Clinical Article

Diagnosis and treatment of osteochondral defects of the ankle

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Abstract

An osteochondral defect of the talus is a lesion involving talar articular cartilage and subchondral bone. It is frequently caused by a traumatic event. The lesions may either heal, stabilise or progress to subchondral bone cysts. The subchondral cysts may develop due to the forcing of cartilaginous or synovial fluid with every step. Malalignment of the hindfoot plays an important role in the development of further degeneration. Plain radiographs may disclose the lesion. Modern imaging technology has enhanced the ability to fully evaluate and accurately determine the size and extent of the lesion, which are fundamental for proper treatment. Asymptomatic or low-symptomatic lesions are treated nonoperatively. For surgical treatment the following types of surgery are in clinical use: debridement and bone marrow stimulation, retrograde drilling, internal fixation, cancellous bone grafting, osteochondral autograft transfer, autologous chondrocyte implantation, and allograft transplantation. Although these are often successful, malalignment may persist with these treatment options. Calcaneal correction osteotomy may be suitable for osteochondral defects in select cases.

Introduction

An osteochondral defect (OD) is a lesion involving the articular cartilage and its subchondral bone. If only cartilage is involved in the pathology, the term chondral defect is used. Many synonyms are used, including osteochondritis dissecans,[1] transchondral fracture,[2] flake fracture,[3] talar dome fracture,[4] osteochondral fracture,[5] osteochondral lesion[6] and osteochondral defect.[7]

In the eighteenth century Monro was the first to report the presence of cartilaginous bodies in the ankle joint.[8] In 1888 König used the term osteochondritis dissecans to describe loose bodies in the knee joint and suggested that these were the result of spontaneous necrosis.[9] Since then, several aetiologies for these lesions have been suggested. Trauma is known to be the most important aetiologic factor, but ischaemia and idiopathic osteochondral ankle lesions do occur.[10,11] The most common location of osteochondral defects is in the knee, followed by the talar dome.[12]

ODs can either heal and remain asymptomatic or progress to deep ankle pain on weightbearing, prolonged joint swelling, recurrent synovitis, diminished range of motion and formation of subchondral bone cysts. The development of an osteochondral lesion may have a sudden onset, but the development of a subchondral cyst is most often a slow process.

Early accurate diagnosis of ODs of the talus is important because optimal ankle joint function requires talar integrity.[13] However, an OD is often not recognised and therefore not adequately treated. The non-recognition is mainly due to the fact that the lesion produces symptoms that cannot be distinguished from the previous trauma, and it cannot always be identified on plain radiographs.

Even though elaborate knowledge exists concerning osteochondral lesions of the talus, its aetiology and pathogenesis are still not fully understood. Increasing attention is paid to invasive and sometimes expensive surgical treatments, while research for pathogenesis of the lesions has been somewhat neglected. In order to treat osteochondral lesions in all its dimensions, more should be known about their natural history.

For the last decade great developments have been made in their surgical treatment. Despite advancements in options like osteochondral autograft transfer system (OATS) or autologous chondrocyte implantation (ACI), arthroscopic debridement and bone marrow stimulation remains the best treatment that is currently available for defects up to 15 mm in diameter.[14,15] In larger (cystic) defects this treatment is less successful, hence there is more debate.[16,17]

In this review the natural history, diagnosis and treatment options for ODs of the talus are summarised.

Aetiology

A traumatic insult is widely accepted as the most important aetiologic factor of an osteochondral lesion of the talus. Trauma causing the lesion may be a single event or consist of a series of repeated, less intense (micro) traumas.[10,18,19] For lateral talar lesions trauma has been described in 93-98% and for medial lesions in 61-70%.[1,15] Because not all patients report a history of ankle injury, a subdivision can be made in the aetiology of non-traumatic and traumatic defects.

Non-traumatic ODs are called osteochondritis or osteochondrosis dissecans.[20] Ischaemia, subsequent necrosis and possibly genetics are aetiologic factors in non-traumatic ODs.[10] Furthermore, ODs in identical twins[21,22] and in siblings[23] have been described. Less reported possible causes are metabolic, vascular, endocrine and degenerative factors, as well as morphologic abnormalities.[2,12,18]

In the aetiology of traumatic ODs ankle sprains play the largest role. When a talus twists inside the ankle mortise, the cartilage lining of the talus can be damaged. This may lead to a bruise and subsequent softening of the cartilage or even a crack in the cartilage with subsequent delamination. Separation may occur in the upper layer, as a result of shearing forces, or may occur in the subchondral bone. Fragments may break off and float loose in the ankle joint, or remain partially attached and stay in position. The subchondral fracture has no soft-tissue attachments and is highly susceptible to subsequent avascular necrosis.[12]

Berndt and Harty clearly described the trauma mechanism in cadaver ankles.[2] They were able to reproduce lateral defects by strong inversion of a dorsiflexed ankle, leading to compression of the lateral border of the talar dome against the face of the fibula. When the lateral ligament ruptured, avulsion of the chip began. With the use of excessive inverting force, the talus within the mortise was rotated laterally in the frontal plane, impacting and compressing the lateral talar margin against the articular surface of the fibula. A portion of the talar margin was sheared off from the main body of the talus, which caused the lateral osteochondral defect. A medial lesion was reproduced by plantarflexing the ankle in combination with slight anterior displacement of the talus on the tibia, inversion, and internal rotation of the talus on the tibia.

The most common location of osteochondral lesions in patients with ankle trauma is on the anterolateral or posteromedial side of the talar dome. The lateral lesions are usually shallow and wafer-shaped, indicating a shear mechanism of injury. Medial lesions in contrast are usually deep and cup-shaped, indicating a mechanism of torsional impaction and axial loading.[1,2,15,24-26] Because of their shape, lateral lesions are more frequently displaced than medial lesions.

Natural history

Normal articular cartilage comprises chondrocytes and an extracellular matrix which consists primarily of collagen and proteoglycans.[27] Cartilage is avascular and is nourished by the intra-articular fluid. The tissue fluid of the cartilage matrix, which comprises about 75% of the total weight of cartilage, functions as a transport medium[28] by its free exchangeability, whether extra- or intra-fibrillar.[29]

The progression of ODs may be the result of repetitive fluid pressure from the damaged cartilage. During ankle trauma, microfractures often arise in the subchondral bone plate.[30] The damaged subchondral bone is less able to support the overlying cartilage.[31] When overlying cartilage is not supported by the underlying bone plate it loses quality due to loss of proteoglycans and glycoproteins.[28,32] In this situation liquid not only flows within the cartilage, but can also enter the subchondral bone through the microfractured area *(Figure 1).* This fluid pressure causes osteolysis. An intermittent or continuous high local pressure can interfere with normal bone perfusion and lead to osteonecrosis, bone resorption and formation of lytical areas.[33-37]

The damaged cartilage may also function as a valve, allowing intrusion of fluid from the joint space into the subchondral bone but not in the opposite direction.[38] On the weightbearing phase of gait there is full contact between the talar and tibial cartilage over the talar shoulders.[39] During this phase, pressures in opposing talar and tibial cartilage are theoretically identical, which may result in the forcing of fluid in the direction of least resistance, i.e. the damaged subchondral bone. During unloading of the joint, joint space fluid may re-enter the articular cartilage. On the next weightbearing cycle, this fluid is intruded in the subchondral bone. This repetitive mechanism would result in a vicious cycle, causing the shift of synovial fluid into the damaged subchondral talar bone, thereby slowly developing a subchondral cyst *(Figure 2).*

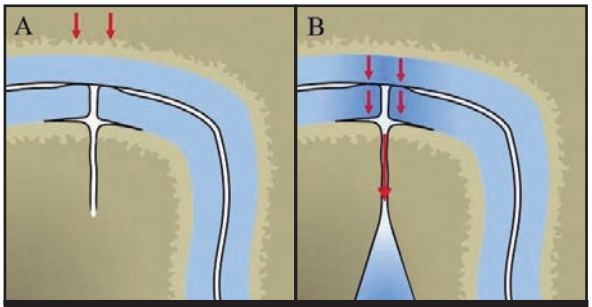

Figure 1:
(A) Fissure in the cartilage and the subchondral bone plate. (B) When loaded the water is forced out of the cartilage into the subchondral bone.

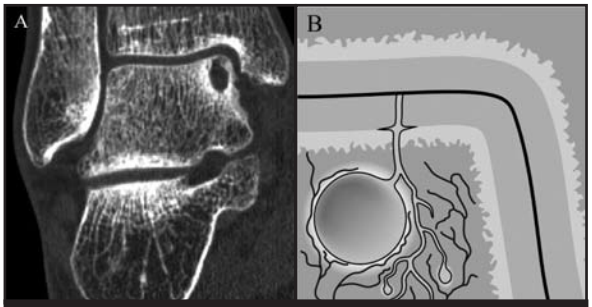

Figure 2:
(A) Coronal CT of a young patient with a long history of deep ankle pain (6 years). The CT shows an opening in the subchondral bone plate. Subchondral osteolysis has caused a subchondral cyst. (B) A schematic situation of the CT image and shows the mechanism of cyst formation. The black lines represent nerve endings in the subchondral bone.

Varus or valgus malalignment of the ankle joint may also play an important role in the natural history by increasing the contact pressure. The ankle joint has a high congruency. A decrease in joint congruence will increase contact pressure per area.[40] More displacement corresponds to increasing contact pressure. Ramsey and Hamilton have shown that a 1 mm lateral talar shift reduces the contact area by 42%, and a 2 mm lateral shift reduces the contact area by 58%.[40] Long-term follow-up studies have demonstrated that patients with persistent displacement of ankle fractures had poorer long-term results than those without persistent displacement.[41] Bruns *et al* demonstrated that in varus and supination the maximum pressure is located on the medial border of the talus, while in valgus and pronation the maximum pressure is located on the lateral talar border.[42] Increased pressure on an existing osteochondral defect may negatively influence the natural history of the lesion.[31] It is therefore important to detect and correct malalignment in patients with an osteochondral defect of the ankle.

Clinical presentation

ODs often cause deep ankle pain on weightbearing, prolonged joint swelling, recurrent synovitis, diminished range of motion, and formation of subchondral bone cysts. A differentiation has to be made between the acute and the chronic situation.[12] In the acute situation, symptoms of an osteochondral lesion of the talus are often unrecognised since the swelling and pain from the lateral ligament lesion prevails. In patients with an isolated ligamentous ankle injury these symptoms usually resolve after functional treatment within two to three weeks. If symptoms have not resolved within 4–6 weeks, an OD should be suspected. Locking and catching are symptoms of a displaced fragment.

Chronic lesions typically present as persistent ankle pain after a prior history of an inversion injury of the ankle. Pain is usually experienced as deep ankle pain, during or after activity. Reactive swelling or stiffness may be present, but absence of swelling, locking or catching does not rule out an OD. Most patients demonstrate a normal range of motion with absence of recognisable tenderness on palpation and absence of swelling.

Diagnosis and classification

The total incidence of symptomatic and asymptomatic localised traumatic articular cartilage damage and ODs is unknown. With the increased awareness and newer diagnostic techniques, the incidence of OD seems to have increased.[43]

Plain radiographs should be the initial investigation of suspected osteochondral lesions of the talus, after careful history-taking and physical examination of the ankle. These consist of weight-bearing anteroposterior mortise and lateral views of both ankles. The sensitivity and specificity of the combination of medical history, physical examination and radiography are 59% and 91%, respectively.[44] The radiographs might not reveal any pathology, or show an area of detached bone, surrounded by radiolucency. Initially, the damage may be too small to be visualised on routine radiography. Only in cases of a large osteochondral defect, the initial X-ray may be positive. By repeating the imaging studies in a later stage, the abnormality sometimes becomes apparent. A heelrise view with the ankle in a plantarflexed position may reveal a posteromedial or posterolateral defect.[44]

Computed tomography (CT) is useful in determining the size, location, shape and degree of displacement of osteochondral fragments.[45] CT is often invaluable in pre-operative planning to define the exact size and location of the lesion.[12,26,45] 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 should be 1 mm. However, CT is limited in its ability to visualise articular cartilage and bone bruises.[10]

Table I: Bernt and Harty (1959)

Grade I:	A small compression fracture
Grade II:	Incomplete avulsion of a fragment
Grade III:	Complete avulsion of a fragment without displacement
Grade IV:	A displaced fragment

Magnetic resonance imaging (MRI) allows multiplanar evaluation and offers the advantage of visualising the articular cartilage and subchondral bone as well as oedema and other features of the surrounding soft tissue. Nevertheless in diagnosing an osteochondral defect, CT has proven to be as valuable as MRI.[44]

In 1959, Berndt and Harty suggested a classification system for staging the lesions based on plain radiographs of the ankle *(Table I)*. 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 avulsion or partial detachment of the 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.[2] Later, classification systems based on MRI, CT and arthroscopic findings were made.[2,5,43,46-48] The use of these classification systems is questionable since none of the systems are dually related to the current treatment options.[15]

Treatment

There are widely published non-surgical and surgical techniques for treatment of symptomatic osteochondral lesions.[49]

Non-surgical treatment

Many authors have suggested that the decision to operate should depend on the grade of the lesion. Berndt and Harty grade I and II lesions should be managed non-surgically for up to 1 year to allow for resolution before resorting to surgery.[24,26,50-52] Nevertheless, a meta-analysis of 14 studies with a total of 201 patients showed only a 45% success rate of non-surgical treatment of grade I, grade II, and medial grade III talar OD (not all injury types were specified).[15] Non-surgical treatment of chronic lesions (>6 weeks) had a success rate of 56%.[15]

Asymptomatic or low symptomatic ODs should be treated with rest and/or restriction of (sporting) activities, non-steroidal anti-inflammatory drugs (NSAIDs), or cast immobilisation for 3 weeks up to 4 months.[15] The aim is to either give the bruised talus rest so oedema can resolve and necrosis is prevented, or stimulate reattachment of the (partly) detached fragment to the surrounding bone.

Surgical treatment

For years there has been an ongoing debate about the optimal surgical treatment regimen. Debridement of the lesion has been performed progressively since the 1950s. This method was later combined with bone marrow stimulation, by means of drilling or microfracturing, with favourable results.[50]

Until the mid-1980s, surgical treatment of talar ODs consisted of open procedures. In the case of posteromedially located ODs, most surgeons performed an osteotomy of the medial malleolus to identify and treat the lesion.[24,53-55] The introduction of arthroscopy has led to less invasive operative procedures and has gained much popularity.[6,7,56,57]

In general, failure of non-surgical management for symptomatic lesions necessitates surgical intervention. Various surgical techniques for symptomatic ODs will now be discussed.

Bone marrow stimulation

This is the treatment of choice for most lesions. With this technique all unstable cartilage including the underlying necrotic bone is removed. Any cysts underlying the defect are opened and curetted. After debridement, multiple connections with the subchondral bone are created. They can be accomplished by drilling or microfracturing. The objective is to partially destroy the calcified zone that is most often present and to create multiple openings into the subchondral bone. Intraosseous blood vessels are disrupted and the release of growth factors leads to the formation of a fibrin-clot *(Figure 3)*. The formation of local new blood vessels is stimulated, marrow cells are introduced in the OD and fibrocartilaginous tissue is formed.[58] In case of a large OD a cancellous bone graft can be placed.

Verhagen *et al* reported that debridement and bone morrow stimulation of the lesion by arthroscopy was successful in 87% and by open procedures in 84% of cases.[15] These good results were confirmed more recently.[59,60]

Retrograde drilling

Retrograde drilling is done for primary ODs when there is relatively intact cartilage with a large subchondral cyst. The aim is to induce subchondral bone revascularisation and subsequently to accomplish new bone formation. A cancellous graft may be placed to fill the defect. Taranow *et al* reported successful outcome in 13 of 16 patients (81%).[48]

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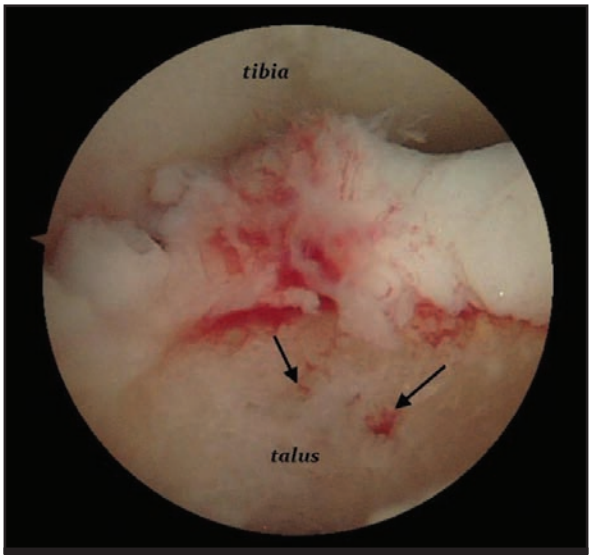

Figure 3:
Arthroscopic view of the result of debridement and microfracturing of an osteochondral defect of the talus. The arrows indicate microfracture holes; bleeding of the subchondral bone to create a fibrin clot is also visible.

Internal fixation

With this technique, the loose fragment is not removed but fixed to the underlying bone by a screw, Kirschner wires, absorbable fixation, or fibrin glue.[61,62] DeLee proposed that internal fixation is indicated when the injury occurs acutely and the fracture is larger than one-third the size of the respective dome.[63] Stone *et al* suggested that the lesion should be at least 7.5 mm in diameter and that the patient should be young for surgical fixation.[26] A meta-analysis of three studies with a total of 11 patients showed a 73% success rate of internal fixation with a variation from 40 to 100%.[15]

Autologous chondrocyte implantation (ACI)

ACI is the implantation of in vitro cultured autologous chondrocytes using a periosteal tissue cover after expansion of isolated chondrocytes. ACI has been popularised by Brittberg and Petersen since 1994.[64,65] Since that time, ACI has been performed in over 25 000 patients: 95% in the knee, 3% in the ankle, and 2% in other joints.[12] Based on promising early results with ACI in the knee, surgeons have now started using ACI for osteochondral lesions of the talus. For patients with an OD who remain symptomatic after primary surgical treatment, ACI is considered a valuable treatment option. The defect should be focal, contained, and preferably more than 1.5 cm in diameter. Large lesions with subchondral cysts may also be treated with ACI, using the 'sandwich technique', i.e. filling the base of the defect with autologous cancellous bone.[65,66]

Contraindications to ACI are bipolar lesions ('kissing lesions') and diffuse degenerative joint changes. Skeletal malalignment and ligamentous instability are also contraindications, unless they are concomitantly corrected at the time of surgery.[66]

Osteochondral autograft transfer (OATS)

OATS consists of the harvesting of one or more osteochondral plugs in a lesser weight bearing area of the knee and transplanting them into the talar defect.[67,68] The aim is to restore the articular surface with hyaline cartilage. One single graft or several smaller grafts (i.e. mosaicplasty) may be used. The use of several grafts provides a better match to the curvature of the talar dome and surface area of the defect, and may reduce donor site morbidity.[69,70]

Although X-ray evaluation and CT may help to determine the extent of the lesion, indication of OATS is rather based on the size determined after excision of the defect. OATS can also be offered to patients in case of failed primary treatment. An essential aspect of the procedure is insertion of the osteochondral plugs perpendicular to the recipient site. Due to the constrained configuration of the talocrural joint with its highly contoured articular surfaces, the best approach is by means of open arthrotomy, most of the times using a malleolar osteotomy. 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 mini-arthrotomy.[12] In case the knee is precluded as a donor site, the ipsilateral talar articular facet may also be used as a harvest site of small sized grafts (2.7 or 3.5 mm in diameter).[71]

Hangody *et al* reported on the outcomes of the talar mosaicplasty, with the medial or lateral femoral condyle as the donor site. In 36 patients, multiple grafts of 4.5 × 3.5 mm were harvested to reconstitute the talar defects, which averaged 1 cm in diameter. Good/excellent results were achieved in 34 patients (94%) at a follow-up of 2 to 7 years.[70]

Calcaneal correction osteotomy (CCO)

Clinical and basic scientific investigations have shown that loading and motion of the joint can influence the healing of articular cartilage and joints.[31] The ankle joint has a high congruency. A decrease in joint congruence and malalignment will increase contact pressure per area, and may lead to osteolysis and large osteochondral defects.[37,40,72,73]

In general the different treatment options as described above have good results. However, with these treatment options malalignment is not corrected. CCO may be necessary to restore the natural congruency of the ankle joint.

Based on promising early results with ACI in the knee, surgeons have now started using ACI for osteochondral lesions of the talus

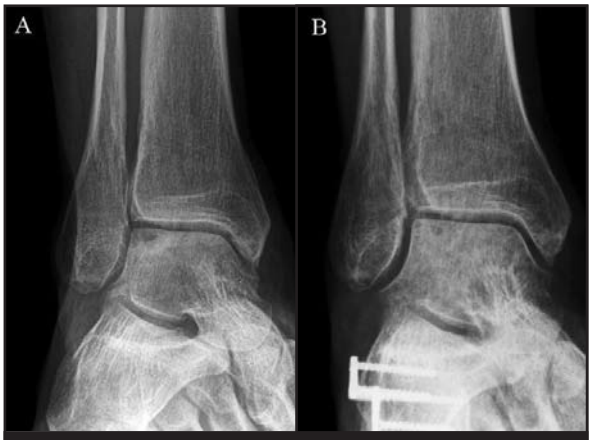

Figure 4:
Weight-bearing anteroposterior radiographs of the right ankle of a 45-year-old male patient with valgus malalignment who had persisting complaints of deep ankle pain over four years. He had been treated by arthroscopy twice. (A) The pre-operative radiograph shows a large osteochondral defect on the lateral talar dome. The lesion was treated by medial displacement of the hindfoot by calcaneal osteotomy with plate-fixation. (B) The situation six months postoperatively, in which the lesion was almost healed.

CCO is an established procedure for acquired adult flatfoot,[74] hindfoot valgus after recurrent pronation trauma and deltoid ligament insufficiency,[75] and malaligned ankle with deformity.[76] We currently perform CCO to treat patients with malaligned hindfeet who have persistent complaints after initial arthroscopic treatment *(Figure 4).*

Conclusion

Initial trauma causes an (osteo)chondral defect. During loading, compressed cartilage forces water into microfractured subchondral bone, which may cause osteolysis and the slow development of a subchondral cyst. To prevent further degeneration early diagnosis and accurate treatment are necessary. There are various treatment options for OD. Arthroscopic debridement and bone marrow stimulation, by nature of the minimally invasive approach, has great advantage in treating typical defects of up to 1.5 cm in diameter. For larger or secondary ODs the optimal treatment may consist of osteochondral autograft transfer, cancellous bone graft and/or autologous chondrocyte implantation. However, with these treatment options malalignment may still exist, while malalignment plays an important role in development of further degeneration in OD. Therefore, calcaneal correction osteotomy may be suitable for ODs in selected cases.

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