Treatment of osteochondral defects of the talus
Traitement des lésions ostéochondrales de l’astragale

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Summary
This review article provides a current concepts overview of osteochondral defects of the talus, with special emphasis on treatment options, their indications and future developments. Osteochondral defects of the talar dome are mostly caused by a traumatic event. They may lead to deep ankle pain on weight-bearing, prolonged swelling, diminished range of motion and synovitis. Plain radiographs may disclose the lesion. For further diagnostic evaluation, computed tomography (CT) and magnetic resonance imaging (MRI) have demonstrated similar accuracy. Computed tomography is preferred for preoperative planning. Treatment options are diverse and up to the present there is no consensus. Based on the current literature, we present a treatment algorithm that is mainly guided by the size of the lesion. Asymptomatic or low-symptomatic lesions are treated nonoperatively. The primary surgical treatment of defects up to 15 mm in diameter consists of arthroscopic debridement and bone marrow stimulation. For large cystic talar lesions, retrograde drilling combined with a bone graft is an important alternative. In adolescents or in (sub)acute situations, in which the fragment is 15 mm or larger, fixation of the fragment is preferred. Osteochondral autograft transfer and autologous chondrocyte implantation (ACI), with or without a cancellous bone graft, are recommended for secondary cases as well as large lesions.

Introduction
An osteochondral defect (OD) is the collective term for focal lesions involving the articular cartilage and 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]. A differentiation should be made between traumatic and nontraumatic origin (i.e. osteochondritis dissecans). A traumatic event may lead to (partial) detachment of an (osteo)chondral fragment, which may further evolve in the formation of a subchondral cyst with or without osteonecrosis. There is sometimes confusion...
between traumatic and nontraumatic because a nontraumatic OD may become symptomatic after trauma.

In 1856, Monro first reported the presence of cartilaginous bodies [8]. In 1888, König used the term osteochondritis dissecans for loose body formation associated with articular cartilage and subchondral bone fracture [9]. He referred to an inflammatory process, although this has never been proved to be involved in the pathology. It was not until 1922 that the first report on osteochondritis dissecans in the ankle was published [10]. The talar dome is the second most common location in the human body; most occur in the knee [11].

An OD is often not recognized and therefore not adequately treated. The nonrecognition is mainly due to the fact that the lesion produces symptoms of previous trauma, and it cannot always be identified on plain radiographs [12]. After standard treatment for acute ankle sprains, residual symptoms are reported in 33% of patients [13]. In these cases the possibility of an OD should be considered.

The talus has a limited reparative capacity because of its restricted vascular supply [14]. Inappropriate treatment of OD may result in chronic ankle pain, functional impairment, subchondral cyst formation, and eventually osteoarthritis of the ankle [14–16].

For the last decade great developments have been made in the surgical treatment. Despite advancements in options like osteochondral autograft transfer system (OATS) or autologous chondrocyte implantation (ACI), arthroscopic debridement and bone marrow stimulation remain the best treatment that is currently available for defects up to 15 mm in diameter [17,18]. In larger (cystic) defects this treatment is less successful, and hence there is more debate [19,20].

The aim of this article is to provide an overview of treatment options and their indications for ODs of the talus, based on the current evidence.

Etiology

In 1985, trauma was described in 98% of lateral lesions and in 70% of medial lesions [1]; more recently, 93% for lateral and 61% for medial lesions were reported [18]. As not all patients report a history of ankle injury, a subdivision can be made in the etiology of nontraumatic and traumatic defects.

Ischemia, subsequent necrosis and possibly genetics are etiologic factors in nontraumatic ODs [14]. Furthermore, ODs in identical twins [21] and in siblings [22] have been described. Less reported possible causes are metabolic, vascular, endocrine and degenerative factors, as well as morphologic abnormalities [2,11,23].

In the etiology of traumatic ODs, ankle sprains play the largest role. A severe ankle sprain may cause a small fracture and subsequent impaired vascularity, leading to the formation of an OD. Alternatively, the cause may not be a single event but may consist of a series of repeated, less intense injuries [14,23]. Microtraumas caused by repetitive surface loading or excessive stress can lead to cartilage cellular degeneration or apoptosis and thickening of the subchondral bone [24].

Mechanism of injury

During an ankle sprain the talus twists inside the ankle mortise, which may lead to a bruise and subsequent softening or even delamination of the cartilage. Separation may occur in the upper layer, as a result of shearing forces, or may occur in the subchondral bone. Osteocartilaginous fragments either remain partially attached or become loose bodies in the ankle joint. The subchondral fracture has no soft tissue attachments and is highly susceptible to subsequent avascular necrosis [11]. The repetitive forcing of synovial fluid into the underlying cancellous bone with every step of walking may create a subchondral cyst [25]. The repetitive fluid pressure may prevent healing of a subchondral cyst.

Berndt and Harty clearly described the trauma mechanism in cadaver ankles [2]. They were able to reproduce a lateral defect by strong inversion of a dorsiflexed ankle, leading to compression of the lateral border of the talar dome against the face of the fibula. Partial detachment of the chip occurred when the lateral ligament ruptured. They reproduced medial lesions by plantar flexion and inversion of the ankle combined with slight anterior displacement and lateral rotation of the tibia upon the talus.

The lateral lesions are typically shallow and wafer-shaped, indicating the shear mechanism of injury [15]. Because of their shape, lateral lesions are more frequently displaced than medial lesions. In contrast, medial lesions are generally deep and cup-shaped, indicating a mechanism of torsional impaction [15]. These lesions are usually larger than lateral lesions [26].

Epidemiology

With the increased awareness and newer diagnostic techniques, the incidence of OD seems to increase [27]. In 1955, Bosien et al. described an incidence of 7% in 113 patients conservatively treated for acute lateral ankle ligament ruptures [13]. Later, van Dijk et al. reported 4% fresh talar dome lesions and 67% fresh chondral lesions of any kind in 30 patients who had operative repair of acute ruptures of lateral ligaments [28]. More recently, an even higher incidence was reported, namely 41% of 86 patients with anterior talofibular ligament disruptions and 71% of 92 patients with distal fibular fractures [29]. However, the majority of these reported lesions were located at the cartilage covering the anterior aspect of the medial malleolus and the opposite medial talar facet or the anteromedial rim of the tibiotalar joint [28,29]. Accordingly, talar ODs were found in 28% [30] and 40% [31] of patients with ankle fractures treated by arthroscopically assisted open reduction and internal fixation. In these series the highest incidence was found in patients with distal fibular fractures [30,31].

Most ODs are localized on the posteromedial (58%) or anterolateral (42%) talar dome (Fig. 1) [18], although anteromedial, posterolateral and central lesions also occur [26]. In a large magnetic resonance imaging (MRI) survey of 428 affected ankles 53% of the lesions were localized centromedially and 26% centrolaterally [26].

In 4–7% of patients the occurrence of the defect is bilateral [2,6,15], suggesting nontraumatic osteochondritis...
Figure 1 Three-dimensional CT-reconstruction of the left ankle of a 26-year-old male patient with a large osteochondral defect typically localized on the posteromedial (PM) talar dome. The usual localization of an anterolateral (AL) defect is also indicated (*).

Clinical presentation

An OD often causes deep pain, swelling, recurrent synovitis, and sometimes locking complaints. A differentiation has to be made between the acute and chronic situation [11]. In the acute situation symptoms of the OD compare to those of acute ankle injuries, including lateral or medial ankle pain, swelling and limited range of motion. In patients with an isolated ligamentous ankle injury these symptoms usually resolve after functional treatment within two to three weeks. If symptoms do not resolve after three to six weeks an OD should be suspected. These patients usually present with persisting symptoms and sometimes a limited range of motion. Locking and catching are symptoms of a displaced fragment. In most patients with a nondisplaced lesion the symptoms in the acute situation cannot be distinguished from the soft tissue damage.

Chronic lesions typically present as persistent or intermittent deep ankle pain, during or after activity. Reactive swelling and stiffness may be present, but absence of swelling, locking, or catching does not rule out an OD. There may be a normal range of motion, with the absence of swelling and absence of recognizable tenderness on palpation.

Diagnosis

Routine radiographs of the ankle should be obtained 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 [12]. The radiographs may not reveal any pathology, or show an area of radiolucency (Fig. 2A). Initially the damage may be too small to be visualized on a routine X-ray. The OD sometimes becomes apparent on radiographs at a later stage. A posteromedial or posterolateral defect may be revealed by a heelrise mortise view with the ankle in plantar flexion (Fig. 2C) [12].

For further diagnostic evaluation MRI or computed tomography (CT) are often used, with similar accuracy [12]. A multislice helical CT-scan is useful for defining the exact size and location of the lesion and is therefore preferred for preoperative planning (Fig. 3) [11,32]. The scanning protocol involves “ultrahigh resolution” axial slices with an increment of 0.3 mm and a thickness of 0.6 mm. Multiplanar coronal and sagittal reconstructions should be 1 mm.

Classification

A number of classifications have been proposed, based on radiography, CT, MRI, and arthroscopy [2,5,27,29,33,34]. The first and most frequently used classification is from Berndt and Harty [2]:

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

Scranton and McDermott later added Stage V, representing cystic lesions [25].

CT-scans are increasingly used in the preoperative workup. A CT-classification was therefore introduced in 1993, resembling the above classification, with stage V representing a radiolucent defect [27]. None of the current grading systems, however, is sufficient to direct the choice of treatment.

Treatment

Various surgical techniques for symptomatic ODs have been published. These are generally based on one of the following three principles [11]:

- debridement and bone marrow stimulation (microfracturing, drilling, abrasion arthroplasty), with or without loose body removal;
- securing a lesion to the talar dome (fragment fixation, retrograde drilling, cancellous bone grafting);
- development or replacement of hyaline cartilage (osteochondral autograft transfer, ACI, allografts).

For years there has been an ongoing debate about the optimal treatment regime. Debridement of the lesion has been performed progressively since the 1950s [2]. This
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Figure 2  Weight-bearing anteroposterior (A) and lateral (B) radiographs of the right ankle of a 36-year-old male patient showing radiolucency in the medial talar dome (arrow), indicating an osteochondral defect. A 4 cm heelrise view (C) of the same ankle reveals the posteroomedial osteochondral defect more clearly (arrow).

method was later combined with bone marrow stimulation, by means of drilling or microfracturing, with favorable results [35]. With the development of ankle arthroscopy, this combined procedure gained much popularity [6,7,36]. Nowadays, arthroscopic debridement and bone marrow stimulation are the mostly performed procedure for OD. Publications on treatment options for talar ODs were bundled in a systematic review performed in our institution in July 1998 [17] and updated in June 2000 [18]. Twenty-one investigations with a total of 272 patients were identified. The success rate of debridement and bone marrow stimulation was superior to other methods. Arthroscopy was successful in 87% and open procedures in 84% of the cases [18]. These good results were confirmed more recently [37,38]. However, OATS and ACI were not included due to few studies, and sizes of the treated lesions were not described.

In the case of a cystic lesion, debridement may be supplemented by cancellous bone grafting [11,38,39]. However, the limited availability of cancellous bone and pain at the donor site remain disadvantages [40]. Large osteochondral fragments can be fixed by screws, with a success rate of 73% (Fig. 4) [17].

Recently, more developments have taken place. OATS and ACI as original procedures for osteochondral lesions of the knee have evolved to suitable treatment methods for certain lesions of the talus [41—43]. Excellent results have been published, although numbers are still fairly small and no long-term follow-up is available [43—46].

In 2005 an expert consensus on the treatment of OD was achieved during the World Consensus Conference of the International Society of Arthroscopy, Knee surgery and Orthopaedic Sports medicine and International Federation of Sports Medicine (ISAKOS—FIMS) [47]. The expert group agreed that debridement and bone marrow stimulation are the first step in the treatment of most symptomatic OD and presented a useful guideline. For failed primary treatment they recommended to consider OATS.

Based on the current literature, a revised treatment guideline is presented (Table 1), which will be further discussed in the Discussion section.

Surgical approach

The size and location of the lesion as well as the type of surgical treatment determine the surgical approach. The preferred approach of most lesions is by means of anterior arthroscopy [48]. Alternative approaches are posterior arthroscopy by means of a two-portal hindfoot approach [49] and open arthrotomy with or without a medial malleolar osteotomy [50,51]. Arthroscopy offers the advantages

Figure 3  CT-scans of the right ankle of a 26-year-old female patient showing a cystic posteroomedial osteochondral ankle defect. A. Axial slice. B. Coronal reconstruction. C. Sagittal reconstruction.
Figure 4  Weight-bearing radiographs of the left ankle of a 13-year-old male patient with progressive complaints of deep ankle pain over 18 months without preceding trauma. Preoperative anteroposterior (A) and lateral (B) radiographs show a large lateral radiolucent area involving more than 50% of the talar dome, representing partial talar necrosis. The lesion was secured to the talus by means of two compression screws. The radiographs C and D represent the situation six months postoperatively in which the lesion has healed. Screw removal was performed three months later.

Table 1  Guideline for treatment of osteochondral defects of the talus.

<table>
<thead>
<tr>
<th>Lesion type</th>
<th>Treatment options</th>
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<tbody>
<tr>
<td>Asymptomatic or low</td>
<td>Conservative</td>
</tr>
<tr>
<td>symptomatic</td>
<td></td>
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<tr>
<td>Symptomatic, &lt; 15 mm</td>
<td>Conservative&lt;sup&gt;a&lt;/sup&gt;, debridement and drilling/microfracturing</td>
</tr>
<tr>
<td>Symptomatic, ≥ 15 mm</td>
<td>Fragment fixation, OATS, ACI, debridement and drilling/microfracturing</td>
</tr>
<tr>
<td>Cystic, ≥ 15 mm</td>
<td>Debridement ± (retrograde) drilling/microfracturing with cancellous bone, OATS, ACI</td>
</tr>
<tr>
<td>Secondary</td>
<td>OATS, ACI, HemiCAP&lt;sup&gt;b&lt;/sup&gt;, TruFit&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Massive lesion</td>
<td>Allograft, ankle arthrodesis, total ankle replacement</td>
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OATS: osteochondral autograft transfer system; ACI: autologous chondrocyte implantation.
<sup>a</sup> A trial period of six months conservative treatment is recommended.
<sup>b</sup> Resurfacing of the medial talar dome by a metal implant (HemiCAP<sup>b</sup>) and biodegradable double-layer implants (TruFit<sup>b</sup>) might become valuable alternatives in the future.

Debridement and bone marrow stimulation

With this technique, all unstable cartilage including the underlying necrotic bone is removed. Any cysts underlying the defect are opened and curetted. After debridement, several connections with the subchondral bone are created by drilling or microfracturing. The objective is to partially destroy the calcified zone that is often present and to create openings into the subchondral bone. Intracapsular 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, marrow cells are introduced in the defect and fibrocartilaginous tissue is formed [53].

Advantages of this technique are the possibility of arthroscopy, the relatively easy procedure, and early rehabilitation. A disadvantage is the formation of fibrous cartilage rather than hyaline cartilage. Although often successful, this may be insufficient for large defects [19].

Preoperatively the approach to the defect should be determined (see section: Surgical approach). In the case of arthroscopic treatment it has to be decided whether to use a 4.0 mm arthroscope and treat the OD in the anterior working area by full plantar flexion of the ankle, or to use a 2.7 mm arthroscope in combination with mechanical distraction [36]. Arthroscopy with the foot in full plantar flexion is the preferred method in most cases, although skill and experience are required [7,48].

The subchondral bone can be perforated using a 2 mm drill, a microfracture awl or a 1.4 mm Kirschner-wire (K-wire). A K-wire has the advantage of flexibility, whereas a drill may break more easily if the position of the ankle is changed during drilling. Microfracturing by means of a microfracture awl offers the possibility to work “around the corner” and results in microfractures of the trabeculae rather than destruction of the bone [54], but any created small bony particles should be carefully removed [55].

Operative technique

The standard anteromedial and anterolateral approaches are created in the fully dorsiflexed position, as described previously [36,48]. Introduction of the 4.0 mm arthroscope
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Figure 5  Arthroscopic images of the right talus of a 17-year-old female patient with a lateral osteochondral defect (OD) that is treated by debridement and drilling. A. The arthroscope is in the anteromedial portal. With the ankle in the neutral position the OD is out of the arthroscopic view. B. By bringing the ankle in plantarflexion the OD can be seen. C. The size of the lesion is identified by palpating the cartilage with a probe (arrows), which is inserted through the anterolateral portal. D. A shaver is introduced for debridement of the defect. E. With the use of a K-wire small holes are drilled in the subchondral bone. F. Arthroscopic view of the treated lesion after switching portals. G. During loosening of the tourniquet sufficient hemorrhage in the defect is checked.

and a 4.5 or 5.5 mm bonecutter shaver is performed with the ankle in the fully dorsiflexed position to prevent iatrogenic cartilage damage. If osteophytes or synovitis are present, they are removed first by a chisel, burr or bonecutter shaver with the ankle in the dorsiflexed position. The completeness of removal is checked by plantarflexing the ankle. During this part of the procedure a soft tissue distractor may be applied (Fig. 5A) [36,56]. It should now be possible to visualize the lesion in the forced plantarflexed position (Fig. 5B) and to identify the defect by palpating the cartilage with a probe or hook (Fig. 5C). Debridement is performed by using the bonecutter shaver or a small closed cup curette (Fig. 5D). It is important to remove all necrotic bone and overlying unstable cartilage [57]. After full debridement, the sclerotic zone is perforated several times at intervals of approximately 3 mm (Fig. 5E and F). Sufficient hemorrhage can be checked by loosening of the tourniquet (Fig. 5G).

Rehabilitation

Active plantar flexion and dorsiflexion are encouraged. Partial weight-bearing (eggshell) is allowed as tolerated. It is the senior author’s practice to allow progress to full weight-bearing within two to four weeks in patients with central or posterior lesions of up to 1 cm. Larger lesions and anterior lesions require partial weight-bearing up to six weeks. Running on even ground is permitted after 12 weeks [11]. Sport is resumed after an average of 15.1 weeks [38]. Full return to normal and sporting activities is usually possible four to six months after surgery [19].

Osteochondral autograft transfer

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 [41,58]. 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 [45,59].

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 (Table 1). 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 arthroscopy, 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 miniarthrotomy [11]. 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) [60].

Operative technique

For medial lesions a medial malleolar osteotomy is usually required. Once the lesion is exposed, all diseased and suspect cartilage is removed by curette and scalpel dissection to a sharply defined rim. After debridement of the bony base of the defect by curettage or abrasion arthroplasty, the sharp cutting edge of the appropriate-sized drill guide helps to determine an ideal filling rate of the defect. The usual size of the drill holes in the talus is 4.5 or 6.5 mm in diameter (Fig. 6).

Upon completion of the recipient site preparation, osteochondral grafts are harvested from the ipsilateral knee. Once the site has been clearly identified, the proper size
holes.

internal fixation is performed utilizing the predrilled screw ency of the mosaicplasty. The osteotomy site is reduced and bodies, and sent through a range of motion to ensure congru-

implanted (Fig. 6), the ankle is lavaged, observed for loose combined step accordingly. After the entire set of grafts is be 3—4 mm deeper than the length of the selected plug.

For each graft, drilling, dilation and delivery are done as a use of a conical dilator, allowing easy insertion of the graft. At this stage the hole is enlarged by 0.1—0.2 mm with the be 3—4 mm deeper than the length of the selected plug. At the end of the graft harvesting a suction drain is left behind in the knee joint.

After the graft harvest the recipient site is again evalu-
ated. The first hole is drilled 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 hole is enlarged by 0.1–0.2 mm with the use of a conical dilator, allowing easy insertion of the graft. For each graft, drilling, dilation and delivery are done as a combined step accordingly. After the entire set of grafts is implanted (Fig. 6), the ankle is lavaged, observed for loose bodies, and sent through a range of motion to ensure congru-

cency of the mosaicplasty. The osteotomy site is reduced and internal fixation is performed utilizing the predrilled screw holes.

Rehabilitation

Patients are kept nonweight-bearing for three weeks; six weeks for those with a malleolar osteotomy. Following this period, partial weight-bearing up to 30 kg for three weeks is allowed to promote integration of the grafts. An ortho-

sis may improve comfort. Range of motion exercises are encouraged. Unprotected weight-bearing is subsequently

malleolar osteotomy is necessary to provide access for the ACI procedure [46]. All pathologic fibrous and cartilaginous tissue is debrided. One should not penetrate the subchondral bone during this step, as this would enable marrow elements to contaminate the cultured chondrocyte population.

The peristeoal graft, oversized by 1–2 mm, is next obtained from the ipsilateral proximal or distal tibia. With the cambium side facing toward bone, the peristeoal graft is placed over the defect and sutured with multifilament absorbable sutures, size 5.0 or 6.0. Fibrin glue is placed at the interface to help seal the graft. A small opening at the interface is left patent. Saline is injected, to confirm a watertight compartment, and subsequently aspi-
rated from the defect. The cultured chondrocytes are then placed into the defect and the insertion site is closed with the last stitch and fibrin glue. The osteotomy is repaired with two malleolar screws inserted through predrilled holes.

Rehabilitation

The patient is kept nonweight-bearing and placed in a well-
padded short leg cast during the immediate postoperative period. At two weeks postoperatively, the patient is placed in a controlled action motion (CAM) walker boot. Partial weight-bearing and gentle ankle range of motion exercises are permitted. Weight-bearing is advanced based on radiographic evidence of osteotomy healing. At six weeks, the patient discontinues the use of the CAM walker. Repeti-
tive impact activities, such as jogging and aerobics, can be resumed after six to eight months. Return to high level sports is permitted after 12 months.

Operative technique

ACI is a staged procedure. The initial surgery consists of ipsilateral knee arthroscopy for cartilage harvesting. Articular cartilage is harvested from nonweight-bearing surfaces such as the intercondylar notch. Approximately 200–300 mg of cartilage is harvested with the use of curettes and sent to the laboratory for chondrocyte isolation and proliferation.

The second stage of the procedure is usually at least four weeks after the harvesting procedure. A medial or lateral malleolar osteotomy is necessary to provide access for the ACI procedure [46]. All pathologic fibrous and cartilaginous tissue is debrided. One should not penetrate the subchondral bone during this step, as this would enable marrow elements to contaminate the cultured chondrocyte population. The peristeoal graft, oversized by 1–2 mm, is next obtained from the ipsilateral proximal or distal tibia. With the cambium side facing toward bone, the peristeoal graft is placed over the defect and sutured with multifilament absorbable sutures, size 5.0 or 6.0. Fibrin glue is placed at the interface to help seal the graft. A small opening at the interface is left patent. Saline is injected, to confirm a watertight compartment, and subsequently aspi-
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Future developments

To overcome the disadvantages of current treatment options various attempts are undertaken, aimed at the improvement of current techniques or the development of alternative methods.

To improve ACI, researchers are experimenting with alternatives. The detached osteochondral fragment has been proposed as a source of osteocytes to result in less inflammation [61]. Furthermore, different scaffolds that can be implanted with cultured chondrocytes have been developed, obviating the need for periosteal grafting for fixation [62–64]. Matrix-induced autologous chondrocyte implantation (MACI) makes use of a collagen type I–III membrane, which serves as the scaffold for implanted chondrocytes. Although this is promising in animal and human knees [64,65], only two short-term ankle cases have been reported [63]. Using Hyalograft C as the scaffold, Giannini et al. performed arthroscopic ACI in 30 patients with good short-term results [20].

Regarding OATS, the postoperative application of pulsed electromagnetic fields has been recently shown to limit graft resorption and cyst formation in sheep [66].

As a tissue engineering technique of cartilage, bone marrow-derived mesenchymal stem cells have been successfully implanted using different scaffolds. The majority of this research, however, is still experimental [67–70]. Jancewicz et al. were the first to report a clinical series in the talus [71]. Demineralized bone matrix has been proposed as an alternative to autologous bone grafts for the treatment of OD [72].

Experimental progress is made with the use of biodegradable composite implants. Müller et al. reported the use of double-layer biodegradable implants consisting of poly-dl-lactide and a polyglactin/polydioxanone fleece [73]. Jiang et al. investigated repair with a biphasic osteochondral composite consisting of b-tricalcium phosphate and dl-polylactide-co-glycolide seeded with autologous chondrocytes using single stage surgery [74]. Commercially available composite implants have become available for the treatment of ODs of the knee (TruFit Plug®, Smith & Nephew, San Antonio, TX, USA) [75].

Our research group is currently investigating the applicability of a novel resurfacing technique of the medial talar dome by means of a contoured metal implant (HemiCAP®, Arthrosurface Inc., Franklin, MA, USA) [76].

Alignment and potential correction osteotomy are important issues. In case of persistent complaints after initial treatment, check the overall alignment and hindfoot alignment. The future role of correction osteotomy in the treatment algorithm of OD has to be established.

Discussion

The choice of treatment depends on several factors, such as the patient’s age, symptoms, duration of complaints, location and size of the defect, and whether it concerns a primary or secondary OD [6,11,19,20].

Asymptomatic or low-symptomatic lesions are treated nonoperatively by rest, ice, temporarily reduced weight-bearing, and an orthosis in case of giving way, for a trial period of six months [7,11]. Although nonoperative therapy yields only 45% successful results [18], a trial period does not adversely affect the outcome of surgery [35]. Cartilage lesions have demonstrated to deteriorate slowly in the ankle joint. Hence, the advice is to be conservative; there is always time to test the effect of debridement and bone marrow stimulation.

Surgical treatment is considered in the case of failure of nonoperative treatment or continuing symptoms after previous surgery (secondary OD). According to reviews of the literature the best currently available treatment for primary ODs is the combination of excision, debridement and bone marrow stimulation [17,18]. According to the ISAKOS–FIMS consensus debridement and drilling or microfracturing are the first step in the treatment of symptomatic osteochondral lesions that are too small to consider fixation [47]. Hence, symptomatic lesions up to 15 mm are treated primarily by debridement and bone marrow stimulation. Subchondral cystic lesions smaller than 15 mm do not influence the postoperative prognosis [77].

In the case of a (cystic) defect sized 15 mm or larger this technique might also be considered as a primary treatment option. In these cases a cancellous bone graft may be placed in the defect after debridement [20,38,78]. Retrograde drilling, combined with cancellous bone grafting if necessary, may be performed if there is a (large) subchondral cyst with intact cartilage [34,79]. Alternatively, the cancellous bone graft may be placed underneath the cartilage flap after debridement of the subchondral bone [78]. A cancellous bone graft is harvested from the ipsilateral iliac crest or locally from the distal tibial metaphysis [39]. Possible drawbacks of this procedure are the limited availability and pain at the donor site [40].

Fragment fixation with one or two screws or K-wires is preferred in (sub)acute situations in which the fragment is 15 mm or larger. In adolescents, fixation of an OD should always be considered in case of failure of a period of conservative treatment (Fig. 4) [32].

In case of failed primary surgical treatment, OATS [41,45,80] and the more recently introduced ACI [43,44] are reasonable options. They both aim at creating a new layer of hyaline cartilage.

OATS was originally developed to treat osteochondral lesions in the knee [81]. After promising early experiences, the indication was extended to the talus [45]. Christel et al. emphasized the difficulty of restoring the curvature of the articular surface and reported less favorable clinical outcome in the ankle compared to the knee [82], whereas other authors published promising results [45,80,83,84].

Although hyaline cartilage of the donor area located in the knee is different from the talar hyaline cartilage, there is no evidence this would represent a negative influence on the results. However, the integration of donor and recipient hyaline cartilage can be impaired because of different mechanical properties and thickness [82,85,86]. Other possible disadvantages are the limited availability of grafts [85] and the risk of donor site morbidity [87]. Furthermore, the use of a medial malleolar osteotomy to approach the lesion has been associated with a worse outcome, i.e. local osteoarthritis and higher morbidity [88]. A randomized controlled trial with two years follow-up comparing OATS with debridement (with or without microfracture) showed similar
results among the methods [89]. However, the debridement and microfracture techniques were recommended because of less postoperative pain. Possible alternatives including artificial osteochondral plugs and metal implants may be used in the future.

The newer ACI offers a promising treatment alternative, but long-term data are lacking. Several authors have reported favorable results at short-term follow-up [11,43,44,90–92]. However, long-term studies are needed to evaluate the efficacy of this technique. Incomplete healing of subchondral cysts has been noted in some patients after ACI, although this did not adversely influence clinical outcomes at short-term follow-up [11]. Other possible disadvantages are insufficient graft integration, two-stage surgery and high costs [85,92,93]. Until further data are available, we cannot advocate ACI as an initial treatment option for most cases of OD. However, in patients who have a large OD or failed prior surgical treatment, the short-term data suggest that ACI can provide good results.

For massive ODs the transplantation of fresh or frozen allografts has been described. A number of experts have expressed their concerns of use in the talus, based upon the gradual deterioration of the hyaline part of such grafts in the knee and resorption and fragmentation of the graft [94]. Therefore, transplantation of osteochondral allografts is only indicated for massive osteochondral lesions [95]. Salvage procedures for massive ODs or recurrent failure of treatment are ankle arthrodesis or total ankle replacement.

Conclusion

The choice of treatment is hindered by the fact that none of the grading systems is related to current treatment options. In Table 1 we present a guideline for treatment that is primarily based on the size of the lesion.

Arthroscopic debridement and bone marrow stimulation, by nature of the minimally invasive approach, have great advantage in treating typical defects of up to 1.5 cm in diameter. For larger ODs, the optimal treatment may consist of OATS, cancellous bone graft and/or ACI. The medium-term encouraging results of OATS and cancellous bone grafts hold promise for these procedures in lasting relief of symptoms and prevention of ankle osteoarthritis. ACI has encouraging short-term results in the ankle joint. Much research is performed to improve this method, and its place in the treatment algorithm remains to be further defined. Tissue engineering techniques, artificial plugs and resurfacing by metal implants might become reasonable alternatives in the future.

References

TREATMENT OF OSTEOCHONDRAL DEFECTS OF THE TALUS


