Chapter 9
Osteochondral Defects in the Ankle Joint

Rogier Gerards, Maartje Zengerink, and C. Niek Van Dijk

Football is a simple game, but the hardest in Football is to play simple.

Johan Cruijff

Abstract Osteochondral defects (OCDs), also known as osteochondritis dissecans, can cause pain and decreased function in patients and offer a significant challenge to the foot and ankle surgeons. An OCD is a lesion involving articular hyaline cartilage, the subchondral bone plate and the subarticular spongiosa. An OCD is mostly caused by a single or multiple traumatic events leading to partial or complete detachment of the osteochondral fragment with or without osteonecrosis. Osteochondral ankle defects can be seen in the tibial plafond but occur predominantly on the talar dome.

9.1 Introduction

Osteochondral defects (OCDs), also known as osteochondritis dissecans, can cause pain and decreased function in patients and offer a significant challenge to the foot and ankle surgeons. An OCD is a lesion involving articular hyaline cartilage, the subchondral bone plate and the subarticular spongiosa. An OCD is mostly caused by a single or multiple traumatic events leading to partial or complete detachment of the osteochondral fragment with or without osteonecrosis. Osteochondral ankle defects can be seen in the tibial plafond but occur predominantly on the talar dome.

Osteochondral defects can occur in any joint; however, the most common location is the knee, followed by the elbow. Of the total number of OCDs, the ankle comprises approximately 4 %, and they occur most frequently in 20- to 30-year-old males [1, 2]. Little is known about the incidence of osteochondral defect in the

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general population. A study following US military personnel (1999–2008) described an average incidence of 27 per 100,000 people. The incidence rate was 16 per 100,000 in 2002, with steady annual increases resulting in an incidence rate of 56 per 100,000 in 2008, corresponding to the years of active involvement in combat operations and thus increased physical activities [3].

The ankle is one of the most frequently injured joints in sporting activities and in the general population [4, 5]. Ankle sprain injuries account for 14.8 % of all reported injuries in the emergency department. These injuries are more common among athletes who participate in sports that involve running on changing terrains, repetitive jumping, or frequent changes in direction, such as in basketball, volleyball, football, American football and cross-country [6–8]. Ankle injuries comprise of 45 % of all basketball injuries, 31 % of all football injuries and 25 % of all volleyball injuries [9, 10].

Ankle sprains are accepted as being the most common cause of osteochondral ankle defects. The incidence of osteochondral lesion after chronic ankle sprains is reported in the literature as 7 % [11, 12]. The results of acute ankle arthroscopy in a series of acute ankle sprains revealed a medial osteochondral talar lesion in 66 % of cases (20 lesions in 30 patients) [13]. In a retrospective study on 108 ankle sprains, the MRI findings showed bone bruises in 39 % [14].

Treatment of an ankle sprain is directed at returning the athlete to the previous level of competition in the shortest possible time. However, after standard treatment for acute ankle sprains, residual symptoms are reported in up to 40 % of patients [11, 15]. In case of persisting symptoms, the possibility of an OCD needs to be considered. The aim of this chapter is to provide an overview of osteochondral ankle defects, their symptoms and specific treatment indications in football players.

9.2 History

Osteonecrosis of subchondral bone was first described by Ambroise Pare in 1558 after finding loose bodies in a patient's knee. Monro was, in 1856, the first to report the presence of cartilaginous bodies in the ankle joint [16]. Paget further described the pathology and named the process "quiet necrosis" when describing two patients with knee pain in 1870 [17]. In 1887–1888, König was given credit for his original description. He suggested that the loose bodies, found in the knee joint, resulted from a spontaneous osteonecrosis secondary to vascular occlusion of the subchondral bone. He used the term osteochondritis to refer to an inflammatory process and dissecans, derived from the Latin word dissecare, to separate [18]. However, throughout the years, inflammation was never revealed as a contributing factor, making the name osteochondritis a misnomer. In 1922, Kappis [19] was the first to describe a similar lesion in the talar dome, but it was not until 1959, when Berndt and Harty were the first to mention trauma as the main aetiological factor of osteochondral ankle defects. They used the term transchondral fracture of the talus to describe the defect and presented a classification system and guidelines for indications for surgery. Since Berndt and Harty's classic paper, indications for surgical treatment have changed, and nowadays, a large variety of treatment options exist for the different forms of osteochondral ankle defects [20].

9.3 Aetiology

Although trauma is mentioned as the main aetiological factor of OCDs, not all patients report a history of ankle injury. A subdivision can be made between traumatic and non-traumatic defects. In the aetiology of traumatic OCDs, ankle sprains play a large role. A severe ankle sprain can cause a small fracture and subsequent impaired vascularity, leading to the formation of an OCD. Microtraumas, caused by repetitive articular cartilage surface loading or excessive stress, can lead to cellular degeneration or death by the disruption of the collagen fibril ultrastructure and thickening of the subarticular spongiosa [21]. In lateral OCD lesions, trauma is described in 98 % of cases; in medial lesions, this is 70 % [22]. In non-traumatic OCDs, ischemia, subsequent necrosis and genetics are possible aetiological factors. OCDs have been described in identical twins and in siblings [23–25]. And in 10–25 % of patients, the occurrence of the defect is bilateral [20, 26, 27].

9.4 Mechanism of Injury

When an inversion trauma occurs, the talus twists inside its boxlike housing, and the cartilage lining and the underlying bone can be damaged. Trauma may lead to a bone bruise and softening of the cartilage or even a crack in the cartilage with subsequent delamination. Due to the shearing forces separation may also occur in the subchondral bone, giving rise to a subchondral lesion. These fragments can detach completely and become a loose body in the ankle joint or remain partially attached. When trauma has caused microfractures in the subchondral plate and subarticular spongiosa, it creates a situation in which liquid from the damaged cartilage can be forced into the subarticular spongiosa during loading. The smaller the diameter of the defect in the subchondral plate, the higher the fluid pressure. This intermittent local rise in high fluid pressure will cause osteolysis and the eventual formation of a subchondral cyst. The ongoing intermittent flow of fluid from the joint through the damaged subchondral bone plate into the spongiosa can prevent healing of the lesion in the subchondral bone plate [28].

In cadaver ankles, Berndt and Harty could reproduce lateral defects by strong inversion of a dorsiflexed ankle [20]. When the foot is inverted, the lateral part of the talar dome is compressed against the fibula (Fig. 9.1). Due to the forces released when the lateral ligament ruptures, an avulsion of the lateral talar border can occur. They were also able to reproduce a medial lesion in a plantar-flexed ankle with a slight anterior displacement of the talus accompanied by an inversion and internal rotation of the talus. Until recently, it was estimated that lateral osteochondral lesions were usually located in the anterior third of the talar dome and medial lesions in the posterior half of the talus. Anteromedial, posterolateral and central lesions were also seen but less frequent. A study to evaluate the location and morphologic characteristics of osteochondral lesions on the talar dome showed that medial lesions are more frequent than lateral lesions. Lateral lesions are typically shallow

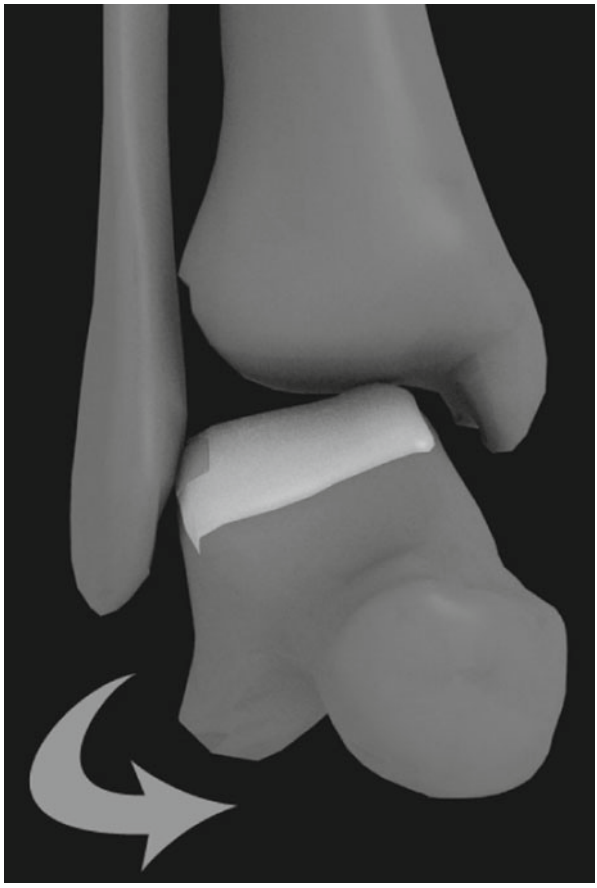


Fig. 9.1 Inversion of the talus; a shear mechanism injury resulting in a lateral osteochondral defect

and wafer shaped, indicating a shear mechanism of injury. In contrast, medial lesions are generally deep and cup-shaped, indicating a mechanism of torsional impaction. Because of their shape, location and trauma mechanism, lateral lesions are more often displaced than medial lesions (Fig. 9.2) [29].

9.5 Clinical Presentation

In the acute situation, after a traumatic incident, an OCD of the talus often remains unrecognised. This is because of swelling and pain from soft tissue injury like lateral ligament lesion. The radiographs taken at the emergency unit may not reveal any pathology. In case of a large OCD, the initial radiographs may be positive. When the symptoms of the ligament injury have resolved after some weeks, symptoms like persistent swelling, limited range of motion and pain on weight bearing

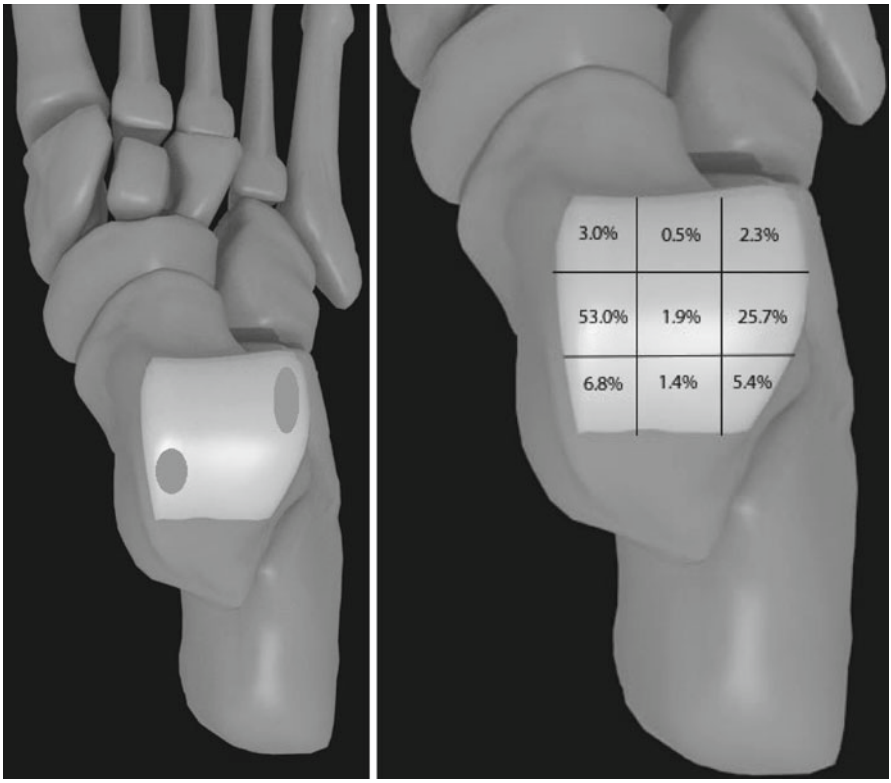


Fig. 9.2 Main shape and locations of OCDs of the right talar bone [29]

may persist. 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 4–6 weeks, an OCD of the talus should be suspected. Locking and catching are symptoms of a displaced fragment.

A differentiation has to be made between the acute and chronic situation. Chronic lesions classically present as deep lateral or medial ankle pain associated with weight bearing. Reactive swelling and stiffness can be present, but the absence of swelling, locking or catching does not rule out an OCD. Recognisable tenderness on palpation is typically not present in these patients, but can be present in case of synovitis. Some patients have a diminished range of motion.

Differential diagnoses are:

- Posttraumatic synovitis
- OCD of tibial plafond
- Sinus tarsi syndrome
- Ligament laxity
- Osteoarthritis
- Subtalar joint pathology

Cartilage has a liquid and a solid component (i.e. collagen and proteoglycans) that enables it to withstand compressive stress. Fluid from the damaged cartilage can be forced into the microfractured subchondral bone plate underneath during loading. The smaller the diameter of the defect in the subchondral plate, the higher the fluid pressure. This intermittent local rise in high fluid pressure will cause osteolysis and the eventual formation of a subchondral cyst. Malalignment of the ankle joint may aggravate this process by increasing the local pressure in specific locations of the ankle. The pain in osteochondral defects is most probably caused by the repetitive high fluid pressure, sensitising the highly innervated subchondral bone [28].

9.6 Diagnosis

After medical history and physical examination of the ankle, routine radiographs are made consisting of weight-bearing anteroposterior (mortise) and lateral views of both ankles. OCDs may be visible on the plain AP radiograph shown as an area of radiolucency (Fig. 9.3), although the findings may be subtle and require very careful attention. Displaced fragments are more likely to be detected on the plain radiograph than those which are undisplaced. However, it is not unusual for the initial radiograph to be normal. Small fragments are rarely visible. A heel-rise view with the ankle in a plantar-flexed position may reveal a posteromedial or posterolateral defect [30].

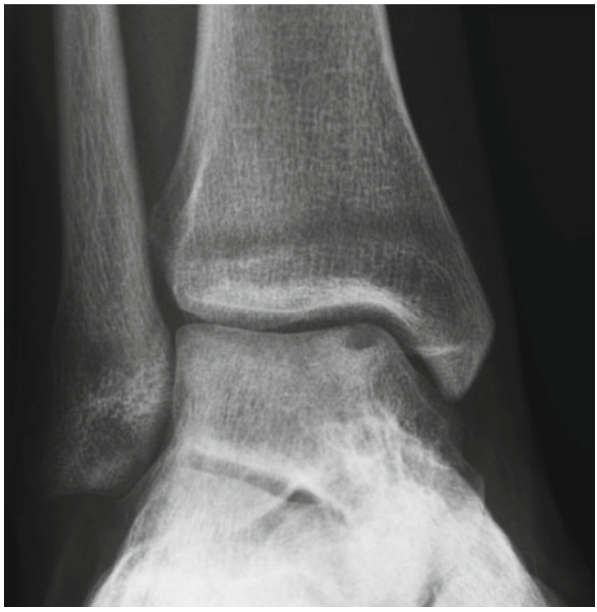


Fig. 9.3 Radiolucency of the medial talar dome indicating an osteochondral defect (x-ray)

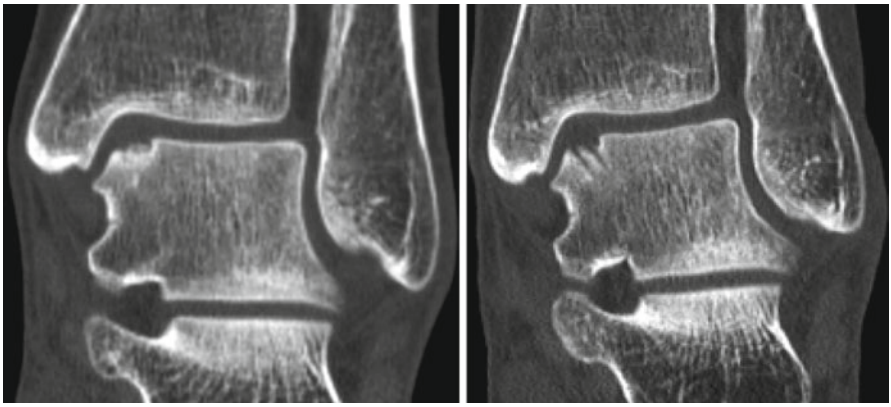


Fig. 9.4 Computed tomography (CT) scan of a medial osteochondral defect before and after excision, debridement of the sclerotic bone and bone marrow stimulation

Table 9.1 Classification and staging of lesions

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

According to Berndt and Harty [20]

The sensitivity of routine radiography is 50–75 %, whereas pickup on bone scan is 99 % sensitive and can differentiate between a symptomatic and asymptomatic lesion. CT scan may be useful for bony anatomy and location of the lesion and is therefore more valuable for preoperative planning (Fig. 9.4) [30].

MRI is indicated if standard radiographic results are normal; it may give information regarding vascularity, healing and cartilage integrity. However, the true extent of the OCDs may be obscured by concomitant bone marrow oedema [31].

9.7 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 [20].

In stage I, there is local compression of the cartilage and subchondral bone, and usually there are no radiographic findings. In stage II, there is avulsion or partial detachment of the osteochondral fragment, but the main part is still attached to the talus. In stage III, there is complete avulsion of an osteochondral fragment without any displacement. In stage IV, the osteochondral fragment is completely detached and displaced inside the ankle joint (Table 9.1 and Fig. 9.5).

Loomer et al. later modified the staging system to include stage 5, subchondral cysts [32]. Ferkel and Sgaglione developed a classification system based on CT:

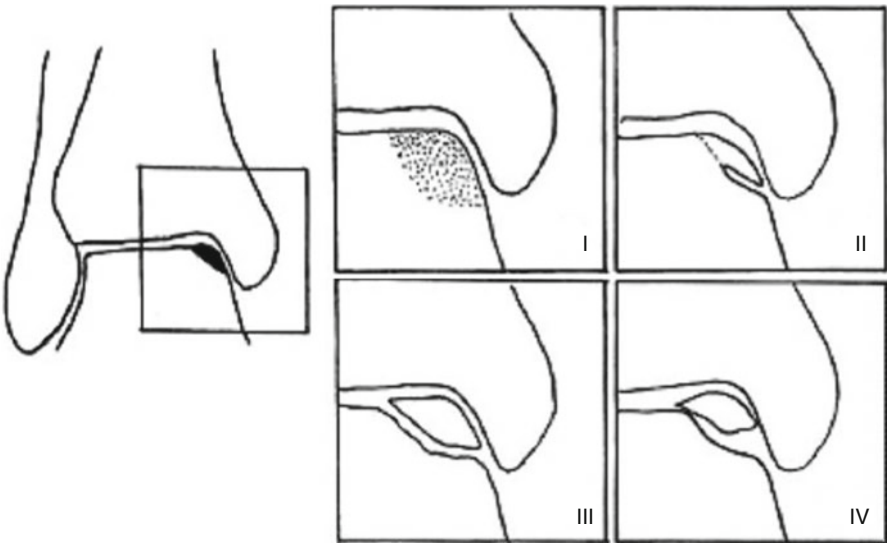


Fig. 9.5 Classification of osteochondral ankle defects [20]

stage I, intact roof/cartilage with cystic lesion beneath; stage IIA, cystic lesion with communication to the surface; stage IIB, open surface lesion with overlying fragment; stage III, non-displaced fragment with lucency underneath; and stage IV, displaced fragment [33]. Hepple et al. revised the MRI classification in 1999 to resemble Berndt and Harty's original classification. Stage 1 represents articular cartilage damage only. Stage 2a represents articular cartilage damage with underlying fracture and bony oedema. Stage 2b is similar to 2a without bony oedema. Stage 3 represents a detached but undisplaced osteochondral fragment. The fragment is displaced in stage 4, and in stage 5, subchondral cyst formation occurs. Pritsch et al. were one of the first to stage talar osteochondral lesions with arthroscopic findings according to cartilage quality [34]. Cheng et al. further developed arthroscopic staging of the lesions. In stage A, the articular cartilage is smooth and intact, but soft. In stage B, the articular cartilage surface is rough. In stage C, fibrillation or fissuring of the cartilage is present. In stage D, an osteochondral flap is present or bone is exposed. In stage E, the osteochondral fragment is detached but undisplaced. In stage F, the osteochondral fragment is detached and displaced. Arthroscopy is useful in staging talar osteochondral lesions, but it is unable to completely assess underlying bony lesions [35].

9.8 Current Treatment Options

There are widely published nonsurgical and surgical techniques for treatments of symptomatic osteochondral lesions.

9.8.1 Nonoperative Treatment

Conservative treatment consists of rest and/or restriction of (sporting) activities, with or without treatment with nonsteroidal anti-inflammatory drugs (NSAIDs) or a cast immobilisation for at least 3 weeks and up to 4 months. A systematic review of treatment strategies for OCDs demonstrated only a 45 % success rate for nonoperative treatment [38]. The treatment aim is to unload the damaged cartilage, so oedema can resolve and necrosis is prevented.

9.8.2 Bone Marrow Stimulation (BMS)

Surgical treatment can include excision, in which the partially detached fragment is excised and the defect itself is left untreated; excision and debridement, in which, after excision of the loose body, the surrounding necrotic subchondral bone is curetted using either an open or arthroscopic technique; and finally excision, debridement and bone marrow stimulation (BMS) (Fig. 9.4). BMS is the current treatment of choice regarding OCDs. Following excision and curettage (debridement), multiple connections with the subarticular spongiosa are created. This can be accomplished by drilling or microfracturing. The objective is to partially destroy the calcified zone that is most present and to create multiple openings into the subchondral spongiosa. Intraosseous blood vessels are disrupted, and the release of growth factors leads to the formation of a fibrin clot. The formation of local new blood vessels is stimulated, bone marrow cells are introduced in the osteochondral defect, and fibrocartilaginous tissue is formed. Diameter of the lesions usually did not exceed 1.5 cm. Treatment was reported to be successful in 85 % of described cases [36]. In the case of large defects, a cancellous bone graft can be placed.

9.8.3 Antegrade (Transmalleolar) Drilling

Transmalleolar antegrade drilling is considered in cases of OCDs that are difficult to approach because of their location on the talar dome. In this technique, a K-wire is inserted about 3 cm proximal to the tip of the medial malleolus and directed across the medial malleolus into the lesion through the intact cartilage. In case an osteochondral lesion is hard to reach because of its location on the talar dome, the defect can be drilled through the malleolus. A K-wire is inserted about 3 cm proximal to the tip of the medial malleolus and directed across the medial malleolus into the lesion through the intact cartilage [36]. This method is not recommended in football players since it damages the intact cartilage of the distal tibia.

9.8.4 Retrograde Drilling

Retrograde drilling is done for primary OCDs when there is more or less intact cartilage with a large subchondral cyst or when the defect is hard to reach via the usual anterolateral and anteromedial portals. For medial lesions, arthroscopic drilling can take place through the sinus tarsi. For lateral lesions, the cyst is approached from anteromedial. A posterior arthroscopic approach is possible by drilling through the posterior talar process. The aim is to induce subchondral bone revascularisation and subsequently to stimulate the formation of new bone. A cancellous graft may be placed to fill the gap [36, 37]. It is the treatment of choice when there is a large subchondral cyst with overlying healthy cartilage.

9.8.5 Osteochondral Autograft Transplantation

Osteochondral autografts have been introduced as an alternative to allografts for the treatment of OCDs. Two related procedures have been developed: mosaicplasty and osteochondral autograft transfer system (OATS). Both are reconstructive bone grafting techniques that use one or more cylindrical osteochondral grafts from the less weight-bearing periphery of the ipsilateral knee and transplant them into the prepared defect site on the talus. Its goal is to reproduce the mechanical, structural and biochemical properties of the original hyaline articular cartilage which has become damaged. It is carried out either by an open approach or by an arthroscopic procedure. Indications involve large, often medial lesions, sometimes with a cyst underneath [36, 37]. Osteochondral grafting of defects yielded 90–94 % good to excellent results at intermediate follow-up; recent studies suggested significant midterm donor-site morbidity at the previously uninjured knee joint [39–41]. On the search for an alternative method that addresses both the osseous and chondral levels and provides intrinsic osteochondral stability without harming another joint, a modified mosaicplasty procedure was developed for severe and recurrent talus OCL: bony periosteum-covered iliac crest plug transplantation. But more research is needed to prove its effect [42]. OATS is not recommended as initial treatment in football players because of the high donor-site morbidity.

9.8.6 Autologous Chondrocyte Implantation (ACI)

Autologous chondrocyte implantation attempts to regenerate tissue with a high percentage of hyaline-like cartilage. The ACI technique involves placing cultured chondrocytes under a periosteal patch that covers the lesion. It is done for lesions larger than 1 cm^2 in the absence of generalised osteoarthritic changes. Harvesting is

first accomplished from either the knee or ankle from the region on the perimeter of the talus lesion. A second procedure is performed after the cells have been cultured for 6–8 weeks. An osteotomy of the medial malleolus can be performed for medial defects. The damaged articular surface is curetted to a stable border, and a periosteal patch is harvested from the tibia. The patch is sutured to the defect and sealed with fibrin glue. Finally, cultured chondrocytes are injected under the patch. Matrix-based chondrocyte implantations (MACI) are also available [43]. It differs from traditional ACI in that chondrocytes are not placed under the periosteal patch but embedded in a type I/III collagen membrane bilayer. As with ACI, the membrane is placed in the defect, but sutures are not required. The membrane bilayer is secured using fibrin sealant. MACI is technically easier than ACI and does not require an osteotomy [36, 37].

9.8.7 Fixation

Large fragments are treated surgically with reduction and fixation of the osteochondral fragment. Several types of internal fixation have been reported, including Herbert screws, Kirschner wires, absorbable fixation and fibrin glue. The advantage is that the graft fits anatomically. Fixation is recommended for lesions of more than 15 mm [36].

9.9 Treatment in Primary Lesions

The surgical treatment of osteochondral lesions of the talus remains controversial among orthopaedic surgeons worldwide. The choice of treatment for osteochondral ankle defects depends on symptomatology, duration of complaints, size of defect and whether a primary or secondary OCD. None of the current grading systems are sufficient to direct the choice of treatment [30]. Pure cartilage lesions and asymptomatic and low symptomatic lesions are treated conservatively with rest, ice, temporarily reduced weight bearing or non-weight bearing using a cast and, in case of giving way, an orthosis. Consideration for surgical treatment is failure of nonoperative treatment or continuing or exacerbation of symptoms after 6 months or residual symptoms after previous surgical treatment.

Arthroscopic bone marrow stimulation is the primary treatment in primary OCDs smaller than 15 mm, with good success proven by Level II or III studies with consistent findings (Table 9.2) [36, 44].

The treatment of symptomatic OCDs has difficulties and limitations because of the poor regeneration of articular cartilage and the limited access to the ankle joint. It is important that the surgeon understand the causes of failure as well as the factors influencing the results of BMS and other treatments of OCDs (Table 9.3).

Table 9.2 Primary OCDs in football players, best treatment options

Lesion type	Best treatment
Asymptomatic lesions	Conservative
Symptomatic lesions <15 mm	BMS
Symptomatic lesions >15 mm	Fixation
Talar cystic lesions	Retrograde drilling

Table 9.3 Conceptions and misconceptions regarding results after BMS

Factors influencing the results of BMS of OCDs
Increasing age is not an independent risk factor for poor clinical outcome after the arthroscopic treatment of OCDs
In patients with a large area of more than 15 mm, the clinical failure rate is significantly higher
The existence of a cyst in osteochondral defects has not demonstrated to affect the postoperative prognosis
Patients with an uncontained lesion experienced inferior clinical outcomes as compared with patients with a contained lesion after arthroscopic treatment
Osteochondral transplantation is a viable alternative secondary procedure for treating unstable OCDs that are refractive to arthroscopic treatment

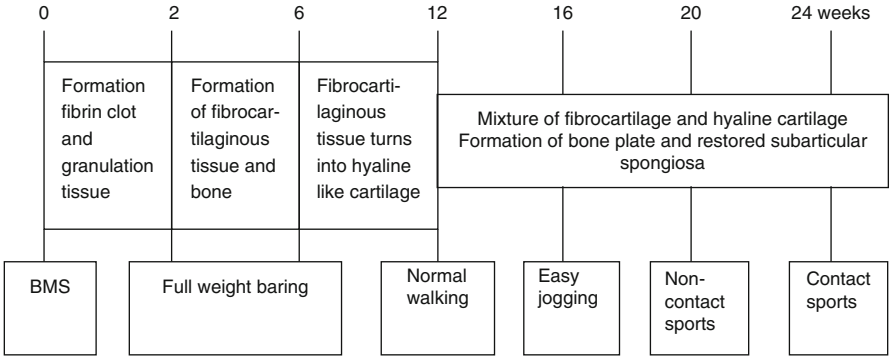
9.9.1 BMS Surgical Technique

Preoperatively, the best approach to the defect is decided. Upon the preference of the surgeon and the location of the lesion, the approach can be either from the anterior, from the posterior or by means of a medial malleolar osteotomy. On the lateral side, a detachment of the anterior talofibular ligament (ATFL) and the calcaneofibular ligament (CFL) can extend the approach to a posterolateral lesion. In case of arthroscopic treatment, we recommend to place the ankle in full plantar flexion. We use a 4.0-mm arthroscope or a 2.7-mm scoop of 11-cm length with a high volume shaft of 4.6 mm. All defects in the anterior half of the talus as well as lesions located in the anterior part of the posterior half can thus be reached and treated.

The procedure is started without distraction. The standard anteromedial and anterolateral approaches are created as described [45]. When introducing instruments for an anterolateral defect, the ankle is in fully dorsiflexed position, and the scoop is introduced through the anteromedial portal and a 4.5- or 5.5-mm shaver through the anterolateral portal. If the OCD is located anteromedially, the arthroscope is moved over to the anterolateral portal, and the instruments are introduced through the anteromedial portal.

If osteophytes are present, they can be removed by chisel, burr or aggressive full-radius resector (bone cutter). Synovitis located anterolaterally (in case of an anterolateral defect) or anteromedially (in case of an anteromedial defect) is removed by a 4.5- or 5.5-mm full-radius resector with the ankle in the dorsiflexed position. The completeness of removal of osteophytes and synovitis is checked by bringing the ankle into plantar flexion. It should be possible to palpate and visualise the OCD

Table 9.4 Timetable recovery after BMS



without disturbance of the synovium or overlying osteophyte. If this is not the case, then a further synovectomy is performed with the ankle in the dorsiflexed position. After sufficient synovectomy, it is possible to identify the lesion with the ankle in the forced plantar-flexed position by palpating the cartilage with a probe. In case of a posteriorly located osteochondral lesion, a full forced plantar flexion is needed for adequate visualisation. A little joint laxity helps to open up the joint. During this part of the procedure, we apply a soft tissue distractor [46]. Debridement is performed by means of the aggressive full-radius resector or a small closed cup curette. It is important to remove all dead bone and overlying unsupported, unstable cartilage. Every step in the debridement procedure is checked by regularly switching portals. A precise and complete debridement with removal of all loose fragments can be performed.

After full debridement, the sclerotic zone is drilled by multiple drill holes using a 2-mm burr or a 1.4-mm Kirschner wire. A K-wire has the advantage of flexibility, whereas a 2-mm drill can break more easily if the position of the ankle is changed during drilling. When a 2-mm drill is used, a drill sleeve is necessary to protect the tissue. Microfracturing by means of a microfracture probe offers the possibility to work "around the corner". Make sure that the calcified area is penetrated.

9.9.2 Aftercare

Aftercare depends on the type of surgical treatment. After arthroscopic treatment of OCDs, a 4-level activity scheme, derived from rehabilitation after Achilles tendon rupture, has been described (Table 9.4) [46, 50].

Level 1: The first level of activity phase is a return to normal walking that commences on the day of the operation with partial weight bearing. Training for ROM is important in this phase. Patients are encouraged to make active plantar-flexed and dorsiflexed ankle movements. The most important factor is the quality and strength of the tissue repair. The formation of granulation and thereafter fibrocartilaginous

tissue starts on the day of the operation. Partial weight bearing provides synovial fluid to nourish chondrocytes. Allowing full weight bearing depends on size and location of the lesion. A lesion of up to 1 cm is allowed to progress to full weight bearing within 2–4 weeks. Larger lesions and anteriorly located lesions require partial weight bearing of up to 6 weeks. After 6–8 weeks, fibrocartilaginous tissue is formed, and full weight bearing is allowed to further stimulate osteoblasts in the formation of bone underneath the cartilage. At the end of this phase, training of proprioception is commenced to regain normal active stability.

Level 2: The next level of activity phase is to resume running on even ground. Progression from walking to running on even ground is permitted between 12 and 16 weeks. Further training of proprioception might be needed, in case active stability has not yet been achieved. The ROM should be normal. By training for 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 ceased after 24 h.

Level 3: The third level of activity phase is a return to noncontact activities. Depending on the size and location, full return to noncontact sporting activities is usually possible 20–24 weeks postoperatively. By means of further training for speed and endurance, running on even ground and sprinting should become possible. At the end of this phase, rope jumping, turning and twisting should also be possible. Some pain may occur after increased activity but should be absent after 24 h.

Level 4: This, the highest level of activity phase, is defined as a return to contact sports. Contact sports are permitted from 24 weeks and up. Final training for speed, muscle strength and endurance should enable running on uneven ground, generating explosive force, changing direction and other sports-specific movements.

The course of rehabilitation after other treatment options, like fixation or OATS, is slightly different. Large fragments are treated surgically with reduction and fixation of the osteochondral fragment. If a fragment is fixed, the period of non-weight bearing is 6 weeks followed by another 4–6 weeks of controlled weight bearing to ensure proper fixation.

After medial malleolar osteotomy, weight bearing is dependent on the surgical treatment of the osteochondral lesion. After OATS, running is not permitted until the graft has been incorporated. Furthermore, the literature describes several factors, like growth factors, PRP, bisphosphonates, hyaluronic acid and PEMF, that can influence the natural recovery of an OCD and, thereby, the speed of rehabilitation and return to sports. Since most factors are investigated in vitro and in animal studies, more research on potentially influencing factors is needed for talar OCDs in humans [46].

9.10 Important Notes Concerning Football Players

The major cause of osteochondral ankle defects is supination trauma. Prevention should therefore be aimed at preventing ankle sprains. A 2011 *Cochrane Systematic Database Review* provides good evidence for the beneficial effect of

ankle supports in the form of semi-rigid orthosis or air-cast braces to prevent ankle sprains during high-risk sporting activities. Football players with a history of previous sprain can be advised that wearing such supports may reduce the risk of incurring a future sprain. However, any potential prophylactic effect should be balanced against the baseline risk of the activity, the cost of the device and – for some – the possible or perceived loss of performance [47]. Proprioceptive training has also been shown to be effective for prevention of ankle sprain recurrences [48]. The majority of lesions can be treated arthroscopically. Many posteromedial lesions do not have to be treated by malleolar osteotomy but can be treated arthroscopically by bringing the foot in hyperplantar flexion although skill and experience are required. Advantages of arthroscopic treatment are low morbidity, low cost, fast recovery and fast mobilisation.

Possible disadvantages of a medial malleolar osteotomy in case of a posteromedial osteochondral ankle defect are persisting ankle stiffness, use of fixation screws, malunion, non-union and degenerative changes due to the osteotomy over the long term [49]. We do not recommend the use in professional football players.

Morbidity at the donor site in OATS is seen in up to 36 % of cases [36]. For a football player, this is a concern since knee pain may prevent the patient from returning to competitive play. For secondary lesions, BMS is still a good option, with a success rate of 75 %. Other options are OATS and ACI.

Rehabilitation in athletes is directed not only at progressing the patient from protected mobilisation to partial and full weight bearing but also at strengthening and proprioceptive activity.

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