Cartilage Lesions of the Ankle

Gian Luigi Canata · C. Niek van Dijk
Editors

Springer

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ISBN 978-3-662-46331-4 ISBN 978-3-662-46332-1 (eBook)
DOI 10.1007/978-3-662-46332-1

Library of Congress Control Number: 2015936186

Springer Berlin Heidelberg New York Dordrecht London

Printed on acid-free paper

Springer-Verlag GmbH Berlin Heidelberg is part of Springer Science+Business Media (www.springer.com)

Preface

The ankle is a complex joint, requiring an optimal biomechanical balance between stability and mobility to guarantee a physiological function. All anatomical structures must be restored if a lesion has occurred. Cartilage must be healthy to ensure good joint kinematics.

Over the last years, knowledge of the anatomical structures and biomechanics of the ankle has considerably increased, and experience has grown; in this booklet, several management and surgical procedures of cartilage lesions of the ankle are exposed.

The booklet begins with a chapter dedicated to MRI evaluation of osteochondral lesions of the ankle, describing different sequences and reporting MR-based classifications of cartilage lesions.

The following chapter discusses the conservative treatment of talar osteochondritis dissecans, giving an in-depth description to diagnose and manage this pathology.

Subsequent chapters explore different approaches to chondral and osteochondral lesions of the ankle. The last chapter is an update on the current rehabilitative approaches.

The result is a complete and detailed overview about this subject to supply the same guidelines and the same language in managing and reporting cartilage lesions of the ankle to orthopedic surgeons from all over the world.

We hope that this book, next to many others as supported by the ISAKOS educational drive, will help to improve the clinical and surgical management of our patients.

For the experienced surgeon, it is a good reference and brings him up to date with the latest developments.

The editorial group is grateful to all the authors involved in the project, who have actively cooperated to develop this educational book.

Turin, Italy — Gian Luigi Canata
Amsterdam, The Netherlands — C. Niek van Dijk

Contents

MRI in the Evaluation of Osteochondral Lesion

1

Sandro Giannini, Paolo Spinnato, and Francesca Vannini

1.1 Introduction

Magnetic resonance imaging (MRI) is nowadays an important noninvasive imaging technique for the evaluation of articular cartilage and subchondral bone alterations. MR examinations for osteochondral lesions of the talus showed high diagnostic performance compared to arthroscopy [1, 2].

The first imaging approach study is usually represented by conventional radiographs (anteroposterior, lateral and oblique projections) that could show positive but non-specific findings especially in chronic lesions, lesions with displacement, osteonecrosis or cystic change of the bone. Conventional radiographs presented low sensitivity, particularly in acute non-displaced lesions, and are unable to evaluate the integrity of articular cartilage surface [3]. Loomer et al. in a prospective study of 92 patients reported that 50 % of the osteochondral lesions of the talus are not detected on plain radiographs [4].

MRI showed higher sensitivity than computed tomography (CT) studies, especially for the evaluation of cartilage damage. However, the accuracy of CT studies increases in arthro-CT examinations. CT could be superior to MRI in bone analysis, particularly when strong bone oedema is present. Moreover, MRI is preferable to

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G.L. Canata, C.N. van Dijk (eds.), *Cartilage Lesions of the Ankle*,
DOI 10.1007/978-3-662-46332-1_1

CT after a negative preliminary conventional radiographic study, in case of persistent ankle pain, because it is able to evaluate at the same time the articular and extra-articular findings such as ligament or tendon injuries [3, 5]. On the other hand, when an osteochondral lesion is detected on plain radiographs, the preferred second-level diagnostic evaluation is CT to determine lesion's further details [6]. CT scan is important for preoperative planning since it shows the exact size and location of the lesion.

The absence of ionizing radiation makes MRI a safe and preferable imaging modality especially for paediatric patients and also for young patients that underwent several follow-up controls.

As previously outlined, several studies have been performed comparing MRI with arthroscopy, confirming high diagnostic performance of MRI. Results of these studies showed that MRI findings correlate closely with arthroscopy [6]. Moreover, MRI seems to be more sensitive than arthroscopy for deep lesions, while arthroscopy seems to be more accurate for superficial lesions.

1.2 Anatomy and Location of Lesions

The majority of osteochondral lesions of the ankle occur on the talar dome, while lesions on the distal tibia are quite rare because of mechanical and anatomic features of articular surfaces. Osteochondral lesions, less frequently, could affect more than one bone articular surface (Fig. 1.1). The talar dome is the third most common site of osteochondral lesions in the human body after the knee and the elbow [7, 8].

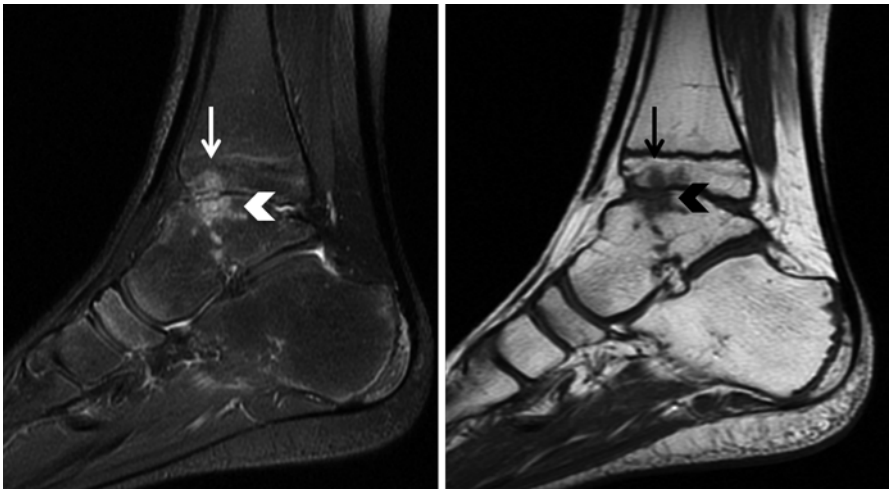

Fig. 1.1 MRI ankle evaluation (sagittal plane, proton density with fat saturation on the *left* and fast spin-echo T1-weighted on the *right*) of an 11-year-old boy showed osteochondral lesions on both distal tibia (*arrows*) and talar dome (*arrowheads*)

Osteochondral lesions of the distal fibula are extremely rare; however, the articular surface of distal fibula is described as a possible location of this kind of lesions [9].

Most of the osteochondral lesions of the talar dome occur in central medial ridge (about 65 %), and the second highest frequency of lesions occur on the central lateral ridge (about 32 %). Only 3 % of lesions are located in the middle of the talar dome. Moreover, the osteochondral lesions in the medial third of the talar dome are usually larger in both surface area and depth than lesions in the lateral third of talar dome [8, 10].

1.3 MRI Findings

To evaluate MRI findings for the diagnosis of osteochondral lesions, it is necessary to understand the normal appearance of both articular cartilage and bone tissue.

Articular cartilage, on MRI spin-echo T1-weighted sequences, is characterized by an intermediate signal intensity, higher than muscle signal intensity and lower than adipose tissue signal intensity. Also on proton density sequences, the articular cartilage presents an intermediate signal intensity between muscle and adipose tissue. On the contrary on spin-echo T2-weighted images, cartilage has a low signal intensity; this low signal intensity is in contrast with the adjacent synovial fluid that presents a very high signal intensity on T2-weighted images. This signal intensity contrast allows an excellent evaluation of cartilage tissue on these sequences. Moreover, the signal intensity of the cartilage on T2-weighted images increases from depth to superficial layers. In the gradient-echo sequences, articular cartilage presents an intermediate signal intensity that decreases in the depth layers near the subchondral bone [11].

Regarding bone tissue, we have to distinguish into cortical bone and medullary bone. Cortical bone has a typical low intensity of signal in all MRI sequences. Signal intensity of medullary bone can vary on the basis of bone marrow changes and presents different kinds of MRI presentations depending mostly on age and skeletal sites. In the adult, in normal bones of the ankle prevails the adipose medullary bone pattern which has high signal intensity on both T1- and T2-weighted images and it decreases significantly on fat-saturated sequences [11].

On T1-weighted images, the signal intensity of areas adjacent to fragment of osteochondral lesions is usually intermediate or low. A characteristic high signal line is detectable on T2-weighted images adjacent to the fragment demarcating it. This sign is usually called ‘rim sign’ and usually indicates an unstable lesion. Chondral defects may be detected on T2-weighted and density proton sequences with exposure of subchondral bone filled with synovial fluid that presents high intensity signal in these sequences. The loose bodies may be diagnosed on T2 images as area of low signal outlined by high signal fluid. The donor defect area is usually filled with articular fluid that is usually hyperintense on T2-weighted images [12]. The presence of subchondral cyst may be detected as regular area of high intense signal on T2-weighted and proton density images and hypointense on T1-weighted images (Fig. 1.2).

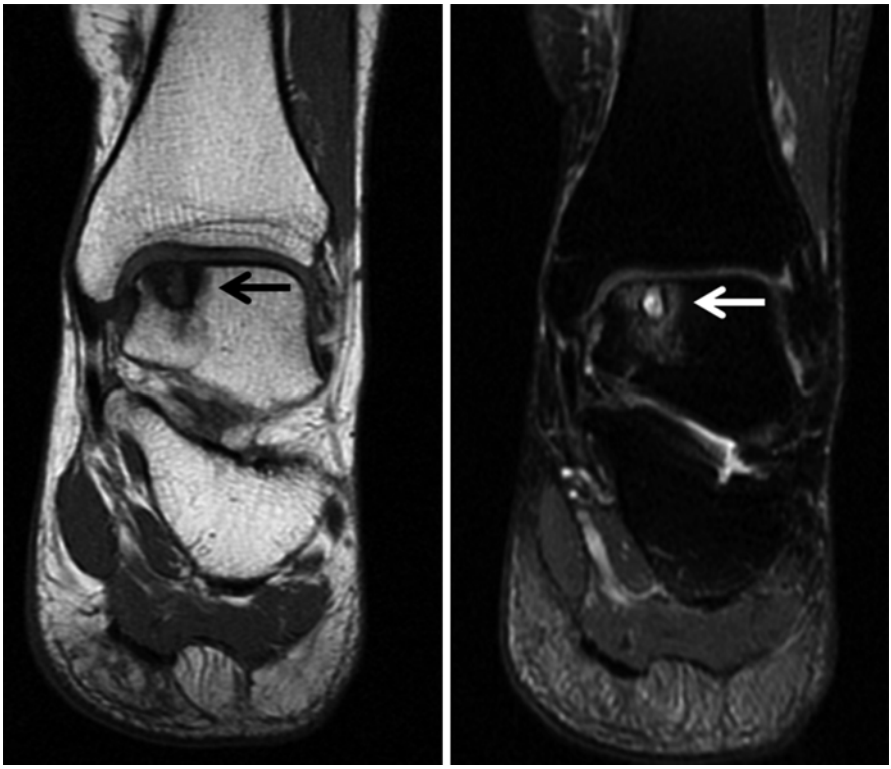

Fig. 1.2 Osteochondral lesion of the medial side of the talar dome (*arrows*) with cystic degeneration of subchondral bone and surrounded by bone oedema. The lesion is shown on T1-weighted MR images (*on the left*) and on proton density fat-suppressed images (*on the right*)

Bone oedema that frequently surround osteochondral lesions is characterized by low signal intensity on T1-weighted images and high signal intensity on T2-weighted fat-suppressed images or STIR sequences.

MRI is also able to help in differentiating between chronic or recent lesions. In fact, a low intensity signal on T1 images represents sclerosis and is a characteristic in chronic osteochondral lesions.

1.4 Technical Notes

Standard MR imaging sequences usually consist of fast spin-echo proton density, T2-weighted fat-suppressed sequences and newer various gradient echo sequences [13]. T1-weighted sequences are important and must be used for a correct evaluation of bone structures.

The best MRI sequence for the detection of osteochondral lesions is still a subject of controversy. Some reports based proposed thin-section 3D Fourier transform

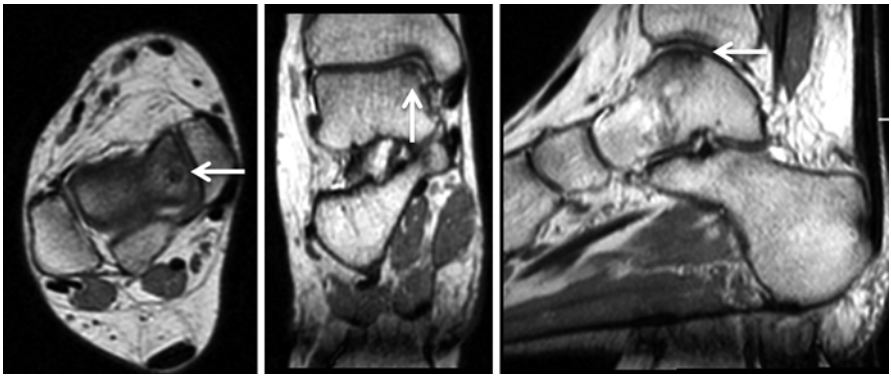

Fig. 1.3 MRI, fast spin-echo proton density 'cube' with thin slices acquired on axial plane (*on the left*) and reconstructed on both frontal (*on the middle*) and sagittal planes (*on the right*), showed a small osteochondral lesion of the talar dome (*arrows*) in a 47-year-old woman with persistent ankle pain and previous negative findings on plain radiographs

spoiled gradient-echo techniques with fat saturation and others fat-saturated fast spin-echo technique. Some authors also recommended intra-articular injection of contrast material (gadolinium) to obtain an improved detection of osteochondral lesions even if this approach is rather invasive [14].

Sagittal and coronal views are the preferred planes to evaluate osteochondral lesions of the ankle that mostly occur in the talar dome.

Three-dimensional sequences can improve the detection of cartilage lesions because of their possibility to obtain thin slices with high spatial resolution. This is particularly important in the ankle that is characterized by thin and incongruent articular surface of the distal tibia and talar dome [15]. Three-dimensional sequences permit also multiplanar reconstruction on coronal, sagittal, axial and also oblique plain, independently from the sequence acquisition plane, similar to CT thin slice reconstructions, and they are also able to show very small lesions (Fig. 1.3).

Recent researches have highlighted the importance of the quantitative MRI evaluations that can detect early articular cartilage damage before morphologic cartilage loss occurs. Amongst these new MRI tools, we report T2 and T2* relaxation time measurements (T2 and T2* mapping) that is the most used and represents a biomarker for the valuation of articular cartilage and cartilage repair tissue. Higher and more heterogeneous T2 values are thought to characterize collagen deterioration and increasing water contents [16].

Diffusion-weighted imaging evaluates water mobility in the articular cartilage. In cartilage with an intact collagen structure, water mobility is restricted. Early cartilage degeneration can be demonstrated on diffusion-weighted images by the increased mobility of water in a deteriorated extracellular matrix [16].

These tools have particularly grown in importance due to the necessity to closely follow up osteochondral lesions after cartilage repair, since the ethical aspects of biopsy in healthy patients are not acceptable anymore.

1.5 Classification

Berndt and Harty in 1959 established a 4-stage classification system of ankle OCLs based on the severity of the lesion on plain radiographs, which has been widespread used for decades and still has an importance on the evaluation of plain radiographs (Table 1.1) [17].

As new imaging modalities emerged, several osteochondral classifications have been created.

In 1993 Loomer et al. completed the Berndt and Harty classification, adding a fifth type based on CT findings, characterized by radiolucent cystic lesion [4].

In the past years, as MRI was emerging as the gold standard imaging tools for the diagnosis of osteochondral lesions, many classifications have been proposed using this imaging technique.

Dipaola et al. proposed a classification for osteochondral lesion of the talus in 1991, using MRI (Table 1.2) [18].

Later, Taranow et al. used MRI to describe the condition of both the cartilage and subchondral bone by employing and describing also the viability of cartilage (Table 1.3) [19].

Table 1.1 Berndt and Harty classification of osteochondral lesions of the ankle (1959)

Stage	Lesion's morphology on plain radiographs
I	Compressed
II	Chip avulsed but attached
III	Detached chip but undisplaced
IV	Detached and displace chip

Table 1.2 Dipaola et al. classification based on MRI findings (1991)

Stage	MRI findings
I	Thickening of articular cartilage and low-signal changes on intermediate/spin density images
II	Articular cartilage breached with low-signal rim behind fragment indicating fibrous attachment
III	Articular cartilage breached, high-signal changes behind fragment indicating synovial fluid between fragment and underlying subchondral bone
IV	Loose body

Table 1.3 Tarantow et al. classification based on MRI findings (1999)

Stage	MRI findings	Grade A	Grade B
I	Thickening of articular cartilage and low-signal changes on intermediate/spin density images	Cartilage viable and intact	Cartilage nonviable
II	Articular cartilage breached with low-signal rim behind fragment indicating fibrous attachment	Cartilage viable and intact	Cartilage nonviable
III	Articular cartilage breached, high-signal changes behind fragment indicating synovial fluid between fragment and underlying subchondral bone	Cartilage viable and intact	Cartilage nonviable
IV	Loose body	Cartilage viable and intact	Cartilage nonviable

Table 1.4 Hepple et al. classification (1999)

Stage	MRI findings
I	Articular cartilage damage only
IIa	Cartilage injury with underlying fracture and surrounding bony oedema
IIb	Stage 2a without surrounding bony oedema
III	Detached but undisplaced fragment
IV	Detached and displaced fragment
V	Subchondral cyst formation

Table 1.5 Mintz et al. classification (2003)

Grade	MRI findings
0	Normal
I	Hyperintense but morphologically intact cartilage surface
II	Fibrillation or fissures not extending to bone
III	Flap present or bone exposed
IV	Loose undisplaced fragment
V	Displaced fragment

Table 1.6 Griffith et al. high-resolution MRI classification (2012) – grades 2b, 4b and 5 are classified as unstable lesions of variable severity

Grade	A	B
I	Bone marrow change (oedema, cystic change) with no collapse of subchondral bone area and no osteochondral junction separation and intact cartilage	Similar to grade 1a, although with cartilage fracture
II	Variable collapse of subchondral bone area with osteochondral separation through intact cartilage	Similar to grade 2a, although with cartilage fracture
III	Variable collapse of subchondral bone area with no osteochondral separation with or without variable cartilage hypertrophy	Similar to grade 3a, although with cartilage fracture
IV	Separation within or at edge of bone component, with intact overlying cartilage	Similar to grade 4a, although with cartilage fracture; unstable lesion, with level of instability related to extent of cartilage fracture
V	Complete detachment of osteochondral lesion; unstable lesion	

Hepple et al. improve the original Berndt and Harty classification, on the basis of MRI findings (Table 1.4) [20].

In the following years, a classification with arthroscopic correlation has been proposed for grading osteochondral lesions of the talus. This classification can help physician identify patients who would benefit from arthroscopic treatment (Table 1.5) [1].

More recently a new classification has been studied based on high-resolution MRI; the research has been carried out with microscopy coil imaging at 1.5 T (Table 1.6) [21].

Conclusions

MRI has demonstrated in the last two decades to be an important noninvasive imaging modality of choice in the evaluation of osteochondral lesions of the ankle, with high diagnostic accuracy if compared with invasive arthroscopy evaluation.

MRI is the diagnostic modality of choice in patients with clinical suspicions of osteochondral lesions with negative plain radiographs. Moreover, MRI is the preferable tool for the follow-up of patients who underwent different kinds of surgical treatments with particular regards on new modality of cartilage reconstruction [22, 23].

MRI can provide information regarding the size, grade and location of osteochondral lesions. Although for preoperative planning, CT scan is often preferred. MRI is able to inform about other fundamental findings that can help physicians in choosing correct treatment strategies such as the condition of the overlying articular cartilage, the congruity of the articular surfaces, the viability of the bone fragment, the stability or degree of healing between the osteochondral fragment and the donor site and the location of the osteochondral fragment if it has become displaced within the joint space [14].

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Conservative Treatment of Talar Osteochondritis Dissecans (OCD)

2

Ezequiel Palmanovich and Meir Nyska

2.1 Introduction

Since 1922 when this syndrome was first described by Kappis [1], numerous terms have been used to describe chondral lesion of the dome of the talus including osteochondral fracture, osteochondritis dissecans, and talar dome fracture. This lack of well-defined terminology clearly demonstrates the unclear cause of the lesion [1]. In 1959, Berndt and Harty [2] proposed trauma as the principle etiological factor. In 1966, Campbell and Ranawat [3] proposed the name osteochondritis dissecans as a pathologic fracture resulting from necrotic changes due to ischemia. However, traumatic etiology plays an important role in the pathogenesis of osteochondral lesions of the talus. The lesion most likely represents the chronic phase of a compressed or avulsed talar dome fracture. Isolated incidents of macrotrauma or repeated cumulative microtrauma may contribute to initiation of the lesion in someone predisposed to talar dome ischemia. Osteonecrosis in a patient may cause the bone to collapse resulting in subchondral fracture and bony collapse. The subchondral cysts with overlying chondromalacia, osteochondral fragments, and loose bodies all represent stages in the progression of this disease [1].

2.2 Clinical Presentation

In acute injury, clinical presentation remains unrecognized. Pain and swelling interfere with the diagnosis. An x-ray performed in the emergency room may not reveal pathology or even a small area of radiolucency. An x-ray may reveal larger lesions. Lesions can heal spontaneously, or chronic symptoms may develop. Initially, the

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G.L. Canata, C.N. van Dijk (eds.), *Cartilage Lesions of the Ankle*,
DOI 10.1007/978-3-662-46332-1_2

patient may experience pain during exercise or weight bearing; then deep joint pains may emerge. If symptoms continue for more than 4–6 weeks after "ligament injury" in the ankle, differential diagnosis of chondral defect must be ruled out. Morning or post-rest stiffness, chronic swelling, and catching [4, 5] are the most common symptoms.

As cartilage is aneural, the pain originates from the subchondral bone exposed beneath the cartilage defect [6]. In chronic cases, there is a delay in clinical manifestation.

2.3 Nonoperative Treatment

Prognosis depends on the patient's age. Lesions which occur during childhood and adolescence tend to heal spontaneously, while the prognosis is poor for older patients and requires treatment [8].

Different treatment options depend on the lesion grade. Using computerized tomography (CT), lesions can be classified according to Ferkel et al. [9] from grade 1 to 4. Grade 1 includes cystic lesions with intact walls; grade 2 includes cystic lesions communicating with the talar dome, or full-thickness lesion with an overlaid fragment; grade 3 includes undisplaced lesions with lucency; and finally grade 4 includes free loose fragments [7].

Goals for treatment of talar OCDs are to decrease pain, improve function, and regenerate articular cartilage or prevent early joint degeneration [7].

For grades 1 and 2, nonoperative treatments are indicated [7].

Treatment in acute cases starts with rest and immobilization. Non-weight bearing physiotherapy is crucial to preserve range of motion.

2.3.1 Hyaluronic Acid (HA)

Treatment with hyaluronic acid, discovered in 1934 is well known as nonoperative treatment for knee and ankle osteoarthritis [9–12] and also for talar chondral lesions [4].

Hyaluronic acid (HA) is a glycosaminoglycan present in many tissues throughout the body and represents the major component of synovial fluid. At the cellular level, it is found in abundance in the extracellular matrices [4]. The rationale for the use of HA is based not only on the findings that the viscoelasticity of the synovial fluid is entirely due to its HA content and that HA forms an integral part of the proteoglycans of articular cartilage, but also on mounting evidence that HA may influence the disease by interacting with components of the synovial fluid and synovial cavity [13].

In 2008, Mei-Dan et al. [4] published results of the first prospective study on patients treated for OCD of the talus. In the study, 15 patients aged 18–60 with talar OCD were followed up 26 weeks after receiving three weekly injections. About 60 % of the patients had grade 3 lesions. On a scale for pain from 1 (no pain) to 10

(severe pain), mean VAS reported a decrease in pain from 5.6 to 3.2. There were also reports of an improvement in stiffness from 5.1 to 2.9 and function from 5.9 to 3.3, from baseline to week 26. Subjective global scores, on a scale from 0 to 100 (100 representing healthy normal function), reported an improvement, on average, from 57.3 at baseline to 74.3 by week 26. In this study, HA provided relief of pain, stiffness, and improved function. Efficacy of the treatment started at 3 weeks from the first intra-articular injection and peaked at 3–5 months with minimal safety concerns.

2.3.2 Platelet-Rich Plasma

Plasma rich in growth factor is a form of PRP [14] and is a biological delivery system of a complex mixture of bioactive proteins essential to natural repair, including anabolic and protective factors for cartilage, such as transforming growth factor-β1 (TGF-β1), platelet-derived growth factor (PDGF), and insulin-like growth factor (IGF-I) [7, 15]. The TGF-β1 is essential for cartilage integrity and is a powerful tool for prevention or repair of cartilage damage [16]. This growth factor is in its latent form while in the platelets and is activated by TSP-1 (thrombospondin), which is also released from platelet a-granules found in PRGF. As a result, intra-articular administration of PRGF could retard or prevent progressive degeneration of joint cartilage [7]

In 2012, Mei-Dan et al. [7] reevaluated the short-term efficacy of PRP compared with hyaluronic acid in reducing pain and disability in a randomized study of 32 patients. In the study, the results showed that the osteochondral lesions of the ankle treated with intra-articular injections of PRP and HA resulted in a decrease in pain scores and an increase in function for at least 6 months, with minimal adverse events. Platelet-rich plasma treatment led to a significantly better outcome than HA. This study showed again the beneficial effect of injection of hyaluronic acid.

2.3.3 Steroid Injections

Corticosteroid injections are common procedures for reducing pain associated with OA [17].

The pathways by which injectable depot corticosteroids mediate symptom relief are not completely understood. Local action of decreasing inflammation in synovial tissues is believed to be the primary effect of depot corticosteroids. The effect is particularly profound on edema as well as the number of lymphocytes, macrophages, and mast cells [18]. In addition to local effects, intra-articular corticosteroids may elicit dose-related systemic effects. Marked improvements in inflammatory markers, such as erythrocyte sedimentation rate (ESR) and C-reactive protein level, can occur in patients with rheumatoid arthritis (RA) [19].

The efficacy and duration of intra-articular steroid injections were reevaluated by Hepper et al. [18] in 2009. This level 1 systematic review for knee osteoarthritis

shows significant pain reduction for about 1 week; however, after 4–6 weeks, there was no difference between the treatment and the placebo. There are no data concerning treatment of OCD by local injection of steroids, but it can be assumed that the results might be similar to those as in osteoarthritis of the knee.

2.3.4 Hyperbaric Oxygen Treatment (HBOT)

One of the main findings of OCD in MRI is bone edema accompanying the lesion. This is believed to demonstrate active lesion and one of the causes for pain. One of the known treatments to reduce bone edema is HBOT. The mechanism of action of HBOT is production of PO_2 gradient between arterial blood and hypoxic tissue. This produces a fluid pump effect in the desired direction for resolving edema [20]. In HBOT, the O_2 dissolved in plasma and tissues is increased, reducing the edema effect. This reduction may be beneficial in the reduction of the edema around the OCD, which lead to reduction in pain. Yet, other known beneficial effect of HBOT on bone healing which might affect the OCD has to be explored.

2.4 Summary

Conservative treatment in symptomatic patients with OCD is poorly described. PRP treatment seems an efficient conservative treatment of OCD. There are however no large-scale randomized trials and no long-term follow-up studies to support the large-scale use of PRP. Treatment with hyaluronic acid may also be an effective mode of treatment. Due to the short duration of efficacy, intra-articular steroid injections are not recommended. HBOT decreases bone edema and may lead to decrease in pain.

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Evolution of Surgical Treatment of Cartilaginous Lesions

3

Sandro Giannini, Marco Cavallo, and Francesco Castagnini

3.1 Introduction

Osteochondral lesions of the talus (OLT) are defects of the cartilaginous surface and the underlying subchondral bone [1].

OLT are usually traumatic in origin, mostly subsequent to ankle sprains or repetitive microtraumas [1, 2]. The non-traumatic etiology encounters for small amount of cases which have been related to various (weak) hypothesis, for example, embolic, hereditary, endocrine, and idiopathic [1–3]. OLT are frequently reported by young patients, often related to acute traumas. Pain and limited range of motion (ROM) are usually reported, with swelling and even locking or catching as well [1]. The diagnosis is usually confirmed by mean of MRI [4] (Fig. 3.1).

OLT may evolve to osteoarthritis as chondral tissue has poor healing abilities; therefore, the damage may be irreversible and lead to chronic symptoms [1, 2]. OLT provides not only a local osteochondral disruption, but it also alters the biomechanics of the surrounding cartilage, predisposing to arthritis. In a work by Choi, in line with the classical theory of Berndt Hardy, a critical size defect was traced at 150 mm^2 OLT, with good healing for defects with lower area [5, 6].

No clear indications exist for OLT treatment: there is a lack of evidence to point out the better treatment strategy for OLT in adults [2, 3]. The aims of the treatments are to provide a stable, smooth joint osteochondral surface, restore function, and prevent the degenerative evolution [1–3, 6].

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G.L. Canata, C.N. van Dijk (eds.), *Cartilage Lesions of the Ankle*,
DOI 10.1007/978-3-662-46332-1_3

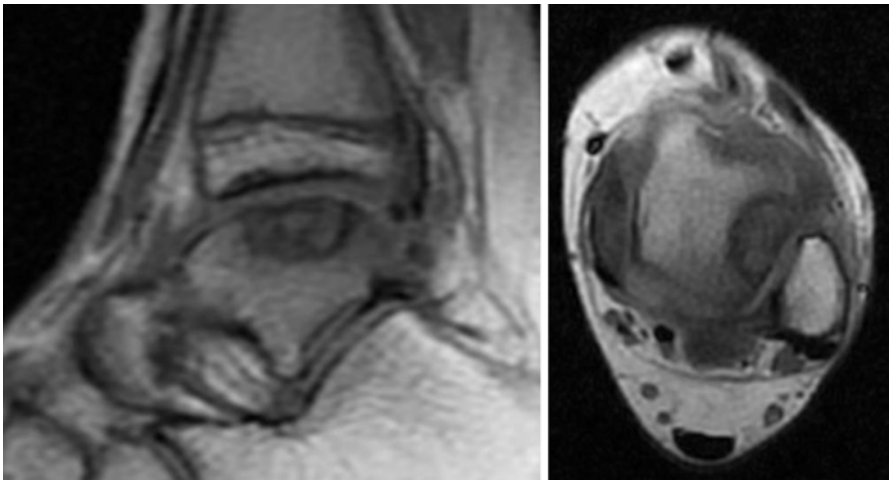

Fig. 3.1 Sagittal and transversal MRI scan of an osteochondral lesions of the talar dome: MRI is useful to confirm the diagnosis

A useful guideline is given by Giannini's classification, focused on arthroscopic/MRI findings and corresponding treatments, considering the area and the depth of the lesions [7].

Acute lesions are divided into two groups, considering the dimensions of the fragment. Debridement and excision are advised in case of acute lesions with fragment dimensions inferior to 1 cm, whereas fragment fixation performed with bioresorbable screws is performed in case of larger OLT [8].

In case of chronic lesions with no chondral disruption (type 0 according to Giannini), retrograde drilling could be effective to treat the modest subchondral bone involvement. The rationale consists in a stimulation of the repair depending on subchondral bone marrow cells [2, 3, 8].

Microfractures are preferred in larger OLT, inferior to 1.5 cm^2. The technique can be easily performed arthroscopically, penetrating the subchondral bone every 3–4 mm, using a dedicated pick [9, 10]. Thanks to bone marrow stimulation, this procedure allows a good and rapid restoration of the osteochondral layer, but it generates fibrocartilage, with lower biomechanical properties and durability [9, 10]. Moreover, microfractures demonstrated to achieve lower scores in large and medial OLT [9].

Due to previous described limits, regenerative techniques were developed to supply hyaline cartilage restoration, treating larger defects as well. To date, mosaicplasty or osteochondral autograft, autologous chondrocytes implantation, and bone marrow-derived cells transplantation are the most used techniques [10–12].

Osteochondral autograft, obtained from non-weight-bearing areas of the knee and, possibly, ankle, is implanted to restore the proper osteochondral layer [11, 12]. It has been used for lesions >1.5 cm^2 with reported good results [12]. Nevertheless, it carries out several drawbacks, such as the need of a malleolar osteotomy and

donor site pathology, technically demanding challenges in the reconstruction of a smooth continuous articular surface [11].

In order to avoid these drawbacks, procedures including cellular supplying like ACI or BMDCT were introduced, marking a milestone in regenerative medicine.

3.2 Autologous Chondrocytes Implantation: Open-Field Procedure

3.2.1 Preliminary Considerations

Similarly to mosaicplasty, the best approach to large osteochondral lesions would be regeneration with hyaline cartilage, possibly avoiding the deleterious effects of donor site morbidity [7, 11, 13–15]. Some clinical and animal experiments reported encouraging results injecting cultured autologous chondrocytes into the defect under a periosteal flap, achieving defect healing with hyaline-type cartilage [13]. ACI was first described in the knee joint, with excellent intermediate results; the application was then extended to the ankle, despite the many biomechanical differences between the two joints [11, 13].

3.2.2 Surgical Technique

Cartilage harvesting is the first step required in ACI [13]. Using arthroscopical standard portals, a small sample of cartilage (15–25 mg cartilage tissue) is harvested from the ipsilateral knee for cell culturing. In order to minimize the harvest site pathology, after the first cases performed in this way, ankle arthroscopy was performed to identify a different cell source. The osteochondral detached fragment in the ankle was proven to be a good source of viable cells for ACI [13, 15].

Alternatively, cartilage may be harvested directly from the lesion margins of the affected ankle in the first step arthroscopy [15]. A direct evaluation and accurate measurement of the osteochondral damage is performed at the same time.

Cartilage is sent to a specialized laboratory for cell expansion, and it is available for implantation 4 weeks later [13, 15]. In the meanwhile, after few days of crutches and joint protection, patients are allowed to weight-bear progressively, basing on pain.

The possible surgical approach to the OLT is transmalleolar, medial, or lateral, depending on the location of the defect [13]. The lesion is exposed and the damaged cartilage and bone are debrided. The lesion is accurately measured, in order to shape and size the periosteal flap to be implanted. A periosteal flap is harvested either from the proximal or distal tibia and fixed over the cartilaginous gap with reabsorbable 6-0 suture threads. The suture is sealed with fibrin glue. The chondrocytes in liquid media are then transplanted through a hole deliberately left open and finally sutured and sealed with fibrin glue. The malleolar osteotomy is finally repaired [13].

3.2.3 Results

The clinical results were excellent at 10-year follow-up. The MRI outcomes showed a smooth cartilaginous layer, with hyaline-like features at biochemical evaluation using T2 mapping. The procedures achieved clinical and radiological scores which were not inferior to ACI in knee defects [13]. Nevertheless, the technical difficulties of open ACI required some improvements towards a simpler procedure [11, 13, 15].

3.3 Autologous Chondrocytes Implantation: Arthroscopic Procedure

3.3.1 Preliminary Considerations

The surgical technique was deeply influenced by the introduction of tissue engineering and a specific instrumentation for an arthroscopic procedure [15, 16]. These developments allowed to perform the procedure arthroscopically, decreasing patients' morbidity and technical drawbacks [15, 16]. The chondrocytes are harvested and loaded on a biodegradable scaffold based on the benzylic ester of hyaluronic acid (HYAFF 11, Fidia Advanced Biopolymers, Italy). The scaffold is a 3D support for cell adhesion and proliferation thanks to a network of 10–15 μm thick fibers with interstices of variable sizes. This peculiar constitution permitted an optimal physical support to allow cell-to-cell contact, cluster formation, and extracellular matrix deposition [15, 16].

3.3.2 Surgical Technique

The surgical technique is completely performed arthroscopically [15, 16]. In the first step, the harvesting of chondrocytes is executed, performing a standard ankle arthroscopy. Then after culturing the chondrocytes in the hyaluronic membrane, a second standard arthroscopy is performed [15, 16]. The lesion is debrided, and after sizing and shaping the biomaterial basing on the lesion, the membrane is delivered. A custom-made-specific instrumentation was developed for this purpose (CITIEFFE, Calderara di Reno, Italy) [15]. This consists of an 8 mm diameter and 111 mm-long stainless steel cannula with a window on one side and a positioner specifically designed to slide inside the cannula delivering the scaffold directly to the site of lesion [15].

In cases with an osteochondral defect deeper than 5 mm (IIA lesions), a bone graft using cancellous bone harvested from the distal tibia is performed before biomaterial positioning [15, 16].

3.3.3 Results

Autologous chondrocytes implantation (ACI) has been intensively applied for OLT with successful clinical outcomes (90 %) [14–16]. Although no clear superiority

has been established, ACI is considered one of the most reliable techniques in regenerative procedures [14]. The clinical results are described as excellent in many case series, at midterm and long-term follow-ups [14, 15]. The hyaline regeneration was confirmed by histological and radiological outcomes [16]. Nevertheless, the high costs, the need of a specialized laboratory phase, and most of all two surgical steps, and the lack of the bone regeneration are the most significant limits of this technique [16, 17].

3.4 Bone Marrow-Derived Cells Transplantation

3.4.1 Preliminary Considerations

Bone marrow-derived cells transplantation (BMDCT) represents the third generation of regenerative technique for bony and chondral layer, based on mesenchymal stem cells [17, 18]. This technique may be performed one step, in a same surgical session, or with more steps, with cells culture and enrichment [17]. In our experience, we have been performing the one-step technique, developed to overcome the two surgical steps and reduce the costs of ACI [17, 18]. Moreover, the osteogenic lineage differentiation of bone marrow-derived cells allows large bony lesion regeneration [18].

3.4.2 Surgical Procedure

3.4.2.1 Platelet Gel Production

In order to provide a supplement of growth factors and stabilize the biomaterial implantation, platelet-rich fibrin (PRF) can be used in this technique [17, 18]. Through the Vivostat system (Vivolution A/S, 3460 Birkerod, Denmark), 120 ml of the patient's venous blood is processed and as far as 6 ml of PRF is obtained [17, 18]. This procedure takes place the day before surgery.

3.4.2.2 Bone Marrow Aspiration

A total amount of about 60 ml of bone marrow aspirate is harvested from the posterior iliac crest, with the patient in prone decubitus and in sterile regimen, under general or spinal anesthesia [17, 18]. A marrow needle (size 11 G 9 100 mm) is inserted into the spongy bone of the posterior iliac crest; 5 ml of bone marrow is aspirated into a 20 ml syringe internally coated with calcium-heparin solution. Then rotation and withdrawing are performed. The procedure is repeated 3–4 times by perforating the iliac crest through the same skin opening, in order to avoid excessive blood dilution and maximize cells harvesting. 60 ml of bone marrow is eventually collected [17, 18].

3.4.2.3 Bone Marrow Concentration

Through a process of separation-concentration using the kit IOR G1 (Novagenit, Mezzolombardo, Italy), 6 ml is obtained from the aspirated bone marrow: they are

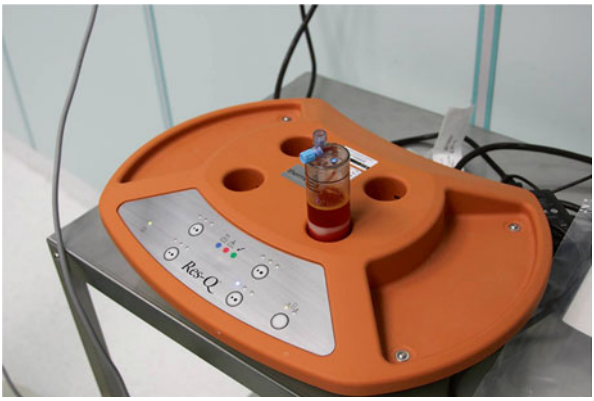

Fig. 3.2 A phase of cell process and concentration is performed. A concentrate of nucleated stem cells is obtained

rich in nucleated cells like stem cells, monocytes, lymphocytes, and bone marrow resident cells [17, 18]. This procedure takes place in the same surgical session [17, 18] (Fig 3.2).

3.4.2.4 Arthroscopic BMDCT

After the bone marrow harvesting phase, a standard anterior ankle arthroscopy is performed, with the patient in the supine position [17, 18]. The joint environment is evaluated; bony and fibrous impingement is removed when present. The OLT is detected and curetted, evaluating the size and shape of the lesion [17, 18]. Then, a scaffold is prepared according to lesion dimensions. It is tridimensional structured, biocompatible, and reabsorbable. In our experience, we used hyaluronate or, more recently, collagenic scaffold, as it seems to be better for bony regeneration [17, 18] (Fig. 3.3). The scaffold is loaded with 2 ml of bone marrow concentrate and 1 ml of PRF. The composite is positioned onto the lesion using the same instrumentation as previously described for the ACI [15–18]. Finally a layer of PRF is added. Multiple sagittal ankle movements are performed under arthroscopic control to verify the stability of the implant (Fig. 3.4).

3.4.2.5 Results

Clinical results at medium-term follow-up are encouraging, with excellent outcomes even in athletes. Hyaline cartilage regeneration has been appreciated in bioptic samples and MRI qualitative scans [17, 18].

3.5 Postoperative Care and Rehabilitation

Regenerative techniques require a specific and long timetable for rehabilitation, due to biological properties of the implanted cells [19]. Chondrocytes require 6 weeks to start incorporating and dividing. Then, extracellular matrix is produced between 3 and 6 months [19]. The integration of the biomaterial with the subchondral bone occurs not before 6 months. Then a process of remodeling and maturation takes

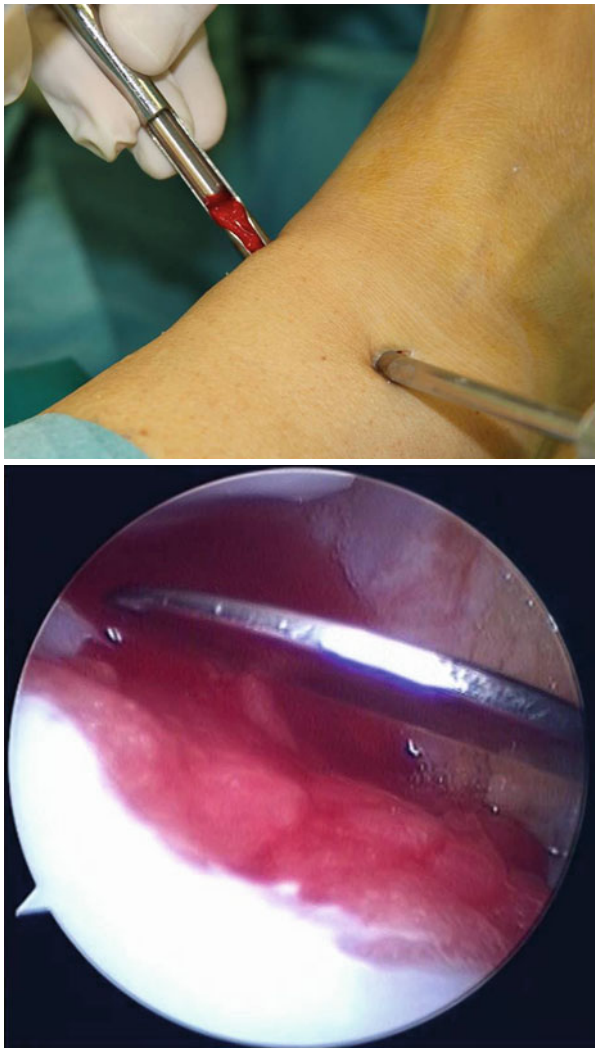

Fig. 3.3 An arthroscopic implantation of the cells is performed using a dedicated instrumentation, which was elaborated for ACI

place, for 2–3 years [17, 19]. The rehabilitation protocol for regenerative techniques should be focused on this process, respecting the chondral layer. For this purpose, a mix of continuous passive motion, delayed progressive weight-bearing, and muscular strengthening is advised [19]. Personalized schemes should be encouraged, mostly when athletes are involved. In our experience, ACI and BMDCT share the same rehabilitation protocol [15, 17, 19]. The day after surgery, continuous passive motion is advised, and a Walker ankle brace is applied [15, 17]. The period of joint protection lasts about 6 weeks, followed by a partial, progressive weight-bearing [15, 17]. After 4 months from surgery, low impact sport activities (swimming, cycling, etc.) can be safely performed [15, 17]. A progressive return to running and high-impact sport activities is not allowed before 10–12 months [15, 17].

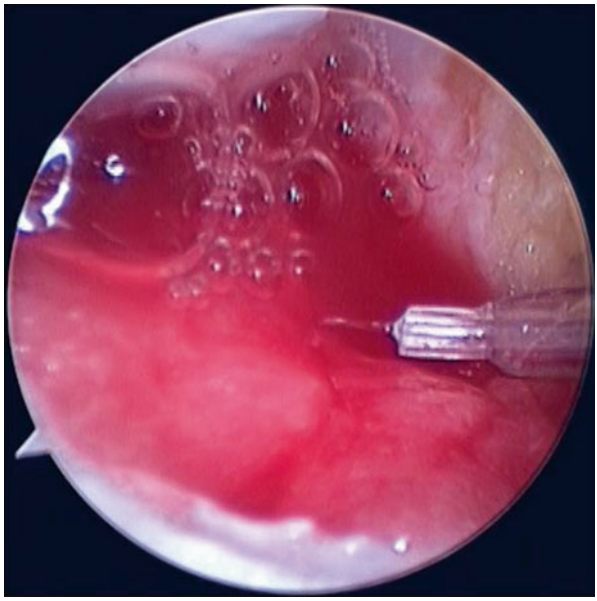

Fig. 3.4 A layer of PRF is sprayed onto the biomaterial, in order to improve stability and add growth factors

Conclusions

The appropriate surgical treatment for osteochondral lesions of the talus is still a controversial topic [2, 3, 9, 11].

Debridement, drilling, and microfractures in chronic osteochondral lesions of the talus are reported to have good results. However, the newly formed tissue is fibrocartilage with poor mechanical quality: nevertheless, these procedures could achieve successful outcomes in all OLT up to 15 mm diameter [2, 3, 9, 11].

Regenerative procedures could be useful to restore the hyaline chondral layer, overcoming the limits of the previous techniques [9, 11]. Osteochondral autografts are currently available for this purpose [12]. Nevertheless, the many drawbacks related to osteochondral autograft implantation gave a significant prompt for other simpler procedures with no donor site morbidity, as ACI or BMDCT [12, 17, 19].

Open-field ACI in the ankle was the first technique to be widely applied [13]. It required an open-field approach, with a periosteum suture flap, which made this procedure technically demanding. Nevertheless, remarkable outcomes were achieved even in large-size lesion, even at long follow-up (10 years) [13].

The development of a specific biodegradable 3-dimensional scaffold for cell support and proliferation allowed the evolution of the surgical technique to be completely arthroscopic [15]. The ACI arthroscopic treatment is fast and effective; it is associated with a very low morbidity rate [15, 17].

The main drawbacks of arthroscopic ACI are the need of two surgical operations, the high costs, and a specialized laboratoristic phase [15–17]. In order to overcome these drawbacks, BMDCT, relying on mesenchymal stem cells, was developed [15–17].

Mesenchymal stem cells represent 2–3 % of the total mononuclear cells in bone marrow and recently have been indicated as a new option for the treatment of articular osteochondral defects, because of their ability to differentiate into various lineages, including osteoblasts and chondroblasts [17]. The transplantation of the entire bone marrow cellular pool, the niche, permits the cells to be processed directly in the operating room (one-step procedure), without the need of a laboratory phase and supplying a large amount of growth factors [17].

Very good results have been achieved with BMDCT, with remarkable clinical and MRI outcomes at midterm follow-up, with hyaline cartilage restoration at histological evaluation [17].

Cartilage repair techniques experienced a dramatic improvement over the last years, and to date, newer one-step treatments are available and capable to regenerate a cartilage with hyaline like features. When choosing a surgical strategy, it is mandatory to consider the size and the depth of the lesion, the time elapsed from the possible traumatic event, and the age and the functional needs of the patient. To correct the associated lower limb deformities, or ankle instabilities, if present, is furthermore a key point to obtain a durable and stable result. Longer follow-ups, and possibly randomized controlled trials, will highlight the reliability of these techniques and the possible drawbacks. Moreover, more biological works about mesenchymal stem cells implantation in large OLT and degenerated joints would be welcomed, in order to determine possible wider indications for BMDCT.

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Arthroscopic Debridement of Osteochondral Lesions of the Talus

4

Gian Luigi Canata and Valentina Casale

4.1 Introduction

An osteochondral ankle defect is a lesion of the talar cartilage and subchondral bone mostly caused by a single or multiple traumatic events; this can lead to partial or complete detachment of the fragment.

The consequences are ankle pain associated with weight bearing, impaired function, limited range of motion, stiffness, catching, locking and swelling [1].

These lesions account for more than 6 % of ankle sprains [2]. However, the incidence of these lesions cannot be estimated properly as they can be subclinical, masked by more obvious associated injuries of foot and ankle or because of limitations in conventional radiological investigations [3].

These injuries may occur in any location on the talar dome, even though they have been typically observed posteromedially or anterolaterally.

Lateral osteochondral lesions are often associated with a traumatic event, while medial lesions are more frequently caused by spontaneous events [4].

In fact, in the absence of trauma, ossification defects, abnormal vasculature, emboli or endocrine disorders may account for focal pathologic subchondral fractures of the talar dome [5].

The Berndt and Harty scale is the most widely recognized classification. It is based on radiographic images or intraoperative findings [4] (Table 4.1). In 2001, Scranton and McDermott added to this classification system a further stage corresponding to those cases in which a subchondral cyst formation develops within the talar dome [6].

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G.L. Canata, C.N. van Dijk (eds.), *Cartilage Lesions of the Ankle*,
DOI 10.1007/978-3-662-46332-1_4

Table 4.1 Radiographic classification of osteochondritis dissecans (Berndt and Harty)

Stage I	Small, subchondral compression of the bone
Stage II	Partial detachment of the fragment from the chondral surface
Stage III	Complete fragment detachment with no displacement
Stage IV	Total detachment of fragment with displacement

4.2 Clinical Evaluation and Diagnosis

The presenting signs and symptoms of osteochondral lesions of the talus can be vague and nonspecific, unless a trauma has recently occurred.

Clinically, there can be a low-grade persistent ankle pain after an ankle sprain. The patient may also describe swelling, catching, clicking, locking and, occasionally, giving way.

On physical examination, lateral or medial tenderness can be present, associated with reduced range of motion and instability of ankle joint [7].

The diagnosis of an osteochondral lesion of the talus can be delayed because plain radiographs often appear normal or only slightly altered. The real onset of symptoms ranges between 4 months and 2 years, as the ankle may appear completely normal during the inspection. For this reason, the diagnosis of this kind of injury requires a high index of suspicion [7].

Recent advances in diagnostic and operative arthroscopy have resulted in new techniques that allow for improved management of osteochondral lesions of the talus.

In addition to plain radiographs, more detailed studies include computer tomography (CT) scans, magnetic resonance imaging (MRI) studies and direct arthroscopy of the ankle.

CT is useful in determining the site, size, location, shape and degree of displacement of osteochondral fragments; still it does not give a good visualization of cartilage or bone bruises.

MRI assesses both articular cartilage and subchondral bony lesions, as well as evaluates surrounding soft tissue abnormalities.

MR arthrography has recently been considered a valuable choice for an accurate visualization of cartilage lesions, especially if compared with standard MR imaging. In 2003, Schmid et al. [8] examined both MR arthrography and CT for the evaluation of cartilage lesions in the ankle joint. They outlined CT superiority in the assessment of hyaline cartilage for being more sensitive than MR arthrography (when detecting articular cartilage lesions).

It has been suggested that plain radiographs should be the initial evaluation in patients presenting acute ankle injuries. Any detected lesion should be evaluated with CT to research more details. In cases with persistent ankle pain despite normal plain radiographs, an MRI may be helpful [3].

4.3 Treatment Options

There is a wide variety of management strategies for osteochondral lesions of the talus. They range from nonsurgical to biological repair and cartilage regeneration.

Management decisions should be based on symptoms, on size (>1,5 cm) and on the grade of the lesion [3].

4.3.1 Surgical Treatment

It is reserved for lesions that fail to respond to conservative measures, but it is also appropriate for patients with unstable lesions and mechanical symptoms: in this case, a long period of conservative management is not recommended, as there is little hope for spontaneous improvement.

Surgical options include excision with or without fibrocartilage growth stimulation techniques, such as microfracture, curettage, abrasion or transarticular drilling.

If a fragment is large enough, it could be reattached to the talar dome through retrograde drilling, bone grafting or *internal fixation*, using screws and pins [9], bone pegs [10] or Kirschner wires. The results are fairly good, although this is one of the most invasive techniques.

Other options include cancellous bone grafting and osteochondral transplantation through osteochondral autografts, allografts or cell culture [3].

In 1999, Kumai et al. [11] reported excellent outcomes after arthroscopic drilling in 18 patients. The same year, Taranow et al. [12] first described the retrograde drilling, effective when a subchondral cyst is present or when the lesion is hard to reach with the usual anteromedial and anterolateral portals. Anterograde drilling can also be effective (Figs. 4.1 and 4.2).

In 2002, Schuman et al. [13] reported 82 % good to excellent results in 38 patients after arthroscopic curettage and drilling.

4.3.1.1 Debridement, Microfracture and Drilling

These procedures are reserved for completely detached talar osteochondral lesions that are not amenable by internal fixation.

Human articular cartilage has a limited reparative capability due to its avascularity. To address this problem, arthroscopic techniques have been developed to stimulate bone marrow reparative properties, including drilling and microfractures [14].

These techniques promote the development of a fibrocartilaginous formation over the defect, as frequently occurs in the case of small lesions.

The principal goal consists in breaching the subchondral plate at multiple intervals to stimulate chondroprogenitor cells of the bone marrow into the lesion site; the release of fatty drops from the new fractures provides a clinical indicator that the depth of the microfracture is adequate. This procedure stimulates the formation of fibrin clots and subsequently fibrocartilage (type I collagen) in the defect.

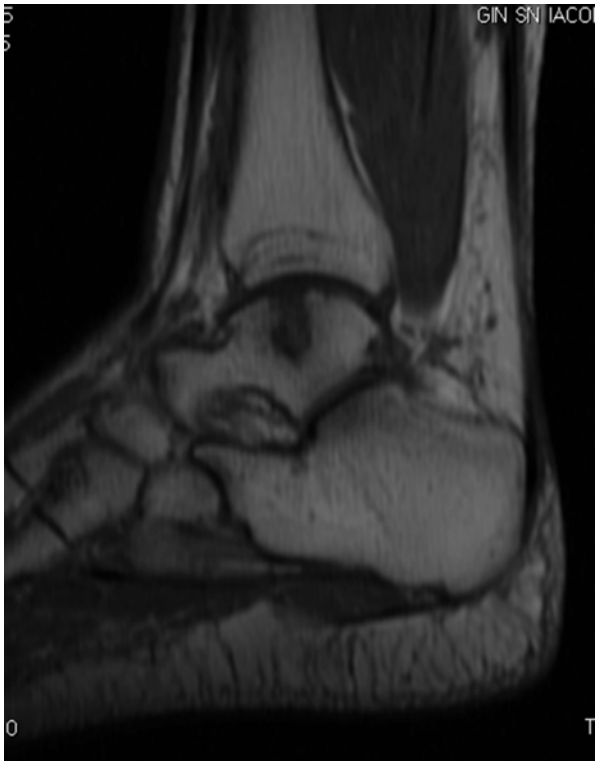

Fig. 4.1 Talar osteochondral lesion with cystic degeneration

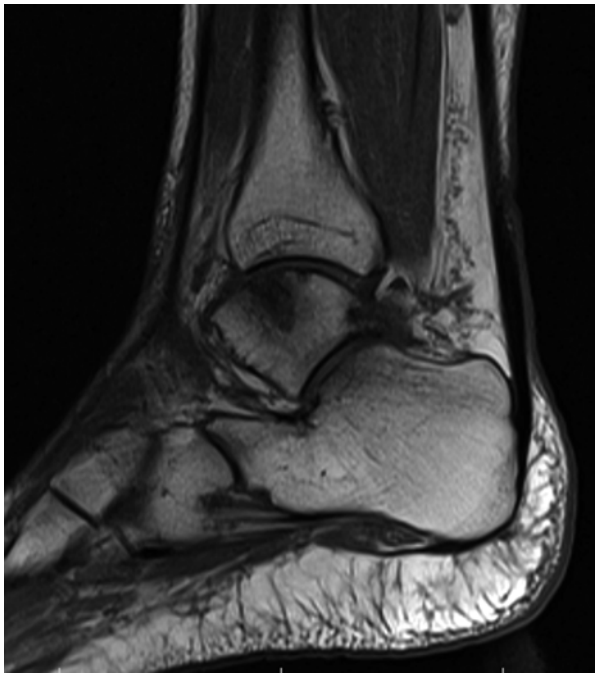

Fig. 4.2 MRI 2 months later. Asymptomatic patient after 6 months (AOFAS score evaluation: 100)

The technique is supplemented by excision and curettage of the damaged tissue for better results [14].

It is well documented that arthroscopic excision, curettage and bone marrow stimulation should be the first treatment of choice for primary osteochondral talar lesions: this procedure is relatively inexpensive; there is low morbidity, a quick recovery and a high success rate [1].

The best indications for bone marrow stimulation techniques are a lesion smaller than 1,5 cm^2 with frayed cartilage, primary surgery, patients younger than 50 years old, a traumatic etiology, lateral lesions and low body mass index.

When advanced fraying of the lesion is present, whether or not the lesion is stable, curettage is indicated and can be eventually combined with penetration of the subchondral bone.

Microfracturing and microdrilling are mini-invasive techniques which require a short period of hospitalization, avoiding risks of thermal damage with easier access to the defects without more invasive steps such as transtibial drilling or osteotomy of the medial malleolus [15].

Drilling, first introduced by Pridie in 1959, was used for decades before microfractures were advocated in the 1990s [16]. These two techniques are thought to be very similar; nevertheless, some differences exist. Chen et al. were the first to compare microfractures (MF) to microdrilling (MD): in particular, they compared acute osteochondral characteristics 24 h after MF or MD procedures in a rabbit model. They found that MF produced a more compact bone around holes than MD did, avoiding marrow cells to reach the new holes and thus limiting the repairing process. On the other hand, MD did not produce the expected heat necrosis: this result was reached using a properly thin drill bit under cooled irrigation [16].

For Takao [17], arthroscopic drilling is less invasive than microfractures. It must be said that no reports of long-term follow-up results have been published yet; therefore, the incidence and degree of complications are not clear.

On the other hand, a disadvantage of the microfracture technique is that the repaired cartilage is fibrous, with inferior mechanical qualities when compared to hyaline cartilage, and it can often degenerate eventually causing osteoarthritis [18].

For this reason, if the cartilage is intact, a retrograde drilling of the subchondral bone without damaging the overlying cartilage can be considered [19].

Retrograde drilling was first described in 1999 by Taranow et al.: it avoids removal of healthy articular cartilage [12]. It has been reported that there is often no difference in functional outcomes between the transmalleolar and retrograde drilling. However, retrograde drilling avoids damage to healthy articular structures [20].

Retrograde drilling can be performed using an anterolateral approach, but there are risks of injuring the talar attachment of the anterior talofibular ligament (ATFL) or mechanoreceptors around the sinus tarsi. The posterolateral approach seems safer.

A disadvantage of this technique is that the surgeon may have difficulty in visualizing the lesion accurately, if it results far from the insertion point. In this respect, a preoperative MRI or CT scan can be important [20].

Concerning microfracturing, attention should be payed to the correct dimensions and placement of the microfracture holes; in fact, there are differences between the use of microfracture awl and the drilling of a Kirschner wire [21]. The flexible Kirschner wire eases drilling at different angles, while the rigid, angled awl gives a better control for creating holes at the desired depth and perpendicular to the surface; nevertheless, awls are difficult to use in posterior lesions.

Arthroscopic debridement combined with drilling stands out as the best treatment for many authors [1].

In 2002, Schuman and Van Dijk [13] compared functional results between two distinct groups of patients, the first one treated with debridement or drilling and the other one treated with a combination of both the techniques.

The results indicated that the second group obtained better scores, and similar outcomes had been reported also by Ogilvie-Harris and Sarrosa in 1999 [22].

In 2003, Robinson et al. sustained that excision and curettage could give equal or even better results compared with those obtained with excision, curettage and drilling [2]. They observed that the drilling technique modifies the normal structure of the medial malleolus; thus, its adoption should be recommended only if strictly necessary.

It is widely assumed in fact that when K-wires are used to drill into hard subchondral bone, they could generate heat and subsequent thermal necrosis of the tissue that should refill the chondral defect [23].

Based on this concept, as described above, in 2009, Chen et al. [16] evaluated acute events at 1 day postoperative in order to observe the state of subchondral bone of a skeletally mature rabbit model after drilling. They demonstrated that microdrilling did not produce apparent heat necrosis.

Among the several techniques available for this kind of lesions, this procedure represents at the moment a valid and effective treatment option (Figs. 4.3 , 4.4, 4.5, 4.6, 4.7 and 4.8).

Doral reported that an additional treatment with intra-articular hyaluronan injections could improve results [24].

4.4 Summary

Good to excellent results can be consistently reached in more than 85 % of patients undergoing arthroscopic debridement, microdrilling and microfracturing [25].

Only symptomatic cases need operative treatment, since minimally symptomatic osteochondral lesions do not appear to progress or worsen over time when treated nonoperatively [26].

Arthroscopic treatment using bone marrow stimulation, debridement and mini-invasive drilling is an effective, less invasive and not expensive strategy to treat small- to medium-sized lesions (less than 2.0 cm^2) [27] (Fig. 4.4).

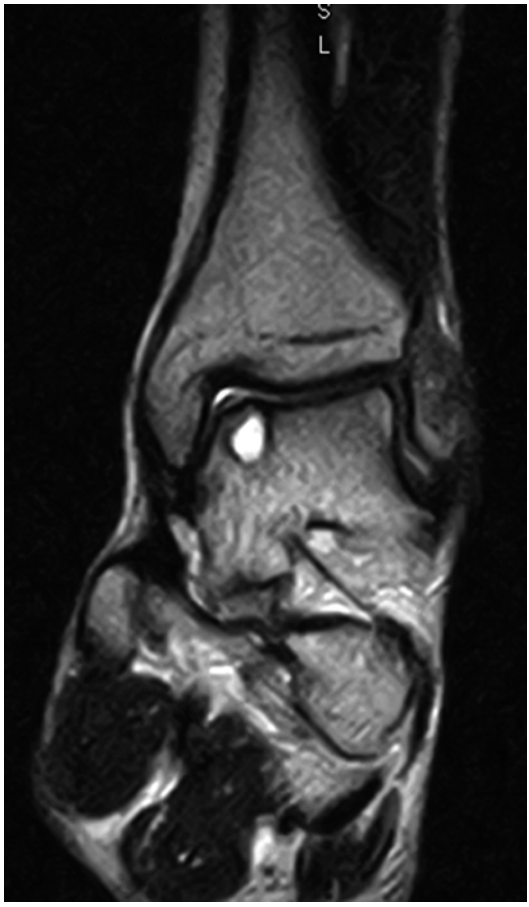

Fig. 4.3 Subchondral cyst in a Stage III osteochondritis

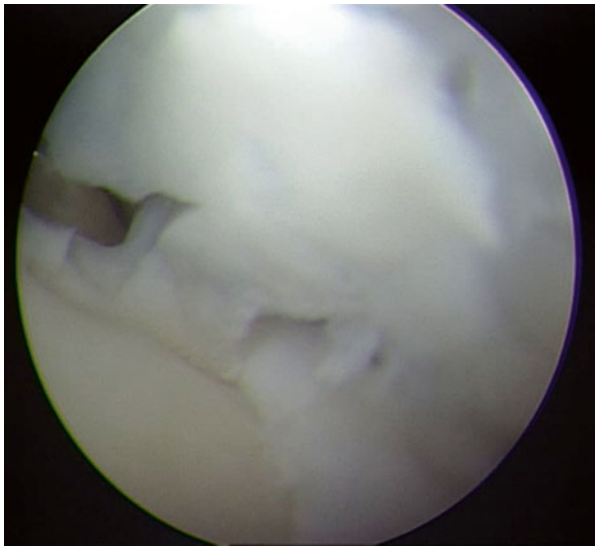

Fig. 4.4 Detached fragment

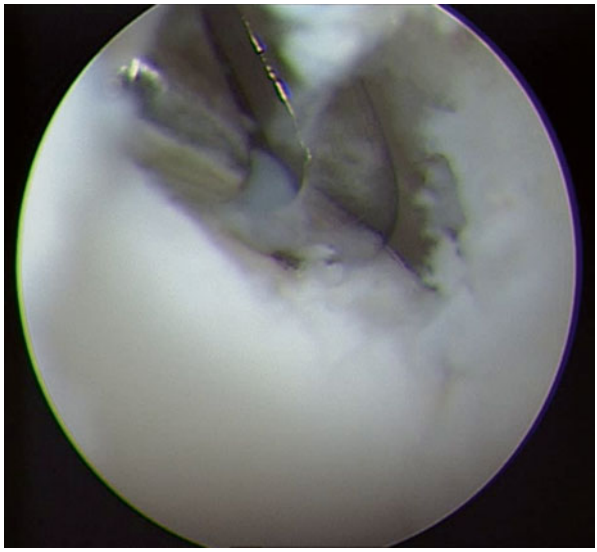

Fig. 4.5 Debridement of the lesion

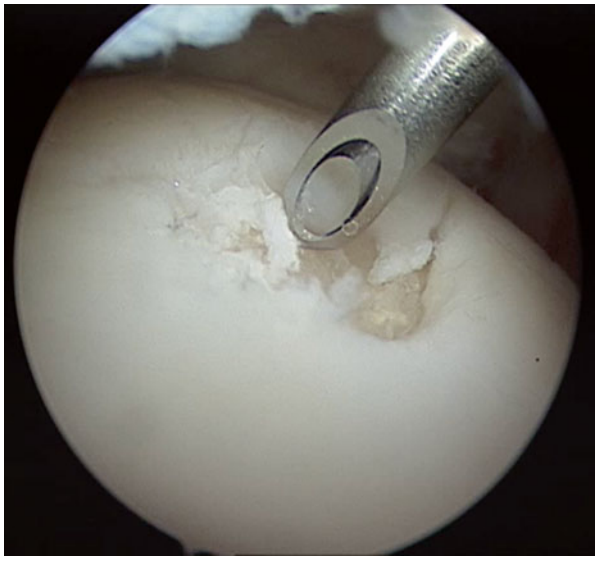

Fig. 4.6 A spinal needle traces the way before a mini-invasive drilling

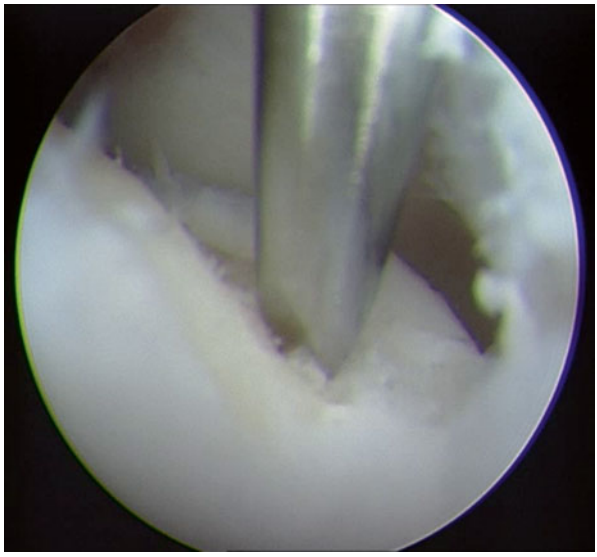

Fig. 4.7 Anterograde drilling

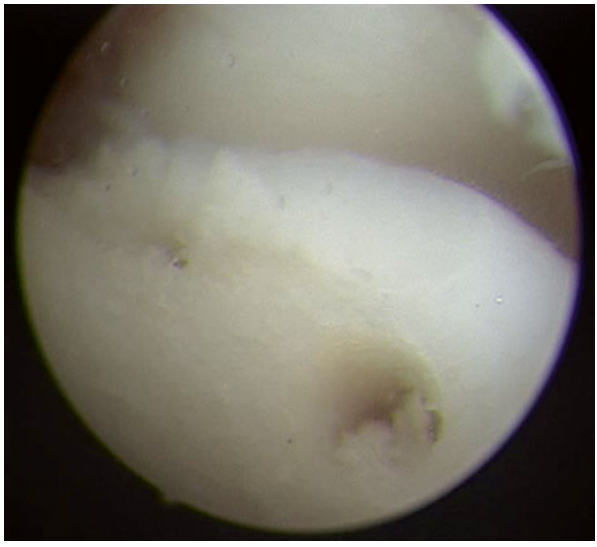

Fig. 4.8 The holes after drilling

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Mosaicplasty of Osteochondral Lesions of the Ankle

5

Károly Pap, Ágnes Berta, and László Hangody

5.1 Historical Background

The presence of cartilaginous bodies in the ankle joint was first reported by Monro in 1856 [1]. Term and nature of osteochondritis dissecans (OCD) were described in classic reports of König and Rendu [2, 3]. Davidson et al., Flick and Gould and Nash and Baker have all discussed the late finding of OCD lesions after an initially diagnosed "sprained ankle" [4–6]. Canale and Bending further emphasised trauma as a causative factor [7]. Lateral lesions cause more symptoms than medial OCDs. Also, lateral lesions have a higher incidence of a previous traumatic event. The head of the talus represents a less frequent location of talar OCD lesions, although recently, this location has also been described [8].

In 1959, Berndt and Harty were the first to mention trauma as the main aetiologic factor of osteochondral ankle defects. They used the term transchondral fracture of the talus to describe the defect and presented a classification system (determined by X-ray appearance) and guidelines for indications for surgery [1]. Since Berndt and Harty's classical paper, the indications for surgical treatment have changed, and nowadays, a large variety of treatment options exist for the different forms of osteochondral ankle defects.

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G.L. Canata, C.N. van Dijk (eds.), *Cartilage Lesions of the Ankle*,
DOI 10.1007/978-3-662-46332-1_5

5.1.1 Introduction

An osteochondral ankle defect is a lesion involving talar articular cartilage and subchondral bone and mostly caused by a single or multiple traumatic event, leading to partial or complete detachment of the osteochondral fragment with or without osteonecrosis.

Many terms are in use, including osteochondral fracture, osteochondral lesion, osteochondritis dissecans, transchondral fracture, flake fracture and intra-articular fracture.

Osteochondral defects can occur in any joint, and the most common location is the knee. Osteochondral defects of the ankle comprise approximately 4 % of the total number of osteochondral defects [9]. It occurs most frequently in 20–30-year-old men [10]. Defects can be found on the medial and lateral sides of the talar dome and are occasionally located centrally.

Ankle sprains are accepted to be the most common cause of osteochondral ankle defects. Ankle injuries are also among the most common injuries seen in sports medicine. It is estimated that one ankle injury per 10.000 people per day occurs [11]. Ankle injuries comprise 45 % of basketball injuries, 25 % of volleyball injuries and 31 % of football injuries [12].

The percentage of osteochondral lesions associated with lateral ankle ligament rupture has been determined by three authors, who routinely inspected the lateral talar dome in a consecutive series of patients operated with lateral ankle ligament rupture. These authors reported 5, 6 and 9 % of lateral talar dome lesions, respectively [13–15]. The percentage of medial dome lesions is unknown, but estimated to be as high as lateral talar dome lesions [14].

After sustaining an ankle sprain, treatment is directed at prevention of future ankle sprains and returning the patient to the previous activity level. However, after standard treatment for acute ankle sprains, residual symptoms are reported in 33–40 % of patients [13]. If symptoms persist after an ankle sprain, the possibility of an osteochondral defect needs to be considered.

5.2 Aetiology

In the ankle, traumatic events are widely accepted as the aetiology of talar osteochondral defects, although not without controversy.

Both trauma and ischemia are probably involved in the pathological process. As not all patients report a history of ankle injury, a subdivision can be made in the aetiology of non-traumatic and traumatic defects.

Idiopathic osteochondral defects have a non-traumatic aetiology, where ischemia, subsequent necrosis and possibly genetics are the aetiologic factors. Osteochondral defects in identical twins and in siblings have been described [16–18]. In 10–25 % of patients, the occurrence of the defect is bilateral [1, 19].

In the aetiology of traumatic osteochondral defects, ankle sprains play the largest role. A severe ankle sprain can cause a small fracture and subsequent impaired

vascularity leading to the formation of an osteochondral defect. Besides, microtrauma caused by repetitive articular cartilage surface loading or excessive stress can lead to cellular degeneration or death by the disruption of collagen fibril ultrastructure and thickening of the subchondral bone [20].

In lateral lesions, trauma is described in 98 % of the cases and in medial lesions in 70 % of the cases [5].

5.3 Mechanism of Injury

When the talus twists inside its box-like housing during an ankle sprain, the cartilage lining can be damaged. It may lead to a bruise and subsequent softening of the cartilage or worse: a crack in the cartilage or delamination. Separation of the cartilage can occur in the upper layer as a result of shearing forces. Alternatively, separation may occur in the subchondral bone giving rise to a subchondral lesion. Fragments can break off and float loose in the ankle joint, or they can remain partially attached and in position. Progression may result in increased joint pressure, leading to forcing synovial fluid into the epiphysis, creating a subchondral cyst. The subchondral cyst and increased joint pressure may prevent healing. The subchondral fracture has no soft tissue attachments and is highly susceptible to subsequent avascular necrosis.

In cadaver ankles, Berndt and Harty were able to reproduce lateral defects by strong inversion of a dorsiflexed ankle [1]. As the foot was inverted, the lateral border of the talar dome was compressed against the surface of the fibula. When the lateral ligament ruptured, avulsion of an osteochondral chip occurred. This chip could be completely detached but remain in place or be displaced by supination. They were able to reproduce a medial lesion by plantar flexing the ankle, slight anterior displacement of the talus upon the tibia and inversion and internal rotation of the talus on the tibia.

Lateral osteochondral lesions are usually located in the anterior third of the talar dome. Medial lesions are mostly located in the posterior half (Fig. 5.1). There are exceptions, however, and anteromedially, posterolaterally and centrally located lesions do occur after trauma. Multiple lesions can also be present. Lateral lesions are typically shallow and wafer-shaped, indicating a shear mechanism of injury (Fig. 5.2). In contrast, medial lesions are generally deep and cup-shaped, indicating a mechanism of torsional impaction. Medial lesions are usually asymmetric, whereas lateral lesions are symmetric. Because of their shape, lateral lesions are more often displaced than medial lesions.

5.4 Clinical Appearance

A differentiation has to be made between acute and chronic conditions. In acute conditions, symptoms of osteochondral ankle defects are similar to those of acute ankle injuries. They include lateral or medial ankle pain, functio laesa and swelling.

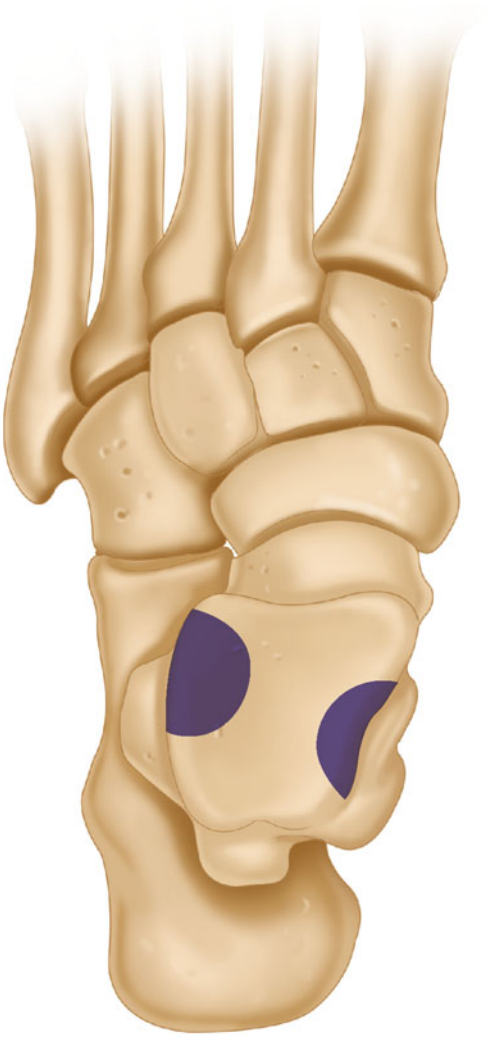

Fig. 5.1 Main locations of osteochondral ankle defects

In patients with an isolated ligamentous ankle injury, these symptoms usually resolve after functional treatment within 2–3 weeks. If symptoms do not resolve after 3–6 weeks, an osteochondral defect of the talus should be suspected. These patients typically present with persisting symptoms and limited range of motion.

Locking and catching are symptoms of a displaced fragment. In most patients with a non-displaced lesion after supination trauma, acute symptoms cannot be distinguished from soft tissue damage.

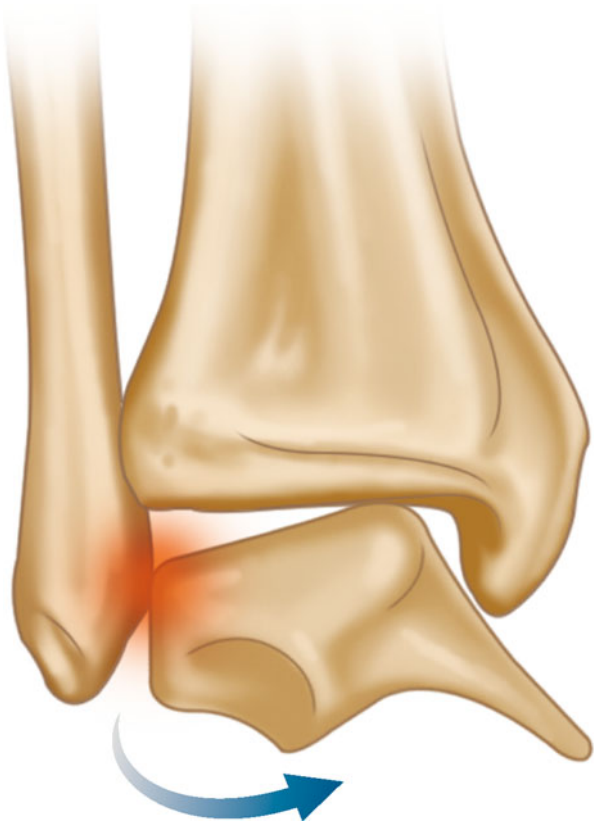

Fig. 5.2 Shear mechanism of injury in lateral osteochondral ankle defects

Chronic lesions classically present as deep lateral or medial ankle pain associated with weight-bearing. Reactive swelling and stiffness can be present, but absence of swelling, locking or catching does not rule out an osteochondral defect. Recognisable pain on palpation is typically not present in these patients. Some patients have a diminished range of motion. See Table 5.1. Differential diagnoses are listed in Table 5.2.

Damaged talar cartilage is responsible for pain during weight-bearing. It is probably the result of edge-loading by the tibia on the cartilage rim of the defect and the subchondral bone underneath. Due to the convex nature of the talus, the edges of the mainly circular defect are more heavily loaded than usual. Part of the healthy cartilage is gone, and the remaining cartilage has to carry the weight transmitted by the tibia. Nerve endings in the subchondral bone in the rim of the defect or underneath the defect are excitated by the increased loading. Purpose of the treatment is to diminish edge-loading, destroying the mechanism that is responsible for increased local hydraulic pressure onto the subchondral area below the defect.

Table 5.1 Possible symptoms

Lateral or medial ankle pain
Functio laesa
Swelling
Locking
Catching
Deep pain on weight-bearing
Stiffness
Diminished range of motion
Typical: no recognisable pain on palpation

Table 5.2 Differential diagnoses

Posttraumatic synovitis
Osteochondral defect of the tibial plafond
Sinus tarsi syndrome
Os trigonum
Ligament laxity
Peritendinitis
Osteoarthritis
Osteoid osteoma
Avascular necrosis of the talus

5.5 Diagnosis

After thorough history taking and physical examination, routine radiographs of the ankle are made consisting of weight-bearing anteroposterior, mortise and lateral views of both ankles.

The radiographs may show an area of detached bone, surrounded by radiolucency (Fig. 5.3). Initially, the damage might be too small to be visualised on routine X-ray. By repeating the images in a later stage, the abnormality sometimes becomes apparent.

A heel-rise view with the ankle in a plantarflexed position may reveal a posteromedial or a posterolateral defect [21]. A bone scan can differentiate between a symptomatic lesion and an asymptomatic lesion. MRI is often used for detection of these lesions. Computed tomography is useful for defining the exact size and location of the lesion and is therefore valuable for preoperative planning (Figs. 5.4 and 5.5).

5.6 Classification and Staging

In 1959, Berndt and Harty suggested a classification system for staging the lesions at the time of surgery based on plain radiographs of the ankle. In grade I, there is local compression of the cartilage and subchondral bone, and usually, there are no radiographic findings. In grade II, there is an avulsion or partial detachment of the

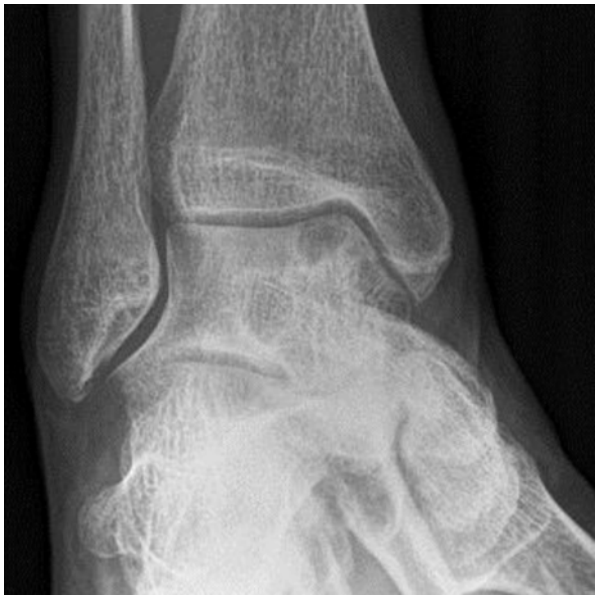

Fig. 5.3 Radiograph: radiolucency of medial talar dome indicating an osteochondral defect

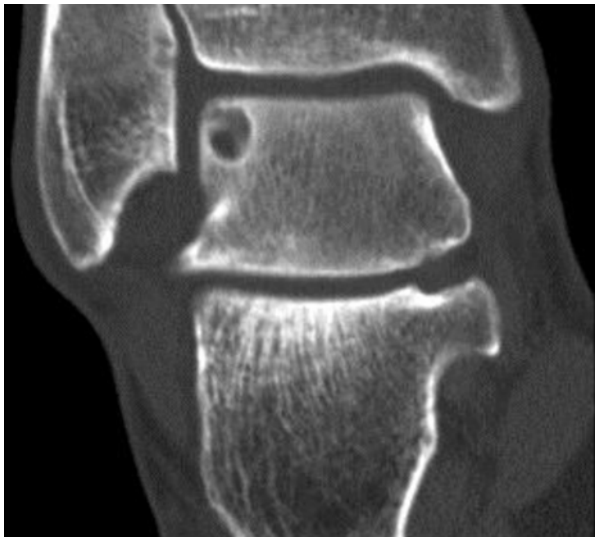

Fig. 5.4 CT scan of a lateral osteochondral defect, coronal reconstruction

osteochondral fragment, but the main part is still attached to the talus. In grade III, there is complete avulsion of an osteochondral fragment, without any displacement. In grade IV, the osteochondral fragment is completely detached and displaced inside the ankle joint (Table 5.3 and Fig. 5.6). Later, classification systems [22] based on CT [23] (Table 5.4), MRI [24] (Table 5.5) and arthroscopic findings were made [25] (Table 5.6). The use of these classification systems is questionable since none of the systems are duly related to the current treatment options [26].

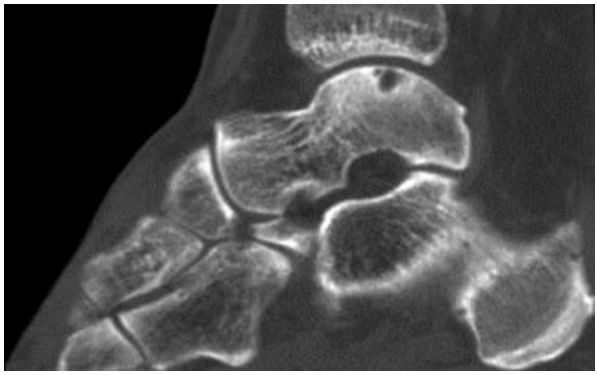

Fig. 5.5 CT scan of a medial osteochondral defect, sagittal reconstruction

Table 5.3 Berndt and Harty (1959)

Stage I	A small compression fracture
Stage II	Incomplete avulsion of a fragment
Stage III	Complete avulsion of a fragment without displacement
Stage IV	Displaced fragment

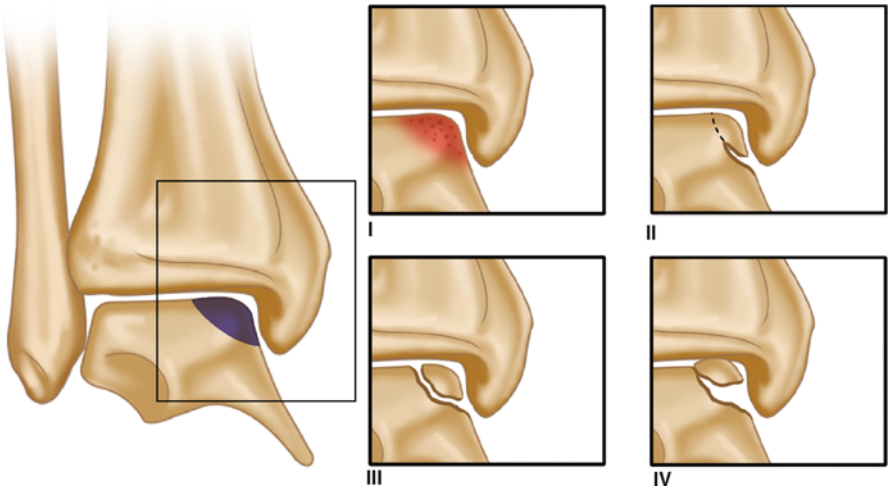

Fig. 5.6 Berndt and Harty classification of osteochondral ankle defects

5.7 Current Treatment Options

There are widely published nonsurgical and surgical methods for treatment of symptomatic osteochondral lesions.

Table 5.4 Talus OCD CT classification of Ferkel and Sgaglione

Stage I	Intact roof/cartilage with cyst lesion beneath
Stage II/A	Cyst lesion with communication to the surface
Stage II/B	Open surface lesion with overlying fragment
Stage III	Non-displaced fragment with lucency underneath
Stage IV	Displaced fragment

Table 5.5 Talus OCD MRI classification of Hepple et al.

Stage I	Articular cartilage injury only
Stage II/A	Cartilage injury with bony fracture and oedema (flap, acute)
Stage II/B	Cartilage injury with bony fracture and without oedema (chronic)
Stage III	Detached, non-displaced bony fragment (fluid rim beneath fragment)
Stage IV	Displaced fragment, uncovered subchondral bone
Stage V	Subchondral cyst present

Table 5.6 Ferkel/Cheng rating: arthroscopic surgical grade based on status of articular cartilage

Grade A	Smooth, intact, but soft cartilage
Grade B	Rough surface
Grade C	Fibrillations/fissures
Grade D	Flap present or bone exposed
Grade E	Loose, undisplaced fragment
Grade F	Displaced fragment

5.7.1 Nonoperative Treatment

This may be rest and/or restriction of (sporting) activities with or without nonsteroidal anti-inflammatory drugs (NSAIDs) or cast immobilisation for at least 3 weeks up to 4 months. The aim is to give the bruised talus a rest to allow the oedema to resolve and to prevent necrosis or for the (partly) detached fragment to become reattached to the surrounding bone.

5.7.2 Operative Treatment

A great variety of surgical treatments have been developed for talar OCD lesions over the years, including lavage, debridement, removal of loose bodies, refixation, abrasion arthroplasty, anterograde and retrograde drilling and bone grafting. Small defects and stable OCD lesions can be treated with minimally invasive arthroscopic procedures (debridement, retrograde drilling, bone grafting, etc.). For larger osteochondral defects and unstable OCD lesions, the ultimate aim would be the long-term replacement and integration of type-specific hyaline cartilage.

Autologous osteochondral transplantation techniques, such as mosaicplasty, are unique procedures that can replace the original surface with hyaline or hyaline-like gliding surface over the affected area. Mosaicplasty allows creating osteochondral autograft substitution by harvesting and transplanting cylindrical osteochondral plugs from the less weight-bearing periphery of the patellofemoral area and inserting them into drilled tunnels in the defective section of cartilage. Use of multiple smaller grafts instead of one large block helps to avoid donor site morbidity and incongruity at the recipient site. Previous experimental trials confirmed the viability of the transplanted hyaline cartilage and fibrocartilage repair of the donor sites [27]. Hyaline cartilage of the donor area is certainly different from talar hyaline cartilage; however, to date, this fact has not been proven to have a negative influence on long-term results.

5.8 Preoperative Considerations

Talar OCD lesions usually reside on the lateral and medial central aspects of the talar dome where the talus accepts the majority of weight-bearing and torsional loads; the caput tali may also be a less frequent location. Preoperative X-rays, bone scan, CAT scan and MRI offer important information about the size and location of the lesion, the blood supply of the talus and any other associated pathology.

Essential aspect of the procedure is insertion of the osteochondral plugs perpendicular to the recipient site. The constrained configuration of the talocrural joint with its highly contoured articular surfaces can make access to these lesions difficult. The recommended approach is through a miniarthrotomy, associated with malleolar osteotomy in some cases. The grafts are usually obtained from the medial femoral ridge and sometimes from the lateral femoral ridge of the ipsilateral knee. These sites are considered to be low weight-bearing surfaces, and the quality of the hyaline cartilage coverage is appropriate for talar requirements. Use of the knee as a donor site may be precluded on the basis of arthritis, patellofemoral symptoms or decreased range of motion. In such cases, use of small-sized (2.7 or 3.5 mm in diameter) autogenous grafts from the anterior talus can be considered.

Although X-ray evaluation, CT scan or MRI can help to determine the extent of the lesion, indication of mosaicplasty is only determined after excision of the defect. Usually the patient is prepared for a mosaicplasty based on X-ray and MRI findings, but the final decision is made during arthroscopy. Based on the history, physical examination and imaging studies, the patient can be advised of the nature of the proposed surgery: whether an osteotomy may be necessary and the projected postoperative course including the duration of non-weight-bearing status. Caution should be exercised in offering mosaicplasty to:

1. Patients over the age of 50
2. Patients who had multiple previous surgeries
3. Patients, regardless of age or previous surgical history, who have of pan-articular arthritis or articular cartilage thinning

5.9 Operative Technique

Under general or spinal anaesthesia, the affected lower extremity is prepared from the upper thigh to toes and the thigh tourniquet is set to 100 mmHg above systolic pressure. The location, size and surgical grade of the lesion are further defined by arthroscopy, and final determination of the surgical treatment course is made during this phase. The ideal findings for mosaicplasty are:

1. ≅10 mm diameter focal osteochondral lesion
2. On the medial or lateral dome
3. Detached osteochondral fragments
4. Otherwise normal articular surfaces of the tibia and talus

Osteoarthritis of the ankle is a contraindication. However, anterior talar and tibial osteophytes, as seen in jumping athletes, do not exclude mosaicplasty. In fact, their removal is an integral part of the operation for establishing better postoperative motion.

For medial lesions, a medial malleolar osteotomy is usually required. To ensure that adequate exposure is made, the line of osteotomy must be made at the junction of the medial plafond. If the lesion is large and central, rotating the ankle into valgus might become necessary (Figs. 5.7 and 5.8). Use of a Steinmann pin may help to achieve eversion of the talus.

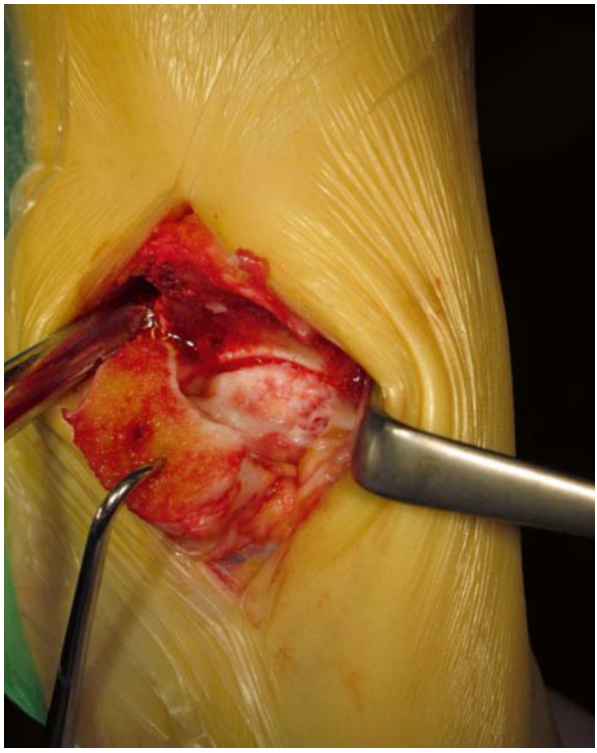

Fig. 5.7 Osteochondral lesion is exposed

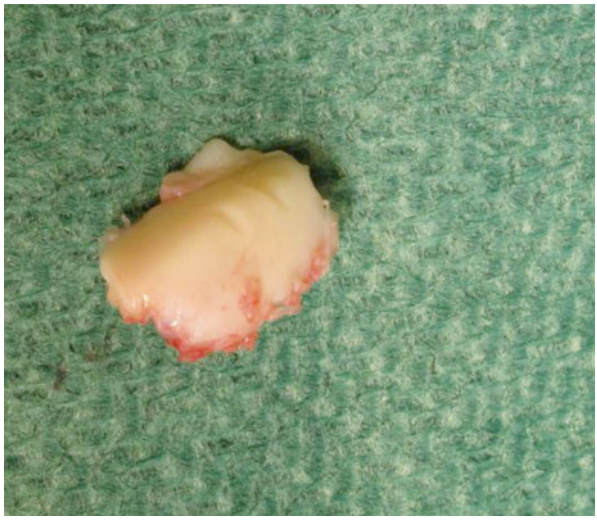

Fig. 5.8 The removed injured osteochondral defect

Once the lesion is exposed, damaged cartilage tissue is removed by curette and knife blade to a sharply defined rim. This vertical rim has the advantage of optimal load sharing between recipient site and transplants. The currently used mosaicplasty instruments (Mosaicplasty™ Complete Instrumentation, Smith & Nephew Inc., Andover, MA) have tools for precisely measuring the intended number and diameter of grafts and the depth of the recipient holes. Size and location of the intended drill holes are etched on the base of the surface. In the talus, the usual size of the drill holes is 6.5 and 4.5 mm in diameter. Smaller sizes – such as 3.5 mm in diameter – can be used to fill the spaces between the previously implanted grafts. These graft sizes allow for contouring and rotating the grafts for the desired surface confluence.

Upon completion of the recipient site preparation, the osteochondral grafts are obtained from the ipsilateral knee. The primary harvest site is the medial upper part of the medial femoral condyle. As a less frequent option, the lateral supracondylar ridge can also be used through a 15–20 mm miniarthrotomy. By flexing the knee through 0–100°, 3–4 plugs can be obtained. The grafts are procured with a double-edged tubular cutting chisels to ensure the precise diameter and length of the grafts. Upon removal of the grafts from the chisels, there will be an anticipated 0.1–0.2 mm expansion in their diameter, a characteristic which helps with the press-fit fixation. Each graft length is recorded. At the end of the graft harvesting, a suction drain is inserted into the knee joint.

After graft harvest, the recipient site is re-evaluated. All accumulated clot and bone debris are lavaged from the base of the lesion and the holes. The first drill hole is made through the tubular drill guide, which also serves as the delivery tube. The depth should be 3–4 mm deeper than the length of the selected plug. At this stage, the first hole is enlarged by 0.1–0.2 mm with the conical dilator. Dilation of the recipient tunnel allows an easy insertion of the graft – without too much insertion force on the hyaline cap of the osteochondral cylinder. It is important to note that dilation of the next recipient hole will impact the surrounding bone to the previously

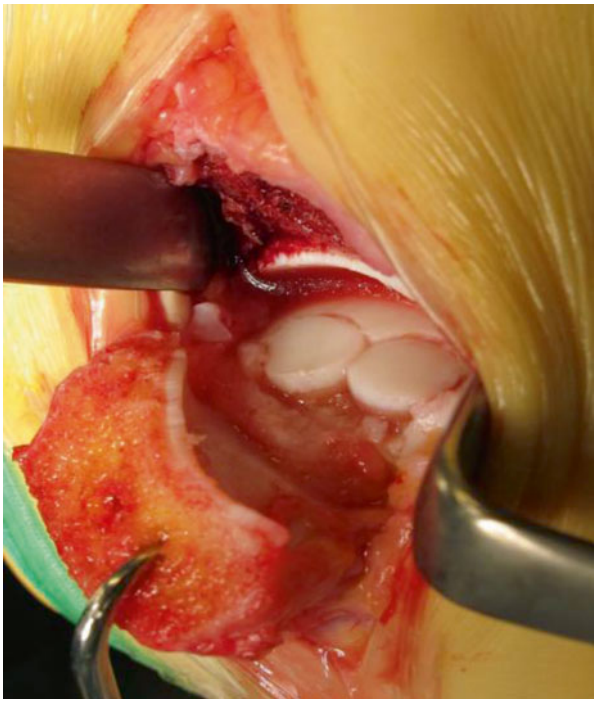

Fig. 5.9 Impacted grafts

implanted graft, resulting in a safe press-fit fixation of the cylinders (Fig. 5.9). The grafts are inserted with the windowed delivery tube. The tube provides protection and visualisation of the graft as it is gently tamped into the recipient area. Judicious use of the tube and its tamp obviates inadvertent recessing of the graft. The resurfacement is a step-by-step procedure, and the same steps are repeated during the implantations. Accordingly, the combination of drilling, dilation and delivery are done for each graft. After the entire set of grafts is implanted (Fig. 5.10), the ankle is lavaged, observed for loose bodies and moved through its range of motion to ensure congruency of the resurfaced area. The osteotomy is fixed with two malleolar screws inserted through predrilled holes.

Lateral OCD lesions most frequently occur in the anterolateral surface of the dome. In most cases, these lesions can be reached through a vertical anterior lateral arthrotomy. Then by rolling the ankle through flexion and extension, perpendicular insertion of the grafts can be performed. For large lesions, which extend posteriorly, Gautier and Jakob have promoted a lateral malleolar osteotomy [8]. We recommend exposure of these large defects through an anterior fibular periosteal flap containing the origin of the anterior talofibular ligament and, if necessary, the calcaneofibular ligament [28]. The talus can then be drawn forwards and rotated downwards with the help of a K-wire driven through the body of the talus. Careful graft harvest and/or precise implantation technique can help to achieve perfect contouring of the talar dome. OCD defects combined with subchondral cystic lesions can also be treated by fine technical modifications.

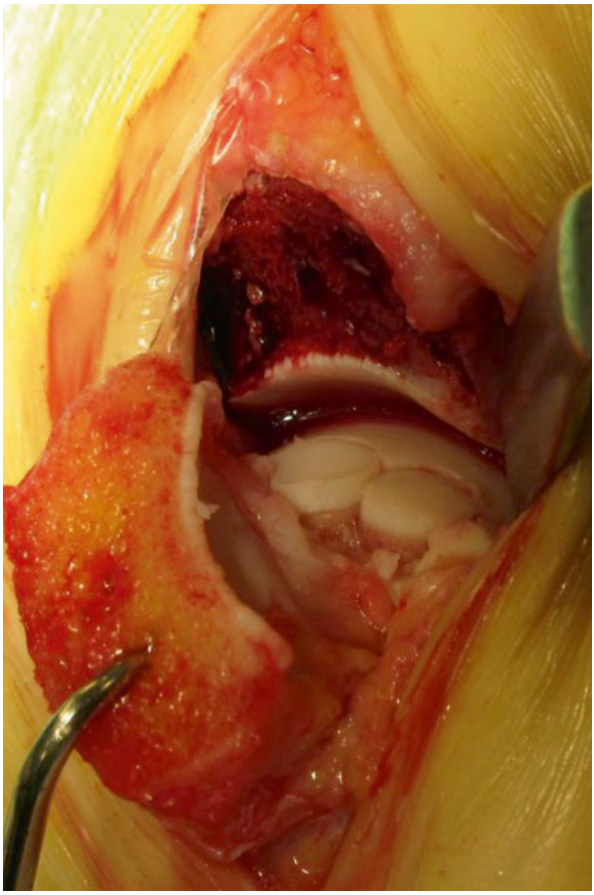

Fig. 5.10 Final levels of the grafts with good congruency

At the end of the procedure, the tourniquet is released, bleeding is controlled and a well-padded compression dressing is applied. The patient is observed for 24 h to administer iv antibiotics and control pain, and the operated extremity should be elevated. Knee drain is removed after 24 h. Promotion of active range of motion and progressive weight-bearing are the most important postoperative goals.

5.10 Rehabilitation

Non-weight-bearing for 3 weeks and partial loading with 30 kg for 3 weeks are the standard protocol during rehabilitation. Patient education and cooperation are essential for the optimal outcome. Use of removable foot and ankle orthosis aids providing controlled range of motion, protected weight-bearing and patient comfort.

Patients are kept non-weight-bearing for 3–6 weeks. If an osteotomy was not performed, patients are kept non-weight-bearing for 3 weeks, and following medial

malleolar osteotomy, patients are kept non-weight-bearing for 6 weeks. Afterwards, partial weight-bearing with 30 kg for 3 weeks is allowed, to promote integration of the grafts [29]. A foot and ankle orthosis may improve comfort. A full range of motion exercises is encouraged. Unprotected weight-bearing is allowed at 6 weeks. Athletic activities may begin at approximately 6 months, depending on the postoperative assessment.

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Management of Cystic Osteochondral Lesions of the Talus

6

Graham McCollum

6.1 Introduction

Osteochondral lesions of the talus can be difficult clinical problems to treat. Their size and severity range from small asymptomatic incidental lesions to massive cysts, which can threaten the structure of the talus. Treatment strategies range from monitoring and watching the lesions in asymptomatic individuals, marrow stimulation, cartilage transplantation and bone grafting to bulk osteochondral allograft, and when osteoarthritis is advanced, arthrodesis and arthroplasty.

The majority of lesions are trauma related. Ankle sprains are very common, affecting 28,000 individuals in the United States every day, and in many sports it is the most commonly injured joint [1]. About 50 % of these will have some form of chondral injury and in up to 70 % of ankle fractures [2]. A smaller percentage of lesions do not have a history of trauma and have metabolic, genetic or local ischaemic aetiologies [3].

Reliable repair techniques are important to reduce pain and return patients to function.

6.2 Pathophysiology of Cystic Osteochondral Lesions

The progression of a chondral or osteochondral lesion to a cyst is not completely understood. Some lesions remain inert and do not progress, whereas others continue to expand, are painful and erode the subchondral bone. One theory postulates that at the initial trauma (sprain or fracture) there is a break in the subchondral bone plate and local bone bruising. The ankle is the most congruent joint in the body, and its cartilage is unique in its inelasticity and low deformability [4]. High hydrostatic

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G.L. Canata, C.N. van Dijk (eds.), *Cartilage Lesions of the Ankle*,
DOI 10.1007/978-3-662-46332-1_6

pressures are generated with weight bearing, forcing intra-cartilage water through the proteoglycan matrix to the point of least resistance, which is the defect in the subchondral bone plate. Cartilage water is a dialysate of synovial fluid and is forced under pressure into the subchondral bone resulting in local osteonecrosis, acidosis and macrophage-mediated osteoclast activation. Cartilage not supported by the subchondral bone changes, losing proteoglycans and glycoprotein, and water flows more freely into the evolving cyst. Remaining intact cartilage can act as a ball valve limiting egress of fluid out the bone when not weight bearing and compound the increasing hydrostatic pressure. Articular cartilage is aneural, but the underlying subchondral bone is maximally innervated by nocireceptors in the immediate subchondral bone. Macrophage-mediated local acidosis and intraosseous hypertension excite these receptors and lead to the typical deep ankle pain associated with cystic lesions. Synovitis, which can increase the intra-articular pressure and cause pain, is not typical with OCLs unless there is instability or a loose body.

This does not explain all cysts, as some are distant to the articular surface. Mucinous degeneration or local avascular necrosis may explain some of these lesions. Corticosteroid-induced local ischaemia, metabolic factors and genetic predisposition make up a minority of cases and can lead to massive cysts. Deep medial lesions are more likely to have a non-traumatic aetiology compared to lateral lesion, which almost exclusively have a trauma history [5, 6].

When bone bruising is distant from the articular surface (reticular), the subchondral bone plate is not usually threatened and uneventful healing without an OCL is usual. Adjacent (geographical) bone bruising may cause softening of the subchondral bone and together with a break in the subchondral bone plate progress to a cystic OCL [7] (Fig. 6.1).

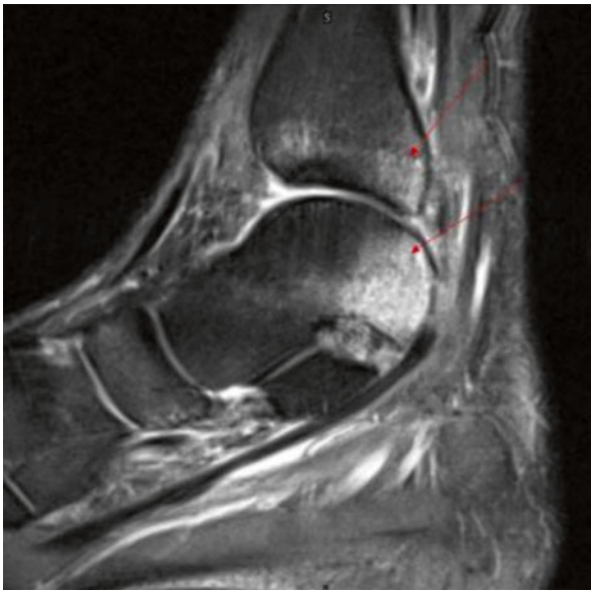

Fig. 6.1 Reticular bone bruising, distant from the articular surface. *Top arrow* indicates bruising of the distal tibia and the *lower red arrow* indicates bruising of the talus

Mechanical malalignment overloads either the medial or lateral boarders of the talus depending if there is tibial or hindfoot varus or valgus [8]. This may affect healing of a defect or retard surgical repair. OCLs in the knee and the ankle have been shown to regress with high tibial realignment osteotomy or calcaneal osteotomies [9]. Similarly, ankle fracture malunion increases the contact pressures [10]. Ankle instability repetitively increases focal load and OLT propagation [11]. This should be addressed in the treatment plan when applicable (Fig. 6.2).

Some lesions enlarge and become massive (>3 $cm^{2)}$ (Fig. 6.3) [12]. These are either central and contained or peripheral and uncontained. Sometimes the subtalar joint is involved. Although OLTs are slowly progressive or static and do not commonly lead to osteoarthritis, when large, point loading may lead to kissing lesions, involvement of the tibial plafond and secondary arthritis [13].

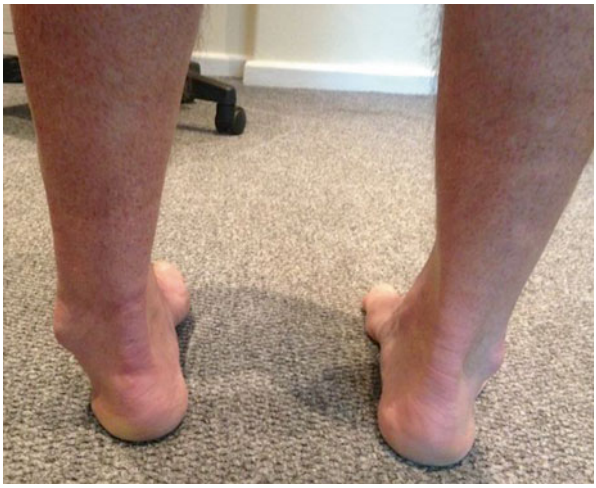

Fig. 6.2 Varus malalignment

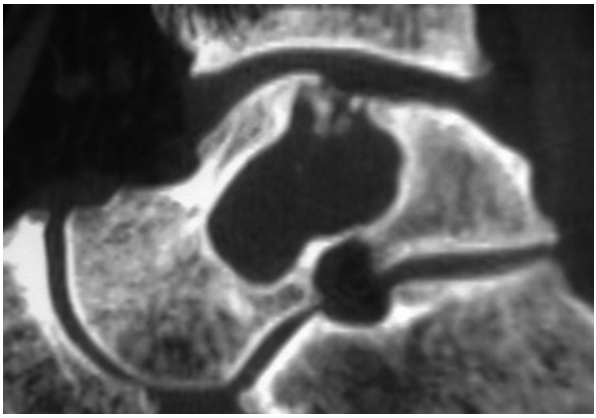

Fig. 6.3 Massive (>3 mm) cystic lesion

6.3 Clinical Presentation

Large uncontained lesions are usually painful as the structure of the talus is threatened. Point loading and loose bodies cause a synovitis, and the mechanics of the joint are altered. Some smaller cystic lesions are incidental findings, remain stable and quiescent and do not require treatment, but should be monitored with serial radiology [14]. Pain from symptomatic OCLs is typically deep, activity related with little swelling unless there is a loose body or cartilage flap. Some describe clicking or other mechanical symptoms in the joint. The majority of patients report a previous ankle sprain or multiple episodes of instability.

6.4 Investigation

Large cystic OTLs are usually visible on plain radiographs, but smaller central lesions can be difficult to see or are not visible (Fig. 6.4). MRI best illustrates the cartilage and surrounding bone oedema, but a CT scan is essential to understand the anatomy of the lesion, see if the cyst is contained and determine the involvement of the ankle and the subtalar joints. A plantarflexion CT is very helpful to plan the surgical approach and the need for an osteotomy in the case of an open procedure [15].

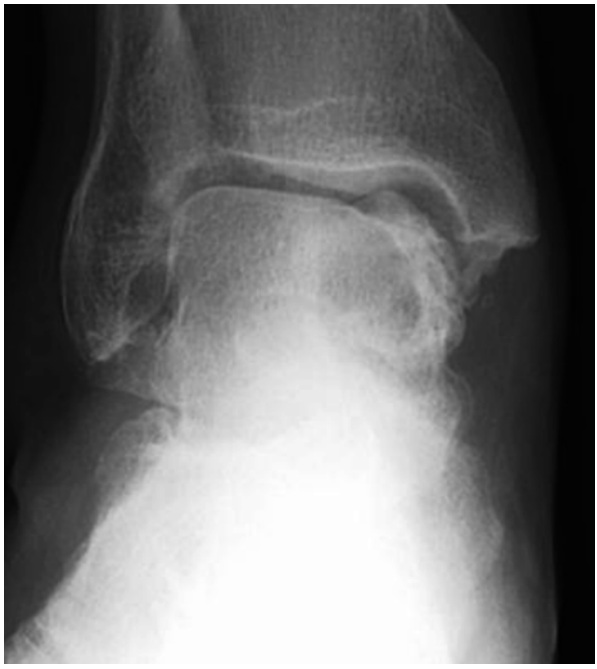

Fig. 6.4 Large cystic lesion on plain radiograph

6.5 Nonoperative Treatment

Rest, restricted sporting activities and casting aim to unload the cartilage and the subchondral bone allowing healing. The success rate, based on a few studies from two decades ago, is low, ranging from 29 to 58 % [16]. In the paediatric population, healing was thought to be superior [17], but a recent study showed that 92 % of initially conservatively treated children required surgery eventually [18]. If the cystic component is large and there is significant reactive bone oedema, nonsurgical treatment is less likely to be successful [3]. Rarely, a large cyst is identified in a relatively asymptomatic individual. This poses a treatment dilemma as an enlarging cyst can threaten the talar structure and be very difficult to manage once they do become symptomatic or the subtalar joint is threatened [19].

6.6 Operative Treatment

The surgical strategies include bone marrow stimulation, bone grafting, tissue transplantation (autologous or allograft), chondrocyte implantation and prosthesis capping. Salvage options in the case of advanced disease with secondary arthritis include arthrodesis and arthroplasty. Cystic lesions where the cartilage layer over the cyst is well supported and remains intact and attached can be treated with retrograde drilling or retrograde bone grafting [20].

The majority of cystic lesions having primary surgery are <2 cm^2, and marrow stimulation techniques should be the first line of surgical treatment. If the cyst is larger than this, is uncontained or is a complex revision case, then another strategy should be attempted, either cartilage transplantation or bone grafting. Some authors have reported good results following repeat marrow stimulation for failed primary surgery, but the success rate is inferior if the lesion is cystic [21].

6.6.1 Arthroscopic Bone Marrow Stimulation

This technique involves breaching the subchondral bone plate multiple times allowing pluripotential stem cells from the resultant bleeding to collect in the lesion (Fig. 6.5). Under the influence of cytokines and growth factors, they differentiate into chondrocyte type cells and produce fibrocartilage containing predominantly type I collagen. Cystic lesions require curettage and penetration of the calcified wall of the cyst. This tissue is mechanically inferior, but the results of the procedure in the short term are generally good with smaller lesion (<1.5 cm^2) [16, 22]. The procedure has a low morbidity and complication rate and low cost and is technically undemanding. Gobbi et al. performed a randomised study of 30 patients comparing chondroplasty with microfracture and autologous osteochondral transplantation [23]. At a mean of 53 months, there was no difference in AOFAS scores and single assessment numeric rating. Lee showed good results selecting lesions <1.5 mm^2 in patients younger than 50 years. Other authors have found age not to affect the clinical outcome [24]. Short- to medium-term results of marrow stimulation have generally been good [25].

Fig. 6.5 Marrow stimulation with microfracture

Of concern, fibrocartilage has inferior mechanical properties compared to hyaline cartilage, and there is a limit to the size of lesion that will fill with repair tissue. Although Van Bergen et al. showed 74 % good to excellent results at 8–20-year follow-up [26], Ferkel et al. showed a 35 % decline in clinical function over 5 years and a grade of worsening of ankle arthritis [27]. Lee et al. showed at relook arthroscopy in 20 patients that 40 % of lesions were incompletely healed with varying degrees of fibrillation and deterioration in the International Cartilage Repair System (ICRS) grading [28]. Shoulder lesions that are not contained have worse clinical results [29]. There is concern that reparative strategies may be a temporary fix, and we are learning more about the limitations of the procedure.

When appropriate, retrograde drilling allows stimulation of the marrow surrounding the cyst and perforation of the cyst wall without disrupting the intact overlying cartilage. Results seem generally favourable with 88 % of patients treated successfully [30, 31]. This was shown to have superior results compared to transmalleolar drilling by Kono et al. [20] leaving the corresponding plafond cartilage intact.

6.6.2 Excision Curettage and Bone Grafting

In this technique autologous cancellous bone is placed in the cyst after curettage and removal of the sclerotic boarder. The aim is to restore the weight-bearing area of the talus in large lesions and to obtain a fibrocartilage-covering surface. If the overlying cartilage is intact, a part can be lifted creating a trapdoor for curettage and placement of the graft. Autograft is usually harvested from the iliac crest or the ipsilateral tibia. Results of this technique are mixed. Kolker et al. showed on a 46 % failure rate in their small series and cautioned the use of the technique [32]. Contrasting this, Draper et al. showed better clinical results with curettage and bone grafting compared to patients just receiving curettage and microfracture [33]. Patients in the microfracture group had smaller lesions.

6.6.3 Osteochondral Autograft Transfer

Osteochondral autograft transfer involves harvesting single or multiple cylindrical cartilage and subchondral bone grafts from the non-weight-bearing part of the ipsilateral knee and transplanting them into the talar defect after preparation. This technique is not possible when the defect is very large (>3 cm^3), as it requires mechanical stability from a press fit of the graft into the talar defect. Shoulder lesions are also more difficult for the above reasons.

Contained talar cysts can be removed and the defect filled with the graft. It is indicated in revision cases when marrow stimulation has not been successful or for primary surgery in cases of contained cystic lesions [16, 34]. The procedure is technically demanding and often requires an osteotomy of the medial malleolus, the fibula or the plafond for access to insert the cylindrical grafts orthogonal to the talus. Congruence of the talar surface is paramount and can be difficult to achieve on the shoulders. Cadaver studies have confirmed increases in contact pressure of the graft if set elevated or increased local pressure on the surrounding cartilage if the graft is recessed too deep [35, 36]. The number of cylinders implanted does not seem to affect the clinical result at 5 years as long as the lesion is well covered by the graft [37].

Cartilage from the knee is thicker than talar cartilage, and the properties of knee cartilage differ from that of the talus. The osseous part of the graft has to sit lower than the bone level in the talus to achieve a flush surface. Donor site knee pain is a concern: Hangody et al. in the largest series of talar mosaicplasty (98) and over 1,000 mosaicplasty cases in other joints had an incidence of long-term knee pain in 3 % of cases [38]. Similarly Kennedy et al. reported an incidence of 4 % [39] but up to 16 % in another series [40]. The number of grafts harvested does not seem to influence the clinical result, but body mass index (BMI) has a negative influence [25]. The procedure provides good outcomes in the short to medium term in upward of 85 % of patients [16]. Replacing the defect with bone and hyaline cartilage and achieving good integration potentially are long-lasting solutions and superior to repeat arthroscopy in cases of failed marrow stimulation [41, 42]. Scranton et al. had a 90 % good to excellent clinical result at a mean of 36 months for cystic, type 5 lesions [43].

The critical size of lesion that dictates whether replacement or repair is the best long-term treatment is yet to be determined. Lesions greater than 8 mm that are more cystic and cases where primary repair has failed are the usual indications [39].

6.6.4 Osteochondral Allograft Transplantation

This replacement procedure transplants viable cartilage and subchondral bone from a fresh cadaver talus to a recipient with a large cystic defect. Some of the advantages of the procedure include the following: (1) It has the ability to match the shoulder of the recipient talus with careful harvesting for uncontained lesions. (2) It can fill massive (>3 cm^2) defects that are not amenable to autograft techniques [12, 13]. (3) Tibial or fibula osteotomy is often not necessary for access as the graft can be put in

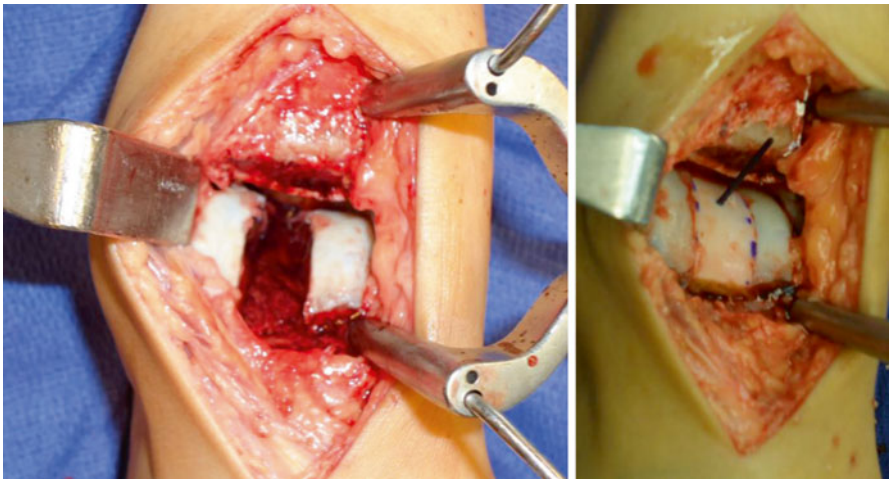

Fig. 6.6 Intra-operative picture of cyst removal and after implantation of the prepared allograft

from the anterior approach—one does not have to be orthogonal to the talus as with mosaicplasty or osteochondral autograft transplant [3].

Fresh allograft is preferred over fresh frozen or cryopreserved as chondrocyte activity is optimal [44]. Implantation should take place as soon after harvesting and screening as possible to preserve the living chondrocyte count. This decreases over time to 70 % at 28 days even if the graft is stored in a temperature-controlled environment of 2–4 °C [45].

After excision of the diseased cystic talus, the donor talus is fashioned to fit the defect perfectly and secured with bioabsorbable or recessed permanent screws or pegs (Fig. 6.6).

Although this treatment has its indications and advantages, there is little supporting evidence for the technique. Raikin et al. reported his results of 15 patients at 2-year follow-up with an improvement in the mean AOFAS scores from 38 to 83 and an improvement in pain scores [12]. There were two failures reported (13 %) requiring ankle arthrodesis, and there tended to be radiographic deterioration over time. Urgery et al. showed similar clinical improvement in 38 patients at a mean follow-up time of 37 months. 4 (11 %) patients required ankle arthrodesis for graft failure and arthritis. They performed MRI scans in 15 patients at a mean of 39 months post surgery showing poor graft incorporation and instability in 5 cases with graft-host boarder signal intensity. Ten showed fair to good graft incorporation, and one demonstrated graft subsidence [46].

This joint-preserving procedure should be used in select cases not amenable to marrow stimulation or autograft procedures. It can relieve pain, improve function and fill massive defects prolonging joint function.

6.6.5 Autologous Chondrocyte Implantation

In the quest to replace osteochondral defects with hyaline cartilage, Brittberg et al. first treated lesions in the knee with chondrocytes harvested from non-weight-bearing parts of the knee, cultured and then placed in the defect, covered with a periosteal flap from the ipsilateral tibia [47]. Another second-generation technique has been developed, eliminating the need for periosteal flaps culturing the chondrocytes on a matrix of porcine collagen membrane or hyaluronic acid-based membrane [48]. This can then be placed into the lesion with or without addition of cancellous bone graft and can be performed arthroscopically. Cell distribution is more evenly distributed within the matrix, and dedifferentiation is reduced [49].

Evidence for the technique is sparse in the ankle compared to the knee. Apart from one meta-analysis [50], there are no other level 1 evidence studies. Niemeyer's analysis identified 16 retrospective studies and 213 cases. The overall clinical success was 89 %, but there were nine different clinical scoring systems used, and the mean study sample size was small at 9 [50]. Giannini et al. in the largest series to date involving 46 patients followed up for 36 months showed improvement in AOFAS scores from 57.2 to 89.5. Three patients had a second-look arthroscopy and biopsy. Macroscopically and histologically the repair tissue was similar to hyaline cartilage [51]. MRI T2 mapping at 24 months post surgery has shown cartilage similar to a normal control group [52]. Both of these studies did not look at large cystic lesions; they were smaller, contained lesions

Results look promising but evidence is lacking compared to research in cartilage repair in the knee. Major limitations of the technique are the need for a second procedure after harvesting cartilage and costs of the procedure. The limit to the size of defect that can be filled by the matrix has not been determined. In these large cystic lesions, there is primarily a bone defect, which this technique will not be able to address.

6.6.6 Metal Resurfacing Implant

Donor site morbidity, availability of allograft and cost of chondrocyte culture can be prohibitive. For these reasons, a modular metallic implant has been developed as an alternative to fill cysts and resurface cartilage defects. Moderate to large medial cystic lesions that have failed previous marrow stimulation techniques are ideal cases for the procedure. Cadaver studies have shown that an implant recessed 0.5 mm reduced the contact pressure under loads of 1,000–2,000 N to less than 0.1 % without loading the surrounding normal cartilage. This is favourable for the longevity of the implant and the corresponding tibial plafond [53]. Evidence to support the technique is limited to a case report [15] and a small series of 20 patients followed up for between 2 and 5 years [54]. The authors report mean improvement of AOFAS scores from 62 to 87 and improvement in numeric rating scale (NRS) for

pain during sitting, walking, running and climbing stairs. There were no cases of loosening or subsidence of the implant, and 12 patients returned to pre-symptom level of sport participation. Five patients required removal of medial malleolar screws, but there were no reported problems with the medial malleolar osteotomy except for one symptom free tibial subchondral cyst. The mean size of the lesion was 15 mm (11–20 mm).

Although an attractive alternative with reports of good short- to medium-term results, further study will be necessary to assess longevity. The technique is limited by the size of the lesion. Although partially modular, the size of the cap is 15 mm in diameter. Larger cystic lesions may only be partially covered by the implant, and conversely with lesions smaller than 15 mm, normal surrounding cartilage is sacrificed. In massive cystic lesions fixation may not be optimal.

Conclusion

Ankle cartilage defects and the clinical implications thereof are increasingly being recognised. As populations continue to participate in sporting activities and remain active, ankle injuries are increasingly common. Recognition by physicians and surgeons with access to better imaging is also identifying an increasing number of cartilage and subchondral bone defects.

Great advances have taken place over the last three decades in the quest to repair or regenerate ankle cartilage, but the surgical choice remains controversial, and high-level evidence is lacking for many techniques. Further research with prospective trials is necessary to provide us with fixed guidelines.

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Osteochondral Ankle Injuries in Footballers

7

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7.1 Introduction

The Catalan Soccer Federation is a sports association in the autonomous region of Catalonia, Spain, with more than 140,000 members. Approximately 22,000 injuries occur annually among members of the Federation, most of which are muscle, ankle, and knee lesions. The *Mutualitat de Futbolistes* is an organization within the Catalan Soccer Federation that provides health insurance coverage for all soccer-related injuries sustained by federated soccer players in Catalonia [31]. The *Mutualitat de*

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G.L. Canata, C.N. van Dijk (eds.), *Cartilage Lesions of the Ankle*,
DOI 10.1007/978-3-662-46332-1_7

Futbolistes has maintained a database of all such injuries attended by affiliated physicians since 1994. Osteochondral lesions of the talus have been related to acute and chronic ligament lesions of the ankle and malleolar fractures [36].

7.2 Review

Ankle injuries are extremely common in the majority of field sports, and sprains are the most common ankle injuries [6, 21]. It is thought that injury to the lateral aspect of the talus is associated with specific traumatic events. The anterolateral aspect of the talus dome collides with the fibula when the ankle is in forced inversion or dorsiflexion [1, 22]. Some works indicate that more than 50 % of acute ankle sprains and fractures develop some form of chondral injury [20]. Osteochondral lesions of the talus are a relatively broad term used to describe an injury or abnormality of the talar articular cartilage and adjacent bone [2]. Osteochondral lesions can lead to mechanical pain and swelling that precludes resumption of sports activity [41]. The ability to return to play at pre-injury level is a good indication that the lesion has resolved satisfactorily [44]. As is true for most sports-related injuries, osteochondral lesions are usually managed conservatively. It is only after this approach has failed that alternative surgical strategies are contemplated. This study reviews the data on all patients undergoing surgery for osteochondral injuries of the talus at the *Mutualitat de Futbolistes* and analyzes the associated lesions, the severity of the injury, and the time required for recovery. The treatment presented has proven to be a good solution for a considerable problem in a population that requires total recovery to resume soccer activity at pre-injury level [43, 44].

7.3 General Concepts

Osteochondral lesions of the talus are uncommon injuries, occupying the third position in order of frequency following knee and elbow involvement, and represent 4 % of all the osteochondral lesions of the body [28]. The portion of the talar dome bearing the medial load is most often affected and less commonly the lateral load area [1, 24, 26]. There are two common patterns of osteochondral lesions of the talus. Anterolateral talar dome lesions result from inversion and dorsiflexion injuries of the ankle at the area impacting against the fibula. Posteromedial lesions result from inversion, plantar flexion, and external rotation injuries of the ankle at the area impacting against the tibial ceiling of the ankle joint [33]. Injuries involving the lateral aspect are usually more severe (Grades III and IV) [29]. Recent studies suggest that after studying the location of injuries by MRI, it seems that most lesions are located medially and centrally on the talar dome [14].

Our group [31] presented a series of 16 patients from a group of soccer players with Tegner activity levels 9 and 10 from the Mutualitat de Futbolistes suffering from osteochondral injuries which after 3 months of conservative treatment, pain persisted and impeded sports activity [31]. They had a mean age of 24.4 years old (range 16–33), and all but one were males and all with a follow-up period of

3.56 years (range 0.66–6.42). In the most recent review of the data at the Mutualitat de Futbolistes, a total of 36 patients had been collected over a period of 20 years (1994–2014) with results similar to those previously published by our group (31). Other series have shown more advanced medial lesions, although their evolution was slower [27]. In this series, 94 % were medial lesions, and in more than 60 %, there was a traumatic causal relationship without injury to the lateral dome, in agreement with a previous study by Burns and Rosenbach in 1989 [4]. In 70 % of cases, there were associated lesions, including soft tissue impingement in 50 %, rupture of the external lateral ligament (lateral collateral ligament) in 13 %, and fracture of the fibula in 6 %.

7.4 Clinical Symptoms

The clinical symptoms of osteochondral talar lesions include pain, recurrent synovitis, joint balance alterations, and obstruction due to the presence of loose bodies. The recurrent synovitis and balance alterations are the likely cause of tibiotalar arthritis [12]. The symptoms are not specific to this condition, and because these are uncommon lesions, they can be mistaken for acute or chronic ankle sprains. For this reason, when the symptoms persist and the initial X-ray shows normal findings, it is advisable to perform CT or MRI to investigate the injury [3]. MRI shows the osteocartilaginous lesion [19], with a high degree of correlation, and can detect lesions that may be missed on plain films [18, 34]. Scintigraphy findings can also be diagnostic, but MRI is superior in diagnostic precision for this purpose. In our series, all patients were assessed preoperatively with radiography and with MRI, confirming the tendency to request an MRI exam even when scintigraphy findings are positive. Prevention of ankle sprains by proprioceptive training exercises is necessary to reduce the susceptibility of the joint to osteocartilaginous lesions [15].

7.5 Treatment

The initial treatment indicated is usually conservative [2]. The common treatment strategies of symptomatic osteochondral lesions include nonsurgical treatment, with rest, cast immobilization, and use of nonsteroidal anti-inflammatory drugs (NSAIDs) [2]. The mere presence of osteocartilaginous injuries on imaging studies does not imply that they will progress or lead to arthritic degeneration. For this reason, treatment is only contemplated in symptomatic cases [22]. Only one case in our series progressed to arthritic degeneration. When the patient's condition fails to improve, arthroscopic surgery obtains results equal to or better than arthrotomy, with the associated advantages of lower morbidity and faster recovery [3, 22]. Some authors consider arthroscopy the treatment of choice, leaving the option of open surgery for cases in which the access is difficult and malleolar osteotomy is required [32]. In addition to enabling treatment of many types of injuries, ankle arthroscopy provides a more accurate assessment of the problem than imaging, thereby facilitating the decision as to the most appropriate therapeutic approach [29]. A meta-analysis of

published studies using different treatments has shown success rates of 78 % in series in which excision and curettage were carried out and 86 % when microfracture was additionally performed [42]. Until randomized studies are conducted and more conclusive results can be obtained, these are the most effective treatments available. Conservative treatment has yielded a success rate of 45 % [35, 39, 42, 45]. Our results, which were excellent in 81.75 % and good in 18.25 %, are in accordance with the reported outcome of most authors [1]. Arthroscopic bone marrow stimulation techniques, such as microfracture and drilling, perforate the subchondral plate with multiple openings to recruit mesenchymal stem cells from the underlying bone marrow to stimulate the differentiation of fibrocartilaginous repair tissue in the defect site [20].

In patients with large osteochondral defects and unstable osteocartilaginous lesions, the optimum therapeutic option in the long term appears to be mosaic grafting with autologous osteochondral material [12], although there are no randomized studies with long-term follow-up to confirm these results. In a randomized study, Gobbi et al. reported similar findings with the use of microfractures or autografts with chondrocytes for osteocartilaginous talus lesions [10]. Hankemeier et al. obtained excellent or good outcome in 89 % of patients with osteochondral ankle lesions treated by debridement and abrasion of the subchondral space [13]. In a prospective study, Takao et al. found that the debridement of the osteochondral bed favored cartilaginous improvement at the site of the lesion in more than 90 % of cases [37]. Thermann and Becher reported a success rate of 93 %, 2 years after performing microfractures, and proved that age was not a criterion favoring a poor prognosis [38]. In contrast, Kumai et al. found that younger patients had better clinical and radiological results, as well as those undergoing surgery in the first months following the start of symptoms with respect to those operated 1 year later [16]. Studies reporting the outcome of mosaic grafting at 3 years of follow-up show success rates of almost 90 % [17] with "second-look" arthroscopic revision showing correct congruence of the grafts in 87.5 % [17]. Giza presented a series of 10 patients, wherein after conservative treatment and arthroscopy with debridement/curettage without good results, they then underwent matrix-induced autologous chondrocyte implantation (MACI) showing improvements in AOFAS and SF-36 scales [9].

In our patients at the Mutualitat de Futbolistes, motorized arthroscopic abrasion and perforations were performed in all cases. The arthroscopic technique starts with a spinal block and placing the patient in supine position with the leg placed in decline on a gynecological leg rest. The tourniquet is applied at the root of the thigh and after the blood has been expressed, sterilization is performed and the surgery starts. Two anterior portals are performed, first the medial portal at the tibiotalar joint, medial to the tibialis anterior tendon. The second with arthroscopic view is performed in the lateral surface and adjacent to the digitorum longus. After reviewing the joint and locating the osteochondral lesion, necrotic tissue is debrided to a bleeding bone bed followed by Steadman microfractures (Figs. 7.1, 7.2, 7.3, and 7.4). Excellent outcome were obtained in 80 % of patients and good results in the remaining cases, with a follow-up of 3.5 years. Open surgery usually requires osteotomies to access the talar lesions, and this procedure can delay consolidation and lead to

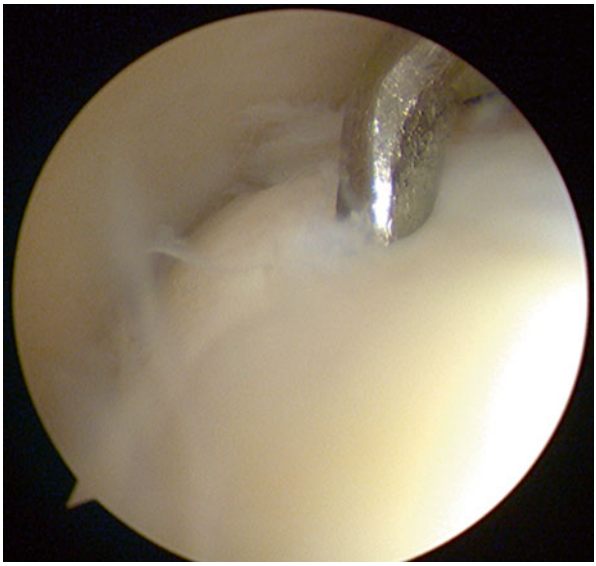

Fig. 7.1 Arthroscopic view of cartilage lesion with palpable defect

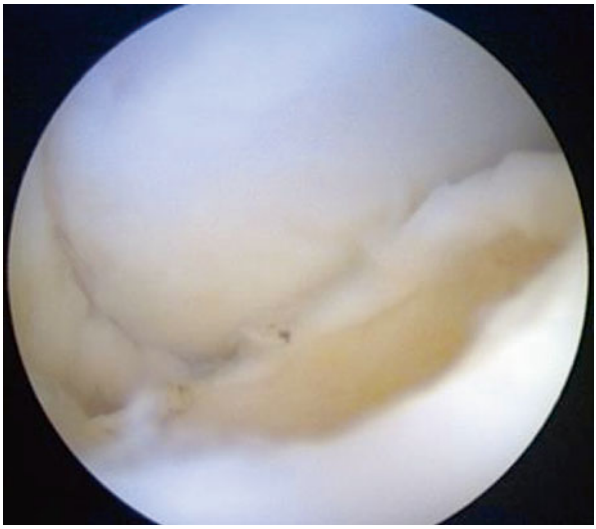

Fig. 7.2 Articular bone bed after curettage of cartilage injury

pseudoarthrosis [22]. Nonetheless, several studies have reported good results in more than 90 % of patients, with no serious complications in large lesions [11, 30]. Other reported treatments include fresh osteochondral allografts [5], autologous transplant of chondrocytes [40], percutaneous cement injection [33], and application of growth factors [8], which, from the biological viewpoint, seek healing and regeneration of injured tissues.

Clinical improvements have been reported in different studies on cartilage lesions with the use of plasma rich in growth factors (PRGF) [47, 48]. When

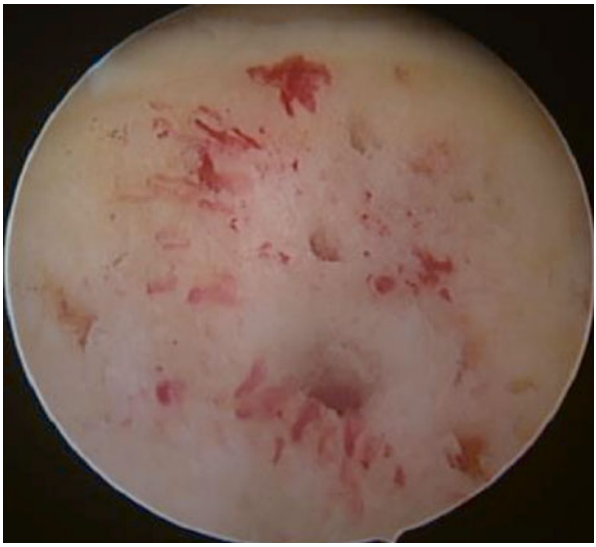

Fig. 7.3 After performing microfractures with bleeding bone bed

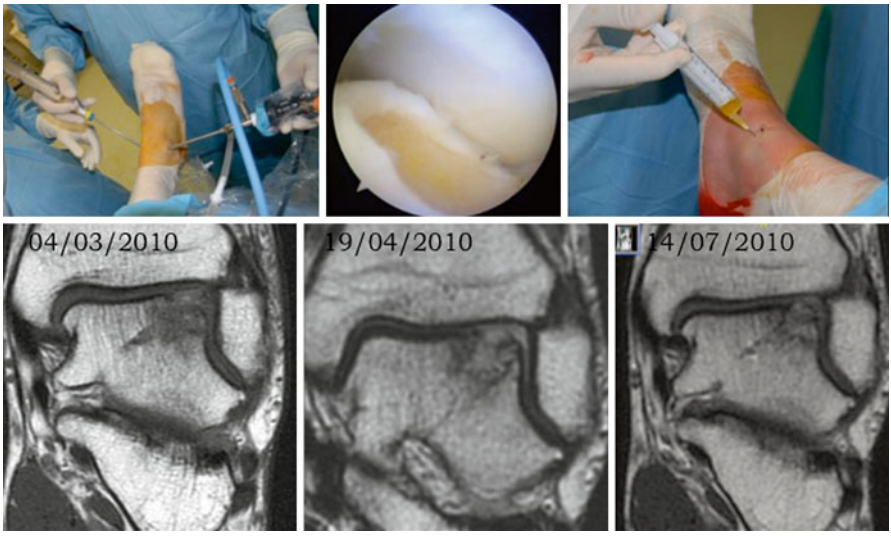

Fig. 7.4 The superior images show the arthroscopic access with the two anterior portals. The central image shows the talar articular surface without cartilage defect and the bone bed. The superior right image shows the application of PRGF-Endoret at the end of the surgery in the joint space. The inferior images show the changes of an osteochondral lesion over a 4-month period treated arthroscopically with curettage, microfractures, and application of PRGF-Endoret. The patient presented at the final follow-up with the MRI, without pain, and had recovered full mobility having returned to normal daily life activities including running and contact sports

working with PRGF, the methodology described by its author should be followed with regard to its preparation and application to ensure traceability [46] and thus render the results comparable to the various studies published. As indicated by Taylor, when obtaining PRP, taking different paths leads to the preparation of different products, with the huge possibility of getting different results [49].

Giannini presents favorable results in a 4-year follow-up with improvements in AOFAS scores and objectified by MRI T2 mapping sequences. Patients were treated in one surgical procedure with bone marrow-derived cell transplantation [7]. The study with meta-analysis by Niemeyer evaluated 16 publications with autologous chondrocyte implantation wherein he concluded that although clinical outcome as described in the studies available seems promising (with regard to a lack of controlled studies), a superiority or inferiority to other techniques such as osteochondral transplantation or microfracturing cannot be estimated [23]. Paul et al. presented a series treated by talar osteochondral transplantation where they found that patients modify their postoperative sports activities, and they noted a reduction of participation in high-impact and contact sports [25].

Conclusions

The series described here is composed of a highly homogeneous population of federated soccer players of similar age. Treatment by arthroscopy and debridement is not extremely aggressive and provides good results in terms of functional recovery. Based on the review of the literature, there are insufficient works with high levels of evidence that recommend a specific type of treatment in cases of osteochondritis of the talus. However, based on the accumulated experience gained at *Mutualitat de Futbolistes* which provides medical assistance to more than 140,000 athletes with more than 22,000 injuries per year, ankle osteochondritis (if symptomatic) is initially treated conservatively by physiotherapy and with biological techniques such as the infiltration of plasma rich in growth factors. If conservative treatment fails, surgical techniques are indicated with ankle arthroscopy and curettage of the lesion down to healthy bone and the application of articular PRGF-Endoret®.

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Lift, Drill, Fill and Fix (LDFF): A Cartilage Preservation Technique in Osteochondral Talar Defects

8

M.L. Reilingh and G.M.M.J. Kerkhoffs

8.1 Introduction

An osteochondral defect (OCD) of the talus is a lesion of the talar cartilage and subchondral bone. Several descriptive terms exist for this type of lesion, including osteochondritis dissecans, osteochondral fracture, transchondral fracture, osteochondral lesion and flake fracture. It is mostly caused by a (single or multiple) traumatic event, leading to partial or complete detachment of the fragment [1]. An OCD can either heal and remain asymptomatic or progress to deep ankle pain on weight bearing. The deep ankle pain is most probably caused by high fluid pressure during activity, resulting in stimulation of the subchondral bone nerves underneath the cartilage defect [2, 3]. Other possible symptoms are limited range of motion, stiffness, locking and swelling.

Treatment strategies for primary OCDs of the ankle have substantially increased over the last decade [1]. Conservative treatment is the first step in the treatment of symptomatic OCDs and may consist of nonsteroidal anti-inflammatory drugs (NSAIDs), restriction of (sporting) activities, rest and/or cast immobilisation. Currently, arthroscopic debridement and bone marrow stimulation is considered the primary treatment in symptomatic lesions up to 1.5 cm in diameter [1, 4–7]. With this technique all unstable cartilage, including the underlying necrotic bone,

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G.L. Canata, C.N. van Dijk (eds.), *Cartilage Lesions of the Ankle*,
DOI 10.1007/978-3-662-46332-1_8

is removed and small holes are drilled or punctured in the subchondral bone to promote revascularization. Consequentially, bone marrow cells migrate to the defect, and new fibrous cartilage is formed [3]. However, Qiu et al. studied OCDs in femoral condyles of rabbits and found that the presence of an advanced and irregular subchondral bone plate was associated with degradation of repaired articular surface [8].

Internal fixation of an osteochondral talar defect is a good alternative technique [9–11]. The advantage of this treatment option is to restore the natural congruency of the subchondral bone and to preserve hyaline cartilage. In this chapter, we provide an overview of internal fixation of osteochondral talar defects, with special emphasis on a new arthroscopic fixation technique: lift, drill, fill and fix (LDFF).

8.2 Indications and Contraindications

The indication for internal fixation is a primary, large OCD of the talar dome (anterior-posterior or medial-lateral diameter >10 mm on computed tomography) in patients with persistent complaints for more than 1 year. Contraindications are loose chondral lesions, ankle osteoarthritis grade II or III [12], advanced osteoporosis, infectious pathology and malignancy.

8.3 Preoperative Planning

Computed tomography is made with the ankle in maximum plantar flexion to determine the accessibility of the OCD [13], size, location, morphology and degree of displacement of osteochondral fragment. The scanning protocol involves 'ultra-high-resolution' axial slices with an increment of 0.3 mm and a thickness of 0.6 mm. Multi-planar coronal and sagittal reconstructions are 1 mm.

8.4 Surgical Technique

8.4.1 Arthrotomy

Internal fixation of an OCD of the talus is until now performed with a medial or lateral arthrotomy often combined with a malleolar osteotomy to allow proper visibility and working access (Fig. 8.1a). After exposure of the OCD, an osteochondral flap is created with a knife and lifted up with the posterior side of the flap left intact (Fig. 8.1b, c). The attached bone of the fragment is debrided, and several sites in the osteosclerotic area of the bed are drilled (Fig. 8.1d). If there is a subchondral cyst, its contents will be curetted and the circumference of its sclerotic wall drilled. After debridement and drilling the defect can be filled with cancellous bone from the malleolar osteotomy or of the distal tibial metaphysic (Fig. 8.1e). After the osteochondral fragment is correctly aligned, a permanent screw or solvable screw can be used

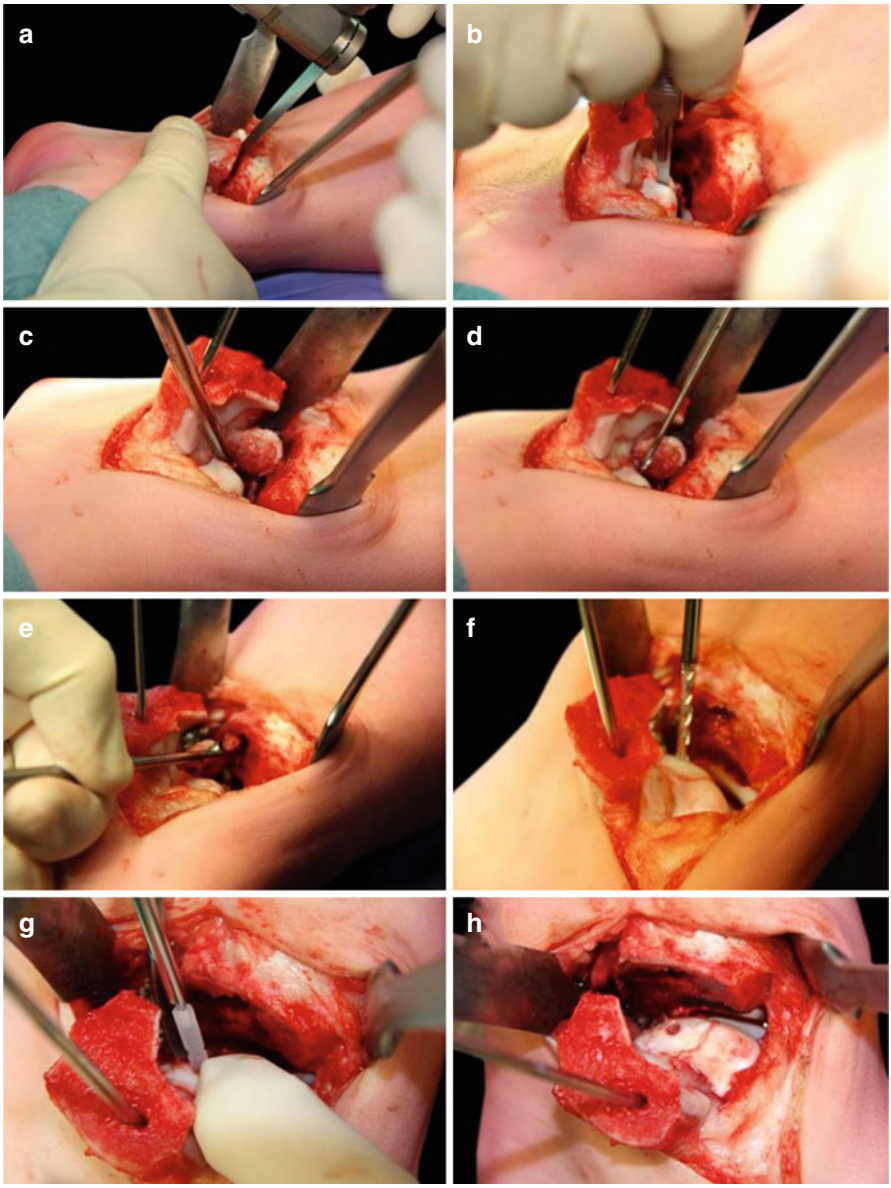

Fig. 8.1 Images of a left talus with a medial OCD that is treated by internal fixation. (**a**) The medial OCD is exposed after a medial malleolar osteotomy. (**b**) The osteochondral flap is created with the use of a knife. (**c**) With a chisel the osteochondral flap is lifted, taking care not to loosen the posterior part of the flap. (**d**) After debridement the osteosclerotic area of the bed and the bone flake of the osteochondral fragment are drilled to promote revascularization. (**e**) Cancellous bone is harvested from the medial malleolus using curette. (**f**) A cannulated system allows a predrilling and tapping of a compression screw (Arthrex Inc, Naples, USA). (**g**, **h**). An absorbable Bio-Compression screw (Arthrex Inc, Naples, USA) is placed 1–2 mm recessed relative to the surrounding surface of hyaline cartilage

to fix the fragment (Fig. 8.1f, g). The screws are placed under the articular surface (Fig. 8.1h). If an osteotomy was created, it will be reduced and the wound will be closed in layers. A short-leg cast is applied at the operation theatre.

8.4.2 Arthroscopy

Arthroscopic LDFF surgeries for osteochondral talar defects can be carried out as an outpatient procedure under general or spinal anaesthesia. Patients are placed in a supine position with slight elevation of the ipsilateral buttock. A support is placed at the contralateral side of the pelvis to prevent the patient from moving when the table is turned sideways for straight ankle positioning. The heel of the affected foot rests on the very end of the operating table. This positioning enabled the surgeon to fully dorsiflex the ankle by leaning against the foot sole and to use the table as a lever when maximal plantar flexion is needed. When indicated a noninvasive soft tissue distraction device is used. The ankle joint is assessed by an anteromedial and an anterolateral portal [14]. The anteromedial portal is made first with the ankle in slight dorsiflexion. A 4 mm 30° angled arthroscope will be introduced with the ankle in full dorsiflexion. In this ankle position, the talar cartilage is covered by the distal tibia and is therefore protected for iatrogenic damage on instrument insertion. Under direct arthroscopic vision, the location of the anterolateral portal is determined with a spinal needle and created. With a shaver the distal tibia rim is removed to facilitate better access to the ankle joint. The arthroscopic portals are interchangeable to allow optimal vision. With a probe the location of the OCD is identified, and a beaver knife is used to allow the making of a sharp osteochondral flap (Fig. 8.2a, b). The posterior side of the flap should be left intact and can be used as a lever, allowing lifting from the anterior with the use of a chisel (lift) (Fig. 8.2c). The attached bone of the osteochondral flap and the osteosclerotic area of the bed are debrided and drilled to promote revascularization (drill) (Fig. 8.2d). All subchondral cysts are debrided and punctured. After debridement and drilling the defect is filled with cancellous bone of the distal tibial metaphysis. Cancellous bone is harvested with a chisel by creating longitudinal particles which are transported into the defect with a grasp (fill) (Fig. 8.2e, f). After correct alignment of the osteochondral flap Bio-Compression screw(s) (Arthrex Inc, Naples, USA) or multiple chondral darts (Arthrex Inc, Naples, USA) are used to fix it (fix) (Fig. 8.2g, h). At the end of the procedure, the skin incisions are sutured with 3.0 Ethilon and a short-leg cast is applied at the operation theatre.

8.5 Postoperative Management

In both fixation techniques, a short-leg, non-weight-bearing cast will be applied for 4 weeks postoperatively. After these 4 weeks the foot is placed in a short-leg walking cast in neutral flexion position and neutral hindfoot position, with full weight bearing allowed. At 8 weeks postoperatively the cast will be removed. If a malleolar

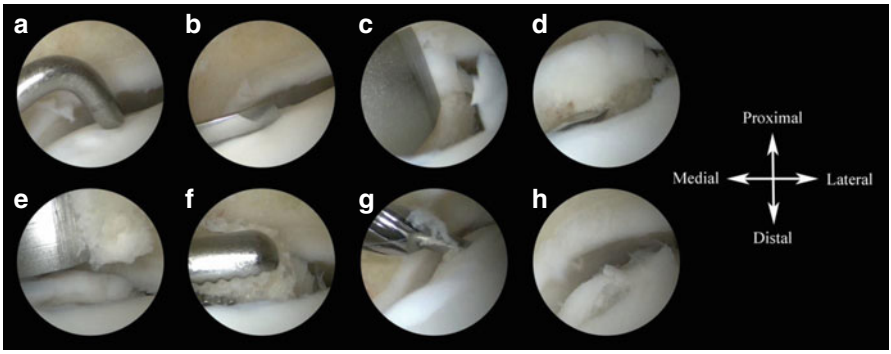

Fig. 8.2 Arthroscopic images of a left talus with a medial osteochondral defect that is treated by LDFF. (**a**) The exact location of the defect is identified by palpating the cartilage with a probe, while the ankle is in plantarflexion. (**b**) An osteochondral flap is created with the use of beaver knife. (**c**) With a chisel the osteochondral flap is lifted, taking care not to loosen the posterior part of the flap. (**d**) The bone flake of the osteochondral fragment is drilled with the use of a K-wire and a shaver blade to promote revascularization, again taking care not to loosen the fragment at its posterior attachment. (**e**) Cancellous bone is harvested from the distal tibia using a 4 mm chisel. (**f**) With an arthroscopic grasper the cancellous bone is transported to the defect until there is sufficient filling. (**g**) A cannulated system allows a predrilling and tapping of a compression screw (Arthrex Inc, Naples, USA). (**h**) An absorbable Bio-Compression screw (Arthrex Inc, Naples, USA) is placed 1–2 mm recessed relative to the surrounding surface of hyaline cartilage. The noncannulated screw is preferred because of its elegant diameter and compression strength

osteotomy was created during surgery, radiographs of the operated ankle are obtained to confirm consolidation. Physical therapy will be prescribed to assist in functional recovery and extend to full weight bearing in approximately 2 weeks.

8.6 Results

We published a retrospective case series of 9 patients after internal fixation of an OCD of the talus with a median follow-up of 4 years [10]. All patients had failed conservative treatment of a primary OCD. Various clinical outcome measures were recorded, including the Berndt and Harty outcome question [15]; Ogilvie-Harris score [16]; numeric rating scales (NRS; 0 to 10) of pain at rest, during walking and during running; American Orthopaedic Foot and Ankle Society (AOFAS; 0 to 100) score [17]; and Short Form 36 (SF-36; 0 to 100) [18, 19]. The Berndt and Harty clinical outcome was good in 7 cases (78 %) and fair in 2 cases (22 %). The Ogilvie-Harris score was excellent in 4 cases, good in 3 cases and fair in 2 cases. The median NRS pain at rest was 0 (range 0 to 6), during walking 1 (range 0 to 7) and during running 1.5 (range 0 to 4). The median AOFAS was 95 (range 77 to 100). The SF-36 physical component scale was 47.6 ± 8.3, and the mental component scale was 47.6 ± 13.9. On the final radiographs there were no progressive degenerative changes seen in all patients. An area of numbness around the scar was reported in one patient after fixation of the fragment.

Recently, we published the short-term clinical outcome of the arthroscopic LDFF technique for primary OCD of the talus, with a mean follow-up of 12 months (SD 0.6) [20]. Pre- and postoperative clinical assessment included the American Orthopaedic Foot and Ankle Society (AOFAS; 0 to 100) score [17] and the numeric rating scales (NRS; 0 to 100) of pain at rest and during walking. At final follow-up, the AOFAS score significantly improved from 63 ± 9.7 to 99 ± 1.6 ($p < 0.001$). The NRS of pain at rest significantly improved from 2.9 ± 1.9 to 0.1 ± 0.4 ($p = 0.004$) and during walking significantly improved from 7.6 ± 0.5 to 0.1 ± 0.4 ($p < 0.001$). On the final radiographs five of seven patients showed remodelling and bone ingrowth after LDFF (Fig. 8.3). All seven patients were satisfied and indicated that they would undergo the procedure again.

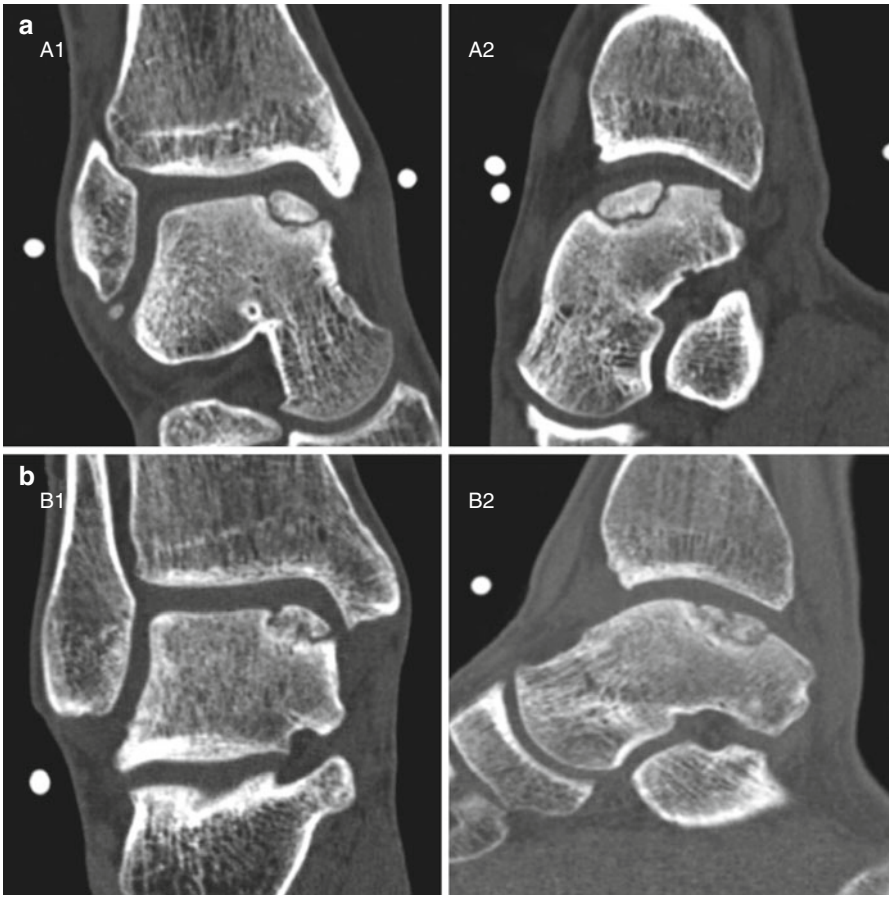

Fig. 8.3 (**a**) Preoperative coronal (A1) and sagittal (A2) computed tomography of a medial osteochondral talar defect of a right ankle in plantarflexion. (**b**) Postoperative coronal (B1) and sagittal (B2) computed tomography of the same ankle after LDFF with progressive bone ingrowth at 12-month follow-up

8.7 Discussions

Currently, arthroscopic debridement and bone marrow stimulation is considered the primary treatment in osteochondral talar defects up to 1.5 cm in diameter with a good clinical outcome of 85 % [1, 5], lasting over the years to have a 76 % satisfactory outcome at the long term [21]. However, after debridement and bone marrow stimulation, the subchondral plate will be irregular which is associated with degradation of repaired articular surface [8]. Furthermore, second-look arthroscopy after 12 months revealed that 40 % of the defects were incompletely healed with fibrocartilaginous tissue [22]. In contrast to native articular hyaline cartilage, bone marrow stimulation induces formation of fibrocartilaginous tissue which has inherently different biological and mechanical properties that are likely to degenerate over time [23]. Progression of ankle osteoarthritis is seen in 33–34 % after arthroscopic debridement and bone marrow stimulation of an osteochondral talar defect at long-term follow-up [21, 24].

Internal fixation is described as an alternative technique in the treatment of an OCD. The theoretical advantages of fixation are restoration of the subchondral bone and preservation of the hyaline cartilage. Different studies reported good clinical results after fixation of the OCD [9–11]. Schuh et al. reported a success rate of 100 % in 20 cases using K-wire fixation [11]. Kumai et al. reported a good clinical outcome of 89 % after fixation using bone pegs in 27 patients [9]. Later our study group reported a good clinical outcome of 78 % and a median AOFAS score of 95 after screw fixation in 9 children [10]. However, in these studies arthrotomies with or without a malleolar osteotomy were performed to fix the OCD.

Recently, we described a new arthroscopic LDFF technique [20]. With this arthroscopic technique, a lower complication rate and a faster rehabilitation are expected as compared to open fixation of an OCD [25]. Clinical and radiological results of 1-year follow-up are promising, but more patients and longer follow-up are needed to draw any firm conclusions and determine if the results stand the test of time.

A successful clinical and radiological outcome of the arthroscopic LDFF technique depends on several factors. The arthroscopic visualisation of the OCD is an important part of the arthroscopic LDFF technique, since adequate fixation of the osteochondral flap depends on the accessibility and quality of vision. Computed tomography with the ankle in maximal plantar flexion can be advised to allow adequate preoperative planning [13]. Furthermore, fixation of the osteochondral flap with Bio-Compression screw(s) (Arthrex Inc, Naples, USA) or chondral darts (Arthrex Inc, Naples, USA) should be performed perpendicular to the articular surface and slightly recessed relative to the surrounding surface to promote bone ingrowth.

In conclusion, internal fixation of an OCD of the talus is a good surgical option after failed conservative treatment. Arthroscopic fixation with the LDFF technique appears to be an elegant new treatment option; we are prospectively documenting all our patients with talar OCDs and will present our long-term outcomes in the future.

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9 Rehabilitation and Return-to-Sports Activity After Debridement and Bone Marrow Stimulation of Osteochondral Talar Defects

R.M. Gerards, I.C.M. van Eekeren, and C. Niek van Dijk

9.1 Introduction

An osteochondral ankle defect is a lesion of the talar cartilage and subchondral bone mostly caused by a single or multiple traumatic events. This can lead to partial or complete detachment of the fragment. The defects cause deep ankle pain associated with weight bearing. Impaired function, limited range of motion, stiffness, catching, locking, and swelling may be present. These symptoms bring the ability to walk, work, and perform sports at risk [1].

An OCD is often missed and can therefore be inadequately treated [2]. Because of advancements in cartilage-sensitive imaging, including magnetic resonance imaging (MRI) [3] and computed tomography (CT) [2, 4], OCDs of the ankle are increasingly detected. OCDs can heal or remain asymptomatic or progress to deep ankle pain on weight bearing. The primary treatment for OCDs up to 15 mm consists of arthroscopic debridement and bone marrow stimulation with an overall expected success rate of 85 % [5]. OCDs of the talus can severely impact quality of life, especially in athletes [6, 7]. As a result, these athletes aren't able to train or compete. For athletes with an OCD, the lapse before resuming high-impact sports (after surgery) can be as much as 3–6 months [8–10]. This chapter gives an

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G.L. Canata, C.N. van Dijk (eds.), *Cartilage Lesions of the Ankle*,
DOI 10.1007/978-3-662-46332-1_9

overview of the levels of activity and the time needed to return to activity and reviews the factors that affect rehabilitation after arthroscopic debridement and bone marrow stimulation of a talar OD.

9.2 Tissue Healing After Debridement and Bone Marrow Stimulation

Both conservative and surgical treatment options for OCDs have been widely discussed in the literature [5]. Conservative treatment consists of rest and/or restriction of (sports) activities, with or without treatment with nonsteroidal anti-inflammatory drugs (NSAIDs) or a cast immobilization for at least 3 weeks and up to 4 months [5]. The primary (surgical) treatment for most OCDs is debridement and bone marrow stimulation. The objective is to remove all unstable cartilage and underlying necrotic bone, opening and curetting the cysts underneath and then performing bone marrow stimulation (Fig. 9.1). Bone marrow stimulation can be performed with a drill, a

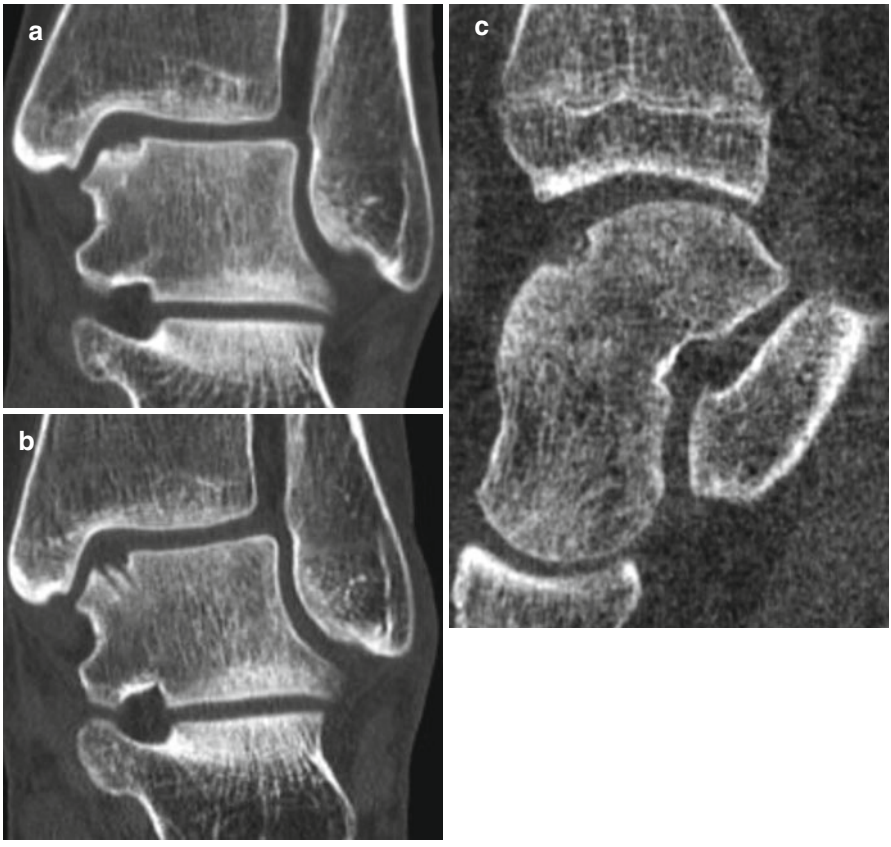

Fig. 9.1 Computed tomography (CT) scan of an osteochondral defect of the talar bone. (**a**) Before debridement and bone marrow stimulation. (**b**) After debridement and bone marrow stimulation. (**c**) CT is made in full plantar flexion to see if the OCD is approachable arthroscopically

Table 9.1 Phases of OCD healing after bone marrow stimulation

Phase		Time in weeks
1 Inflammatory phase	Formation of fibrin clot that releases growth factors and cytokines to format granulation tissue	1–2
2 Remodeling phase	Mesenchymal cells proliferate and differentiate into chondrocyte-like cells producing a matrix containing type II collagen and proteoglycans. Formation of fibrocartilaginous tissue and bone	3–8
	Fibrocartilaginous tissue turns into hyaline-like cartilage Formation of woven bone	8–12
	Mixture of fibrocartilage and hyaline cartilage Formation of bone plate and reformed tide mark with restored subchondral bone	12–48

K-wire, or a microfracture probe. The latter has the advantage that it results in multiple fractures in the trabeculae instead of destruction of the bone (Fig. 9.1b). Interosseous blood vessels are disrupted by this technique, which leads to the release of growth factors and the formation of a fibrin clot. The subsequent classical wound repair cascade comprised of an acute inflammatory response and cell chemotaxis leads to the generation of a vascularized granulation tissue and the proliferation of pluripotent mesenchymal progenitor cells with a capacity to differentiate into multiple mesenchymal cell types [11]. Within 2 weeks, undifferentiated mesenchymal cells have differentiated into osteoblast-like cells and into chondrocyte-like cells. The osteoblasts form new woven bone, while the chondroblasts produce a matrix containing type II collagen and proteoglycans forming fibrocartilaginous tissue within 6–8 weeks [12–14]. After 8 weeks, hyaline-like cartilage can be detected with a high component of type II collagen [12] and, at 12 weeks, the ODs are completely filled with mostly hyaline-like tissue [14]. Initially, the new subchondral bone is woven; then eventually, it becomes lamellated with the subchondral region modified to a compact bone plate and reformed tide mark. The restored bone is, however, of lesser quality than normal bone and it is not identical to the original [15]. When examined at 24 and 48 weeks, no difference in histological analysis of the cartilage is detectable [16]. At 1 year, the chondral repair tissue is a mixture of fibrocartilage and hyaline cartilage, with a considerable component of type I collagen [17] (Table 9.1). If repair or remodeling fails to restore the functional balance of articular cartilage and subchondral bone, it can lead to a disturbed balance and a disordered joint will remain [15].

9.3 Activity Levels

Before returning to activity/sports, it is important to quantify the patient's level of activity. This can be monitored in various ways. Tegner and Lysholm described their activity score in 1985. It was originally tested for knee ligament injuries, but for the past 17 years, it has been used for other joint evaluations as well. The development of already existing and new kinds of sports, differences between knee and ankle loading, and different injury rates provided reasons for developing an ankle-specific activity score. Halasi et al. developed an activity score designed specifically for

ankle joint. It is a single-page, easy-to-survey system that contains 53 sports, 3 working activities, and 4 general activities in categories from 0 to 10. Contact team ball sports and gymnastics are at the top of the list. The score is 10 for top-level, 9 for competitive-level, and 8 for recreational-level athletes [18]. Another activity score was developed in 1972 by Roles and Maudsley [19]. This system measures activity level, pain, and range of motion (ROM), but it is not restricted to the ankle joint. A more practical activity-level score was described for patients rehabilitating from Achilles tendon ruptures [20]. This consists of four levels of activity: walking, running, noncontact sports, and contact sports. The first and most basic level of activity after an injury is to return to normal walking. The second level is to return to running, the third is to return to a noncontact sport and the highest is to return to a contact sport. This system isn't specific for Achilles tendon ruptures and can be applied for any ankle injury and thus also to monitor the rehabilitation after surgery for talar OCDs.

9.4 Rehabilitation

This 4-level activity scheme to return to activity is applicable for determining a return to activity after the debridement and bone marrow stimulation of a talar OCD. Return to activity is divided into four levels of increasing intensity: walking, running, return to noncontact sports, and return to contact sports (Table 9.2). Each of these levels demands specific training and exercises, and each have to be

Table 9.2 Return to activity after debridement and bone marrow stimulation of an osteochondral defect

Level	Goal	Training	End terms
1	Return to normal walking	Proprioception Passive and active sagittal ROM Force	Active stability Near normal Force, 25 % L/R → Normal walking
2	Return to running on even ground	Force Technical skills Endurance	Force <12 % L/R Sideward movement → Easy jogging
3	Return to noncontact sport	Speed Force Endurance	Running even ground Sprinting Force normalized Turning/twisting Rope jumping → Noncontact sports
4	Return to contact sports	Speed Force Endurance	Running uneven ground Explosive force Changing direction Sports-specific movements → Contact sports

L/R left/right, *ROM* range of motion, → indicates end of phase

mastered before the next level can be attempted [20]. The patient's activities are systematically expanded and carefully monitored. Before progression to the next phase and increasing training components like speed, force, and endurance all end, terms should be met. The four phases are outlined as follows:

Level 1: The first level of activity commences on the day of the operation with active and passive dorsi- and plantar flexed motions and partial weight bearing. Early mobilization will prohibit joint stiffness. The classical wound repair cascade will start on the day of the operation. Partial weight bearing provides synovial fluid to nourish chondrocytes. After 6–8 weeks, fibrocartilaginous tissue is formed and full weight bearing is allowed to further stimulate osteoblasts in the formation of bone matrix. At the end of this phase, training of proprioception is commenced to regain normal active stability.

Level 2: The next level of activity is to resume running on even round. In case active stability has not yet been achieved, further training of proprioception might be needed. The ROM should be normalized. By training of force, endurance, and technical skills, the aim is to achieve controlled sideways movement, with the lower-leg force increasing to a left/right difference of less than 12 %. After increased activity, pain and swelling should have subsided within 24 h.

Level 3: The third level of activity is a return to noncontact sports. Training focuses on speed and endurance. Running or sprinting on even ground shouldn't be problematic. At the end of this phase, rope jumping, turning, or twisting should be possible. Some pain may occur after intensifying activity but should be absent after 24 hours.

Level 4: This, the highest level of activity phase, is defined as a return to contact sports. Final training for gaining muscle strength, improving speed and endurance should make it possible to run on uneven ground, generating explosive force in sprinting, twisting, and turning and/or other sports-specific movements.

9.4.1 Level 1 (Walking)

In the literature, returning to normal weight bearing after debridement and bone marrow stimulation varies directly from post surgery to 8 weeks after surgery [10, 21–25]. Ogilvie-Harris et al. [25] allowed immediate full weight bearing according to comfort, whereas Chuckpaiwong et al. [27] splinted their patients for 1–2 weeks, after which they commenced ROM exercises and full weight bearing in walking boots. Barnes and Ferkel et al. [26] prefer to keep patients non-weight bearing for 4 weeks when the lesion is less than 1.5 cm in diameter and for 8 weeks when the lesion is larger. Saxena and Eakin [10] prevented weight bearing using a below-knee cast boot for up to 6 weeks, although patients with small lesions (<3 mm in diameter) were allowed to partially bear weight after 3 weeks. All patients were allowed passive ROM exercises at 3 weeks and at 6 weeks, active ROM exercises were

allowed. In the study of Guo et al. [22], patients were allowed to advance to full weight bearing 8 weeks after surgery, while Lee et al. [23] had a non-weight-bearing period of 6–8 weeks and partial weight bearing after 8 weeks [23]. Seijas et al. [24] reported weight bearing after an average of 8 weeks (4–14 weeks).

Lee et al. [30] compared early versus delayed weight bearing. Early weight bearing was defined as partial weight bearing in the first 2 weeks followed by full weight bearing as soon as tolerated. In the delayed group, all patients were kept non-weight bearing for 6 weeks. Lee et al. showed no significant difference between the two groups.

Patients in our own clinic, Academic Medical Center, Amsterdam, are allowed to progress from partial weight bearing (eggshell) to full weight bearing within 4–6 weeks depending on the size of the lesion. Active plantar flexed and dorsiflexed ankle movements are encouraged [7, 27, 28].

9.4.2 Level 2 (Running)

Impact activities, such as running, were first allowed at 12 weeks by the following authors: Saxena and Eakin [10], Seijas et al. [24], and Ogilvie-Harris and Sarrosa [31]. No other studies mentioned return to running [21–23]. It is also the present senior author's practice to allow running on even ground after 12 weeks.

9.4.3 Level 3 (Noncontact Sports)

Chuckpaiwong et al. [21] allowed patients to return to sports after 4–6 months, depending on muscle strength. Guo et al. [22] allowed sports after 6 months. In the study of Lee et al. [23], the ankle activity score by Halasi et al. significantly improved from 3 [1–5] to 6 [3–8] and showed that 63 % of their patients were returned to their pre-injury sporting level. Ogilvie-Harris and Sarrosa [25] reported in their study that 79 % of patients were able to return to unrestricted sports, 18 % were able to play but at a lower level, and 3 % were unable to return to any sports.

9.4.4 Level 4 (Contact Sports)

Saxena and Eakin [10] reported a significantly faster return to activity after treatment with microfracturing in high-injury-prone patients, such as soccer and basketball players, when compared with patients treated with bone grafting (15.1 ± 4.0 weeks vs 19.6 ± 5.9 weeks). Arthroscopically treated patients had a faster return to activity, compared with patients treated with an arthrotomy (15.8 ± 4.8 weeks vs 17.5 ± 5.5 weeks), but the difference was not statistically significant. Seijas et al. [24] reported a return to competition soccer within an average of 20 weeks. In our clinic, a full return to sports activities is usually possible 4–6 months after surgery [7, 27].

9.4.5 Influencing Factors

9.4.5.1 Age

Animal studies show that cartilage proteoglycans from immature animals are larger compared to those of mature animals. It is likely that OCDs in young individuals heal more effectively than in mature and elderly individuals [31]. This was supported by two studies, where younger patients with talar ODs had a better functional and clinical outcome after debridement and bone marrow stimulation. Kumai et al. reported a clinical good result (12 out of 13 ankles) in patients under the age of 30 after bone marrow stimulation, but in patients of 50 years and older, only 1 out of 5 patients managed the same results. Chuckpaiwong et al. also showed significant improvements in the outcome of surgical treatment when the patient is younger, has a lower BMI, and has a shorter duration of symptoms [32, 21]. However, other clinical studies failed to show that older age is an independent predictor for clinical failure after arthroscopic treatment [29, 33, 34].

9.4.5.2 Body Mass Index

Although Chuckpaiwong [21] reported that patients with a lower body mass index (BMI) have a better functional outcome after debridement and bone marrow stimulation of ODs in the talus, this isn't supported by all the literature [23]. The general consensus is that a lower BMI is beneficial.

9.4.5.3 Defect Size

An animal study has demonstrated that larger defects are less likely to recover completely [35]. Chuckpaiwong et al. [21] reported good to excellent results in 100 % of patients with lesions <15 mm in diameter ($n=73$). However, they found a poor outcome in all but one of the 32 patients with lesions exciding 15 mm in diameter. The effect of the size of the lesion on the clinical outcome was further assessed by Choi et al. [36]. Of the 25 patients with lesions >15 mm in diameter, 80 % had a poor outcome. Clinically, the larger the defect size, the less likely a functional outcome for patients after debridement and bone marrow stimulation of the talus [21, 22]. It is concluded that the cutoff point is approximately 15 mm [36].

9.4.5.4 Hyaluronic Acid

Hyaluronic acid (HA) is widely used as a nonsurgical treatment option for ankle and knee osteoarthritis [37, 38], but it is also used in the treatment of talar OCD [39]. Intra-articular injection of HA reduces pain and inflammation and at the same time supplements the endogenous joint fluid. Some studies suggested that HA treatments facilitate a biological activation based on the lasting benefits of HA treatment long after the presence of HA after injection [40].

9.4.5.5 Mobilization

Immobilization has a great influence on the ankle joint. One week of ankle immobilization resulted in a loss in plantar flexion strength, balance, and walking gait in asymptomatic volunteers [41]. In animal studies, mobilization leads to thicker and

stiffer cartilage with a greater concentration of endogenous proteoglycan [42–46]. In our clinic, we promote active dorsi- and plantar flexed movements, without weight bearing, direct post operatively.

9.4.5.6 Platelet-Rich Plasma

In vitro data shows a higher rate of proteoglycan synthesis and accumulation as well as collagen synthesis with treatment of platelet-rich plasma (PRP) [47]. PRP treatment also enhances mesenchymal stem cell (MSC) proliferation [48]. In animal studies, PRP treatment leads to more neochondrogenesis and glycosaminoglycans in OCDs after 4 weeks and to more hyaline tissue after 12 weeks. Although there is no literature on the use of PRP as an adjunct to surgical treatment, Mei-Dan et al. [39] recently performed a prospective clinical trial comparing PRP with hyaluronic acid (HA) injection for the treatment of osteochondral lesions of the talus. There was a significantly better clinical improvement after PRP treatment than after HA injection.

9.4.5.7 Insulin-Like Growth Factor

In vitro data shows that insulin-like growth factor (IGF-1) stimulates the extracellular matrix and decreases matrix catabolism [49]. In animal models, IGF-I has led to enhanced repair of extensive cartilage defects and protection of the synovial membrane from chronic inflammation [50–54].

9.4.5.8 Bone Morphogenic Proteins

Bone morphogenic protein-7 (BMP-7) has been investigated for its capacity to regenerate articular cartilage and currently appears to be the gold standard growth factor for cartilage repair. In animal studies, BMP-7 appears effective in regeneration of osteochondral or focal chondral defects [55].

9.4.5.9 Platelet-Derived Growth Factor

Evidence to support the use of platelet-derived growth factor (PDGF) is extrapolated from its role in wound healing and stimulation of matrix synthesis in growth plate chondrocytes [52]. In an animal study, rats were injected into the knee and no adverse effects were noted in the cartilage or synovial membrane [56]. Presently, the most commonly used form of PDGF is as a component of platelet-rich plasma (PRP).

9.4.5.10 Transforming Growth Factor-b1

In vitro, TGF-b1 stimulates de novo synthesis of matrix macromolecules as well as stimulation of chondrogenesis of synovial lining and bone marrow-derived MSCs [57, 58].

9.4.5.11 Pulse Electromagnetic Fields

In vitro studies show improved bone development, increased chondrocyte proliferation, and increased proteoglycan synthesis with downregulation of IL-1 and stimulation of TGF-b and IGF-1 after treatment with PEMFs [60–67]. In an animal study,

PEMFs stimulated osteoblast activity during the healing process of an OD [68]. Clinically, PEMF treatment improves the functional recovery of patients after arthroscopic treatment of chondral lesions in the knee and reduces the use of non-steroidal anti-inflammatory drugs [69]. A double-blind, randomized controlled trial started in 2008 that will provide information about the efficiency of treatment with PEMF in patients with an OD in the talus [59].

9.4.5.12 Shock Wave

In vitro, the expression of TGF-b1 in defect tissues is increased after shock wave treatment [70]. An animal study showed more hyaline-like cartilage with more proteoglycans and rich blood vessels at the bottom of an OD after bone marrow stimulation with shock wave therapy, compared with bone marrow stimulation alone [71].

9.4.5.13 Bisphosphonate

An animal study showed acceleration of subchondral bone repair during the early stages and better cartilage quality after treatment with bisphosphonates (alendronate) [72].

Conclusion

There isn't much published on return to activity or sports after debridement and bone marrow stimulation of a talar OCD, and the literature shows no consensus.

We propose early mobilization after surgery and 4–6 weeks to return to full weight bearing. Further rehabilitation depends on the desired level of activity. For the return to activity after surgery, we propose a four-level activity scale. To return to contact sports, patients first have to achieve the level of normal walking, followed by running and return to noncontact sports. As most patients with an OCD are young and athletic, a faster rehabilitation will improve the quality of life in these patients. Potential factors reducing the rehabilitation time are a younger age, lower BMI, smaller defect size, mobilization, and treatment with growth factors, PRP, bisphosphonates, hyaluronic acid, and PEMF. More high-level research on potentially influencing factors is needed for talar OCDs

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