** examination of the skull.

Medical management: with NSAIDs and antimicrobial therapy resolves most cases due to upper respiratory tract inflammation.

- Temporary relief can be achieved by **catheterizing** the affected guttural pouch or pouches.
- **Surgical treatment** is required for permanent resolution and should be performed promptly to prevent complications, such as empyema and bronchopneumonia.
- Goal is to create **permanent means of evacuating air**, either through the unaffected guttural pouch or through the guttural pouch opening (i.e by

GUTTURAL POUCH EMPYEMA

- Empyema of the guttural pouches is an **accumulation of purulent material** in one or both guttural pouches, which can become inspissated or form chondroids (pus stones), independent of duration of infection.
- Upper respiratory tract infections (especially those caused by ***Streptococcus equi*** subspecies ***equi*** can cause abscessation and rupture of retropharyngeal lymph nodes into the guttural pouch)
- Less common causes are **infusion** of irritant drugs, **fracture** of the stylohyoid bone and **pharyngeal perforation** by a nasogastric tube.

Diagnosis

Clinical signs: include **intermittent nasal discharge**, **swelling** of submandibular and pharyngeal lymph nodes, parotid swelling and pain, **extended head carriage**, **loud respiratory noise**, and difficulties in swallowing and breathing

Endoscopic examination: a purulent discharge can be seen at the pharyngeal orifice of the affected side.

Radiographs: fluid lines within the guttural pouch are suggestive of liquid or inspissated contents, and discrete round masses indicate chondroids.

Aspirates or saline washings from the guttural pouch are submitted for culture and sensitivity testing and for polymerase chain reaction analysis.

Treatment

Treatment of this condition usually includes combinations of **lavage** and **systemic antibiotics**.

Prognosis

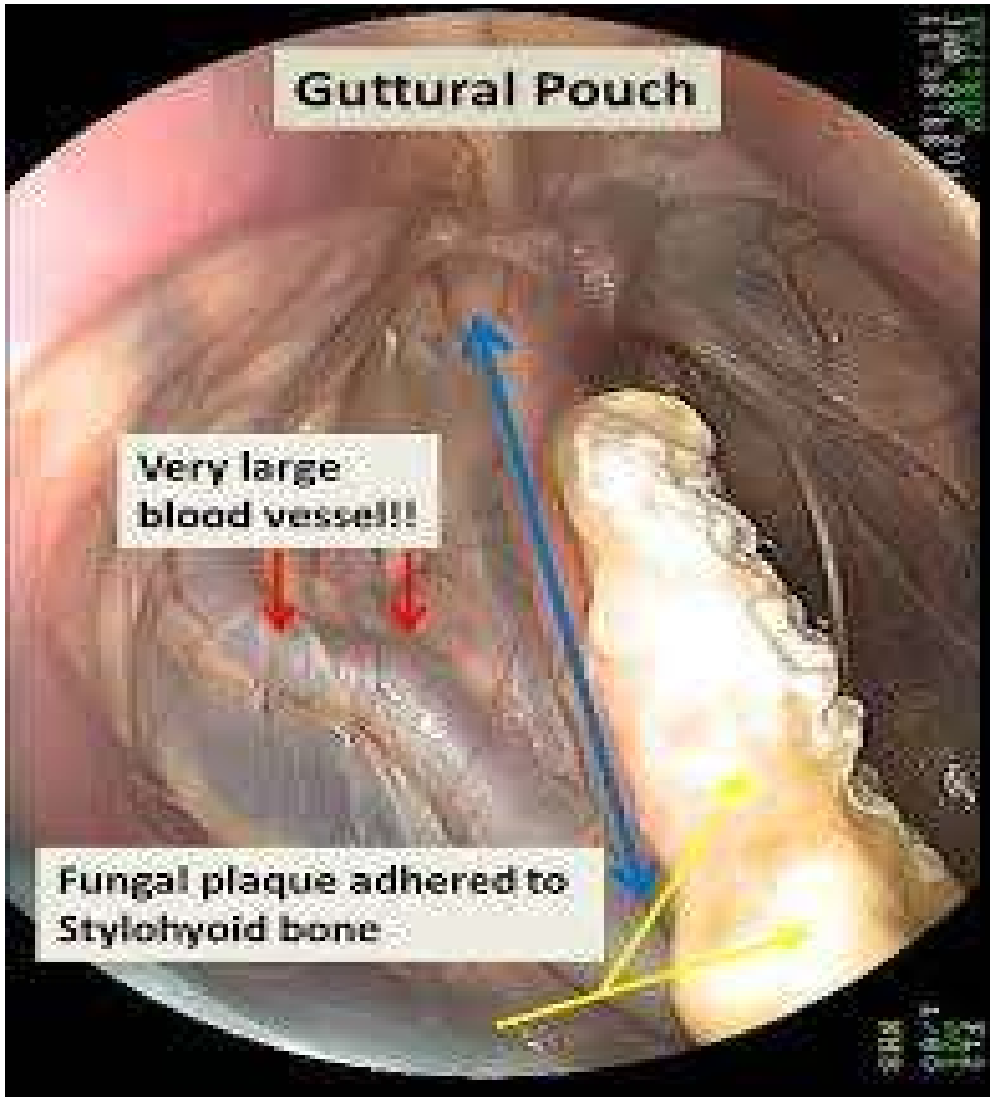
Response to medical treatment and surgery is usually satisfactory

GUTTURAL POUCH MYCOSIS

- Guttural pouch mycosis affects the roof of one (or rarely both) guttural pouch.
- There is not any apparent age, sex, breed, or geographic predisposition.
- The fungus ***Aspergillus fumigatus*** has been identified as the most common causal agent.
- Guttural pouch mycosis typically forms a **diphtheritic membrane** of variable size, composed of necrotic tissue, cell debris, different bacteria, and fungal mycelia.

Diagnosis

- The most common clinical sign is **moderate-to-severe epistaxis**.
- It is caused by **fungal erosion** of the **internal carotid artery** (ICA) in most cases and of the **maxillary artery** (MA) in approximately one-third of cases.
- Several bouts of hemorrhage usually precede a fatal episode.


Guttural Pouch
Very large
blood vessel!!
Fungal plaque adhered to
Stylohyoid bone

- **Mucus and dark blood continue to drain from the nostril** on the affected side for days after acute hemorrhage ceases.
- The second most common clinical sign is **dysphagia** caused by damage to the pharyngeal branches of the **vagus** and **glossopharyngeal nerves**, which can lead to **aspiration pneumonia**.
- Abnormal respiratory noise can be caused by pharyngeal paresis or laryngeal hemiplegia, the latter caused by **recurrent laryngeal nerve damage**.
- **Horner syndrome** from damage to the cranial cervical ganglion and postganglionic sympathetic fibers causes **ptosis**, **miosis**, and **enophthalmos**, patchy sweating, and congestion of the nasal mucosa.
- Less common signs are parotid pain, mucopurulent nasal discharge, abnormal head posture, head shyness, sweating and shivering, corneal ulcers, colic, blindness, etc.

Endoscopy (mycotic plaques are visibel), combined with history and clinical signs, is critical for diagnosis.

Medical Treatment

Antifungal agents

- **Amphotericin B** at 0.38 to 1.47 mg/kg diluted in 1 L of 5% dextrose has been given intravenously (IV) daily for up to 40 days
- **Itraconazole** at 5 mg/kg orally every 24 hours or Topical infusion of 30 to 60 mL of 10 mg/mL itraconazole through the biopsy channel of the endoscope every other day for 10 days.
- Daily guttural pouch irrigations with a **0.08% clotrimazole emulsion** in 500 mL water for 14 days prevented recurrence.

Surgical Treatment

- Removal of the diphtheritic membrane
- Surgical occlusion of the affected artery

Prognosis

- 50% mortality rate reduced by occlusion of artery and Laryngeal hemiplegia is one of the more common clinical signs

Non-infectious Disorders of Upper Respiratory Tract

Nasal Septum

- Most nasal septal disorders are congenital abnormalities that remain undetected until the horse is exercised.
- Traumatic injury to the bridge of the nose as a juvenile can produce nasal septal deviation and thickening.
- Other less common diseases of the nasal septum include amyloidosis, fungal infection, and squamous cell carcinoma
- Thickening or deviation of the nasal septum causes low-pitched stertorous breathing during exercise. Facial deformity may be seen. Septal abnormalities may be detected by palpation, visual inspection, and endoscopic examination.
- Histologic examination of any nodules or discrete lesions on the septum will identify tumors, amyloidosis, or fungal infections.
- Surgical resection of the nasal septum is the only treatment option in most cases

Nasal Polyps

- Nasal polyps are pedunculated growths that arise from the mucosa of the nasal cavity, nasal septum, or tooth alveolus.
- Polyps are usually unilateral and single but can be bilateral and multiple.
- They form in response to **chronic inflammation** by hypertrophy of the mucous membrane or exuberant proliferation of fibrous connective tissue.
- **Clinical signs:** are poor airflow through the affected nasal passage; inspiratory dyspnea; unilateral, malodorous, mucopurulent nasal discharge; and low-volume epistaxis.
- **Diagnosis**: Polyps are detected via endoscopic and radiographic examination, and histopathologic evaluation of biopsy samples provide a definitive diagnosis.
- **Treatment**: Surgical excision is performed via an incision in the false nostril, a trephine opening, or a bone flap.

Diseases of the Paranasal Sinuses in Horses

- Most diseases of the paranasal sinuses cause mucopurulent or bloody nasal discharge.
- Drainage is unilateral, in contrast to disease of the lungs, pharynx, and guttural pouches.

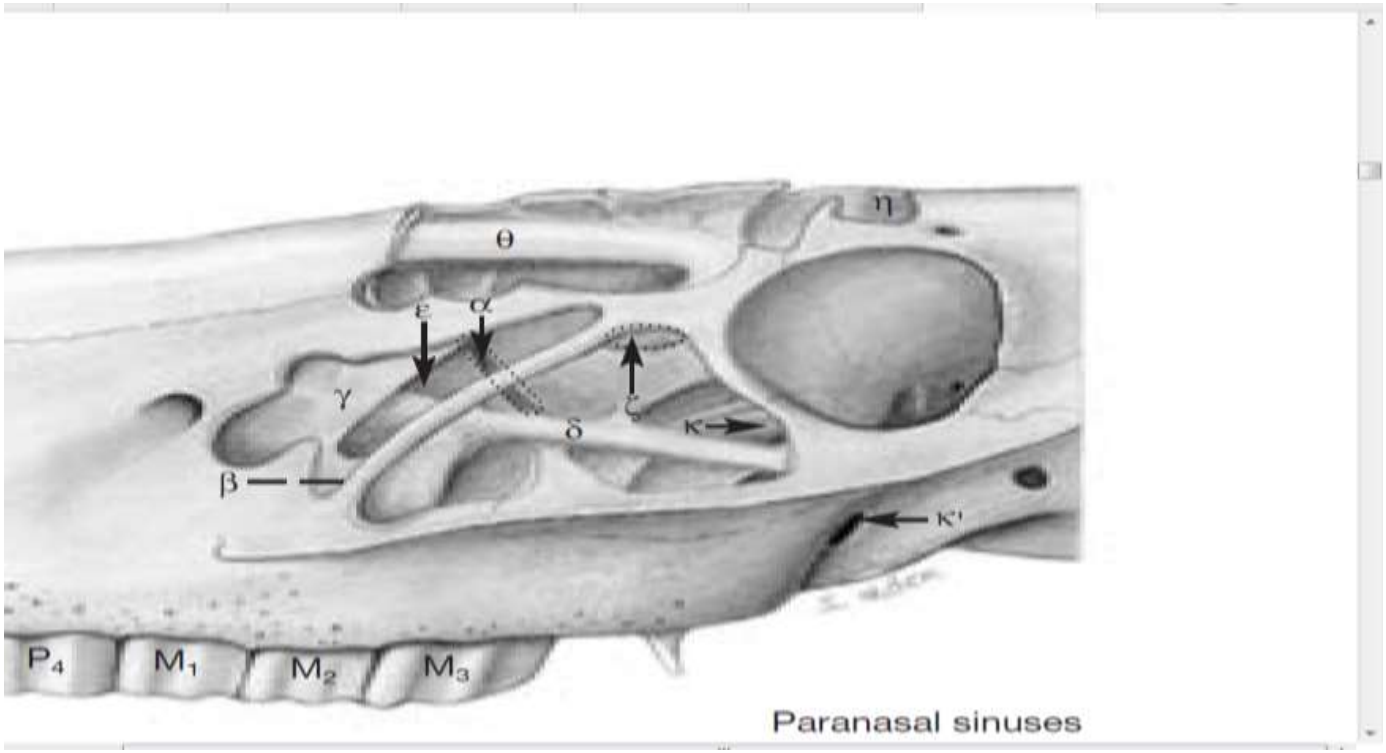
Sinusitis

Primary sinusitis occurs subsequent to an upper respiratory tract infection that has involved the paranasal sinuses.

Secondary sinusitis can result from tooth root infection, fracture, or sinus cyst. The first molar, fourth premolar, and third premolar are the most likely to develop tooth root abscesses.

Clinical signs: of secondary sinusitis closely resemble those of primary sinusitis, including unilateral mucopurulent nasal discharge and facial deformity. Tooth root abscesses typically produce a fetid nasal discharge.

Treatment: of primary sinusitis involves lavage of the sinus cavity and systemic antimicrobial therapy based on culture and sensitivity results



Nasomaxillary aperture α
Septum between rostral and caudal maxillary sinuses β
Rostral maxillary sinus γ
Caudal maxillary sinus δ
Ventral conchal sinus ε
Frontomaxillary opening ζ
Frontal sinus η

Sinus Cysts

- Sinus cysts are single or loculated fluid-filled cavities with an epithelial lining.
- They develop in the maxillary sinuses and ventral conchae and can extend into the frontal sinus.

Clinical signs: are facial deformity, nasal discharge, and partial airway obstruction. Radiographs are more likely to identify a sinus cyst than endoscopic examination.

Treatment: involves radical surgical removal of the cyst and associated conchal lining.

Strangles

❖Strangles is an acute, highly contagious disease characterized by fever, inflammation of the **upper respiratory tract** mucous membranes, purulent nasal discharge, and **abscesses** of the **mandibular** and **retropharyngeal** lymph nodes.

- Strangles is caused by infection with **Streptococcus equi subsp equi**, a β-hemolytic, Lancefield group C Streptococcus.
- Strangles is most severe in young horses.
- *S equi enters* the horse through the **mouth or nose** and immediately attaches to **tonsillar** tissues in the oropharynx and nasopharynx.
- Penetration is followed by multiplication of the organism in the follicular tissues of the **tonsil**.

Clinical signs

- After an incubation period of **3 to 11 days**, abrupt onset of fever is followed by swelling of one or more lymph nodes.
- Shedding of *S equi* in nasal discharges in most horses ceases 2 to 3 weeks after the onset of clinical signs.

- **Lymphadenopathy progresses to abscesses** that, if allowed to mature, generally rupture and drain a tenacious purulent material.
- Depending on the direction of rupture(retropharyngeal lymph nodes can drain into the GP or pharynx) purulent material may drain via the nasal passages or directly externally.
- Cough is occasionally present and may be worsened by secondary pharyngitis or laryngitis.

Complications

- Chronic carriers, metastatic abscessation (**bastard strangles**-infections occur in sites remote to the usual head and neck involvement), and immunologic complications.

DIAGNOSIS

Classic diagnosis has relied on **culture** of various samples, **ideally purulent discharge** collected from an abscess.

PCR testing has more recently become important for the diagnosis of strangles particularly in chronic shedders.

TREATMENT

- When managing an outbreak situation, it has been suggested that **immediate treatment** with antimicrobials at the first evidence of fever (and before the development of lymphadenopathy) prevents lymph node abscesses from developing.
- Nonsteroidal antiinflammatories, such as **phenylbutazone** or **flunixin meglumine**, helps in addressing this issue and boost appetite.
- Encouraging the development and maturation of any abscesses present speeds resolution.
  - **Hot packing** and **topical application** of a drawing or **softening agent** (eg, ichthammol ointment) have been recommended.

- Metastatic abscessation typically requires a combination of long-term antimicrobial therapy and local drainage
- **Dexamethasone** at an initial dose of **0.1 mg/kg** every 24 h with tapering.

PREVENTION

❖**Immunity**: Most horses develop a durable immunity to disease for around 5 years after recovery.

❖**Vaccination**: vaccines are available, but none can be considered fully protective against disease.

❖**Biosecurity:** measures to prevent infection and its spread from solitary cases.

Exercise-Induced Pulmonary Hemorrhage (EIPH)

- Exercise-induced pulmonary hemorrhage (EIPH) is a common condition of **intensely exercising horses** and occurs in up to 75% of horses that race.
- **Epistaxis** is seen in a small proportion (~5%) of horses with EIPH.
- Condition is most commonly **identified in racing** Thoroughbreds, and to a similar extent in Standardbreds.

EFFECT ON PERFORMANCE

- Exercise-induced pulmonary hemorrhage has a negative effect on racehorse performance.
- In a study, it was determined that horses with either mild EIPH or no evidence of blood in the large airways were four times as likely to win than horses with moderate or severe EIPH.

RISK FACTOR

EIPH affects most of the **intensely exercising horses** to some degree.

- **Lifetime starts**: horses with more than 50 lifetime starts are 1.8 times

Etiology

- The proposed pathophysiologic mechanism for pulmonary hemorrhage includes **high pulmonary vascular pressures** during maximal exercise, with **resultant thickening of pulmonary vein walls** and **decreased luminal diameter** and **increased intravascular pressure** at the level of the pulmonary capillaries.

Diagnosis

- **Endoscopic observation** of blood in the airways 30–90 min after exercise provides definitive evidence of EIPH. Other sources of hemorrhage in the upper airway, particularly guttural pouch mycosis and ethmoid hematoma must be excluded during endoscopic examination.
- **Cytologic examination of bronchoalveolar lavage** (**BAL**) fluid for semiquantitative assessment of **hemosiderophages** is diagnostic.
- **Thoracic radiography** demonstrates alveolar or mixed alveolar-interstitial **opacities** in the caudodorsal lung fields

CLINICAL SIGNS

- Horses with EIPH **do not** typically manifest signs of systemic disease, and on physical examination, the only detectable abnormality may be **epistaxis (0.15% horses)**.

Treatment and Control

- **Furosemide** (**proven efficacy**) reduces the incidence and severity of EIPH in Thoroughbred racehorses.

Others without proven efficacy are as

- **Sildenafil**: Sildenafil citrate is a phosphodiesterase-5 inhibitor that mediates reductions in both systemic and pulmonary vascular pressures
- Aminocaproic Acid
- Pentoxifylline
- Clenbuterol (bronchodilator) etc

Noninfectious Inflammatory Diseases of the Lower Airway

- **Inflammatory airway disease** and **recurrent airway obstruction** are 2 **nonseptic diseases** with a shared cause of exposure to particulate matter (DUST).
- Both inflammatory airway disease (IAD, also known as Allergic Airway Disease) and recurrent airway obstruction (RAO, also known as heaves or Chronic Obstructive Pulmonary Disease-COPD) are **inflammatory** but **not septic** diseases of the equine respiratory system.
- Both IAD and RAO share similarities and occur due to exposure to particulate matter (Dust).

Inflammatory airway disease/Allergic Airway Disease

CAUSE OF INFLAMMATORY AIRWAY DISEASE

- ❖Exact cause not yet known, however, there are different causes that likely contribute to the clinical signs that are recognised in this disease.

The most commonly invoked contributors

- o high levels of particulates in the environment,
- o viral disease (previous exposure)
- o air pollution
- o genetic predisposition
- o bacterial infection

- ❖Environment has long been associated with airway inflammation in horses with conditions such as poorly ventilated stables, organic dusts and molds are primarily to blame.
- ❖Role of ammonia in inducing airway inflammation is being increasingly examined as well

Airborne particulate matter in the stable environment is largely **organic**, including

- plant debris
- mold spores primarily from hay
- β-glucans
- live and dead microbes
- proteases
- animal dander

Inorganic particulates are of less importance, but still contribute

- **silicates** (from dusty arenas or oil fly ash from diesel machinery being used inside large barns)

PATHOGENESIS OF INFLAMMATORY AIRWAY DISEASE

❖IAD is caused by environmental exposures, little is still known about the actual pathogenesis of IAD.

- elevated numbers of **mast cells** in bronchoalveolar lavage fluid (BALF) of horses (in horses with poor performance and the association of BAL mastocytosis are suggestive of a degree of allergic response)

IAD vs ROD

Inflammatory airway disease is a noninfectious inflammatory disease of the lower respiratory tract (small airways) that can affect horses of any age.

Clinical signs are **nonspecific** and vary in severity, and include some combination of coughing, nasal discharge, poor performance, or prolonged recovery following exercise.

The disease is characterized by **excessive mucus accumulation**, accumulation of **inflammatory cells** (e.g., neutrophils, eosinophils, or mast cells) in the peripheral airways.

Cytology profiles (e.g., high numbers of neutrophils vs. high numbers of mast cells).

In contrast to horses with active RAO, **the pattern of breathing at rest in those with IAD is unchanged, and lung auscultation is typically normal**.

Diagnosis (can be challenging)

- Different criteria have been used to diagnose IAD, and include combinations of clinical signs, visual assessment of tracheal mucus, tracheal wash fluid cytology, BAL cytology, and pulmonary function testing.
- Video-endoscopic evaluation of the trachea: because mucus accumulation is associated with reduced performance in racehorses and sport horses.

Treatment

- Combination therapy with **corticosteroids** and **bronchodilators** is used.
- **Interferon-α** administration (in combination with rest) has shown some benefit in IAD-affected racehorses, @ 90 U, orally , every 24 hr.

RECURRENT AIRWAY OBSTRUCTION CAUSE

- Horses with RAO are hypersensitive to dust in hay.
- Susceptibility is hereditary, yet the disease only manifests in mature adult horses (>5years).
- The disease is characterized by two clinical phases: active disease and remission.
  - Active disease develops after inhalation of hay dust, which acts as an immunologic trigger inciting a pulmonary response characterized by bronchoconstriction, mucus production, and bronchoalveolar neutrophilic inflammation.
  - Remission occurs when the inciting cause of dust is removed.
- Horses maintain lifelong susceptibility, and any reexposure to hay dust reinitiates active disease

- Horses thus tend to **vacillate** between active and remission states, and commonly are presented with chronic history of waxing and waning respiratory disease.
- Common owner complaints typically include **coughing**, **nonpurulent nasal discharge**, **reduced performance**, and **difficulty breathing** at **rest**.

Clinical examination findings

- During active disease, **expiratory wheezes** and **early inspiratory crackles** may be auscultated when the horse is at rest (or elicited with use of a rebreathing bag), and **gurgling** or **rattling** produced by mucus movement may be auscultated in the **distal cervical portion** of the trachea.
- A **hallmark of active disease** is an **abnormal breathing pattern** at **rest**; the rate and effort are increased (the abdominal effort is particularly visible), and the rhythm becomes more regular.

Diagnosis of RAO

- Moderate to severe RAO is relatively easy to diagnose on the basis of clinical signs, signalment, and a history of **recurrent bouts of coughing**, increased respiratory effort, and exposure to hay or other inciting dust.
- bronchoalveolar lavage (BAL)
- cytologic findings of nondegenerative **neutrophilic inflammation** (>20% neutrophils) support a diagnosis of RAO.
- An RAO-susceptible horse in remission has normal findings on physical examination, BAL and Cytology.

Treatment

- Successful treatment of the disease can be challenging because effective therapy fundamentally requires **permanent management modifications to reduce exposure** to **hay dust** and **improve air quality.**
- **Corticosteroids:** reduce inflammation and consequently improve pulmonary function**. Dexamethasone is best choice, @ 0.05-0.1 mg/kg IV, IM**
- **Bronchodilators:** largely provide symptomatic relief**. Albuterol (β2 agonist), Short acting @ 360-720 µg, Inhalation. Clenbuterol (β2 agonist), Long acting**

Pneumonia and Pleuropneumonia in the Adult Horse

- Bacterial infections of the lower respiratory tract are common in adult horses.
- **Bronchopneumonia**: Infection involving both the bronchi and the lung parenchyma is bronchopneumonia.
- **Pleuropneumonia**: When the infection extends from the **pulmonary parenchyma** to the **pleural space**, **pleuropneumonia** occurs.

ETIOLOGY/PATHOGENESIS

- Adult horses most commonly acquire **bacterial pneumonia** by aspiration of microorganisms that are **commensal** to nasopharynx or oral cavity.
- The most common bacterial pathogens isolated from adult horses with bronchopneumonia is β-Hemolytic streptococci, particularly ***S equi*** subspecies ***zooepidemicus.***
- ***Synergy*** between **aerobic**, facultative aerobic, and **anaerobic** bacteria results in many **mixed bacterial infections**.

- Among Mycoplasma, ***M. equirhinis*** and ***M. felis*** are the common species isolated.
- Opportunistic bacteria can **colonize the lungs** when the pulmonary defense mechanisms are compromised.
- **Pulmonary defense mechanisms** can be altered by numerous factors, including
  - stress
  - viral infections
  - malnutrition
  - exposure to dust or noxious gases
  - Immunosuppressive therapy
  - immunodeficiency disorders, and
  - general anesthesia
  - Infections with **influenza virus** and **equine herpesvirus-4** have been shown to significantly decrease mucociliary clearance

- Bacterial invasion induces **infiltration with neutrophils** and other **inflammatory cells into the airways** and **pulmonary parenchyma** resulting in various degrees of **consolidation** or **focal abscesses**.
- These lesions **interfere with gas exchange** and, if severe enough, the resulting ventilation-perfusion mismatch **leads to hypoxemia** and clinical signs of respiratory disease.
- In animals with severe bronchopneumonia, inflammation extends to the pleural space. During the exudative stage, sterile fluid fills the pleural space in response to inflammation.
- If appropriate antimicrobial therapy is not initiated, the bacteria from the lung parenchyma **invade the pleural fluid**, resulting in **septic exudate** and the **fibrinopurulent stage**.
- **Fibrin** is deposited in continuous sheets covering both the visceral and parietal pleura resulting in loculation

DIAGNOSTIC EVALUATION

Physical evaluation

Clinical signs may include

- fever
- anorexia
- Bilateral nasal discharge
- cough
- weight loss
- tachypnea
- respiratory distress.
- Nasal discharge is usually mucopurulent but may be hemorrhagic with pulmonary infarction and necrotizing pneumonia

❖Horses with acute pleuropneumonia often exhibit pleurodynia (by grunting, evading pressure applied to the thorax, pawing, moving with a stiff forelimb gait, standing with abducted elbows)

Hematology and Biochemistry

- Bacterial bronchopneumonia frequently results in a **leukocytosis** and absolute **neutrophilia** with or without a left shift (**Note**: however, a normal leukogram does not rule out bacterial bronchopneumonia).

Diagnostic Imaging

- Radiographic abnormalities in horses with mild disease may range from normal to a mild bronchointerstitial pattern.

Tracheobronchial Aspirate

- Tracheobronchial aspirate (TBA) or transtracheal wash for cytologic examination and bacterial culture is one of the most helpful diagnostic procedures available

Thoracocentesis

- Thoracocentesis should be considered in horses with pleural effusion (to differentiate septic effusion from non-septic one)

TREATMENT

Antimicrobial therapy

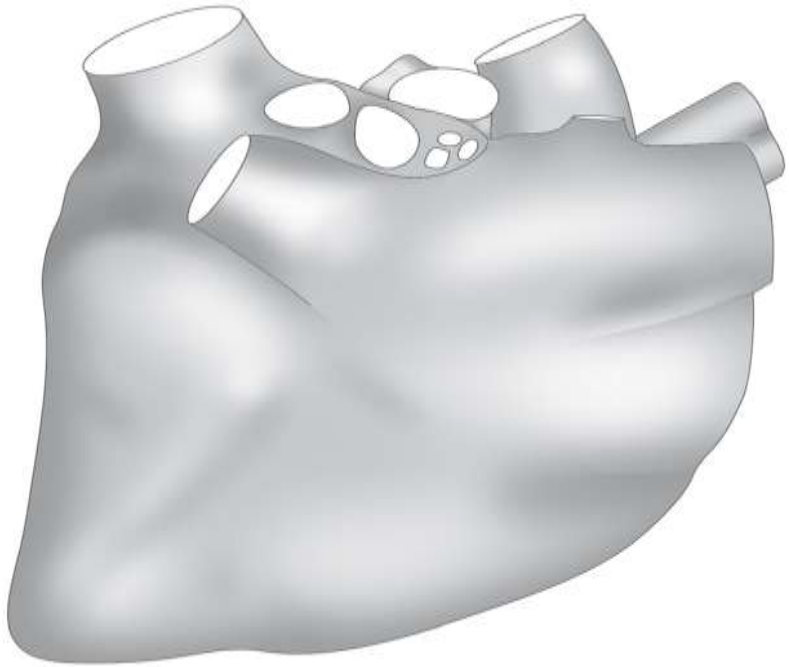
- Ceftiofur (broad spectrum) @2.2–4.4 mg/kg, IM BID.
- Oxytetracycline @ 6.6 mg/kg, **dilute** and Give **Slow IV**.
- Gentamicin @6.6 mg/kg, OD, IV or IM.

Nonsteroidal antiinflammatory agents

- flunixin meglumine or phenylbutazone may also be used for their anti-inflammatory, analgesic, and antipyrexic effects.

Oxygen supplementation

O_2 may be required in persistently severely hypoxemic horses.



Equine Cardiovascular system

Acquired cardiac disease

- Acquired cardiovascular disease is significantly more common in the horse than congenital heart disease.
- The majority of conditions involve the heart valves and degenerative valve disease is common.

VALVULAR DISEASE

- Acquired valvular disease may be a result of degenerative changes, damage or rupture of chordae tendinae, or bacterial endocarditis.
- Degenerative valve disease is most common. Aortic and left AV valve changes have been reported commonly.

Left atrioventricular valve disease

- Murmurs associated with left AV valve disease are relatively common in the horse
- it is the most likely valvular insufficiency to lead to congestive heart failure.

Etiology/Pathophysiology

- Structural changes in the **left AV valve** may contribute to **regurgitation**. Valvular thickening, fenestration, and cystic changes have been reported.
- With valvular insufficiency, **blood leaks** from the left ventricle to the left atrium during systole. This increases the volume load on the left atrium. With time, **atrial dilatation** may occur, which predisposes to the development of AF.
- This **progresses to pulmonary hypertension** and **right-sided pressure overload**.

Clinical presentation

- Typically, no clinical signs are apparent and the **murmur** is detected during routine examination.
- With severe regurgitation, respiratory signs such as labored breathing

Diagnosis

- Murmur on cardiac auscultation
- Echocardiography is required for diagnosis

Management/prognosis

- There is no treatment for correcting left AV valvular regurgitation.
- Clinical signs of heart failure should be treated as follows
  - Initially, administration of furosemide (0.5–2 mg/kg i/v, i/m, or p/o q8–12h) is indicated to decrease pulmonary fluids.
  - Bronchodilators (e.g. salbutamol) may improve respiratory function. Salt supplementation should cease
  - Digoxin (positive inotropic)
  - ACE inhibitors

Infectious Disorders of Blood and the vessels

Equine Infectious Anemia (EIA)

- Equine infectious anemia is also known as **swamp fever**.

ETIOLOGY

- EIAV is classified within the **Lentivirus** genus of the subfamily **Orthoretrovirinae** in the family **Retroviridae**
- EIAV infects cells of the **monocyte/ macrophage** lineage.

EPIDEMIOLOGY

- EIAV has a worldwide distribution.
- The virus appears to infect all equids, but clinical responses depend on viral, individual host, and host species factors.
- EIAV is a **blood-borne virus** that is naturally transmitted mechanically (no insect viral replication step) by blood-feeding insects especially members of the **Tabanidae** (horse flies and deer flies).
- For transmission to occur **feeding** must be **interrupted**, with the fly seeking and finding a second host to feed to repletion.
- **Transit time**: Virus can remain viable on mouthparts for at least **30 minutes**

PATHOGENESIS

- Clinical disease is initially caused by **proinflammatory cytokines** that include tumor necrosis factor a (TNFα), interleukin-1α and -β (IL-1α, IL-1β), interleukin 6 (IL-6), and transforming growth factor β (TGFβ).
- IL-6 and TNFa induce febrile responses (increases body temperature) while TNFα/TGFβ contribute to thrombocytopenia and TNFα promotes anemia by downregulating erythropoiesis.
- During later stages of the disease adaptive immune responses may also contribute to pathogenesis by immune-mediated destruction of **antibody coated platelets** and **phagocytosis.**

CLINICAL SIGNS

- EIA can have 3 distinct clinical phases designated **acute** (first disease episode), **chronic** (multiple sequential disease episodes), and **inapparent**.
- Disease can occur following a 1- to 4-week incubation period depending upon virulence and inoculum size of the infecting EIAV strain.

Acute Clinical Signs: include **fever**, **thrombocytopenia**, lethargy, and inappetance. In severe cases, petechiation, hemolytic anemia, and epistaxis can occur.

Chronic Clinical Signs: include **Multiple fever sequential episodes**, **anemia**, **thrombocytopenia**, weight loss, dependent edema, and occasionally neurologic signs (ataxia and/or encephalitis)

Inapparant (months to years): No overt signs

Pathology

- Hemosiderin granules in macrophages of liver, spleen, and lymph nodes
- Splenomegaly
- Hepatomegaly
- Glomerulonephritis (immune complex deposition)

DIAGNOSIS

- As no pathognomonic signs or lesions exist for EIA, reliance on laboratory tests is imperative.
- The agar gel immune-diffusion (AGID) test, also known as the **Coggins test**, is the test prescribed by the OIE.
- Confirmation is by PCR

THERAPEUTIC STRATEGIES

- Supportive therapy may be administered to aid in recovery from febrile episodes and associated signs.
- Treatment with corticosteroids is contraindicated because of the resultant increase in viral load and clinical disease.
- Recommend the humane destruction of EIAV test–positive equids.

CONTROL STRATEGIES

- There are currently no vaccines against EIAV in clinical use

EPIZOOTIC LYMPHANGITIS

- Epizootic lymphangitis (EZL) is caused by the fungal agent ***Histoplasma capsulatum var. farciminosum*** and affects horses, mules, and donkeys.

Etiology

- The fungus has 2 distinct phases: the **mycelial form** is present in the environment and the **yeast** is the pathogenic phase found in lesions.
- The mycelial phase favors humid, moist environments and is thought to persist in the environment where the disease is endemic.
- Cross-sectional studies have reported the overall prevalence of EZL in Ethiopia to be between **18.8%** and **30.1%**.

Pathogenesis and Clinical Signs

- Following invasion, H capsulatum var. farciminosum disseminates through the **lymphatic system** to **regional lymph nodes**, and in severe cases to organs.
- **Nodular lesions** develop in the **skin** along the lymphatics and in the lymph nodes.
- These nodules ulcerate, and produce a thick, purulent discharge containing yeast cells.
- **Four** different clinical presentations of EZL have been described: the **cutaneous form**, **ocular form**, **respiratory form**, and **asymptomatic carriers**.

Cutaneous form: Disease characterized by chronic suppurative, ulcerating pyogranulomatous dermatitis and lymphangitis.

- Lesions originate around the **lower limbs**, **chest**, **neck**, and **head**.

Ocular form: Characterized by a kerato-conjunctivitis with a serous to mucopurulent discharge.

- Intradermal swellings present within the palpebrae. There may be characteristic button ulcers on the outer margins of the conjunctivae.

Respiratory form: Nodules can present around the mucocutaneous junction of the nose and at **postmortem** are commonly seen to extend from the **nasal passages**, through the **trachea** and into the **lung parenchyma**.

- There is often an accompanying viscid mucopurulent nasal discharge and in advanced stages, animal produces **stertuous** noise during respiration.
- This form causes severe debility, cough, and progressive weakness

Therapeutic Strategies

- **Amphotericin B** is the listed drug of choice for the treatment of clinical cases of EZL by the OIE.
- On initial presentation, the animal is sedated and all nodules are **incised** and **flushed** with **topical 4% tincture of iodine**.
- **Potassium iodide** (KI, **30 g**) in solution is administered by nasogastric tube (for a horse of 200–250 kg).
- Oral KI is given at the **same dose daily,** for **5 days**, and then **every other day** for a further **3 to 4 weeks**, or as long as there is compliance from the owner.

Control Strategies

- Prophylactic vaccine have been developed and used by some countries like chine and Iran**.**
- Total Stamping out

PIROPLASMOSIS (Babesiosis)

- Equine piroplasmosis (EP) is an infectious, noncontagious, tick-borne disease caused by the hemoprotozoan parasites, ***Theileria equi*** (formerly ***Babesia equi***) and ***Babesia caballi***.
- This disease affects all equid species, including horses, mules, donkeys, and zebras.
- For *T. equi*, the **reservoir** of infection is the persistently **infected equid**; however, for B. caballi, both the infected horse and the primary tick vector serve as reservoirs for transmission.

Pathogenesis and Clinical Signs

- In both parasites, regardless of tick species variation, infectious **sporozoites** are transmitted through the tick saliva to the equid host, causing **erythrocyte lysis** resulting in variable degrees of hemolytic anemia.
- There are 3 presentations of equine piroplasmosis: **acute**, **chronic**, and the inapparent **carrier**.

Acute equine piroplasmosis

- Initially characterized by nonspecific signs of infection, including pyrexia, lethargy, decreased appetite, and peripheral edema.
- Petechiations on mucous membranes due to profound thrombocytopenia may be observed
- Signs of hemolytic anemia follow, with **pale/icteric mucous membranes**, tachycardia, tachypnea, weakness and pigmenturia.

Chronic equine piroplasmosis

- Infected horses usually exhibit signs of **nonspecific** chronic inflammatory conditions, including weight loss, poor body condition, partial anorexia, malaise, and decreased performance

Diagnosis

Diagnosis of acute equine piroplasmosis can be made on the basis of clinical signs and the careful examination of blood smears.

Treatment: Imidocarb dipropionate @ 2.2 to 4.4 mg/kg, IM, once.

Anthrax

- Anthrax is a serious (zoonotic) and commonly fatal septicemic condition resulting from infection with the bacterium ***Bacillus anthracis***.
- These bacteria release highly resistant spores, which contaminate the environment and help to spread the disease.
- Horses are less susceptible than cattle or sheep.
- Infection in cattle, sheep, or horses usually is the result of grazing on infected pasture land.

Virulence Factors/Toxins

- **Factor-I**: Edema Toxin
- **Factor-II**: Protecting Antigen
- **Factor-III**: Lethal Factor

Pathogenesis

- Following ingestion of anthrax spores, they turn into vegetative form in the intestine, followed by gaining access to lymphatic and blood circulation causing septicemia and death.

Clinical signs

- The incubation period of the disease ranges between **2 to 10 days**.
- In horses, the symptoms are generally acute in nature and follow a septicaemic course.

Peracute form: in this form animal may show **sudden death** without manifestations of clinical signs. Horse may die while at work or grazing field.

Acute form: is characterized by **high fever**, rapid pulse rate and **respiratory distress**. In some cases, **acute colic signs** with the passage of **bloody feces** may be seen. In addition, spasms of limbs may be seen. Death occurs due to **asphyxia** within 5-10 hrs of the attack.

Sub-acute form: is characterized by **edematous swelling** on the lower parts of the abdomen, thorax, shoulder and legs. In addition, intermittent colic may be seen.

Lesions

- ❑Carcass decomposes rapidly with gas formation and abdominal distension.
- ❑Blood is dark red and does not clot.
- ❑Spleen is enlarged

Diagnosis

Laboratory examination

- **Giemsa staining**-McFadyean's Reaction; bacteria appears blue rods with pink capsules
- **Cultural examinations**: medusa head appearance of colonies
- **Animal inoculation**: Guinea pig (suspension injected into the thigh)
- **Serological Test**-Ascoli's Test

Treatment

- ❑Treatment is effective in initial stages of the disease. Antibiotics particularly Penicillin group is effective.