AMERICAN ORTHOPAEDIC FOOT & ANKLE SOCIETY

Foot & Ankle International®
1–12

DOI: 10.1177/1071100716677746
fai.sagepub.com

Current Concepts Review Update: Osteochondral Lesions of the Talus

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Level of Evidence: Level V, expert opinion.

Keywords: osteochondral lesions, talus, ankle, microfracture, autologous chondrocyte implantation, bone marrow aspirate concentrate

Introduction

Osteochondral lesions are pathologic entities affecting the articular cartilage and subchondral bone. In 1887, Franz König first hypothesized the potential etiologies for loose bodies coming from the articular surfaces of various joints.[43] These lesions were originally referred to as osteochondritis dissecans. König stated that these injuries were most commonly a result of severe trauma, though they may occasionally be due to spontaneous compromise of cartilage and the underlying subchondral bone. Osteochondritis dissecans of the ankle, now commonly referred to as osteochondral lesions of the talus (OLT), was first described by Kappis in 1922.[38] Since the initial description, these entities have been increasingly studied and understood, and it is currently estimated that the incidence rate of these lesions is 27 per 100 000 person-years among the active military population.[64]

Initially establishing the diagnosis of OLT can be challenging. Patients may present with prolonged pain, swelling, and catching following traumatic injuries to the ankle or after seemingly innocuous incidents. However, many OLTs also arise without specific trauma, and may be related to repetitive injury. Others are asymptomatic and found incidentally on plain radiographs or advanced imaging. In patients who are symptomatic, identification of OLTs through radiographs can be challenging because these lesions are not always immediately evident, and thus further imaging modalities are often required to confirm the diagnosis.[86] Computed tomography (CT) or magnetic resonance imaging (MRI) can help identify both the location and severity of the lesion.[86] Because of the increasing awareness of these entities, as well as recent advances in both nonoperative and operative approaches to care, we present an updated current concepts review of the literature on diagnosis and evidence-based recommendations for the treatment of osteochondral lesions of the talus.

Etiology

Osteochondral lesions of the talus can arise from a number of potential causes. Although exact causal mechanisms can be difficult to determine, most OLTs are likely caused by either an acute traumatic insult or repetitive chronic loading of the ankle joint.[63,90] However, up to 38% of medial talar OLTs are not associated with a specific injury and may result from localized ischemia of the talus or repetitive localized microtrauma.[81,85] It has been estimated that 50% of acute ankle sprains result in some form of chondral injury, with ankle fractures reported to cause cartilage damage in 73% to 81% of cases.[60,78] Interestingly, the mechanism of insult has been shown to be predictive of location and characteristics of these lesions. Lateral OLTs are typically related to trauma from a shearing-type mechanism. The resulting OLTs tend to be smaller in diameter, shallower in depth, more anterior, and less common compared to medial OLTs.[13,21] However, more recent literature notes

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Table 1. Berndt and Harty's Original Classification System for Osteochondral Lesions of the Talus.

Stage	Radiographic Findings
I	Subchondral compression
II	Partial detachment of osteochondral fragment
III	Completely detached fragment without displacement
IV	Detached and displaced fragment

that the location of a lesion may not be a reliable predictor of mechanism of injury.[19,66]

Despite the initial etiology, these lesions all share the common features of injury to the subchondral bone with or without articular cartilage involvement, and are potentially reversible if they are not associated with cartilage displacement. In cases of nontraumatic OLTs, a proposed pathoanatomic cascade involves initial softening of the articular cartilage with intact articular surface overlying the injured subchondral bone followed early by articular cartilage separation, partial detachment of the lesion, and finally osteochondral separation with intraarticular loose bodies.[9] Despite the known focal articular damage, these lesions are not necessarily associated with eventual progression to ankle osteoarthritis (OA), as Bauer et al[8] reported that only 2 of 30 patients with OLTs had developed OA at an average 21-year follow-up.

Diagnosis

The clinical presentation of OLTs can be somewhat nebulous, though a thorough history and physical examination may provide clues that point to the correct pathology. Common symptoms described by patients include generalized ankle pain as well as clicking or catching of the joint during motion.[29] On examination, pain is typically elicited through palpation of the anteromedial or anterolateral edges of the ankle joint, though osteochondral lesions may be present without palpable tenderness.[29] Range of motion may be limited, and dorsiflexion or plantarflexion may cause clicking or catching.

Imaging may be obtained to further aid in the diagnosis of these lesions. Plain radiographs should be obtained in patients with ankle pain and swelling to provisionally evaluate for OLTs and other possible osseous pathology. Weight-bearing radiographs should include anteroposterior (AP), mortise, and lateral weight-bearing views of the ankle.[84] However, studies have reported that up to 50% of OLTs are not detected with plain radiographs.[86] If visible on plain radiographs, OLTs can be classified based on the original classification system based on plain radiography by Berndt and Harty[9] (Table 1).

Patients with OLTs found on radiographic imaging, or in patients with a presumed lesion without radiographic confirmation may then undergo MRI to assess for the presence, size, and stage of lesions. MRI is the preferred imaging modality for determining the integrity of the overlying cartilage in nondisplaced lesions.[77] The addition of T2 mapping with a 3.0-Tesla magnet can be further beneficial to determine the cartilage status in a more premature fashion.[55]

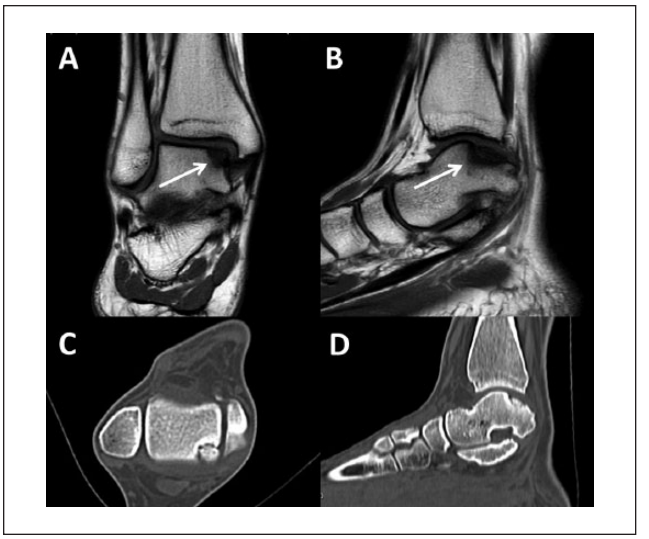

Figure 1. MRI and CT images of OLT in a 16-year-old girl. (A) Coronal and (B) sagittal MR images showing a displaced OLT (arrows). (C and D) CT images showing the extent of the lesion in the posteromedial corner of the talar dome. CT, computed tomography; MRI, magnetic resonance imaging; OLT, osteochondral lesion of the talus.

If a symptomatic OLT is readily identified on plain radiography, the use of computed tomography (CT) can also be utilized to provide details of the lesion. The use of CT has been reported to be more accurate for determining lesion size.[77] Verhagen et al[86] performed a prospective study showing that helical CT, MRI, and diagnostic arthroscopy were significantly better than history, physical examination, and standard radiography alone for confirming or excluding the presence of an osteochondral lesion (Figure 1). Mintz et al[59] performed a retrospective review of 54 patients to correlate arthroscopic findings with MRI results. The authors showed that MRI correctly identified intact cartilage versus osteochondral lesions in 100% of cases. Furthermore, using a 5-point scale from normal cartilage (0) to a displaced fragment (5), MRI correctly graded 83% of OLTs. As MRI technology continues to evolve, this imaging modality will allow for earlier diagnosis of OLTs and may provide more prognostic information than currently available.

Treatment of Osteochondral Lesions of the Talus

When OLTs are identified following appropriate examination and imaging, several treatment options may be available

Table 2. Level of Evidence and Grades of Recommendation.

Level of Evidence
Level I: High-quality prospective randomized clinical trial
Level II: Prospective comparative study
Level III: Retrospective case-control study
Level IV: Case series
Level V: Expert opinion
Grades of Recommendation (given to various treatment options based on Level of Evidence supporting that treatment)
Grade A: Treatment options are supported by strong evidence (consistent with Level I or II studies)
Grade B: Treatment options are supported by fair evidence (consistent with Level III or IV studies)
Grade C: Treatment options are supported by either conflicting or poor quality evidence (Level IV studies)
Grade I: When insufficient evidence exists to make a recommendation

Table 3. Summary of Grades of Recommendation.

Treatment	Grades of Recommendation
Nonoperative management	B
Microfracture	B
Subchondral drilling	B
Open reduction internal fixation (ORIF)	C
Osteochondral autograft transplantation (OAT)	B
Fresh osteochondral allograft transplantation	C
Autologous chondrocyte implantation (ACI)	B
Autologous matrix–induced chondrogenesis (AMIC)	I
Particulated cartilage products	I
Matrix-associated stem cell transplantation (MAST)	I

based on size and stage of the lesion, patient activity level, and chronicity of the lesion. The following sections are an updated, evidence-based (Table 2) review of the treatment of OLTs. A summary of specific evidence-based recommendations can be found in Table 3.

Nonoperative Treatment

Nonoperative treatment is initially indicated for (1) asymptomatic patients, (2) minimally symptomatic patients with nondisplaced lesions or early stage lesions without loose intraarticular fragments, or (3) patients who are clinically improving.[42] Many of these lesions heal without operative intervention. In a case series of 48 patients (Level IV evidence), Klammer et al[42] recently showed that nonoperative management of asymptomatic or minimally symptomatic OLTs resulted in no substantial progression in staging or lesion size. In addition, 86% of all ankles treated were pain-free or less painful at a minimum 2-year follow-up.[42] Multiple predictors of whether an OLT will heal spontaneously have been reported. One of these factors, the location of the lesion, has been shown to be important, as lateral lesions have demonstrated a lower probability of healing with conservative treatment.[13,21]

In children with OLTs, Heyse et al[35] found that a higher age and a grade III lesion (completely detached fragment without displacement) were predictive of failure of conservative treatment. Kijowski et al[40] determined criteria (known as the De Smet criteria) which predict instability of OLTs. The group found a 100% sensitivity of unstable lesions in both populations when the following criteria were grouped together: high T2 signal intensity rim, surrounding cysts, high T2 signal intensity cartilage fracture line, and a fluid-filled osteochondral defect. Of note, the grouped criteria were only 11% specific for instability in juvenile lesions but were 100% specific in adult lesions. These criteria are likely to carry important prognostic information for patients with OLTs and may predict failure of conservative treatment in these patients.

Nonoperative treatment options for OLT may include rest/restriction of sport activities with or without the use of nonsteroidal anti-inflammatory drugs (NSAIDs) or immobilization for 3 weeks to 4 months depending on the persistence of the symptoms.[81] In a systematic review of patients with Berndt and Harty stage I, II, or medial stage III lesions (Table 1), nonoperative treatment was successful in 45% of patients, with rest/restriction of activities resulting in a 59% success rate compared to 41% good/excellent results with cast immobilization.[81]

Biologic treatments such as platelet-rich plasma (PRP) and hyaluronic acid (HA) injections have also been utilized in patients who have failed more conservative approaches and desire to avoid operative intervention. Mei-Dan et al[57] compared HA versus leukocyte-poor PRP in a nonblinded, randomized controlled trial (Level II evidence). They reported on 29 patients with 30 OLTs that were randomized to receive either 3 weekly injections of 1% sodium hyaluronate or 3 biweekly (1 injection every 2 weeks) injections of 2 mL of leukocyte-poor PRP. Patients were assessed at

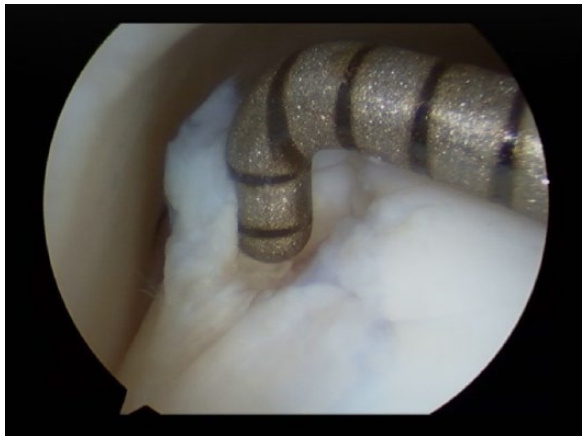

Figure 2. Intraoperative arthroscopic image of an OLT. A probe is placed in the lesion to assess the severity of the lesion. OLT, osteochondral lesion of the talus.

baseline and 4, 12, and 28 weeks postinjection with the modified Ankle-HindFoot Scale (AHFS), subjective global function and disability (1%-100% scale), and visual analog scales (VASs) for pain, stiffness, and function. Both groups demonstrated significant improvement in all 5 parameters at all time points compared to baseline, though the PRP group demonstrated significantly greater improvement compared to the HA group in terms of mean AHFS scores, subjective global function, and VAS for stiffness and function. However, with only 30 OLTs included and the lack of a control group, the conclusions that can be drawn from this study are limited.

Since the last current concepts review was published by McGahan and Pinney,[56] the level of evidence supporting a trial of nonoperative treatment for nondisplaced lesions remains fair (Grade B recommendation). There is insufficient evidence to support one form of treatment over another, including the use of biologic treatments such as PRP or HA.

Operative Treatment Options

Operative treatment of OLT was first described by Ray and Coughlin[72] in 1947 and consisted mainly of removal of loose bodies and debridement of remnant cartilage. Various operative procedures have been used to treat osteochondral lesions of the talus, including bone marrow stimulation (microfracture), osteochondral autograft and allograft transplantation, autologous chondrocyte implantation (ACI), and bone marrow aspirate concentrate transplantation. Some of these procedures may be performed arthroscopically, in which case the OLT may be visualized and assessed immediately prior to definitive operative treatment (Figure 2).

When operative treatment is indicated, many surgeons recommend debridement and marrow stimulation as the initial treatment modality for most OLTs. However, to date, few comparative studies and no cost-effectiveness analyses have been performed for OLT treatments. Further research will benefit both patients and surgeons alike to arrive upon the most appropriate treatment option.

Microfracture. Bone marrow stimulation, more commonly referred to as microfracture surgery, is performed by perforating the subchondral plate at the area of the chondral defect (Figure 3). This results in a blood clot with growth factors and progenitor cells from bone marrow that stimulates healing with regenerative fibrocartilage growth.[58] Open microfracture surgery on osteochondral lesions of the talus was first described by Alexander and Lichtman in 1980.[2] Arthroscopic microfracture was described shortly thereafter by Parisien in 1986,[67] as well as by Pritsch et al[70] in the same year. As noted by McGahan and Pinney,[56] consistently positive results from numerous Level IV studies constitute fair evidence (Grade B recommendation) to support the use of marrow stimulation in the management of painful OLTs.

Since the initial implementation of microfracture, newer operative techniques have been developed for treatment of OLT, including subchondral drilling, cartilaginous autografts and allografts, autologous chondrocyte implantation (ACI), and bone marrow aspirate concentrate (BMAC) transplantation in a biological scaffold[12] or under an osteochondral autograft sleeve.[39] However, an advantage of microfracture over other treatment methods for OLT is that successful outcomes can be achieved with early weight bearing and a resulting shorter recovery time. Recently, Lee et al[50] demonstrated good clinical results with partial weight bearing for the first 2 postoperative weeks, followed by transition to full weight bearing in a walking boot when tolerated. In a separate study,[52] full weight bearing with a figure-of-8 ankle splint was allowed immediately following microfracture surgery with excellent outcomes (Level IV evidence).

It has also recently been shown that the presence of subchondral cysts does not affect outcomes with microfracture surgery for OLT.[51] Lee et al[51] performed a prospective cohort study (Level II evidence) on patients with and without subchondral cysts and found significant improvements in American Orthopaedic Foot & Ankle Society (AOFAS) scores (cyst: 91.8, noncyst: 91.3), VAS for pain (cyst: 2.3, noncyst: 2.2), and Ankle Activity Scores (AAS) (cyst: 6.7, noncyst: 6.5) in both groups compared to preoperative scores. At a mean follow-up of 48 months, no significant difference in any of these 3 scores was found between groups. Average lesion size was 100.9 mm^2 and 99.3 mm^2 in the cyst and noncyst groups, respectively.

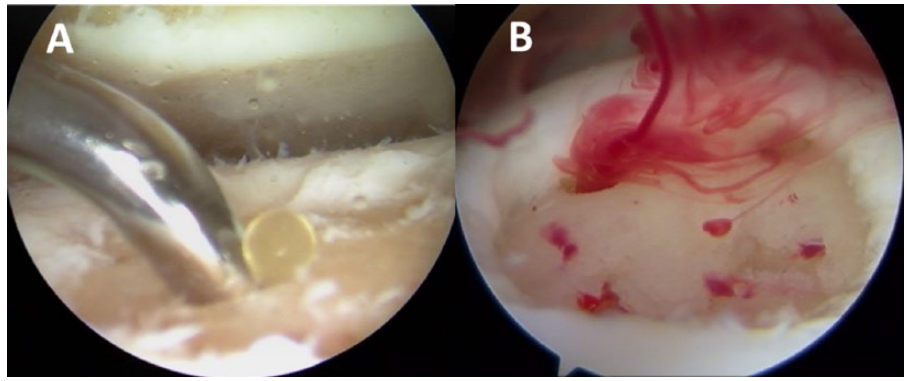

Figure 3. Intraoperative arthroscopic images of microfracture for OLT. (A) A microfracture awl is used to perforate the subchondral plate at the area of the chondral defect. An adipose droplet can be seen coming out of the perforation. (B) After completion of the microfracture, notice the bone marrow elements (which contain growth factors and stem cells) flowing from the subchondral perforations. OLT, osteochondral lesion of the talus.

Biological augmentation of microfracture procedures of the talus has been proposed in an attempt to promote healing of cartilaginous tissue and improve patient outcomes. Two randomized clinical trials have recently been published to assess outcomes following microfracture surgery with adjunctive injections of platelet-rich plasma (PRP). In the first,[26] microfracture with a single-dose injection of PRP was shown to significantly improve patient-reported clinical outcomes compared to microfracture alone or microfracture with injection of hyaluronic acid (HA) (Level I evidence). Both AOFAS and VAS scores for pain showed significant improvement in the PRP group compared to HA and control. Guney et al[30] performed a randomized clinical trial comparing microfracture with PRP to microfracture alone (Level II evidence). At an average follow-up of 16 months, the PRP group showed a superior response in terms of AOFAS scores, VAS for pain, and Foot and Ankle Ability Measure (FAAM) overall pain domain. A randomized controlled trial (Level I evidence) that compared microfracture alone to microfracture with postoperative HA injection reported that patients in the second group had significantly improved outcomes at 2 years postoperatively.[18] These 3 studies support the use of biological adjuncts when used in conjunction with microfracture.

Subchondral antegrade and retrograde drilling. Using the same concept as microfracture, antegrade and retrograde drilling approaches have also been performed to treat OLTs. Retrograde drilling is reserved for lesions where the cartilage is intact.[37,44] For talar dome lesions that are difficult to approach directly using arthroscopy, antegrade or transmalleolar drilling may be performed by drilling a Kirschner wire (K-wire) proximal to the medial malleolus and into the lesion through intact cartilage.[44] Ferkel et al[20] showed good or excellent clinical outcomes in the majority of patients at a mean 71 months following transmalleolar drilling (Level IV evidence). Interestingly, no correlation was made between clinical results and lesion stage according to plain radiographs, CT, or MRI. However, significant correlation was found when classified according to the arthroscopic stage. Thus, in addition to staging OLTs, the method of staging is also an important prognostic indicator.

Depending on the location of an OLT, retrograde drilling may be performed through the sinus tarsi[37] or through the posterolateral talus lateral to the Achilles tendon[44] in cases of intact articular cartilage. This approach carries the advantage of protecting the intra-articular talar hyaline cartilage, though drilling must be stopped when the cartilage starts fibrillating to avoid articular cartilage damage.[48] Typically, 2- or 3-dimensional fluoroscopy has been used to confirm the drilling site, though a "fluoro-free" approach has been shown to be as precise as 2D fluoroscopy.[27] A small cohort study reported that retrograde drilling resulted in improved arthroscopic findings of OLT grades at second-look arthroscopy 1 year postoperatively compared to antegrade drilling[44] (Level III evidence), though no difference has been reported to occur between techniques in terms of patient-oriented outcomes and radiographic healing.[31]

Based on a retrospective cohort study (Level III evidence), no differences have been shown between microfracture and subchondral drilling for small- to medium-sized OLTs.[15] Furthermore, Backus et al[4] showed no difference in pain reduction between arthroscopic debridement and microfracture/drilling at 6 months postoperatively (Level III evidence).

Primary repair with open reduction and internal fixation. Open reduction and internal fixation (ORIF) is another option for addressing select osteochondral lesions (Figure 4). Specifically, it is indicated for OLTs with large, loose fragments

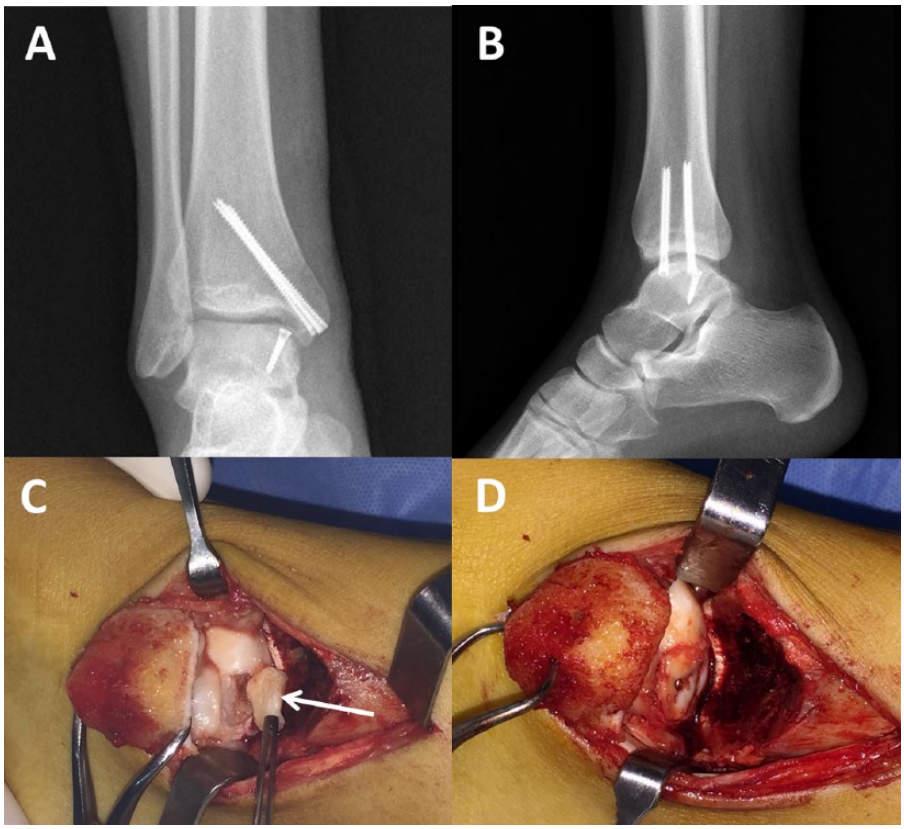

Figure 4. Open reduction and internal fixation of an OLT. (A and B) Postoperative radiographs. To reach the site of the lesion, a medial malleolus osteotomy was performed and then fixed with screws. (C) Intraoperative view of the lesion showing a chondral fragment (arrow). (D) Fragment reduction was completed with a metallic screw concealed in the cartilage layer.

that can be reattached to the underlying bone.[5] This treatment option is often reserved for acute injuries, as ORIF typically fails in chronic lesions with sclerotic borders.[5] Arthroscopy may be used to visualize the lesion while a transmalleolar bone tunnel is created.[61] A K-wire is used to drill from the anterior to posterior edge of the OLT to a depth of at least 10 mm. A pin is then drilled over the K-wire with the tip of the pin at the subchondral level. Cortical bone pegs from the distal tibia may also be used for fixation.[46] It is important to maintain ankle immobilization during this procedure in order to avoid breaking the K-wire or pin. Unlike microfracture or drilling, ORIF preserves the patient's native cartilage. Kumai et al[46] showed good clinical outcomes in 24/27 patients and good radiologic outcomes in 22/27 patients at an average follow-up of 7.0 years (Level IV evidence). Three patients with poor radiologic outcomes demonstrated progressive collapse or depression of the osteochondral fragment postoperatively.

Osteochondral autograft transplantation. Osteochondral autograft transplantation (OAT) is performed by first measuring the size of the osteochondral lesion in the talus, and then harvesting osteochondral "plugs" from healthy cartilage sites such as the femoral condyles or the trochlear notch[24] for later transplantation into the talar lesions. The indications for OAT as treatment for OLTs include failed debridement and microfracture, and larger lesion sizes. The decision to perform an OAT procedure as initial surgical management of an OLT must balance the extended recovery time and potential donor site morbidity, with the tendency for larger (>1.5 cm^2) lesions treated with microfracture to have less predictable results.[17]

A medial malleolar osteotomy is typically required to gain access to medial talar OLTs. A transmalleolar approach has been described for lesions in the posteromedial portion of the talar dome.[74] Adequate access to lateral talar dome lesions can often be achieved through an arthrotomy and plantarflexion. When further exposure of the lateral talar dome is required, a biplanar osteotomy of the anterolateral tibia has been described.[80] Recently the use of intraoperative external fixation has been described as a means to improve lateral talar dome exposure with complications

limited to temporary postoperative pin site pain.[65] Injection of bone marrow aspirate concentrate into the base of the graft site has been performed for promotion of biological integration at the graft/host interface.[39]

A drawback of OAT is donor site morbidity in a previously healthy knee, as plugs cannot be harvested from the ankle given its small size and weight-bearing requirements. The donor site morbidity associated with osteochondral harvest from an asymptomatic knee may be significant and can interfere with activities of daily living postoperatively.[73] Donor site morbidity may be worse in patients with a high body mass index[68] or those older than 40 years at the time of surgery.[89] Another limitation is that the autograft transplantation cannot be performed arthroscopically. Finally, this technique does not provide a "matched fit," as the contour of the knee is different than that of the talus.

Kim et al[41] performed a case series (Level IV evidence) on 52 ankles having undergone OAT with a mean follow-up of 34.1 months. The authors found significant improvements in VAS for pain, AOFAS score, and Tegner activity scale, with no association between clinical outcome and defect size or location, duration of symptoms, or the existence of a subchondral cyst. Using the Karlsson-Peterson Ankle Score, Scranton et al[76] also found good clinical outcomes in a series of 50 patients with type V cystic lesions (Level IV evidence).

Woelfle et al[89] found no association between clinical outcome and defect size or location in a series of 32 patients after the OAT procedure (Level IV evidence). In a separate study by Woelfle et al[88] (Level IV evidence), abnormal postoperative MRI findings were seen in 14 of 28 patients, most commonly irregular and/or hyperintense graft cartilage or incongruity of the articular surface. However, no association was found between abnormal MRI findings and VAS and AOFAS scores. Thus, MRI findings do not necessarily associate with clinical outcomes following osteochondral autograft transplantation.

Paul et al[69] examined postoperative sports activity in a series of 131 patients following OAT for OLTs (Level IV evidence). Although patients did not significantly change their time spent doing sporting activities following surgery, Tegner score decreased significantly from 5.9 preoperatively to 5.0 after surgery, and patients engaged in less contact and high-impact sports.

A randomized controlled trial (Level I evidence) by Gobbi et al[24] in 2006 compared chondroplasty, microfracture, and osteochondral autograft transplantation with a total sample size of 33 ankles. Both Ankle-Hindfoot Scale (AHS) scores and Subjective Assessment Numeric Evaluation (SANE) showed significant improvement in all treatment groups from preoperative to at least 12 months' follow-up. No significant differences were found between groups in terms of AHS scores at 12 and 24 months or in SANE ratings at 53 months. An inverse relationship was found between lesion size and outcome in the microfracture and OAT groups.[24] However, a separate outcomes analysis was not performed between groups in this study on patients with larger lesions or cystic lesions.

Combined, the Level IV studies presented above constitute a Grade B recommendation for the use of OAT in the treatment of large and cystic osteochondral lesions.

Fresh osteochondral allograft transplantation. Similar to osteochondral autograft transplantation but without the donor site morbidity, allografts can be used from a cadaveric donor. Potential indications for fresh osteochondral allograft transplantation include a patient who has failed prior arthroscopic techniques or cartilage restoration, a large OLT that involves the shoulder region of the talus, an OLT with a large cystic component, and a lesion greater than 1.5 cm^2.[28]

In addition to avoiding donor site morbidity, another advantage of allograft use is shorter operative time.[79] Furthermore, the allograft can be taken from the same location on a donor talus to approximate the patient's native anatomy. To best match the recipient defect, a CT of the patient's contralateral (uninjured) talus should be performed and used as a template to size the donor graft needed. These dimensions are then sent to the graft agency that will attempt to match the request with a donor graft.[28] Relative to osteochondral autografts, limitations to this technique include cost, limited availability, possibly lower healing rates,[1] potential disease transmission (although this represents a small risk), and immunologic reaction.

As a result of increasing safety concerns over potential infection, allografts are now hypothermically stored for a minimum of 14 days, allowing for extensive microbiologic and serologic testing. The viability of chondrocytes has been shown to decrease after 28 days postmortem. Consequently, fresh osteochondral allografts should be used between 15 and 28 days postmortem, and ideally the transplant should be performed as soon as the 14-day testing period is complete, at day 15-16 to maximally preserve chondrocyte viability.[49]

For very large lesions that are not amenable to treatment with an osteochondral plug, fresh osteochondral allografts may be fixated in place with pins or screws.[33] Hahn et al[33] showed improved pain and activity level in a series of 13 patients who underwent talar osteochondral allograft transplantation with internal fixation (Level IV evidence). In another case series (Level IV evidence) of 17 ankles with large OLTs (at least 15 mm in 1 dimension), Haene et al[32] performed talar allograft fixation with Herbert screws or bioabsorbable pins. At a mean follow-up of 4.1 years, only 4 of the 17 ankles were symptom-free, with 5 ankles considered failures at follow-up. Raikin[71] also performed talar allograft fixation with titanium compression screws in a series (Level IV evidence) of 15 patients with a minimum lesion volume of 3000 mm^3. At an average follow-up of 54

months, 2 ankles had subsequently undergone conversion to an ankle arthrodesis. Overall, 11 of 15 patients rated the result as excellent or good.

Autologous chondrocyte implantation. Autologous chondrocyte implantation (ACI) was first described in the talus in 2005 by Whittaker et al.[87] ACI is a 2-stage procedure. The first procedure consists of obtaining a biopsy of viable chondrocytes, such as from the femoral intercondylar notch,[47] the proximal tibia,[22] or the anterior talus.[3] Some authors believe that cartilage should not be harvested from the ankle because even small cartilage biopsies may result in dysfunctional mechanics of the ankle joint.[54] Following the first procedure, cells obtained from the biopsy specimen are cultured over a period of 2 to 6 weeks and then implanted into the lesion site during the second procedure.[54] ACI is indicated for full-thickness, large (>1 cm^2) contained defects of the talus. By using a source of viable chondrocytes, ACI has been shown to result in growth of regenerative tissue with biomechanical properties close to normal hyaline articular cartilage.[10]

Chondrocyte implantation can be performed with periosteum or with a scaffold to hold the cells in place over the defect region. Currently, most ACI procedures are performed using a scaffold, as a periosteal patch may be difficult to place in the ankle and periosteal patch hypertrophy has been reported to result in higher reoperation rates.[25] As such, hyaluronan,[22] porcine collagen,[3] and bovine collagen[53] scaffolds have been used. The AOFAS score has been used in several studies[3,22,47,53] to track outcomes, with all studies showing significant improvement from preoperative to various postoperative follow-up times up to 87 months (Level IV evidence). However, it is important to keep in mind that the AOFAS score is not a validated rating scale and has poor responsiveness and reliability.[36] In addition to subjective outcome scores, T2-mapping with MRI is a noninvasive method of characterizing the nature of repair tissue postoperatively. Preliminary results using this technique have shown normal hyaline cartilage and fibrocartilaginous tissue covering an average of 69% and 17% of the lesion area, respectively (Level IV evidence).[7]

Kwak et al[47] evaluated 29 patients undergoing ACI for OLT, with an average lesion size of 198 mm^2 (range, 80-500 mm^2) (Level IV evidence). Cells were implanted into lesion sites using a periosteal graft from the tibia to cover the cell layer, with fibrin glue used to seal the graft over the chondral lesions. Postoperatively, patients were immediately allowed partial weight bearing before transitioning to full weight bearing between 6 and 12 weeks postoperatively. At a mean follow-up of 70 months, patients demonstrated a significant improvement in average Tegner score compared to preoperative scores. No correlation was found between lesion size and follow-up AOFAS score. Twenty-four patients underwent postoperative MRI, with 22 demonstrating >75% repair tissue fill and 2 patients demonstrating 50% to 75% repair tissue fill. In terms of surface regularity, 0 patients were graded as smooth, 18 were graded as mildly irregular, and 6 were graded as moderately irregular.

Giannini et al[22] performed a study on 46 patients undergoing ACI with a mean lesion size of 1.6 cm^2 (Level IV evidence). The rehabilitation protocol used for this study differed from that of Kwak et al[47] in that patients were placed on non–weight bearing restrictions for 4 weeks postoperatively, followed by partial weight bearing from 4 to 8 weeks postoperatively. Patients with a history of previous surgery on the lesion site showed worse AOFAS scores at each follow-up period compared with patients who had never been treated previously. Patients affected by lateral lesions had significantly better AOFAS scores at 12 and 36 months follow-up compared with patients with medial lesions. Among 29 athletes, 20 resumed sports at the same level, whereas 4 patients gave up sports following surgery.

As mentioned above, collagen scaffolds may also be used to perform ACI, in which case these procedures are referred to as matrix-associated autologous chondrocyte implantation (MACI). Anders et al[3] performed a case series (Level IV evidence) of 22 patients with full-thickness OLTs of an average size of 1.94 cm^2. Patients underwent MACI with chondrocytes seeded onto a porcine collagen type I/III scaffold. The AOFAS score improved significantly from a preoperative mean score of 70.1 to 95.3 at a mean 63.5 months follow-up ($P < .001$). Pain (as assessed by a VAS) also significantly improved from 5.7 to 0.9 ($P < .001$). Similarly, Magnan et al[53] reported on 30 ankles treated with MACI (Level IV evidence) using a bovine collagen matrix (Hyaff 11; Verigen, Leverküsen, Germany). Mean AOFAS score increased from 36.9 preoperatively to 83.9 at an average follow-up of 45 months ($P < .01$). In addition, the MOCART score improved from 6.3 preoperatively to 3.8 at final follow-up, representing good integration of the cartilage graft.

Unfortunately, because of a lack of controlled trials, the effectiveness of ACI in comparison to other treatment methods for osteochondral lesions of the talus is yet unknown. Still, consistently good results in several Level IV studies constitute a Grade B treatment recommendation.[3,22,47,53,62] Furthermore, the increased cost of ACI imposes a significant limitation on the use of this technique in comparison to microfracture/debridement or drilling.[90]

Autologous matrix-induced chondrogenesis. Autologous matrix-induced chondrogenesis (AMIC) is a slightly different procedure from ACI. AMIC is accomplished by performing microfracture of the lesion site followed by implantation of a collagen matrix. The matrix offers a protective environment for cell differentiation and new cartilage formation following the microfracture procedure. An advantage of this technique over ACI is that it only requires

a single procedure and can be performed arthroscopically.[82] Valderrabano et al[83] showed excellent clinical results in a case series (Level IV evidence) of 26 patients with use of AOFAS score and VAS for pain, though complete filling of the defect was only shown by MRI in 35% of patients, with a hypertrophic cartilage layer in 50% of patients.

Particulated cartilage products. The DeNovo Natural Tissue (NT) graft (Zimmer, Inc, Warsaw, IN) was recently reported to treat OLT.[14] This is a cartilaginous tissue graft obtained from allograft donors under 13 years of age. Because of the young age of the donors for this graft, the cellular density is higher than in mature articular cartilage samples. Of note, the DeNovo NT graft is a single-stage procedure, whereas autologous chondrocyte implantation requires 2 procedures. Following arthroscopic debridement of the osteochondral lesion, graft particles are placed to cover the OLT, with layers of fibrin glue placed below and above the graft and allowed to dry prior to arthroscopy portal closure. To date, limited case reports and case series[16,45] (Level IV evidence) have demonstrated promising results. In one case series including 24 ankles, only one partial graft delamination was seen at 16 months' follow-up.[16]

Matrix-associated stem cell transplantation. Another 1-stage procedural option for OLT is cell transplantation with bone marrow aspirate concentrate (BMAC).[23] When a biological scaffold is used to assist with cell delivery, the procedure is known as matrix-associated stem cell transplantation (MAST). The percentage of stem cells in a BMAC sample is less than 0.001%. Thus, the regeneration process of BMAC treatment is due mainly to growth factors and inflammatory-blocking proteins contained within the sample. As with ACI, MRI T2-mapping has been performed following BMAC transplantation in a case series (Level IV evidence) to evaluate the repair tissue.[6] Using this technique, normal hyaline cartilage has been found to cover 78% of talar lesions at 2-year follow-up, with fibrocartilage accounting for less than 10% of repair tissue.

Giannini et al[23] performed MAST in a case series (Level IV evidence) with bone marrow harvested from the iliac crest. Scaffolds were created using BMAC and either porcine collagen powder or a hyaluronic acid membrane cut to the size and shape of the osteochondral lesion. Based on a case series of 48 patients,[23] the AOFAS score significantly improved from preoperative to 48 months follow-up ($P < .0001$). The type of scaffold used did not significantly affect AOFAS score results. In 5 patients, a second-look arthroscopy was performed at a minimum follow-up of 12 months, demonstrating integration of regenerated tissue with healthy cartilage in all patients. In 2 of these patients, hypertrophic regenerated tissue was observed during second-look arthroscopy and excess cartilage was shaved. With further follow-up and additional patients, the group found that AOFAS scores at 72 months follow-up were still 15 points higher than preoperatively.[11]

Cadossi et al[12] have described a similar approach of MAST, though in this study patients were randomized following surgery (Level II evidence) to either receive biophysical stimulation with pulsed electromagnetic fields (PEMFs) for 60 days or no additional treatment (control group). At 6 and 12 months' follow-up, the experimental group demonstrated significantly higher AOFAS scores and significantly lower VAS pain scores. No significant difference was found in Short-Form 36 scores between groups.

Buda et al[10] performed a prospective comparative study (Level II evidence) to analyze outcomes between autologous chondrocyte implantation versus MAST with use of a hyaluronic acid membrane. Postoperative AOFAS scores up to 48 months' follow-up revealed no significant differences in scores between the 2 groups. However, when measured by percentage improvement in AOFAS score compared to a preoperative baseline score, the MAST group demonstrated greater improvement at 12, 36, and 48 months follow-up compared to ACI. Radiographically, both groups had similar outcomes in terms of the MRI MOCART score at 48 months follow-up.

Hannon et al[34] recently performed a retrospective review (Level III evidence) to compare bone marrow stimulation (BMS) with versus without BMAC for OLTs. No biological scaffold was used in this study. Rather, the BMAC group received 3 mL of bone marrow aspirate injected directly into the defect site. At an average follow-up of 48 and 77 months in the BMS with and without BMAC groups, respectively, the Foot and Ankle Outcome Score (FAOS) as well as the SF-12 Physical Component Score significantly improved for both groups. No significant difference was found between groups in terms of the amount of change in either score. However, the MOCART score was significantly higher in the group with BMAC supplementation.

Summary

- Nonoperative treatment is initially indicated for patients with early-stage osteochondral lesions of the talus without loose intraarticular fragments, nondisplaced lesions, asymptomatic or minimally symptomatic patients, and those that are improving with physical therapy. Many of these lesions may heal without operative intervention.
- For patients with higher-grade lesions or those who do not respond to nonoperative management, operative treatment is indicated. For osteochondral lesions of the talus <1.5 cm^2, microfracture surgery is typically sufficient to provide satisfactory results (Grade B recommendation).[17]
- Salvage procedures may be performed for patients who do not respond to microfracture or OAT. These

may include repeat microfracture,[75] OAT (Grade B recommendation), osteochondral allograft transplantation (Grade C recommendation), or newer, less invasive procedures such as ACI or MAST.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

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