Osteochondral Defects of the Ankle

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81.1 Introduction

Osteochondral defects (OCDs) are also known as osteochondritis dissecans. OCDs are lesions involving articular hyaline cartilage and subchondral bone. These lesions may cause pain and disability and offer a challenge to foot and ankle surgeons.

OCDs can occur in every joint and are most common in the knee and the elbow. Of all OCDs, only 4 % occurs in the ankle joint, with a peak incidence in 20–30-year-old males [1, 2]. In the general population, little is known on the incidence of OCDs. Although, Orr et al. [3] showed an increase in incidence in military personnel corresponding with an increase in physical activity.

OCDs occur in most cases in the talar dome, but may also occur in the tibial plafond. Most often the OCDs are located at the posteromedial (58 %) or anterolateral (42 %) side of the talar dome [4].

Ankle sprains are the most common cause of OCDs. Treatment of these sprains is mainly conservative. Residual symptoms occur in up to 40 % of patients after an ankle sprain. In case of residual symptoms, an OCD must be considered as the cause of symptoms [5, 6].

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81.2 Aetiology

Ankle trauma is reported as the main etiologic factor for developing an OCD [7]. Not all patients, however, describe a history of ankle trauma. Therefore, OCDs are categorized as traumatic or non-traumatic defects [5, 6].

Previous trauma is reported in 98 % of laterally located OCDs and in 70 % of medially located OCDs [8–10]. Ankle sprains play the most important role in developing a traumatic OCD [5, 6]. A severe ankle sprain may cause a small fracture in the talus and subsequently impaired vascularization. This, in turn, may lead to the formation of an OCD [10]. Microtraumas, caused by repetitive articular cartilage surface loading or excessive stress, can lead to cellular degeneration or necrosis. This is due to disruption of the collagen fibril ultrastructure and thickening of the subarticular spongiosa [10, 11].

OCDs occur in up to 70 % of sprains and fractures involving the ankle and up to 7 % of supination trauma and acute ankle ligament ruptures [9]. These traumatic events can lead to partial or complete detachment of an osteochondral fragment, with or without necrosis [7, 12]. Of all OCDs, 93 % is located laterally and 61 % is located medially [7].

Inadequate treatment of OCDs may lead to osteoarthritis of the ankle [10, 13]. In case of non-traumatic OCDs, genetic, metabolic, vascular, endocrine and degenerative factors, as well as morphologic abnormalities, ligamentous laxity,

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spontaneous necrosis, steroid treatment and embolic disease, may contribute to the development of an OCD [9, 14, 15]. A significantly higher incidence of OCDs found in siblings and bilateral lesions also suggests a congenital or hereditary cause [13, 16].

81.3 Injury Mechanism

Lateral OCDs are mainly caused by a combination of inversion and dorsiflexion, whereas medial lesions are caused by a combination of inversion, plantar flexion and internal rotation [7, 14].

In case of an inversion trauma, the talus twists inside its box-like housing formed by the calcaneus, tibia and fibula, and the lateral part of the talar dome is compressed against the fibula (Fig. 81.1). Forces are released when the lateral ligaments rupture, which may cause an avulsion of the lateral talar border [17]. Traumas may lead to bone bruises and softening of cartilage. Cracks in the cartilage may occur with subsequent delamination. Shear forces may also damage subchondral bone, creating subchondral lesions. Fragments may remain partially attached to the talus or completely detach and become loose bodies.

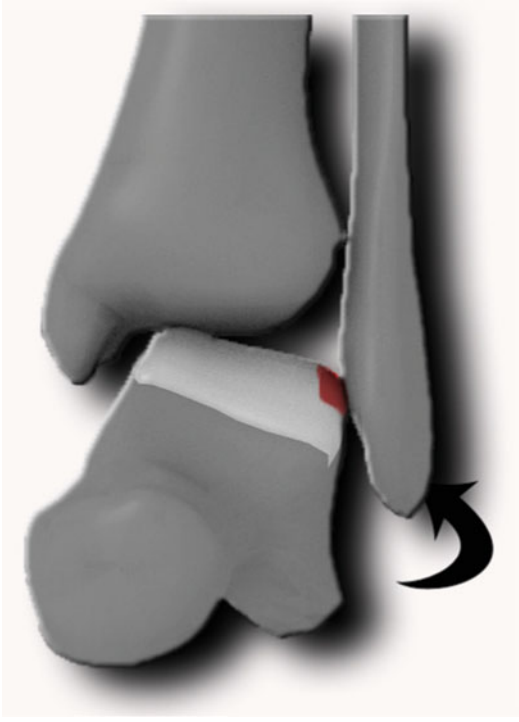


Fig. 81.1 Inversion of the talus, an injury mechanism leading to a lateral osteochondral defect

In case of microfractures in the subchondral plate and subarticular spongiosa, caused by trauma, fluid from the damaged cartilage may be forced into the subarticular spongiosa during loading [10]. The smaller the diameter of the lesion, the higher the fluid pressure. The intermittent local rise in high fluid pressure may cause osteolysis and eventually formation of a subchondral cyst. The intermittent flow of fluid and pressure build-up in the joint through the damaged subchondral bone plate into the spongiosa may prevent healing of the lesion [10, 18].

Overall medial lesions are more frequent compared to lateral lesions. Lateral lesions are typically shallow and wafer shaped, caused by a shear injury mechanism. Medial lesions are generally deep and cup shaped, indicating torsional impaction injury. Lateral lesions are more often displaced compared to medial lesions, which can be explained by their shape, location and trauma mechanism (Fig. 81.2) [17].

81.4 Clinical Presentation

After a traumatic incident, a talar OCD of the talus may remain unrecognized, due to pain and swelling from the soft-tissue injury. Standard radiographs taken at the emergency unit may also fail to reveal an OCD. Size increase enhances the chance of visibility on an X-ray (Figs. 81.3 and 81.4). After a few weeks, symptoms of soft-tissue injuries have resolved, and patients experience persistent or intermittent deep ankle pain during weight bearing and during or after activity. Sometimes this is accompanied by swelling and limited range of motion [7]. Symptoms of isolated ligamentous ankle injury should have resolved within 2–3 weeks after conservative treatment. If symptoms still persist after 4–6 weeks, a

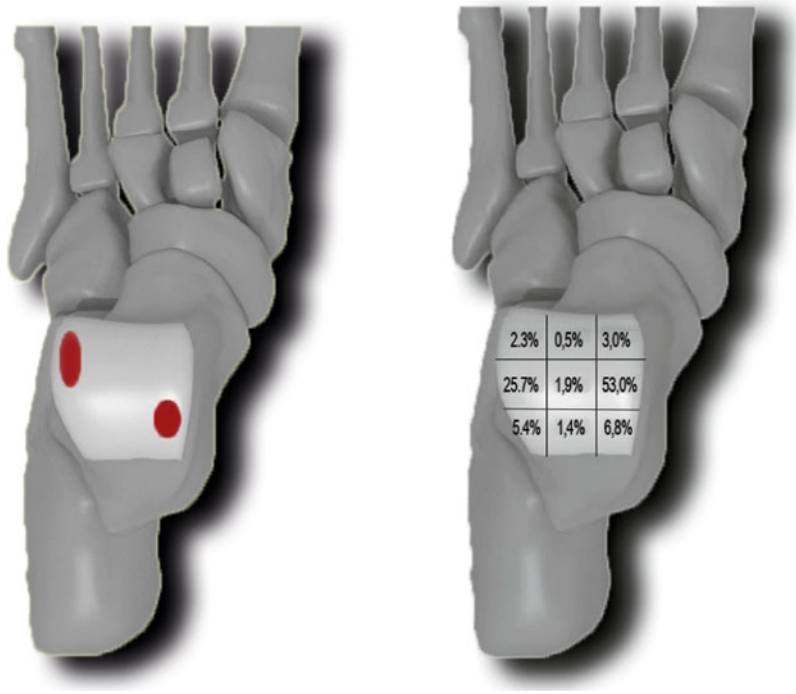


Fig. 81.2 Main shape and locations of the talus [17]

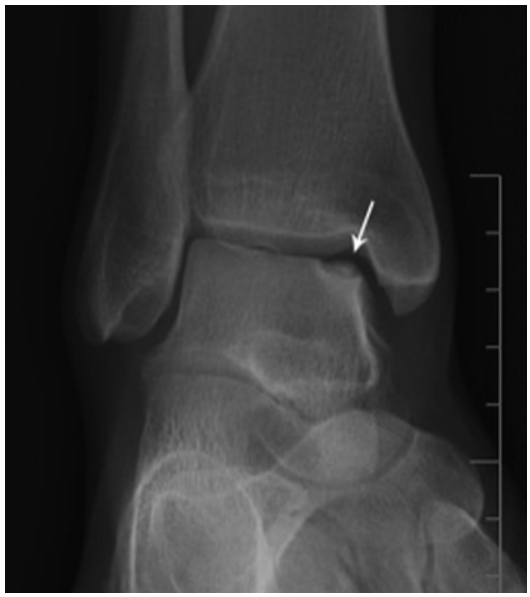


Fig. 81.3 Radiolucency of the medial talar dome indicating an osteochondral defect

talar OCD should be suspected. Locking and catching of the ankle joint can give rise to high suspicion of an OCD with a displaced fragment.

Differentiation must be made between acute and chronic lesions. Chronic lesions classically present as deep lateral or medial ankle pain associated with weight bearing. Reactive swelling and diminished range of motion can be present. Absence of swelling, locking or catching does not rule out an OCD. Generally, no recognizable tenderness is found on palpation, but may be present in case of secondary synovitis [7, 19, 20].

81.5 Clinical and Diagnostic Examination

In case of an ankle injury, evaluation generally consists of taking a medical history and performing regular physical examination. On clinical examination, few abnormalities can be found. Affected ankles may be presented with a normal range of motion, absence of swelling and no recognizable tenderness on palpation [7, 19, 20].

For diagnostic examination, often, routine radiographs of both ankles are taken, consisting of a weight-bearing anteroposterior and

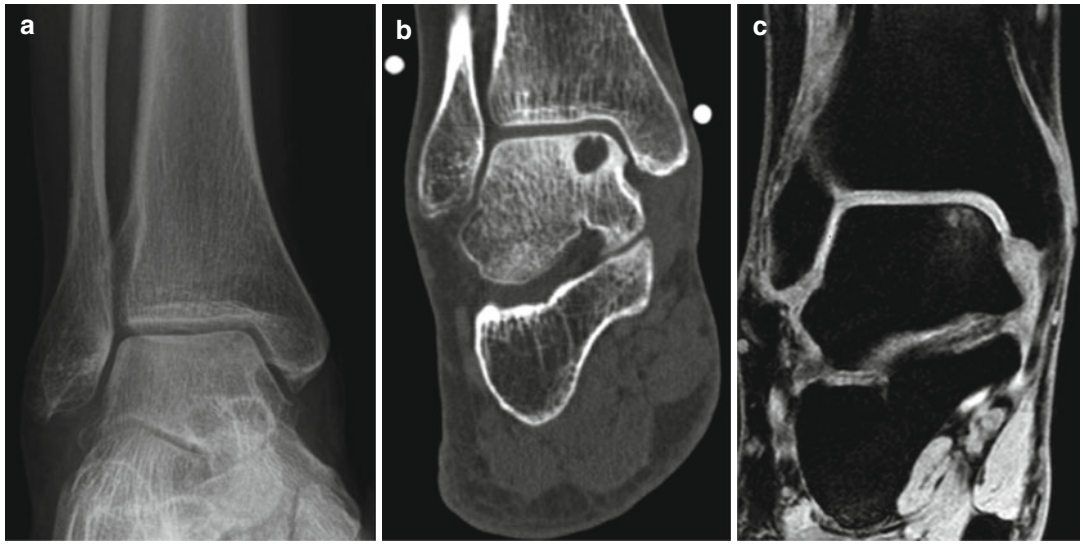


Fig. 81.4 (**a**) X-ray. (**b**) CT scan. (**c**) MRI of a medially located OCD. On the X-ray the OCD is not clearly visible and will be missed by routine screening. On the CT scan, a subchondral cyst is visible, secondary to the OCD. The MRI image is inconclusive in regard to the diagnosis of OCD. The image may also be indicative of a bone bruise. For surgical planning, the CT scan gives essential information on location and size of the defect

Box 81.1: Differential Diagnoses
- Posttraumatic synovitis
- OCD of tibial plafond
- Sinus tarsi syndrome
- Ligament laxity
- Osteoarthritis
- Subtalar joint pathology

lateral view [7]. OCDs may be visible as an area of radiolucency. Conventional radiography, however, only has moderate sensitivity (0.50–0.75) for these lesions, and visualization may be difficult (Fig. 81.4) [21]. In case of fragment displacement, it is more likely lesions will be visible. Routine radiographs fail to detect 30–50 % of OCDs [21]. Using a heel-rise view, developed to visualize the posterior lesions, instead of standard radiographs doubles the chance of diagnosing an OCD [22, 23].

The sensitivity and specificity for detecting an OCD using a helical CT scan, respectively, 0.81 and 0.99, are high, especially compared to standard radiographs [22]. A CT scan cannot visualize cartilage. The relevance of detecting the exact extend of damage to the cartilage, however, is unclear. Pain in OCDs is caused by involvement of bony tissue. Without bone involvement, lesions remain asymptomatic [21]. Additionally, a CT scan is used for preoperative planning. A CT helps determine the extent of the injury, detection of bony fragments, and in plantar flexion, assessment of the accessibility of the OCD can be made [24].

An MRI has shown to have a high accuracy for diagnosing OCDs. Verhagen et al. [22] showed a sensitivity and specificity of 0.96. Mintz et al. [25] reported a sensitivity of 0.95 and a specificity of 1.00 in patients after performing both an MRI and arthroscopy. It has to be taken in consideration the true lesion can be overestimated using an MRI, due to bony oedema, as lesion size is important for the treatment decision. Additionally, an MRI can give information concerning vascularization, healing and cartilage [26]. Using a stronger magnetic field may improve visualization of subchondral defects and cartilage [27].

81.5.1 Classification and Staging

In 1959, Berndt and Harty were the first to suggest a classification system to stage OCD

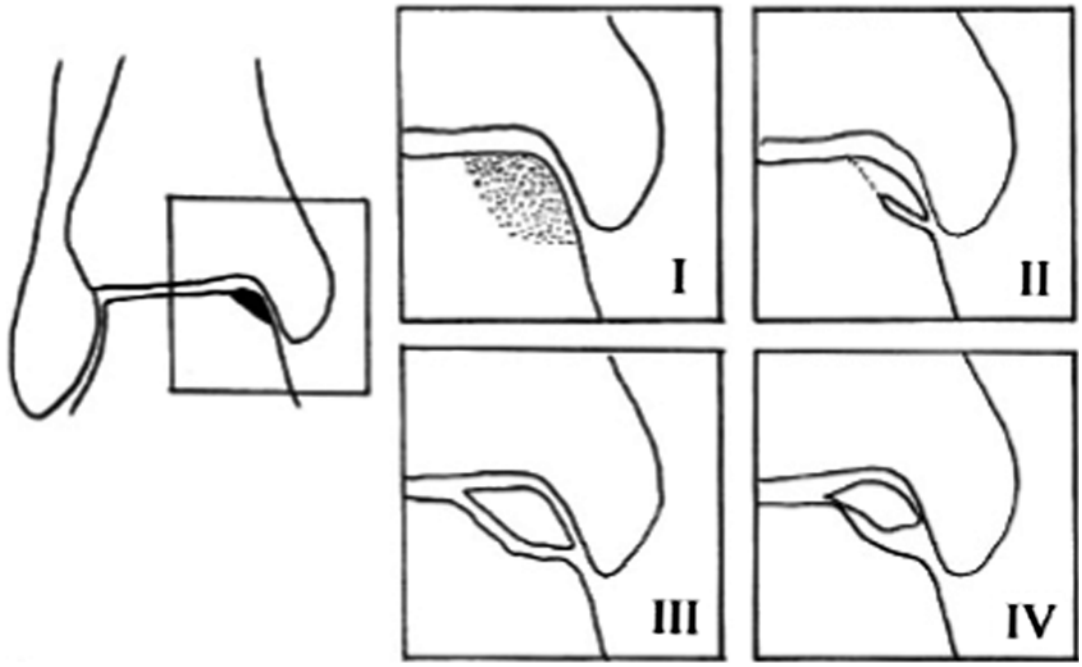


Fig. 81.5 Classification of osteochondral ankle defects by Berndt and Harty [14]

lesions at the time of surgery based on plain radiographs and surgical exploration of the ankle (Table 81.1, Fig. 81.5) [14, 21]. As this classification is based on both radiographic findings and surgical exploration, these findings might not fully correspond. Grade I, for example, describes local compression of cartilage and subchondral bone, which is usually not visible on conventional radiographs. Scranton and McDermott [28] added stage V: cystic lesions.

Ferkel et al. [29] developed a CT-based staging system that corresponds to the Berndt and Harty classification, emphasizing bony characteristics and the cystic component of the defect (Table 81.2). Additionally, to the classification system designed by Berndt and Harty, Ferkel et al. consider fragment separation, the presence of subchondral cysts and the extent of osteonecrosis. Loomer et al. [30] later included stage V: subchondral cysts.

Hepple et al. [31] created an MRI classification to grade OCDs, resembling the classification designed by Berndt and Harty (Table 81.3). None of these current grading systems is sufficient to direct treatment choice [7].

Table 81.1 Classification and staging of lesions according to Berndt and Harty [14]

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

Table 81.2 CT staging system according to Ferkel and Sgaglione [29]

Stage	Description
I	Cystic lesion within dome of talus with an intact roof on all views
IIa	Cystic lesion communication to talar dome surface
IIb	Open articular surface lesion with overlying non-displaced fragment
III	Non-displaced lesion with lucency
IV	Displaced fragment

In 1986 Pritsch et al. [32] were one of the first to grade talar OCDs according to cartilage quality assessed by arthroscopy. Cheng et al. [33] later further developed the arthroscopic staging of OCDs (Table 81.4).

Table 81.3 MRI staging system according to Hepple et al. [31]

Stage	Description
I	Articular cartilage damage
IIa	Articular cartilage damage with underlying fracture and bony oedema
IIb	Articular cartilage damage with underlying fracture without bony oedema
III	Detached, but undisplaced, osteochondral fragment
IV	Displaced fragment
V	Subchondral cyst formation

Table 81.4 Arthroscopic staging system based on cartilage quality according to Pritsch et al. [32] and Cheng et al. [33]

Stage	Description
A	Articular cartilage smooth and intact, but soft
B	Articular cartilage surface is rough
C	Fibrillation or fissuring of the cartilage present
D	Present osteochondral flap or exposed bone
E	Detached, but undisplaced osteochondral fragment
F	Detached and displaced osteochondral fragment

81.6 Treatment Strategy

Various treatments, both conservative and surgical, have been published for the treatment of symptomatic OCDs. Surgical techniques are mainly based on (1) debridement and bone marrow stimulation (microfracturing, drilling, abrasion arthroplasty), (2) securing a lesion to the talar dome (fragment fixation, retrograde drilling, bone grafting) or (3) development or replacement of hyaline cartilage (autologous chondrocyte implantation (ACI), osteochondral autograft transplantation (OAT), mosaicplasty, allografts). The preferred treatment depends on the patient's age, symptoms, duration of complaints and location and size of the lesion, as well as whether it concerns a previously treated OCD [7, 15].

81.6.1 Nonoperative Treatment

Asymptomatic or non-severe lesions are primarily treated conservatively for a period of 6 months, consisting of rest, ice, temporarily reduced weight bearing, restriction of (sporting) activities, use of non-steroidal anti-inflammatory drugs (NSAIDs) and, in case of giving way, an orthosis [9, 15]. Conservative treatment yields a success rate of 45 %. Nonoperative treatment may relieve symptoms for a short term; however, they often recur due to inadequate healing of the lesion. A trial period of nonsurgical treatment does not adversely affect surgery outcome. The treatment aims to unload the damaged cartilage, so oedema can resolve and necrosis is prevented [4, 9, 34, 35].

81.6.2 Debridement and Bone Marrow Stimulation

Surgical treatment may include excision of a (partially) detached fragment, leaving the defect untreated, excision and debridement or excision, debridement and bone marrow stimulation (BMS) using either an open or arthroscopic technique [36].

Symptomatic lesions are primarily treated by debridement and BMS in adolescents and in children if conservative treatment fails [37]. During debridement unstable cartilage is removed, including underlying necrotic bone, and cysts are opened and curetted. The mostly present sclerotic-calcified zone is perforated by drilling or microfracturing into the vascularized subchondral bone (Fig. 81.5). As the underlying intraosseous blood vessels are disrupted and growth factors are released, a fibrin clot is formed in the created defect. Formation of new blood vessels is stimulated, marrow cells are introduced into the OCD and multiple connections with the subarticular spongiosa are formed [36, 38]. In case of a cystic defect of ≥15 mm in diameter, a cancellous bone graft may be placed in the defect [39].

Transmalleolar antegrade drilling can be considered in case the OCD is difficult to reach because of its location on the talar dome. The defect can be drilled through the malleolus using a Kirschner (K)-wire about 3 cm proximal to the tip of the medial malleolus. The K-wire is

directed through the medial malleolus into the lesion, through the intact cartilage [36]. Whenever possible, transmalleolar drilling should be considered due to damage to the tibial plafond cartilage opposite the talar OCD [10].

Treatment by debridement and bone marrow stimulation is, with 78–86 % of good or excellent results, superior to other techniques for treating an OCD and is the current treatment of choice. Even though OAT showed similar results in an RCT, microfracture and chondroplasty are preferred because of less postoperative pain, lower costs, comparable results and avoidance of donor site morbidity [4, 39–41] (Fig. 81.6).

81.6.3 Securing a Lesion to the Talar Dome

Fragment fixation with one or two lag screws is preferred in an acute or semi-acute situation with a fragment ≥15 mm. Materials that can be used for fixation are Herbert screws, K-wires, absorbable fixation and fibrin glue [36]. Following failure after a period of 6 months of conservative treatment, fixation of an OCD in adolescents should always be considered [7].

In case of intact cartilage with a large subchondral cyst in primary OCDs, retrograde drilling, combined with cancellous bone grafting when necessary, may be the treatment of choice [7]. Retrograde drilling is also used in lesions that are hard to reach through the standard anterolateral and anteromedial portals. For medially located lesions, arthroscopic drilling can be done through the sinus tarsi, and for lateral lesions, the cyst is approached from anteromedial. By drilling through the posterior talar process, a posterior arthroscopic approach is possible. The aim of retrograde drilling is to induce revascularization of subchondral bone and subsequently stimulate formation of new bone. Here as well a graft may be placed in the defect. Retrograde drilling is the treatment of choice in case of large subchondral cysts with healthy overlying cartilage [36, 42].

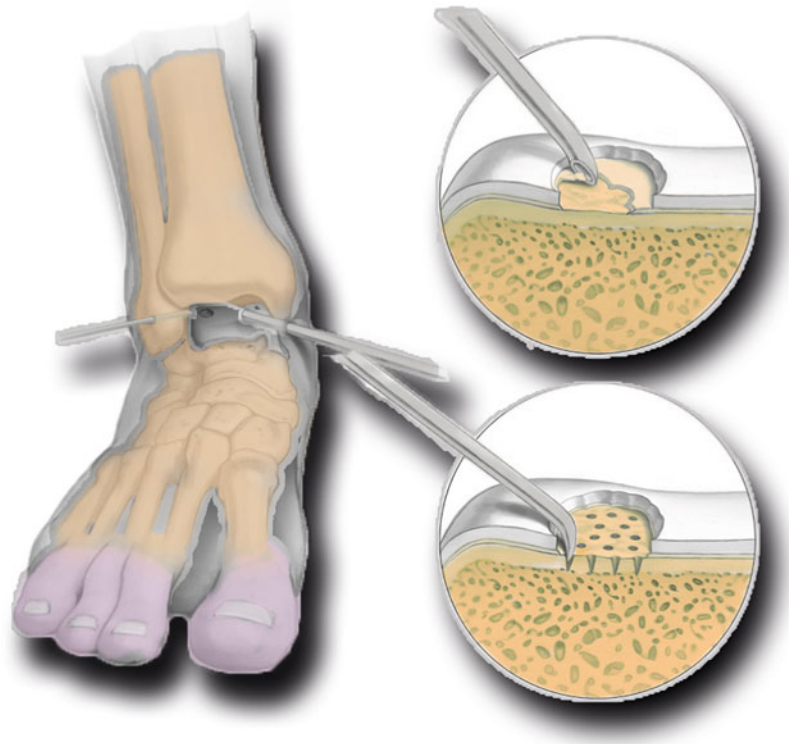


Fig. 81.6 Microfracture of an OCD

81.6.4 Development or Replacement of Hyaline Cartilage

When primary treatment fails, OAT and ACI are the options. For both techniques good results have been reported [39, 43, 44].

OAT has been introduced as an alternative to allografts in the treatment of OCDs. Two procedures have been developed: mosaicplasty and osteochondral autograft transfer system (OATS). These are reconstructive bone grafting techniques that consist of harvesting one or more osteochondral plugs from a lesser weight bearing area of the knee and transplanting them into the talar defect [45]. The grafts are subsequently transplanted into the prepared defect site on the talus. These techniques aim to reproduce (bio) mechanical and structural properties of the original hyaline cartilage. This procedure as well can be performed through an open approach and an arthroscopic procedure. The main indications for OAT involve large, often medial lesions, sometimes with a cyst underneath [36, 42]. OAT yields good to excellent results in 90–94 % at intermediate follow-up. However, this technique is associated with donor site morbidity, and often a medial osteotomy is required [39, 45–47].

ACI is the implantation of in vitro-cultured autologous chondrocytes, using a periosteal tissue cover after expansion of isolated chondrocytes. This technique aims to regenerate tissue with a high percentage of hyaline-like cartilage. Cultured chondrocytes are placed under a periosteal patch that covers the lesion. The technique is applied in lesions >1 cm^3 and no generalized osteoarthritic changes. Chondrocytes are harvested from either the knee or the region on the perimeter of the talar lesion. After the cells have been cultured for 6–8 weeks, a second procedure is performed. A stable border is created by curettage of the damaged articular surface and a periosteal patch is harvested from the tibia. The periosteal patch is sutured to the defect and sealed with fibrin glue. Subsequently the cultured chondrocytes are injected under the periosteal patch [39, 44, 48]. Matrix-based chondrocyte implantation (MACI) is also used. It differs from standard ACI in chondrocytes being embedded in a type I/III collagen membrane bilayer. The membrane is placed in the defect, as with ACI, but MACI requires no sutures. The membrane is secured using fibrin sealant. MACI is technically easier compared to ACI and does not require an osteotomy [49]. Disadvantages include the two-staged surgery, high costs and donor site morbidity [39, 44, 48].

81.6.5 Treatment Choice

Surgical treatment of talar OCDs remains controversial among orthopaedic surgeons. None of the current grading systems is sufficient to direct treatment choice [22]. Treatment should be graded by size of the lesions, location of the lesions and whether it concerns primary or secondary treatment. Age also plays a role. We tend to be more conservative in young patients [10].

In case of pure cartilage lesions, asymptomatic and low symptomatic lesions, conservative treatment is started for 6 months. Surgical treatment should be considered in case of failure of conservative treatment, or continuing or exacerbation of symptoms after 6 months, or in case of residual symptoms after previous surgical treatment (Table 81.5). Arthroscopic BMS is the treatment of choice in primary OCDs <15 mm. Defects of >15 mm have shown less good results compared to OCDs <15 mm [10, 36].

81.7 Surgical Technique BMS

The size and location of an OCD determine whether a standard 4.0-mm arthroscope is used during an anterior approach combined with maximal plantar flexion of the ankle or if a 2.7-mm arthroscope is used in combination with

Table 81.5 Best treatment options based on the talar OCD

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

mechanical distraction. In patients with unlimited plantar flexion, all anteriorly located lesions and lesions at the anterior part of the posterior half of the talus can be reached through an anterior approach [12, 50]. If lesions cannot be approached from anterior, a two-portal hindfoot approach or a medial malleolar osteotomy may offer a solution [34, 51].

The 4.0-mm scope is routinely used in combination with a 4.5- or 5.5-mm bone cutter shaver. In case of synovitis, a local synovectomy is performed with the ankle in dorsiflexion. The lesion is identified in forced plantar flexion by palpating the cartilage with a probe. A soft-tissue distractor can be applied if needed. The full-radius resector as bonecutter is introduced into the defect. In some cases, identifying the defect by introducing a spinal needle, probe or curette can be useful before introducing the resector. Identifying the anterior part of the defect and removing unstable cartilage and subchondral necrotic bone are important. Checking every step in the debridement procedure is done by regularly switching portals. After full debridement, the sclerotic zone is penetrated by a microfracture probe or a Kirschner wire. Postoperatively, a compression dressing is applied [7]. A hyaluronic acid injection after microfracture might improve clinical outcomes [52]. Overall arthroscopic treatment showed excellent to good results in 80–87 % of patients [22, 53].

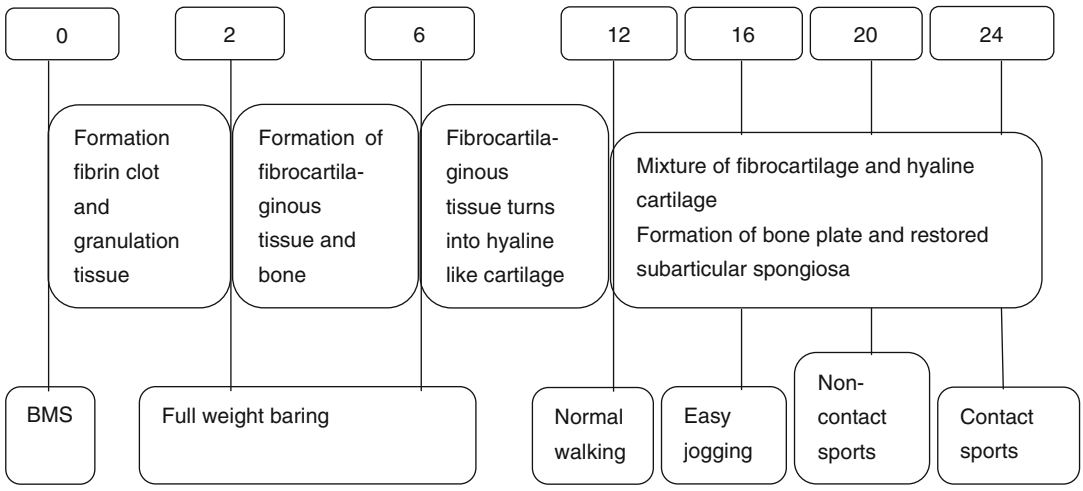
81.8 Rehabilitation

After BMS active plantar and dorsiflexion are encouraged. Partial weight bearing is allowed as tolerated. Progression to full weight bearing is allowed in 2–4 weeks in patients with central or posterior lesions up to 1 cm. Larger lesions and anterior lesions require partial weight bearing up to 6 weeks. Running on even ground is permitted after 12 weeks. Full return to normal and sporting activities is usually possible after 4–6 months of surgery [7]. A four-level activity scheme has been described (Table 81.6) [54].

The first phase aims to return to normal walking, which commences the day of the operation allowing partial weight bearing. Training active range of motion is important. Active plantar- and dorsiflexion is stimulated. Partial weight bearing provides nourishment by synovial fluid for chondrocytes. Full weight bearing stimulates osteoblasts in the formation of bone underneath the cartilage. At the end of the first phase, proprioception training is commenced to regain normal stability.

The second phase aims to resume running on even ground. Progression from walking to run-

Table 81.6 Rehabilitation scheme after bone marrow stimulation



ning on even ground is permitted between 12 and 16 weeks. Sometimes more proprioception training is needed. The range of motion should be normal. Controlled sideways movement is achieved by force, endurance and technical skill training. Pain and swelling should have ceased after 24 h of increased activity.

The third level of the activity phase is a return to non-contact activities. Full return to non-contact sports, depending on the size and location, is usually possible 20–24 weeks postoperatively. Training for speed, endurance, running and sprinting is continued. By the end of this phase, rope jumping, turning and twisting should be possible, without increased pain for more than 24 h.

Phase four 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, generation of explosive force, changing direction and other sports-specific movements.

Rehabilitation after other treatment options, like fixation or OATS, is slightly different. After fragment fixation, the non-weight-bearing period is 6 weeks followed by another 4–6 weeks of controlled weight bearing to ensure proper fixation.

After medial malleolar osteotomy, weight bearing depends on the surgical treatment of the osteochondral lesion. After OATS, running is not permitted until the graft has been incorporated [54].

Conclusion

Osteochondral defects are defects involving hyaline cartilage and subchondral bone. The lesions can differ in size and location. In case of an ankle OCD, there is often a history of ankle trauma, reporting an inversion injury.

Performing clinical examination, an ankle with an OCD may show little abnormality. Physicians must be aware of reported deep ankle pain, which cannot be provoked by joint line palpation. Conventional radiographs might be insufficient to show the lesion, whereas a CT scan may show talar or tibial OCDs.

BMS provides the solution in lesions <15 mm. Lesions >15 mm have shown less good results and fixation is advised. In case of asymptomatic lesions or lesions in younger patients, a conservative approach is advocated.

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