Osteochondral talar lesions and ankle biomechanics
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Chapter 2

Current concepts: treatment of osteochondral ankle defects

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Introduction
An osteochondral lesion of the talus is a lesion involving talar articular cartilage and subchondral bone mostly caused by a traumatic event, leading to partial or complete detachment of the osteochondral fragment, with or without osteonecrosis. Many terms are in use, including osteochondral fracture, osteochondral lesion, osteochondritis dissecans, transchondral fracture, flake fracture, and intra-articular fracture. Osteochondral defects (OCDs) can occur in any joint. The most common location is the knee. Osteochondral defects of the ankle account for approximately 4% of the total number of osteochondral defects [19]. Osteochondral defects of the ankle occur most frequently in 20- to 30-year-old men [52]. Defects can be found on the medial and lateral sides of the talar dome, and occasionally are located centrally [27, 77].

In 1856, Monro first reported the presence of cartilaginous bodies in the (ankle) joint [53]. In 1870, Paget further described the defects [56], and in 1888, König [46] first used the term osteochondritis dissecans for loose body formation associated with articular cartilage and subchondral bone fracture in the knee. König [46] suggested that these loose bodies were the result of 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. Involvement of an inflammatory process in the pathology, however, has never been proved [77]. Rendu [60], in 1932, also described the condition. Davidson et al. [18], Flick and Gould [23], and Nash and Baker [55] discussed the late finding of an OCD after an initially diagnosed “sprained ankle”. Canale and Belding [15] further emphasized trauma as a causative factor.

Concerning trauma, it is estimated that one ankle injury per 10,000 people per day occurs [43]. Ankle injuries compose 45% of basketball injuries, 25% of volleyball injuries, and 31% of football injuries [26]. The percentage of osteochondral lesions associated with lateral ankle ligament rupture was determined by three investigators who routinely inspected the lateral talar dome in a consecutive series of patients who were operated for lateral ankle ligament rupture. Bosien et al. [11], van Dijk [73], and Lippert et al. [49] respectively reported 5%, 6%, and 9% lateral talar dome lesions. The percentage of medial dome lesions is unknown but estimated to be as high as lateral talar dome lesions [73, 77].

Lateral lesions cause more symptoms than medial ones. When the lesion is large and the affected piece is dissected, the joint mechanics are altered, which may lead to osteoarthritis. In 1959, Berndt and Harty [8] gave the first classification determined by radiographic appearance (Box 1); however, the diagnostic approach is based on MRI or CT findings, and different classifications have been made [3, 22, 24, 40]. An osteochondral lesion of the trochlea tali is often not recognized as such and, therefore, not adequately treated. Nonrecognition is mainly due to the fact that the lesion can remain asymptomatic or produce symptoms of inversion–distorsion. To a lesser degree, nonrecognition is also due to the fact that an osteochondral defect often cannot be identified on a plain radiograph.
After standard treatment for acute ankle sprains, residual symptoms are reported in 33 to 40% of patients [11]. When symptoms persist after an ankle sprain, the possibility of an osteochondral defect needs to be considered.

Therapeutic results can be improved by earlier diagnosis and more adequate treatment of the condition. During the past 10 years, great development has been seen in the field of OCD treatment.

The aim of this article is to provide an overview of osteochondral ankle defects, including symptoms and specific treatment indications. Three operative techniques are highlighted: debridement and bone marrow stimulation, osteochondral transplants, and autologous chondrocyte implantation.

**Etiology**

Traumatic insult is more widely accepted as the etiology of talar OCDs, although not without controversy. It is likely that trauma and ischemia are both involved in the pathology. Because not all patients report a history of ankle injury, a subdivision can be made in the etiology of nontraumatic and traumatic defects [77].

The nontraumatic etiology concerns idiopathic osteochondral defects. In these defects, ischemia, subsequent necrosis, and possibly genetics are etiologic factors. Osteochondral defects in identical twins and in siblings have been described [2, 20, 76]. In 10 to 25% of patients, the occurrence of the defect is bilateral [8, 15, 77].

In the etiology of traumatic osteochondral defects, ankle sprains play the largest role. A severe ankle sprain can cause a small fracture and subsequent impaired vascularity, leading to the formation of an osteochondral defect. In addition, microtraumas caused by repetitive articular cartilage surface loading or excessive stress can lead to cellular degeneration or death by the disruption of collagen fibril ultrastructure and thickening of the subchondral bone [25, 77].

In lateral lesions, trauma is described in 98% of cases; in medial lesions, trauma is described in 70% [23].
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Mechanism of injury

When the talus twists inside its boxlike housing during an ankle sprain, the cartilage lining can be damaged. It may lead to a bruise and subsequent softening of the cartilage or worse: a crack in the cartilage or delamination. Separation of the cartilage can occur in the upper layer as a result of shearing forces. Alternatively, separation may occur in the subchondral bone, giving rise to a subchondral lesion. Fragments can break off and float loose in the ankle joint or they can remain partially attached and in position. Progression may result in increased joint pressure, resulting in the forcing of synovial fluid into the epiphysis, creating a subchondral cyst. The subchondral cyst and increased joint pressure may prevent healing. The subchondral fracture has no soft tissue attachments and is highly susceptible to subsequent avascular necrosis [77].

Figure 1 Main locations of osteochondral ankle defects: the anterolateral and posteromedial talar dome.

In cadaver ankles, Berndt and Harty [8] reproduced lateral defects by strongly inverting a dorsiflexed ankle. As the foot was inverted on the leg, the lateral border of the talar dome was compressed against the face of the fibula. When the lateral ligament ruptured, avulsion of the chip began. This chip could be completely detached and remain in place or be displaced by supination. 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 a lateral osteochondral defect. These investigators were able to reproduce a medial lesion by plantar flexing the ankle, by performing a slight anterior displacement of the talus on the tibia, by inversion, and by internal rotation of the talus on the tibia [77].
Lateral osteochondral lesions are usually located in the anterior third of the talar dome, and medial lesions are mostly located in the posterior half (Fig. 1); however, there are exceptions, and anteromedial, posterolateral, and centrally located lesions may occur after trauma. An individual may have multiple lesions. The lateral lesions are typically shallow 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. Medial lesions are usually asymmetric, whereas lateral lesions are symmetric. Because of their shape, lateral lesions are more often displaced than medial lesions [77].

**Clinical presentation**

A differentiation has to be made between the acute and the chronic situation. In the acute situation, symptoms of osteochondral ankle defects compare with those of acute ankle injuries. They include lateral or medial ankle pain, swelling, and limited range of motion. In patients who have an isolated ligamentous ankle injury, these symptoms usually resolve after functional treatment within 2 to 3 weeks. If symptoms do not resolve after 3 to 6 weeks, an (osteo)chondral defect of the talus should be suspected. These patients typically present with persisting symptoms and a limited range of motion.

Locking and catching are symptoms of a displaced fragment. In most patients who have a nondisplaced lesion after supination trauma, the symptoms in the acute situation cannot be distinguished from the soft tissue damage.

Chronic lesions classically present as deep lateral or medial ankle pain associated with weight-bearing. Reactive swelling and stiffness can be present, but absence of swelling, locking, or catching does not rule out an osteochondral defect. Recognizable pain on palpation is typically not present in these patients. Some patients have diminished range of motion [77].

**Diagnosis**

After careful history taking and physical examination of the ankle, routine radiographs of the ankle are taken, consisting of weight bearing anteroposterior, mortise, and lateral views of both ankles. The radiographs may show an area of detached bone surrounded by radiolucency (Fig. 2). Initially, the damage may be too small to be visualized on routine radiography. By repeating the imaging studies in a later stage, the abnormality sometimes becomes apparent.

A heelrise view with the ankle in a plantar-flexed position may reveal a posteromedial or posterolateral defect [74]. A bone scan can differentiate between a symptomatic lesion and an asymptomatic lesion. MRI is often used for detection of these lesions. CT is useful for better defining the exact size and location of the lesion and, therefore, more valuable for preoperative planning (Fig. 3 and Fig. 4). In diagnosing an osteochondral defect, CT has proved to be equally as valuable as MRI [74, 77].
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**Figure 2** Plain antero-posterior radiograph of the ankle: radiolucency of the medial talar dome indicating an osteochondral defect.

**Figure 3** CT scan of a lateral osteochondral ankle defect; coronal reconstruction.

**Figure 4** CT scan of a medial osteochondral ankle defect; sagittal reconstruction.
Operative treatment options

There are widely published surgical techniques for treatment of symptomatic osteochondral lesions. Generally, these techniques are based on one of the following three principles [1, 5, 13, 32, 48, 57, 64, 68]:

1. Debridement and bone marrow stimulation, potentially in combination with loose body removal (microfracture, abrasion arthroplasty, or drilling)
2. Securing a lesion to the talar dome (retrograde drilling, bone grafting, or internal fixation)
3. Stimulating the development of hyaline cartilage (osteochondral autografts [mosaicplasty], allografts, or autologous chondrocyte implantation [ACI])

Assessment of the lesion and possible surgical approaches are significant issues when dealing with talar OCDs. In Parisien’s [57] comprehensive report on ankle arthroscopy techniques, he described portal approaches for synovectomy, debridement, loose body removal, curettage, abrasion, and drilling in the treatment of OCD. The high scores of 88% excellent and satisfactory results have been confirmed by the experience of Van Buecken et al. [71] and others [63, 70] who have promoted wide and modified use of these techniques.

Allografts, fresh and frozen, have been used to treat large lesions. Based on the gradual deterioration of the hyaline part of such grafts in the knee, a number of investigators have expressed their concerns with the use of allografts in the talus. Because of this concern, transplantation of osteochondral allografts is indicated only for massive osteochondral lesions, which are relatively rare in the talocrural joint [36, 44].

Transplantation of free rib perichondrial flaps and periosteal flapping of osteochondral lesions in the ankle joint is still experimental, but there are a few promising clinical reports about implantation of autologous chondrocytes into OCDs [36, 58]. Petersen et al. [58] described good early results by using the autologous chondrocyte implantation method they popularized for the treatment of chondral and osteochondral defects of the knee.

In contrast to the good clinical outcome, the length of the rehabilitation, high laboratory costs, and technical problems appear to be disadvantageous elements of this technique.

The type of surgical treatment also influences the exposure. Most lesions can be treated by means of arthroscopy. Many posteromedial lesions do not have to be treated by malleolar osteotomy but can be treated arthroscopically by bringing the foot into hyper-plantar flexion, although skill and experience are required [63].
Canale and Belding [15] recommended medial malleolar osteotomy when dealing with medial lesions due to their central posterior sites. In an effort to avoid osteotomy, Flick and Gould [23] suggested the use of an anteromedial approach combined with “grooving” of the anteromedial distal tibial articular surface, whereas Thompson and Loomer [69] recommended a combined anteromedial and posteromedial approach. Recently, Jakob et al. [42] described a technique to treat lateral defects by osteotomy of the lateral malleolus.

Bone marrow stimulation

_Curettage and drilling or microfracturing_

After debridement, multiple connections with the subchondral bone are created. The connections 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. The formation of local new blood vessels is stimulated, marrow cells are introduced in the osteochondral defect, and fibrocartilaginous tissue is formed. In the case of large defects, a cancellous bone graft can be placed [77].

Preoperative considerations

Preoperatively, it has to be decided how to approach the defect. Depending on the preference of the surgeon and the location of the lesion, the approach can be from the front, from the back, or by means of a malleolar osteotomy. In the case of arthroscopic treatment, it has to be decided whether to use mechanical distraction in combination with a 2.7 mm arthroscope or to use a 4.0 mm arthroscope and treat the osteochondral defects in the anterior working area by full plantar flexion of the ankle [72]. In patients who have unlimited plantar flexion, all defects in the anterior half of the talus and lesions that are located in the anterior part of the posterior half can thus be reached and treated. The procedure is started without distraction. Introduction of the instruments is performed in the fully dorsiflexed position using the standard anteromedial and anterolateral portal [63, 77].

Operative technique

The standard anteromedial and anterolateral approaches are created as described by Van Dijk and Scholte [72]. A 4.0 mm arthroscope and a 4.5 or 5.5 mm shaver are introduced. When the osteochondral defect is located anteromedially, the 4.0 mm arthroscope is moved over to the anterolateral portal and the instruments are introduced through the anteromedial portal.

For an anterolateral defect, the arthroscope remains in the anteromedial portal and the instruments are introduced through the anterolateral portal. When osteophytes are present, they are removed first by chisel, burr, or aggressive full-radius resector (Bone Cutter Dyonics, Smith & Nephew, Andover, Massachusetts).
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Figure 5 Arthroscopic view of osteochondral ankle defect in left ankle. (A) View after distraction. The 30° arthroscope is in the anteromedial portal, instruments introduced through the anterolateral portal. Lat., lateral; Med., medial; OCD, osteochondral defect. (B) The defect is lifted by a hook, showing its true size. OCD, osteochondral defect. (C) Excision of osteochondral defect. (D) Arthroscope inserted through anterolateral portal. Direct view of defect after debridement and drilling.

Synovitis located anterolaterally (in the case of an anterolateral defect) or anteromedially (in the case of an anteromedial defect) is removed first 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 now be possible to palpate and visualize the osteochondral defect without disturbance of the synovium or overlying osteophyte. If this is not the case, then a further synovectomy is performed in the dorsiflexed position. After sufficient synovectomy, it should be possible to identify the lesion in the forced plantar flexed position by palpating the cartilage with a probe or hook. In cases of a posterior located osteochondral lesion, palpating the lesion demands a full plantar flexion. A little joint laxity helps to open up the joint. During this part of the procedure, a soft tissue distractor is applied (Fig. 5A)
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[72]. Not only can the lesion be palpated with a probe but it should also be possible to visualize at least the anterior part of the lesion and possibly lift it (Fig. 5B). If possible, the 4.5 or 5.5 mm aggressive full-radius resector is now introduced into the defect. In doubtful cases, before introduction of the resector, it can be useful to identify the defect by introducing a spinal needle, thereby penetrating the defect area. When there is any doubt about the direction and the extent of the defect, the arthroscope is moved over to the portal opposite of the defect (the anteromedial portal in the case of an anteromedial osteochondral defect) and the completeness of the debridement is assessed. The arthroscope is then brought back to the opposite portal and further debridement is performed by means of the aggressive full-radius resector or a small closed-cup curette (Fig. 5C). 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 thus be performed. Introduction of the instruments and the arthroscope is performed with the ankle in the fully dorsiflexed position, thus preventing iatrogenic cartilage damage. After full debridement, the sclerotic zone is drilled by multiple drill-holes using a 2 mm burr or a 1.4 mm K-wire. A K-wire has the advantage of flexibility; 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”. The surgeon must make sure that the calcified area is penetrated (Fig. 5D) [77].

Rehabilitation

After arthroscopic debridement and drilling, patients are encouraged to make active plantar flexed and dorsiflexed ankle movements. Partial weight-bearing is allowed. Full weight-bearing is dependent on the size and location of the lesion. A lesion of up to 1 cm is allowed to progress to full weight-bearing within 2 weeks. Larger lesions and anterior located lesions require partial weight-bearing of up to 6 weeks. Running on even ground is permitted after 12 weeks [77]. Full return to normal and sporting activities is usually possible 4 to 6 months post surgery.

Materials and methods

The authors report on two studies of a consecutive group of patients, treated by one of us (C. Niek van Dijk) for osteochondral ankle defects by means of debridement and drilling. Between April 1988 and June 1997, 43 consecutive patients had arthroscopic treatment for an osteochondral ankle defect. Patients were seen at a minimum follow-up of 2 years; 5 were lost to follow-up. Sixteen patients had had previous surgery for the same condition. In the other 22 patients, no previous intervention had taken place (primary group). Between October 1997 and May 1999, another group of 29 consecutive patients who had osteochondral ankle lesions was treated by means of arthroscopic debridement and drilling and prospectively followed. This group was part of a larger group of patients who had chronic ankle pain and presented at the outpatient department of the Academic Medical Center in Amsterdam,
the Netherlands, and were consecutively included into a diagnostic protocol. The study was part of a prospective study design on different diagnostic strategies in patients suffering chronic ankle pain. Patients were seen for a late follow-up at 2 years after the intervention. The outcome in both groups was assessed using the Ogilvie-Harris score, which includes pain, swelling, limping, stiffness, and activity. The patient grades each item as excellent, good, fair, or poor, with the lowest evaluation for each item determining the final score. These assessments were made before operation and at follow-up, as were the radiologic appearances using a scoring system to determine degenerative changes [15].

Results
The mean interval between the initial symptoms and surgery in the patients treated between 1988 and 1997 was 21 months (range 6 to 60 months). The mean age at time of surgery was 29.3 years (range 15 to 78 years) and the mean period of follow-up was 4.8 years (range 2 to 11 years). Good or excellent results were found in 86% in the primary group and in 75% in the revision group. In the primary group, 9% had fair and 5% had poor results; in the revision group, 6% had fair and 19% had poor results. Only one patient in the revision group developed progression of degenerative changes that were seen at a follow-up after 10 years. In none of the other patients was there a change in radiologic grading [63]. Between 1997 and 1999, 35 osteochondral lesions were identified in 29 patients. Among these patients, 25 (86%) had a history of ankle trauma, of which 23 had a history of inversion trauma and 2 sustained an ankle fracture. Preoperative radiologic findings showed that 16 ankles had grade 0 osteoarthritic changes, 9 had grade I, and 4 had grade II. All patients returned for a 2-year follow-up. At 2 years’ follow-up, the results were good or excellent in 24 of 29 patients (83%), there were no changes in grading according to the degenerative scoring scale [74].

Autologous osteochondral grafting
Single block transplants
Single block transplants involve grafting a plug from the lesser–weight bearing femoral condyle into the osteochondral defect on the talar dome. Single plug grafts result in reduced ingrowth of the fibrocartilage, although donor site morbidity may be greater because of harvesting a single, larger plug [51, 66]. Similar to what is practiced in knees, the use of multiple smaller grafts (i.e. mosaicplasty) is preferred, which provides a better match to the talar dome contour and surface area of the defect.

Autologous osteochondral mosaicplasty
Autologous osteochondral grafting seems to be more popular than any other “new technique” that aims to promote a hyaline-type resurfacing of the defected area. Extended experimental trials have confirmed the viability of the transplanted hyaline cartilage and fibrocartilage repair of the donor sites [9, 10, 34, 37, 39]. Use of multiple smaller grafts instead of one large block may help to
avoid donor site morbidity. Furthermore, the congruency in the specific application of the talus may be improved. During the past 10 years, autologous osteochondral mosaicplasty became a popular treatment option for full-thickness femoral condylar lesions, and the indication had already been extended to talar lesions as early as 1992.

Preoperative considerations
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, access to these lesions may be a challenge. Experience has taught that they are best approached through a miniarthrotomy, at times associated with a malleolar osteotomy. The grafts are usually obtained from the medial femoral ridge and sometimes from the lateral femoral ridge of the ipsilateral knee because these are minimal weight bearing surfaces. When the knee is precluded as a donor site, use of small (2.7 or 3.5 mm diameter) autologous grafts from the anterior talus can be considered.

Although radiographic evaluation, CT scan, or MRI may help to determine the extent of the lesion, the size of the mosaicplasty is determined after excision of the defect. Usually the patient is prepared for a mosaicplasty based on radiographic and MRI findings, but the final decision is made during arthroscopy. Preoperatively, the patient is informed thoroughly about the possible treatments and the after-treatment. Caution should be exercised in offering the mosaicplasty to patients over age 50 years, patients who have undergone multiple previous surgeries, and patients who, regardless of age or previous surgical history, demonstrate evidence of panarticular arthritis or articular cartilage thinning.

Operative technique
Under general or spinal anesthesia, the affected lower extremity is prepared from upper thigh to toes and the thigh tourniquet is elevated to 100 mm Hg above systolic pressure. An arthroscopic survey is undertaken to further define the size, location, and surgical grade of the lesion. A final determination of the surgical treatment course is made. The ideal findings for mosaicplasty include an approximately 10 mm diameter focal osteochondral lesion, the medial or lateral dome, detached osteochondral fragments, and otherwise normal articular surfaces of the tibia and talus. Osteoarthritis of the ankle is a contraindication; however, anterior talar and tibial osteophytes do not preclude mosaicplasty consideration. Removal is an integral part of the operation.

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

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

After refreshing the bony base of the defect by sharp curettage or abrasion arthroplasty, the sharp cutting edge of the appropriate-sized drill guide helps to determine an ideal filling rate of the defect. Tapping in this bevel and removing it will mark the bony base and helps to plan the filling. Primarily, the 6.5 mm size can be used to fill the defect, whereas the 4.5 mm and 3.5 mm sizes may be used to fill the remaining spaces. After completion of the recipient site preparation, osteochondral grafts are harvested from the ipsilateral knee. The primary harvest site is the medial upper part of the medial femoral condyle. As a less frequent option, the lateral supracondylar ridge can also be used through a 15 to 20 mm miniarthrotomy. By flexing the knee from 0 to 100°, three to four plugs can be obtained. When the site has been clearly identified, the proper-sized tubular chisel is located perpendicular to the articular surface and driven by hammer to the appropriate depth. Minimal graft length should be at least twice its diameter, but as a rule, plugs 25 mm in length should be taken for osteochondral defects. The grafts are procured with double-edged tubular cutting chisels that ensure the precise diameter and length of the grafts. After removal of the grafts from the chisels, there is an anticipated 0.1 to 0.2 mm expansion in their diameter, a characteristic that helps in the press-fit fixation. Each graft length is recorded. At the end of graft harvesting, a suction drain is inserted into the knee joint.

Figure 6 Osteochondral defect on the medial talar dome. (A) Perpendicular approach by medial malleolar osteotomy. (B) Filling of the previous defect by three grafts (4.5 mm in diameter).

After the graft harvest, the recipient site is re-evaluated. Accumulated clot and bone debris is lavaged from the lesion base and holes. The first drill hole is made through the tubular drill guide, which also serves as the delivery tube. The depth should be 3 to 4 mm deeper than the length of the selected plug. At this stage, the first hole is enlarged by 0.1 to 0.2 mm with the use of a conic
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dilator. Dilation of the recipient tunnel allows easy insertion of the graft. Accordingly, drilling, dilation, and delivery are done as a combined step for each graft. After the entire set of grafts is implanted, the ankle is lavaged, observed for loose bodies, and sent through range of motion to ensure congruency of the mosaicplasty and fluid kinematics (Fig. 6). The osteotomy is repaired with two malleolar screws inserted through predrilled holes (Fig. 7).

Figure 7 Postoperative fixation of the medial malleolar osteotomy by two screws allows immediate range-of-motion exercises.

Lateral OCDs most frequently occur in the anterolateral surface of the dome. In most of the cases, these lesions can be reached through a vertical anterior lateral arthrotomy. By rolling the ankle through flexion and extension, perpendicular insertion of the grafts can be performed. For large posteriorly extended lesions, Gautier et al. [27] and Jakob et al. [42] promoted lateral malleolar osteotomy. Hangody and Kish [36] recommended exposure of these large defects through an anterior fibular periosteal flap containing the origin of the anterior talofibular ligament and, if necessary, the calcaneofibular ligament. The talus can then be drawn forward and rotated downward with the help of a K-wire “joy stick” driven through the body of the talus.

Figure 8 Osteochondral defect on the lateral talar dome. (A) Perpendicular access in equine position by rolling down of the talus. (B) Filling of the previous defect by two grafts (6.5 mm in diameter).
Perfect contouring of the talar dome may represent technical challenge, but careful graft harvest and precise implantation technique can result in perfect congruency (Fig. 8). An osteochondral defect that has a combined subchondral and cystic lesion can also be treated by fine technical modifications.

At the conclusion of the procedure, the tourniquet is released, bleeding is controlled, and a well-padded compression dressing is applied. The patient is observed for 24 hours to keep the extremity elevated, to administer intravenous antibiotics, and to control pain. The knee drain is removed after 24 hours.

Rehabilitation
Patients who do not undergo osteotomy are kept non–weight-bearing for 3 weeks, and those who undergo medial malleolar osteotomy are kept non–weight-bearing for 6 weeks. Following this period, partial weight-bearing of 70 lb for 3 weeks is allowed to promote integration of the grafts. An orthosis may improve comfort. Full range-of-motion exercises are encouraged. Unprotected weight-bearing is allowed at 6 weeks. Athletic activities may begin at approximately 6 months, depending on postoperative assessment.

Materials and methods
Since 1992, the authors (László Hangody and Imre Szerb) performed talar mosaicplasty in more than 80 patients. This report reviews the results of 63 consecutive patients treated between March 1992 and August 2001. All patients have been followed for a minimum of 1 year. The period of follow-up ranged from 1 to 9 years, with an average of 5.8 years.

Postoperatively, all 63 patients were assessed by clinical evaluation and scoring with the Hannover and Bandi scoring systems. Postoperative radiographs were also obtained on all patients. In addition, three-dimensional CT scans were obtained on 31 ankles and MRI on 42 ankles. Second-look arthroscopies were performed in 16 cases, and six biopsy samples were obtained postoperatively between 12 and 41 months. Biopsy specimens were analyzed histologically using the following stains: hematoxylin-eosin, picrosirius red, toluidin blue, and orcein. Histologic examination for polarization, collagen type, and enzyme histochemistry were performed. Finally, cartilage stiffness was measured during four of the second-look arthroscopies using a computerized indentometric device (Artscan 1000; Artscan Oy, Helsinki, Finland).

Results
All 63 patients were available for follow-up. The average patient age was 25.2 years (range 16 to 47 years). The average follow-up for the entire series was 5.8 years (range 1 to 9 years). The average-sized defect treated with mosaicplasty was 1 cm² (range 0.5 to 2.5 cm²), and the average number of grafts per patient was 3 (range 1 to 3 grafts). In most of the cases, 6.5 or 4.5 mm diameter grafts were used, whereas in one third of the cases, 3.5 mm grafts were also implanted.
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All patients achieved full range of motion within 12 weeks following the surgery. No patients showed loosening or subsidence of the osteochondral graft. There were no infections or thromboembolism, but in 1 case, limited range of motion was observed due to arthrofibrosis. According to the Bandi scoring system, in 61 patients, there was no long-term morbidity at the ipsilateral knee donor site. Two patients reported slight or moderate complaints in the patellofemoral area with strenuous physical activity after the first postoperative year. According to the Hannover scoring system, 47 cases were rated excellent, 11 were rated good, 3 were rated moderate, and 2 were rated poor (these 2 were at 4 and 6 years post surgery). By radiographic examination, the transplanted grafts were observed to incorporate into the recipient bed. All osteotomy sites (one on the lateral side and all the others on the medial side) healed without problems. Three-dimensional CT scans in 31 patients showed the recipient defects filled with a congruent surface. Most of the postoperative MRI studies showed congruency of the articular surface with a similar appearance to the surrounding articular cartilage and bone, but in 3 patients the grafted bone showed incomplete incorporation. The second-look arthroscopies demonstrated normal and congruent-appearing surfaces. Biopsy specimen staining showed type II specific normal articular cartilage collagen and articular cartilage proteoglycans. In five patients in whom arthroscopy was also performed postoperatively at the donor site knees, a complete fibrocartilage coverage of the defect was found to be congruent with the surrounding hyaline articular cartilage surface area. No degenerative changes in the donor areas were noted. Articular cartilage stiffness measurements showed values of 2.5 to 3.5 N compared with normal hyaline cartilage measurements of 3.5 to 4.0 N.

**Autologous chondrocyte implantation/transplantation**

*Autologous chondrocyte implantation*

ACI is defined as implantation of in vitro cultured autologous chondrocytes using a periosteal tissue cover after expansion of isolated chondrocytes. In 1965, Smith was the first person to isolate and grow chondrocytes in culture [65]. ACI was popularized by Brittberg et al. [12] whose original article in the *New England Journal of Medicine* described the early results of treatment of osteochondral lesions in the knee. At 2 year follow-up, good or excellent outcomes were reported in 14 of 16 patients. Since then, ACI has been performed in over 25,000 patients. Of these cases, 95% have been in the knee, 3% in the ankle, and 2% in other joints. Based on promising early results with ACI in the knee, surgeons have begun to look at outcomes after ACI for treatment of osteochondral lesions of the talus.

*Preoperative considerations*

Evaluation of patients who have a talar OCD begins with a thorough history and physical examination in addition to plain radiographs including antero-posterior, mortise, and lateral views. Weight-bearing and stress radiographs are obtained as needed. CT scans in the coronal and axial planes with sagittal reconstructions to measure the exact size of the lesion and assess the
cortical outlines are used. MRI is helpful to assess quality of the overlying articular cartilage and to look for subchondral cysts but may overestimate the size of the lesion. The authors classify talar OCDs according to a recognized CT classification system [22]. At The Southern California Orthopedic Institute, the authors (R.M. Dopirak and R.D. Ferkel) presently initially recommend nonoperative treatment for acute and chronic CT scan stage I and II lesions. When patients remain symptomatic after an extended course of conservative management, surgical intervention is recommended. For lesions that are CT scan stage III or IV, ankle arthroscopy is recommended as the initial treatment. Patients who have open growth plates receive an initial course of conservative care for grade I to III lesions and surgical intervention for grade IV lesions. Initial arthroscopic treatment of an osteochondral talus lesion typically consists of marrow-stimulating techniques [7].

For patients who have an osteochondral talus lesion and remain symptomatic after ankle arthroscopy with excision, curettage, and drilling, ACI is considered a viable treatment option. The defect should be focal, contained, and greater than 2 cm². Large lesions with subchondral cystic changes may also be treated with ACI, using the “sandwich technique”. It is the authors’ preference to offer ACI only to patients under age 55 years because articular chondrocytes become senescent with age [7, 14].

Relative contraindications to ACI are bipolar lesions or diffuse degenerative joint changes. ACI is not typically offered as an initial treatment to patients who have a talar OCD. Patients who have a OCD and a significant cyst, however, may be candidates for ACI as the initial operative treatment because these lesions do poorly with marrow-stimulating techniques alone. Advanced osteoarthritis is an absolute contraindication to ACI. Skeletal malalignment and ligamentous instability are also absolute contraindications unless they are concomitantly corrected at the time of surgery.

Operative technique

ACI is a staged procedure. The initial surgery consists of ipsilateral knee arthroscopy for cartilage harvesting. Articular cartilage is harvested from non–weight bearing surfaces such as the intercondylar notch. Approximately 200 to 300 mg of cartilage is harvested with the use of curettes. The specimen is sent to Genzyme Tissue Repair Laboratories (Cambridge, Massachusetts) for chondrocyte isolation and proliferation [7]. Recently, Giannini et al. [29] reported good results using the detached osteochondral fragment as the source of cells. Others have advocated taking a biopsy sample from a non–weight bearing part of the normal portion of the talus.

Ankle arthroscopy is also performed at the time of chondrocyte harvest to assess the size of the OCD and the status of the surrounding articular cartilage and to treat other pathology not amenable to treatment through the planned malleolar osteotomy. Arthroscopy is performed in the supine position, using a thigh holder to flex the hip to 45°. The tourniquet is inflated after exsanguination, and the foot is placed in noninvasive distraction.
Standard anteromedial, anterolateral, and posterolateral portals are created. In most cases, the 2.7 mm 30° and 70° arthroscopes are used, although in some cases, a 1.9 mm arthroscope is necessary due to limited working space in the ankle. A 21-point diagnostic examination is performed to assess the OCD and to evaluate the remainder of the joint for concomitant pathology [21].

After cell preparation, the patient returns to the operating room for the second stage of the procedure, which is typically at least 4 weeks after the harvesting procedure. A medial or lateral malleolar osteotomy is necessary to provide access for the ACI procedure. The level of the osteotomy is determined intraoperatively with the assistance of fluoroscopy and preoperative scans [7] (Fig. 9). It is essential that the osteotomy is carried out medial or lateral enough to provide adequate access to the OCD [7]. Drill-holes for malleolar fixation are created before osteotomy. The osteotomy is initiated with a saw and completed with an osteotome under direct visualization. The medial malleolus is hinged inferiorly on the deltoid ligament, and the fibula is hinged posteriorly after release of the anterior inferior tibiofibular and anterior talofibular ligaments.

The malleolus is retracted to provide direct visualization of the osteochondral lesion. All pathologic fibrous and cartilaginous tissue is debrided (Fig. 10). A no. 15 blade is used to make a vertical incision at the periphery of the defect. The subchondral bone should not be penetrated during this step because this would enable marrow elements to contaminate the cultured chondrocyte population.
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Figure 9 ACI osteotomy. Correct malleolar osteotomy allows for adequate exposure of the OCD. (From Bazaz R, Ferkel RD. Treatment of osteochondral lesions of the talus with autologous chondrocyte implantation. Tech Foot Ankle Surg 2004;3(1):47; with permission.)

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Current concepts

Figure 10 Curettage of osteochondral lesion of the right medial talar dome. Note the medial malleolus has been osteotomized and is retracted inferiorly to allow exposure.

The periosteal graft is next obtained from the ipsilateral proximal or distal tibia. A template of the OCD is created to guide the shape and size of the periosteal resection. The periosteal graft is typically oversized by 1 to 2 mm. The graft is harvested using sharp dissection; electrosurgery is not used because of the potential for tissue necrosis. After incising the periphery of the graft with a scalpel, an elevator is used to gently separate the graft from the underlying bone. Excess soft tissue is debrided to avoid harvesting a graft of excessive thickness. The noncambium layer is marked and the graft is stored in moist sponge to prevent graft shrinkage [7]. The tourniquet is released and hemostasis is obtained.

With the cambium side facing toward bone, the periosteal graft is placed over the defect and secured with 5.0 or 6.0 Vicryl. The sutures are spaced 3 mm apart and the knots tied over the graft (Fig. 11). Fibrin glue is placed at the interface to help seal the graft. A small opening at the interface is left patent so that an angiocatheter can be placed into the OCD to inject the chondrocytes. Saline is injected to confirm that a watertight compartment has been created and is subsequently aspirated from the defect. It is critical to ensure that the periosteal graft does not adhere to the surface of the defect during saline removal. The cultured chondrocytes are now placed into the defect, and the insertion site is closed with the last Vicryl stitch and fibrin glue.

When there is a cystic defect in the subchondral bone, a sandwich procedure may be necessary. The cartilage of the defect is prepared as previously described. The cystic lesion is debrided using curettes and burr. Autogenous bone graft is obtained from iliac crest, proximal tibia, or the calcaneous. After the OCD base is drilled, the bone graft is impacted into the defect to the level of subchondral bone. In the sandwich procedure, two periosteal grafts are necessary. The first graft is placed over the bone graft with the cambium side facing toward the articular surface [7]. This graft is secured with 6.0 Vicryl stitch and fibrin glue. The second periosteal patch is sewn over the cartilage defect with the cambium side down, and the remainder of the procedure is completed as previously described.
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After completion of the ACI, the osteotomy site is reduced, and internal fixation is performed using the predrilled screw holes. The authors use three 4.0 Arbeitsgemeinschaft für Osteosynthesefragen (AO) cannulated screws for fixation of the medial malleolus. The lateral malleolar osteotomy is stabilized with a 1/3 tubular plate and two lag screws. Titanium implants are preferred in all cases to allow future MRI compatibility. The medial and lateral capsule/ligaments are repaired as needed, and routine wound closure is performed. The patient is placed in a well-padded short leg cast.

Rehabilitation

The patient remains in a well-padded short leg cast during the immediate postoperative period and is kept strictly non-weight bearing. At 2 weeks post surgery, the sutures are removed and the patient is placed in a controlled action motion walker boot. Partial weight bearing, limited to 30 lb, is permitted at this time. Gentle ankle range of motion exercises also begin at this time and are performed four to five times per day. Weight bearing is advanced based on radiographic evidence of osteotomy healing. At 6 weeks, the patient discontinues the use of the controlled action motion walker boot and transitions to a lace-up figure-eight brace that may be worn with a comfortable shoe. Formal physical therapy in a pool and on land begins at 6 weeks and consists of four phases: early phase (<8 weeks), transition phase (8 to 12 weeks), midphase (3 to 5 months), and final phase (6 to 12 months). Low-impact athletic activities such as cycling and skating may be started at 4 to 6 months. Repetitive impact activities such as jogging and aerobics can be resumed at 6 to 8 months. Return to high-level sports such as basketball and football is permitted at 12 months [7].
Materials and methods

The authors performed ACI on 31 patients who had an osteochondral talar lesion and formally evaluated the first 11. All 11 patients failed an initial course of conservative management. All 11 patients also had prior surgery for the OCD. Four lesions involved the right ankle, and seven lesions involved the left ankle. The OCD was medial in 9 patients and lateral in 2 patients. The average size of the lesion was $13.1 \times 20.7$ mm (range 8 to 28 mm). The average patient age was 33 years (range 21 to 47 years). Six of the 11 patients had significant cystic subchondral extension that was treated with the sandwich procedure. No patient had a bipolar lesion. No patient had extensive degenerative changes in the joint. Second-look arthroscopy with hardware removal was performed in 10 patients. Mean follow-up of these patients was 38 months (range 24 to 60 months).

Results

Ten of 11 patients reported that they were improved after the surgery; 1 patient was unchanged. At latest follow-up, outcomes were classified as good or excellent in 82% of patients (9/11). Outcomes were fair in 18% of patients (2/11). No patient had a poor outcome. The preoperative Tegner Activity Level was $1.3 \pm 1.0$. Postoperatively, this value improved to $4.0 \pm 1.6$. The American Orthopaedic Foot and Ankle Society score improved from 47.4 preoperatively to 84.3 postoperatively [54].

Figure 12 Forty-seven-year-old woman who had a failed OCD surgery. (A) Preoperative sagittal MRI. (B) Preoperative arthroscopic picture of loose medial OCD. (C) Postoperative sagittal MRI 28 months after ACI. (D) Second-look arthroscopy performed 18 months after ACI.
Second-look arthroscopy was performed in 10 patients at an average 14.2 months post surgery (range 9 to 24 months). At the time of repeat arthroscopy, complete coverage of the defect was seen in all 10 patients. The cartilage at the repair site was noted to be softer than the surrounding native articular cartilage; however, it was observed that there was a correlation between firmness of the graft and total length of time from tissue implantation to second-look arthroscopy. The grafts seemed to become stiffer as a function of time; the more mature grafts felt similar to the surrounding native articular cartilage on palpation (Fig. 12). Periosteal overgrowth was noted in 2 of the 11 patients. No donor site morbidity or any complications were experienced in this study group.

Discussion
The choice of treatment for osteochondral ankle defects depends on symptomatology, duration of complaints, size of the defect, and whether it concerns a primary or secondary OCD. None of the current grading systems is sufficient to direct the choice of treatment [75]. Pure cartilage lesions, asymptomatic lesions, and low-symptomatic lesions are treated conservatively with rest, ice, temporarily reduced weight bearing, and in case of giving way, an orthosis. Consideration for surgical treatment is failure of nonoperative treatment or continuing symptoms after previous surgical treatment (secondary OCD) [77]. In recent reviews of the literature, the best current available treatment for primary osteochondral ankle defects is excision, debridement, and drilling [67, 70, 75]. In these reviews, osteochondral transplantation and autologous chondrocyte implantation play a minor role because results of these techniques are not yet widely published. According to the recent International Society of Arthroscopy Knee Surgery and Orthopaedic Sports Medicine—International Federation of Sports Medicine (ISAKOS—FIMS) consensus, debridement and drilling/microfracturing is the first step in the treatment of symptomatic osteochondral lesions that are too small to consider fixation [16, 77]. In the present article, the authors report 86% favorable results at 2-years’ follow-up and 83% good to excellent results at 2 to 11 years’ follow-up.

Fixation with 1 or 2 lag screws is preferred in (semi)acute lesions in which the fragment is 15 mm or larger. In adolescents, refixation of an osteochondral defect should always be considered, even in fragments that are smaller than 15 mm. Large talar cystic lesions can be treated by retrograde drilling and filling the gap with a bone graft. In case of failed primary treatment, an osteochondral transplant or cultured chondrocyte transplant can be considered.

Autologous osteochondral mosaicplasty was originally developed to treat small and medium-sized (1.0 to 4.0 cm²) focal chondral and osteochondral defects of the femoral condyles and patellotrochlear surfaces. After promising early experiences in the knee, the indication has been extended to osteochondral lesions of the ankle. A perpendicular approach to the defect often requires a relatively aggressive approach such as medial malleolar osteotomy. On the lateral side, an osteotomy can usually be avoided. Central and caput tali lesions can be treated by demanding special approaches. According to Christel et al. [17], talar mosaicplasty represents a more difficult
Autologous osteochondral mosaicplasty was originally developed to treat small and medium-sized drilling and filling the gap with a bone graft. In case of failed primary treatment, an osteochondral larger. In adolescents, refixation of an osteochondral defect should always be considered, even in cases of previous failed procedures. Recently, several other reports confirmed the advantageous initial clinical experiences with the talar mosaicplasty technique [31, 45, 62, 66].

The treatment technique involves only one operation but two incisions, with one at the ankle and one at the knee. The donor area for the graft harvest is the ipsilateral knee, the lesser–weight bearing edges of the femoral condyles at the level of the patellofemoral joint. As has been demonstrated in other publications, these donor site holes eventually fill with cancellous bone and are covered by fibrocartilage, which is acceptable support for the stresses of this relatively lesser–weight bearing portion of the patellofemoral joint [5, 19, 20]. Although hyaline cartilage of the donor area located in the knee is certainly different from the talar hyaline cartilage, there is no evidence to date that this represents a negative influence in the long-term results.

Newer techniques such as ACI offer a promising treatment alternative, but long-term data are lacking. ACI has been shown to yield favorable long-term results in the knee [59], but these findings cannot be directly extrapolated to OCDs of the talus. To date, only short-term data are available on the treatment of talar OCDs with ACI. The authors noted an improvement in 10 of 11 patients at a mean follow-up of 36 months. Other investigators have similarly reported favorable results at short-term follow-up [30, 47, 58]. Long-term studies, however, are needed to evaluate the efficacy of this technique.

In addition, the biologic and mechanical properties of the regenerative tissue after ACI remain undefined. Histologic data are needed to determine whether the repair tissue formed in the talus is the same hyaline-like tissue that is formed in the knee. Incomplete healing of subchondral cysts after ACI was noted in some of the authors’ patients; although this did not seem to adversely influence clinical outcomes at short-term follow-up, the long-term effect of these cysts is unknown. Until further data are available, the authors cannot advocate ACI as an initial treatment option for most cases of osteochondral talar lesions. In patients who have failed prior surgical treatment for the osteochondral lesion and in patients who have large subchondral bone defects, however, the short-term data suggest that ACI can provide good results.

Outside of the United States, newer techniques have recently been developed using scaffolds implanted with cultured chondrocytes to treat talar OCDs [6, 33, 61]. The membrane/matrix ACI (MACI) technique makes use of a bovine collagen membrane that serves as the scaffold for implanted chondrocytes. The chondrocyte-populated membrane may be implanted directly into the OCD arthroscopically or by way of a miniarthrotomy, obviating the need for a malleolar osteotomy. Periosteal graft harvest is not necessary with the MACI technique because the membrane may be directly sutured into place or sealed with fibrin glue. Ronga et al. [61] and Guillen et al. [33] reported on outcomes after MACI for the treatment of OCD; short-term results in these small cohorts of patients are encouraging.
Guideline for treatment of osteochondral talar lesions

<table>
<thead>
<tr>
<th>Lesion</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type 1: asymptomatic lesions, low symptomatic lesions</td>
<td>Conservative</td>
</tr>
<tr>
<td>Type 2: symptomatic lesions ≤ 10 mm</td>
<td>Debridement and drilling/microfracturing</td>
</tr>
<tr>
<td>Type 3: symptomatic lesions 11-14 mm</td>
<td>Consider debridement and drilling, fixation, osteochondral graft, or ACI</td>
</tr>
<tr>
<td>Type 4: symptomatic lesions ≥ 15 mm</td>
<td>Consider fixation, graft, or ACI</td>
</tr>
<tr>
<td>Type 5: large talar cystic lesions</td>
<td>Consider retrograde drilling ± bone transplant, or ACI with sandwich procedure</td>
</tr>
<tr>
<td>Type 6: secondary lesions</td>
<td>Consider osteochondral transplant</td>
</tr>
</tbody>
</table>

For types 4 through 6, debridement and bone marrow stimulation can always be considered a treatment option.

Because none of the current grading systems is sufficient to direct the choice of treatment [67], the authors propose that, as a guideline, the size of the lesion be used as main indicator for treatment (Table 1).

Summary
An osteochondral talar lesion often causes pain, recurrent synovitis, and obstruction from loose bodies. It is a possible precursor of ankle osteoarthritis due to altered joint mechanics and recurrent synovitis. Current diagnostic strategies usually include MRI or CT. Recent research has shown that MRI and CT have the same diagnostic accuracy in the diagnosis of a talar OCD. For preoperative planning, a CT scan gives more precise information.
Arthroscopic procedures like debridement and drilling, by nature of their minimally invasive approach, have great advantage in treating typical defects of up to 1 cm in diameter. For larger osteochondral defects, the optimal treatment result is the long-term replacement and integration of type-specific hyaline cartilage. In principle, mosaicplasty autologous osteochondral transplantation fills these criteria. The early and medium-term encouraging results, complete with confirmatory radiographs and histology, hold promise for this procedure in lasting relief of symptoms and prevention of ankle arthritis. ACI is a relatively new but promising treatment.
Choice of treatment is thwarted by the fact that none of the current grading systems is dually related to current treatment options. Table 1 presents a guideline for treatment that is primarily based on the size of the lesion. Because of the many different operative treatment strategies that have reported satisfactory results and because of the fact that there are only few comparative studies, there is a need for randomized, prospective studies to define and validate available treatment.

Acknowledgments
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