

Osteochondral Defects the Talus: Concepts For Diagnosis and Management

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Abstract

Talar osteochondral lesions (OLTs) are among the most challenging pathologic entities to cure. A solid strategy is needed for them. In order to get a good outcome from a lesion, it's important to consider its size, location, chronicity, and other features including displacement and subchondral cysts. Patients whose OLTs have migrated or who have not shown improvement after three to six months of nonoperative treatment are the ones who are typically considered for surgical intervention. The three main categories of surgical procedures for treating cartilage damage are regeneration, replacement, and repair. In order to reduce the morbidity caused by OLTs, further study is required to determine which of the numerous viable treatment approaches is better.

Keywords: Osteochondral Defects, Management, Talus

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Introduction

Talar osteochondral lesions (OLTs) affect both the talar articular cartilage and the subchondral bone beneath it. Osteochondritis dissecans, osteochondral deformities, and osteochondral fractures are all included in the term as a result. Ankle sprains and fractures are the most common causes of OLTs, with trauma being the main culprit. Over half of all ankle sprains and fractures result in OLTs.^{44,61} Appropriate treatment is essential to decrease morbidity and enable patients to resume sports or ADLs because of the high prevalence of ankle injuries that may cause cartilage damage, making OLTs a significant issue.

OLTs continue to be a major obstacle for orthopedic surgeons in terms of treatment efficacy for a variety of reasons. First, articular cartilage has a weak regenerative capacity and the talus has a poor blood supply, so the lesions don't have much of a chance of healing. In order to circumvent this limited

healing capacity, numerous surgical treatments have been devised. Unfortunately, there is a lack of consensus on the best course of treatment due to the paucity of comparative outcome studies evaluating these various options. When nonoperative treatment fails to alleviate symptoms after three to six months or when displaced OLTs are chronic, surgical intervention may be necessary. The three main types of surgical therapies for cartilage damage are regeneration, replacement, and repair. Osteochondral lesions of the talus are a common type of bone injury, and this article will go over the symptoms, signs, and treatment options for these lesions. It will also touch on some of the debates and potential future research on the subject.

Causes and effects

There is no one pathophysiology for OLTs because they include so many different diseases. Focal, idiopathic subchondral bone lesions can cause instability and even rupture of the neighboring articular cartilage in cases of osteochondritis dissecans.²⁰ Subchondral bone osteonecrosis is believed to contribute to damage to the underlying cartilage, and the dysfunction of the subchondral bone is believed to be vascular in nature. The precise etiology of the vascular insult is unclear; however, it may result from recurrent microtrauma or developmental disturbance of the anastomosing arteries between subchondral bone and cartilage.⁴⁷ After the knee and the elbow, the ankle is the joint that osteochondritis dissecans most commonly affects. About 0.09 percent of the population experiences osteochondritis dissecans of the talus, a condition that primarily manifests in middle age and older.⁴ Pain and mechanical symptoms like locking or catching when cartilage detaches from the underlying faulty subchondral bone are frequent in osteochondritis dissecans, while some patients may not experience any symptoms at all.

As a result of previous trauma, the majority of OLTs, such as osteochondral defects and open osteochondral fractures, develop. Ankle sprains alone account for more than 2 million cases annually, and cartilage injury occurs in almost half of all cases and as many as three quarters of all ankle fractures.^{44,61,70} According to traditional wisdom, trauma is the most common cause of lateral OLTs (94% of lateral lesions and 62% of medial lesions), but it is less common for medial lesions.⁶⁶ Traumatic impaction of the medial talar dome occurs when axial stress is paired with plantarflexion, inversion, and potentially external rotation, whereas axial load with inversion and dorsiflexion causes impaction of the lateral talar dome.⁸ The majority of lesions were localized in the centromedial and centrolateral zones, according to several large investigations that evaluated the anatomical characteristics of OLTs.^{22,27} total It is plausible that factors other than trauma contribute to the formation of certain medial talar lesions, as these investigations also discovered that medial lesions were typically deeper and linked to subchondral alterations.⁸

Due to a number of anatomical factors, talar bone and cartilage are at a higher risk of developing osteochondral lesions. Because of its lack of blood vessels, the cartilage that covers more than 60% of the talus has a low intrinsic regenerating ability. Therefore, the subchondral bone and synovial fluid are the primary sources of sustenance for cartilage. The already reduced capacity of talar cartilage to

recover from damage is made much worse by the talus's inadequate blood supply. The talus is supplied with blood via an intricate anastomotic network that branches off of the peroneal, posterior tibial, and anterior tibial arteries. One cadaveric investigation indicated that the posteromedial, posterolateral, and mid-medial regions of subchondral bone on the 9-section anatomical grid had comparatively low perfusion, which is caused by the complicated network of numerous arteries leading to watershed areas.⁴⁵ In addition, the talar dome and the cartilage above it get blood in a retrograde fashion, which makes it more vulnerable to specific types of injuries. Finally, when compared to other subtalar joints that bear weight, talar cartilage is noticeably thinner. According to research by Shepherd and Seedhom⁶⁵, cadaveric cartilage in the ankle was noticeably thinner than in the knee and hip. In contrast to the 1.5 to 2.6 mm observed in the knee, they discovered a thickness of 0.7 to 1.2 mm in the ankle. The talus is at increased risk for osteochondral lesions because to all of these reasons.

Evaluation and Diagnosis

Many factors, such as the aetiology and severity of the lesion, influence how patients with OLTs present themselves. Pain, edema, and stiffness are the most typical OLT symptoms, but they don't tell you much about the condition. Bearing weight can make these sensations worse. Mechanical symptoms like locking and catching, which are commonly linked to cartilage defects in other joints, also happen seldom with OLTs. Because first evaluations often produce broad differential diagnoses, doctors should always be on the lookout for these abnormalities. Some examples of these conditions include impingement, peroneal tendonopathy, lateral ankle instability, syndesmotic injury, concealed fractures, and subtalar or ankle arthritis.¹⁹

In order to rule out OLT, it is important to inquire about any ankle injuries the patient may have had in the past. Effusion, soreness with palpation of the lesion, reduced mobility, and discomfort upon inversion or dorsiflexion are all symptoms that could be detected during a physical examination.⁴⁹ It is important to compare the affected ankle to the other side while doing provocative tests like the anterior drawer and talar tilt. A thorough radiographic evaluation of the ankle joint is necessary. This should include weight-bearing anteroposterior, lateral, and mortise views. Lesions that aren't seen on radiographs or that can't be well described by them may require more advanced imaging using a computed tomography (CT) or magnetic resonance imaging (MRI) scan.

The initial line of imaging for evaluating OLTs should be weight-bearing anteroposterior, lateral, and mortise radiography images of the afflicted ankle. In 1959, the first OLT categorization using radiography was described by Berndt and Harty⁸ (Table 1). An area of subchondral compression is classified as stage I, a partially detached osteochondral fragment as stage II, a fully detached fragment that has not displaced from the fracture bed as stage III, and a detached and displaced fragment as stage IV, according to their original classification.⁸

Table 1. Staging Systems of Osteochondral Lesions of the Talus.

Radiography: Berndt and Harty ⁸	CT: Ferkel et al ²⁶	MRI: Hepple et al ³⁸	Arthroscopy
I. Subchondral Compression	I. Intact roof/cartilage with underlying cystic lesion	I. Articular Cartilage injury only	A. Smooth and intact but soft
II. Partially detached osteochondral fragment	IIA. Cystic lesion with communication to surface	IIA. Acute cartilage injury with bony fracture	B. Rough surface
III. Completely detached fragment without displacement	IIB. Open surface lesion with overlying fragment	IIB. Chronic cartilage injury with bony fracture	C. Fibrillation/fissures
IV. Detached and displaced fragment	III. Nondisplaced fragment with lucency beneath	III. Detached, nondisplaced bony fragment	D. Flap present or bone exposed
	IV. Displaced fragment	IV. Displaced fragment, uncovered subchondral bone	E. Loose, nondisplaced fragment
		V. Subchondral cyst present	F. Displaced fragment

It is necessary to conduct more advanced imaging, such as an MRI or CT scan, if the results of the radiographs do not support the suspicion of an osteochondral lesion. There are benefits and drawbacks to using any of these imaging techniques to assess osteochondral lesions. If radiographs come up negative, the next best study for finding OLTs is magnetic resonance imaging (MRI) due to its greater sensitivity. Researchers Verhagen et al.⁶⁹ discovered that MRI had a sensitivity of 0.96 for detecting OLTs, while CT scan had a sensitivity of 0.81.

The articular surface and soft tissues can be clearly seen with MRI, making it a valuable tool for describing OLTs.¹⁷ Nevertheless, due to its ability to show swelling in both bone and cartilage, it can exaggerate the size of OLTs or make it hard to determine the actual bone condition and precise osteochondral lesion dimensions.¹⁹ Contrarily, computed tomography (CT) scans provide a clearer picture of the subchondral bone's condition as well as the size and location of any cysts present.⁷³ In light of the significance of these criteria in deciding on the best course of therapy, computed tomography (CT) scans are useful for preoperative planning and are usually the go-to study when radiographs show an osteochondral lesion that might need surgery. It is important to tailor advanced imaging to each individual patient's needs; for example, some lesions may be best detected, evaluated, and treated with a combination of magnetic resonance imaging (MRI) and computed tomography (CT).

Modern imaging is the basis of various classification systems. Although they seldom direct treatment, they are historically significant and provide a standard way to describe lesions. Because of its correlation with clinical success, lesion size is a crucial component in selecting the most effective treatment; however, this dimension is ignored by most staging schemes. In 1999, Hepple et al.³⁸ created an MRI-based categorization system. Articular cartilage injury is the only type in stage I of this classification system. Stage II injuries include bone fractures, which are further classified as acute or chronic depending on whether there is edema. Stage III injuries include a detached, nondisplaced bone fragment. Stage IV injuries involve displaced bone fragments with exposed subchondral bone. Finally, stage V lesions involve a subchondral cyst (Table 1).

In 1990, a CT-based classification system was created by Ferkel et al. 26. According to this system, there are four stages of lesions: stage I corresponds to an intact roof or cartilage with a cystic lesion underneath, stage IIA to cystic lesions that communicate with the surface, stage IIB to an open surface lesion with an overlying fragment, stage III to nondisplaced fragments with lucency underneath, and stage IV to displaced fragments (Table 1).

Arthroscopy, with its ability to directly see and probe the lesions, offers the most comprehensive evaluation to guide therapy, making it the most effective staging technique even with imaging advancements. According to Table 1, the arthroscopy grading system involves the following categories: A for a smooth and intact but soft surface; B for a rough articular surface; C for fibrillations/fissures; D for a flap present or exposed bone; E for a loose, nondisplaced fragment; and F for a misplaced fragment.⁵⁵

The severity, duration, and symptoms of an OLT determine the course of treatment. Serial radiographs are typically taken to track the course of asymptomatic lesions. Lesions that are symptomatic upon presentation or develop symptoms are the ones that are treated. Nonoperative treatment options are often explored for patients with nondisplaced acute symptomatic lesions. After a brief immobilization phase of 6 weeks in a walking boot or short leg cast, patients gradually ease back into their normal activities.^{4,8,54} total About half of these instances respond well to nonoperative therapy.^{11,48} percent When nonoperative treatment fails to alleviate a lesion's symptoms and the lesion persists after 3 to 6 months, surgical intervention may be necessary. However, due to the decreased likelihood of spontaneous symptom remission, surgical intervention should be considered for acute lesions with displaced pieces prior to a trial of conservative treatment.¹¹ Various surgical procedures have been detailed for the treatment of OLTs. Methods for repairing, regenerating, and replacing cartilage can be classified according to Table 2.

Table 2. Classification of Operative Treatment Options for Osteochondral Lesions of the Talus.

Cartilage Repair	Cartilage Regeneration	Cartilage Replacement
A. Microfracture	A. Autologous chondrocyte implantation	A. Osteochondral allograft transfer
B. Retrograde drilling	B. Matrix-induced autologous chondrocyte implantation	B. Osteochondral allograft
	C. Autologous matrix-induced chondrogenesis	C. Particulated juvenile cartilage allograft transplantation

Cartilage Repair Strategies

Retrograde drilling and stimulation of the bone marrow (microfracture) are two methods for repairing cartilage. After nonoperative treatments have failed, bone marrow stimulation is typically said to be the initial line of defense. Figure 1 shows the approach that involves perforating the subchondral bone so that bone marrow progenitor cells can infiltrate the area. Fibrocartilage is produced within the defect as a result of these cells' stimulation of repair. Natural cartilage has superior biomechanical and structural properties than fibrocartilage because it is mainly made of type I collagen instead of the type II collagen found in most hyaline cartilage.^{52,58} But in 65–90% of patients, this method reliably improves clinically, meaning less pain and more function.^{29, 57, 60, 63}

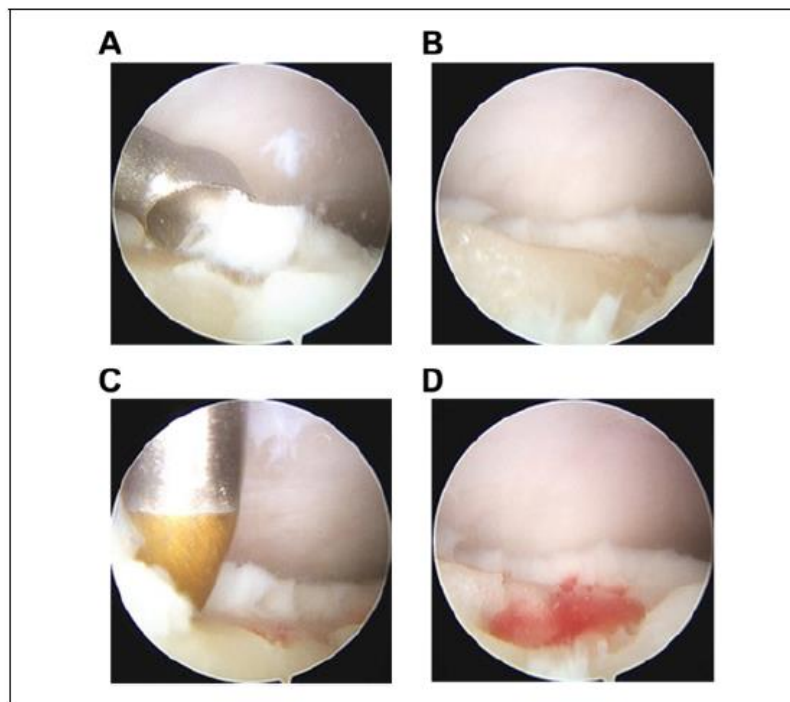


Figure 1. Microfracture technique. (A, B) Debridement of medial talar dome osteochondral lesion of the talus (OLT) to stable margins. (C) Microfracture performed. (D) Marrow elements seen exiting microfracture perforations.

The presence of subchondral cysts or related joint degeneration, as well as the patient's age, lesion chronicity, size, location, and containment, are among the variables that have been investigated as potential predictors of the success of bone marrow stimulation. The association between OLT size and result following microfracture is inverse. A total of 105 ankle osteochondral lesions (tibial and talar) that underwent ankle arthroscopy, debridement, and microfracture were examined by Chuckpaiwong et al.¹⁵ A favorable result was strongly associated with the extent of the lesion. Lesions with an average longitudinal and transverse dimensions of less than 15 mm did not result in treatment failures, but

just one patient (3% of the total) had a positive outcome when the lesion was 15 mm or bigger. In support of this, Choi et al.¹⁴ found that a good clinical outcome was achieved with an MRI cutoff of less than 150 mm². Lesions smaller than 1.5 cm² are best treated with microfracture.

Furthermore, compared to more acute, isolated lesions, older lesions and those associated with ankle arthritis do not reliably ameliorate symptoms with microfracture.^{39,41} There is conflicting evidence about the association between lesions with underlying cysts and poorer outcomes following microfracture. Microfracture for cystic lesions had a poor clinical outcome in 53% of investigations, while no difference in outcome ratings was detected between patients with cystic and noncystic lesions in 2 additional research.^{36,57,61} total Similarly, microfracture seems to have poorer clinical results for individuals with talar shoulder lesions that are not confined.¹² In conclusion, there is no evidence that lesion site (medial or lateral) or patient age correlate with clinical results, according to multiple investigations.^{12, 13, 7, 12,}

Retrograde drilling is preferable to microfracture as a reparative treatment for talar osteochondral lesions when there are subchondral bone defects or cysts with intact overlying cartilage. This is because it can treat the pathology without disturbing the overlying, healthy cartilage. Retrograde drilling has shown better functional outcomes for patients with these lesions, and second-look arthroscopies have not revealed lesion degradation.^{over 3,40,41}

Cartilage Regeneration Strategies

Methods for regenerating cartilage include transplanting cells generated from bone marrow, matrix-induced autologous chondrocyte implantation, and autologous chondrocyte implantation (ACI). When microfracture fails or when dealing with larger lesions that aren't good candidates for microfracture, these methods are usually used.

The initial stage of ACI, which consists of two stages, involves harvesting hyaline cartilage from either the front of the talus or a part of the knee that does not bear weight.^{6,51} times Chondrocytes, cultivated from this cartilage, have a potential shelf life of more than a year.⁵⁰ The second step of the process involves delivering the chondrocytes to the osteochondral lesion and then sewing a periosteal patch over the defect to keep them in place (Figure 2). The thinking behind this is that these chondrocytes can repair the chondral lesion by producing new hyaline cartilage to fill it.

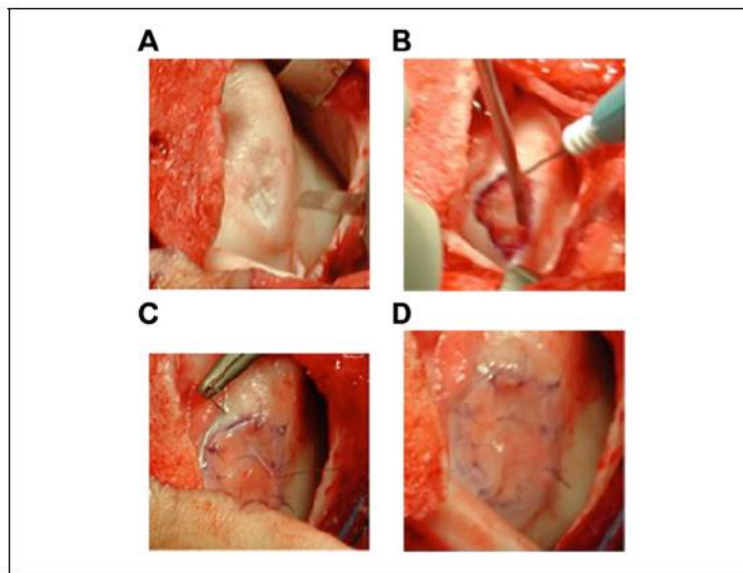


Figure 2. Autologous chondrocyte implantation (ACI). (A) Osteochondral lesion of the talus (OLT) (osteotomy performed). (B) OLT being debrided and chondrocytes being placed. (C) Periosteal patch being sutured over ACI. (D) Fibrin glue overlying ACI.

Some have had success with ACI. Functional results and patient-rated outcomes have been found to improve in multiple groups.⁵ minus 31, 42 plus 51 plus 7 It is worth mentioning that a number of these studies documented these favorable results in treating lesions that had not responded to microfracture in the past. The effectiveness of ACI is unaffected by lesion size, in contrast to microfracture. Lesion sizes larger than 137 mm² and ages younger than 26 were linked to substantially superior modified magnetic resonance scoring system (MOCART) scores in 1 study that evaluated lesions with second-look arthroscopy and MRI.⁴³ Lesions with a stable cartilage rim and a focal location are better candidates for ACI treatment. Therefore, ACI is frequently employed for lesions larger than 1 to 1.5 cm² that do not reach the shoulder.

MACI is an improved version of ACI that uses a matrix instead of a periosteal patch to hold the autologous chondrocytes that are implanted in situ. Using the matrix shortens the operation and lessens the risk of complications, and it should potentially allow for more uniform dispersion of chondrocytes inside the defect, even if the surgery is still two stages long. MACI has also shown promising results, with several case series showing improvements in functional outcome scores following MACI administration and a meta-analysis indicating an 89% mean success rate.^{33,62} Also, postoperative magnetic resonance imaging (MRI) has shown that individuals having MACI rather than microfracture had more normal-appearing cartilage tissue, and second-look arthroscopy has shown repaired articular surfaces.^{47, 53, 62}

Being two-stage procedures is one of the main downsides of ACI and MACI. Similar to ACI and MACI, autologous matrix-induced chondrogenesis (AMIC) use cells taken from bone marrow or other sources in an effort to accomplish the same aim in a single step. This method aims to create hyaline cartilage in the defect instead of the fibrocartilage that microfracture alone produces by combining microfracture with autologous iliac crest bone marrow aspirate concentrate (BMAC) or platelet-rich plasma (PRP) administered on a collagen matrix scaffold. There is evidence that AMIC improves functional outcomes and arthroscopically shows growth of more normal-appearing hyaline cartilage compared to microfracture, although evaluating data on AMIC is challenging because it incorporates so many different procedures.⁵⁶ patients were given ACI, while 25 were given 1-step AMIC; Giannini et al.²⁹ compared the two groups' outcomes. Both the MRI and second-look arthroscopy results were comparable, and their group did not notice any difference in the amount of improvement in the outcome ratings.

Cartilage Replacement Strategies

Osteochondral autograft transfer (OAT), which is exclusive to Arthrex in Naples, FL, osteochondral allograft, and particulated juvenile cartilage allograft transplantation (PJCAT) are all methods for substitute cartilage. Shoulder lesions without a solid cartilaginous rim or larger than 1 to 1.5 cm² typically require these therapies. Osteochondral lesions of the talus can be treated using hyaline cartilage that is taken from the patient's own talus or the nonweightbearing area of the knee using OAT procedures. One advantage of this surgery is that it restores a more typical cartilage surface to the lesion while preserving the grafts' type II collagen.³⁰ Even when applied to flaws bigger than 3.5 cm², this remains valid.³⁴ Using this method has improved pain and functional outcome scores in multiple studies.^{23,34, 37,64, 67} The biggest drawback of this method is the fact that donor site morbidity can occur.^{49,59}

Large, uncontained lesions of the talus shoulder are usually treated with osteochondral allograft transplantation. Bulk allograft and allograft plugs, a substitute for autograft plugs, are the two main components of osteochondral allograft transplantation. Licensed tissue banks use either fresh or frozen grafts taken from human cadavers for this procedure. Research has demonstrated that compared to fresh-frozen grafts, fresh grafts had better chondrocyte survival and less cartilage disintegration. Consequently, fresh grafts are deemed superior.^{74, 25, 24} The recipient talus's CT scan is used to size-match the donor allograft. One benefit of this method is that it can cure big cystic lesions without causing donor site morbidity, and another is that it can restore many dimensions of cartilage loss (Figure 3). Evidence suggests it may be useful; trials have demonstrated improvements in functional outcome scores and patients have reported good outcomes with lesions as big as 6 cm².^{1,21} times Nevertheless, problems such as graft instability, nonunion, resorption, and collapse are prevalent. As a result of these issues, salvage arthrodesis has a failure rate ranging from 13% to 33% in various case series.^{approximately 10,35,56}

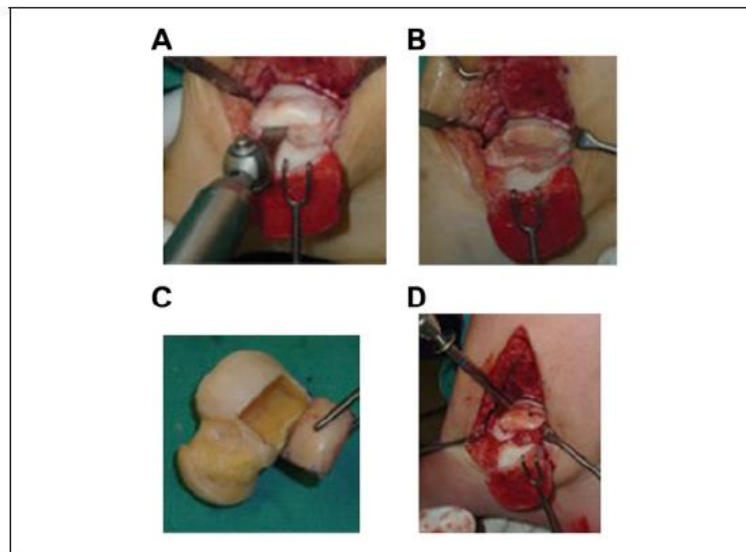


Figure 3. Osteochondral allograft transplantation. (A) Excision of talar shoulder lesion (osteotomy performed). (B) Talus with shoulder lesion removed. (C) Donor allograft with prepared graft removed. (D) Securing graft with 2 screws.

The process of PJCAT entails transferring new fragments of juvenile cartilage that contain living cells in their native extracellular matrixes. The donors of the cartilage are people who have passed away at any point in their lives, from infants up to adolescents. In other cases, an osteotomy is not necessary since the allograft's particle structure allows for its distribution through a narrower approach compared to osteochondral transplant. It can even be done completely by the use of arthroscopy.² There is no donor site morbidity in this one-stage process that involves securing the graft to the osteochondral lesion using fibrin glue (Figure 4). One group reported a case series of 23 patients treated with PJCAT by open and arthroscopic methods; their lesion sizes averaged 125 mm² and 7 mm in depth.¹⁶ Similar to previously published outcomes of patients treated with microfracture, ACI, and MACI, they reported outcomes at 16 months of follow-up for visual analog scale (VAS) pain, American Orthopaedic Foot & Ankle Society (AOFAS) ankle-hindfoot, and Foot and Ankle Ability Measure (FAAM) scores. Lesions that have not responded to prior bone marrow stimulation or those with a diameter larger than 15 mm are the ones that these authors utilize PJCAT on, despite the procedure's official indications have not been established. In cases when substantial bone grafting is required, the authors recommend structural allograft rather than the technically possible bone grafting under particulated juvenile cartilage. At present, there is a scarcity of allograft, there is a potential possibility of disease transmission, and there are no long-term data that support PJCAT.

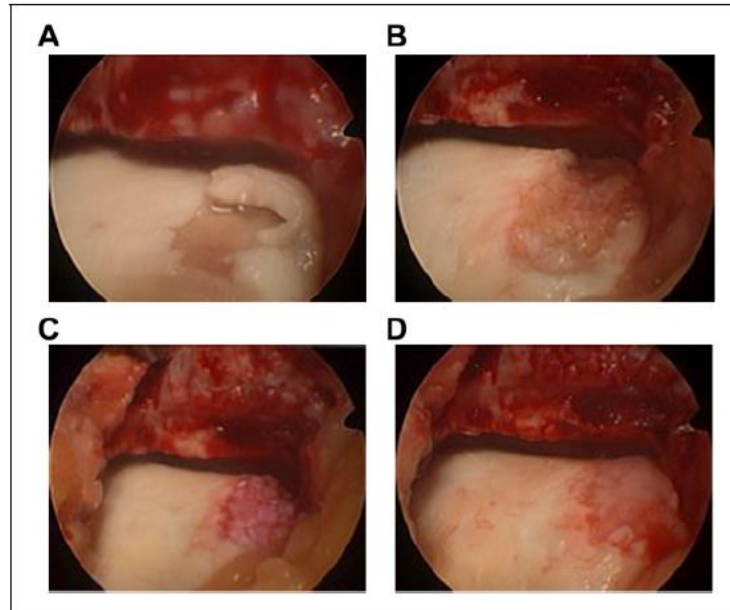


Figure 4. Particulated juvenile cartilage allograft transplantation. (A) Unstable medial talar dome osteochondral lesion of the talus. (B) Lesion debrided to stable margins. (C) Particulated juvenile cartilage allograft pieces were placed into the lesion bed. (D) The cartilage pieces were covered with fibrin glue.

The data supporting these therapies primarily come from smaller case series rather than comparative outcome studies or randomized controlled trials, although the several surgical procedures listed above do show promise in lowering the morbidity associated with osteochondral lesions of the talus. Treatment must be customized to each individual lesion and patient because there are no specific criteria to help orthopaedic surgeons determine the best course of action. The most objective metric is the growth of the lesion, and larger lesions typically call for more sophisticated replacement or regenerative methods than repairative ones. The extent to which the lesion penetrates the shoulder and the existence of subchondral cysts are additional crucial factors to take into account. Figure 5 lays out our methodology for selecting surgical treatments. To better understand which treatments work best for certain lesions and patients, additional randomized controlled trials and comparative research are required.

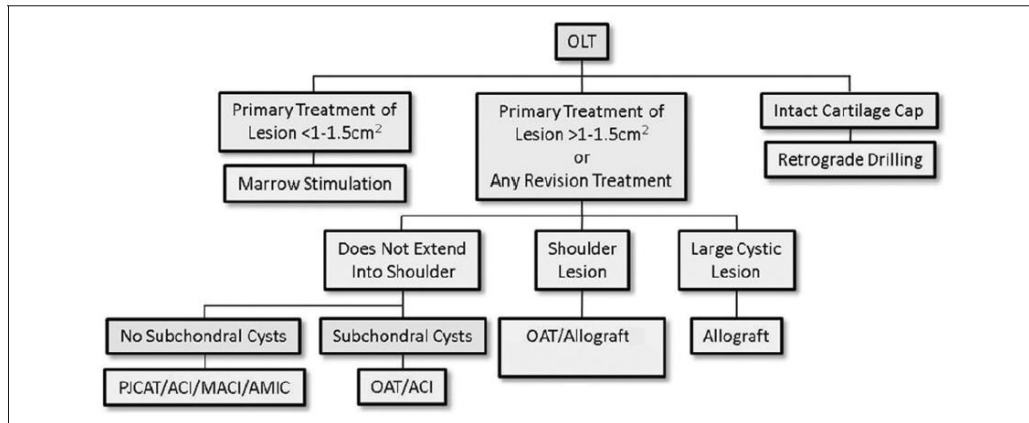


Figure 5. Algorithm for treatment of osteochondral lesions of the talus (OLTs). ACI, autologous chondrocyte implantation; AMIC, autologous matrix-induced chondrogenesis; MACI, matrix-induced autologous chondrocyte implantation; OAT, osteochondral allograft transplantation; PJCAT, particulated juvenile cartilage allograft transplantation.

Conclusion

Many different diseases can manifest as osteochondral lesions of the talus, which can lead to serious health complications for patients. Although trauma is the leading source of these lesions, OLTs can also occur in non-traumatic ways. Typical nonspecific symptoms seen by patients include discomfort, restricted range of motion, effusion, and inflammation. This means that clinicians need to be very suspicious in order to diagnose these lesions, and that sophisticated imaging methods are frequently necessary. Although protected weightbearing is the standard conservative treatment for nondisplaced OLTs at first, many patients will continue to experience symptoms and ultimately need surgical correction. The treatment of OLTs can be accomplished using a number of different surgical methods. Cartilage regeneration, cartilage replacement, and cartilage healing are the three main categories into which these approaches fall. Many of these methods have shown potential in lowering lesion-related symptoms in research. Because there hasn't been enough data on comparative results, orthopedic surgeons don't have any clear criteria to use when deciding whether treatment is best for their patients. To better understand which treatments work best and on what kinds of lesions, additional studies are required.

References:

1. Adams SB Jr, Viens NA, Easley ME, Stinnett SS, Nunley JA. Midterm results of osteochondral lesions of the talar shoulder treated with fresh osteochondral allograft transplantation. *J Bone Joint Surg Series A*. 2011;93(7):648-654.
2. Adams SB Jr, Demetracopoulos CA, Parekh SG, Easley ME, Robbins J. Arthroscopic particulated juvenile cartilage allo- graft transplantation for the treatment of osteochondral lesions of the talus. *Arthrosc Techn*. 2014;3(4):e533-e537.
3. Anders S, Lechler P, Rackl W, Grifka J, Schaumburger J. Fluoroscopy-guided retrograde core drilling and cancellous bone grafting in osteochondral defects of the talus. *Int Orthop*. 2012;36(8):1635-1640.
4. Bauer M, Jonsson K, Linden B. Osteochondritis dissecans of the ankle: a 20-year follow-up study. *J Bone Joint Surg Br*. 1987;69(1):93-96.
5. Baums MH, Heidrich G, Schultz W, Steckel H, Kahl E, Klingner HM. Autologous chondrocyte transplantation for treating cartilage defects of the talus. *J Bone Joint Surg Series A*. 2006; 88(2):303-308.

6. Baums MH, Heidrich G, Schultz W, Steckel H, Kahl E, Klin-ger HM. The surgical technique of autologous chondrocyte transplantation of the talus with use of a periosteal graft. Sur- gical technique. J Bone Joint Surg Am. 2007;89(suppl 2, pt 2): 170-182.
7. Becher C, Thermann H. Results of microfracture in the treat- ment of articular cartilage defects of the talus. Foot Ankle Int. 2005;26(8):583-589.
8. Berndt AL, Harty M. Transchondral fractures (osteochondritis dissecans) of the talus. J Bone Joint Surg Am. 1959;41A: 988-1020.
9. Brittberg M, Peterson L, Sjogren-Jansson E, Tallheden T, Lin- dahl A. Articular cartilage engineering with autologous chon- drocyte transplantation: a review of recent developments. J Bone Joint Surg Am. 2003;85A(suppl 3):109-115.
10. Bugbee WD, Khanna G, Cavallo M, McCauley JC, Gortz S, Brage ME. Bipolar fresh osteochondral allografting of the tibiotalar joint. J Bone Joint Surg Am. 2013;95(5):426-432.
11. Canale ST, Belding RH. Osteochondral lesions of the talus. J Bone Joint Surg Am. 1980;62(1):97-102.
12. Choi WJ, Choi GW, Kim JS, Lee JW. Prognostic significance of the containment and location of osteochondral lesions of the talus: independent adverse outcomes associated with uncon- tained lesions of the talar shoulder. Am J Sports Med. 2012; 41(1):126-133.
13. Choi WJ, Kim BS, Lee JW. Osteochondral lesion of the talus: could age be an indication for arthroscopic treatment? Am J Sports Med. 2012;40(2):419-424.
14. Choi WJ, Park KK, Kim BS, Lee JW. Osteochondral lesion of the talus: is there a critical defect size for poor outcome? Am J Sports Med. 2009;37(10):1974-1980.
15. Chuckpaiwong B, Berkson EM, Theodore GH. Microfracture for osteochondral lesions of the ankle: outcome analysis and outcome predictors of 105 cases. Arthroscopy. 2008;24(1): 106-12.
16. Coetzee JC, Giza E, Schon LC, et al. Treatment of osteochon- dral lesions of the talus with particulated juvenile cartilage. Foot Ankle Int. 2013;34(9):1205-1211.
17. De Smet AA, Fisher DR, Burnstein MI, Graf BK, Lange RH. Value of MR imaging in staging osteochondral lesions of the talus (osteochondritis dissecans): results in 14 patients. AJR Am J Roentgenol. 1990;154(3):555-558.
18. Donnenwerth MP, Roukis TS. Outcome of arthroscopic debri- dement and microfracture as the primary treatment for osteo- chondral lesions of the talar dome. Arthroscopy. 2012;28(12): 1902-1907.
19. Easley ME, Latt LD, Santangelo JR, Merian-Genest M, Nunley JA. Osteochondral lesions of the talus. J Am Acad Orthop Surg. 2010;18(10):616-630.
20. Edmonds EW, Shea KG. Osteochondritis dissecans: editorial comment. Clin Orthop Relat Res. 2013;471(4):1105-1106.
21. El-Rashidy H, Villacis D, Omar I, Kelikian AS. Fresh osteo- chondral allograft for the treatment of cartilage defects of the talus: a retrospective review. J Bone Joint Surg Am. 2011; 93(17):1634-1640.
22. Elias I, Jung JW, Raikin SM, Schweitzer MW, Carrino JA, Morrison WB. Osteochondral lesions of the talus: change in MRI findings over time in talar lesions without operative inter- vention and implications for staging systems. Foot Ankle Int. 2006;27(3):157-166.
23. Emre TY, Ege T, Cift HT, Demircioglu DT, Seyhan B, Uzun M. Open mosaicplasty in osteochondral lesions of the talus: a prospective study. J Foot Ankle Surg. 2012;51(5):556-560.
24. Enneking WF, Campanacci DA. Retrieved human allografts: a clinicopathological study. J Bone Joint Surg Am. 2001;83A(7): 971-986.
25. Enneking WF, Mindell ER. Observations on massive retrieved human allografts. J Bone Joint Surg Am. 1991;73(8):1123-1142.
26. Ferkel RD, Zanotti RM, Komenda GA, et al. Arthroscopic treatment of chronic osteochondral lesions of the talus: long- term results. Am J Sports Med. 2008;36(9):1750-1762.
27. Flick AB, Gould N. Osteochondritis dissecans of the talus (transchondral fractures of the talus): review of the literature and new surgical approach for medial dome lesions. Foot Ankle. 1985;5(4):165-185.

28. Giannini S, Buda R, Battaglia M, et al. One-step repair in talar osteochondral lesions: 4-year clinical results and t2-mapping capability in outcome prediction. *Am J Sports Med.* 2012; 41(3):511-518.
29. Giannini S, Buda R, Cavallo M, et al. Cartilage repair evolution in post-traumatic osteochondral lesions of the talus: from open field autologous chondrocyte to bone-marrow-derived cells transplantation. *Injury.* 2010;41(11):1196-1203.
30. Giannini S, Buda R, Grigolo B, Vannini F. Autologous chondrocyte transplantation in osteochondral lesions of the ankle joint. *Foot Ankle Int.* 2001;22(6):513-517.
31. Giannini S, Buda R, Ruffilli A, et al. Arthroscopic autologous chondrocyte implantation in the ankle joint. *Knee Surg Sports Traumatol Arthrosc.* 2014;22(6):1311-1319.
32. Giannini S, Buda R, Vannini F, Cavallo M, Grigolo B. One-step bone marrow-derived cell transplantation in talar osteochondral lesions. *Clin Orthop Relat Res.* 2009;467(12): 3307-3320.
33. Giza E, Sullivan M, Ocel D, et al. Matrix-induced autologous chondrocyte implantation of talus articular defects. *Foot Ankle Int.* 2010;31(9):747-753.
34. Gobbi A, Grancisco RA, Lubowitz JH, Allegra F, Canata G. Osteochondral lesions of the talus: randomized controlled trial comparing chondroplasty, microfracture, and osteochondral autograft transplantation. *Arthroscopy.* 2006;22(10): 1085-1092.
35. Gross AE, Agnidis Z, Hutchison CR. Osteochondral defects of the talus treated with fresh osteochondral allograft transplantation. *Foot Ankle Int.* 2001;22(5):385-391.
36. Han SH, Lee JW, Lee DY, Kang ES. Radiographic changes and clinical results of osteochondral defects of the talus with and without subchondral cysts. *Foot Ankle Int.* 2006;27(12): 1109-1114.
37. Hangody L, Fules P. Autologous osteochondral mosaicplasty for the treatment of full-thickness defects of weight-bearing joints: ten years of experimental and clinical experience. *J Bone Joint Surg Am.* 2003;85A(suppl 2):25-32.
38. Hepple S, Winson IG, Glew D. Osteochondral lesions of the talus: a revised classification. *Foot Ankle Int.* 1999;20(12): 789-793.
39. Kelberine F, Frank A. Arthroscopic treatment of osteochondral lesions of the talar dome: a retrospective study of 48 cases. *Arthroscopy.* 1999;15(1):77-84.
40. Kono M, Takao M, Naito K, Uchio Y, Ochi M. Retrograde drilling for osteochondral lesions of the talar dome. *Am J Sports Med.* 2006;34(9):1450-1456.
41. Kumai T, Takakura Y, Higashiyama I, Tamai S. Arthroscopic drilling for the treatment of osteochondral lesions of the talus. *J Bone Joint Surg Am.* 1999;81(9):1229-1235.
42. Kwak SK, Kem BS, Ferkel RD, Chan KW, Kasraeian S, Applegate GR. Autologous chondrocyte implantation of the ankle: 2- to 10-year results. *Am J Sports Med.* 2014;42(9): 2156-2164.
43. Lee KT, Lee YK, Young KW, Park SY, Kim JS. Factors influencing result of autologous chondrocyte implantation in osteochondral lesion of the talus using second look arthroscopy. *Scand J Med Sci Sports.* 2012;22(4):510-515.
44. Leontaritis N, Hinojosa L, Panchbhavi VK. Arthroscopically detected intra-articular lesions associated with acute ankle fractures. *J Bone Joint Surg Am.* 2009;91(2): 333-339.
45. Lomax A, Miller RJ, Fogg QA, Jane Madeley N, Senthil Kumar C. Quantitative assessment of the subchondral vascularity of the talar dome: a cadaveric study. *Foot Ankle Surg.* 2014;20(1):57-60.
46. Magnan B, Samaila E, Bondi M, Vecchini E, Micheloni GM, Bartolozzi P. Three-dimensional matrix-induced autologous chondrocytes implantation for talus osteochondral lesions. *J Orthop Traumatol.* 2012;13:S76-S77.
47. McCoy AM, Toth F, Dolvik NI, et al., Articular osteochondrosis: a comparison of naturally-occurring human and animal disease. *Osteoarthritis Cartilage.* 2013;21(11): 1638-1647.
48. McCullough CJ, Venugopal V. Osteochondritis dissecans of the talus: the natural history. *Clin Orthop Relat Res.* 1979;144: 264-268.
49. McGahan PJ, Pinney SJ. Current concept review: osteochondral lesions of the talus. *Foot Ankle Int.* 2010;31(1):90-101.
50. Mitchell ME, Giza E, Sullivan MR. Cartilage transplantation techniques for talar cartilage lesions. *J Am Acad Orthop Surg.* 2009;17(7):407-414.

51. Nam EK, Ferkel RD, Applegate GR. Autologous chondrocyte implantation of the ankle: a 2- to 5-year follow-up. *Am J Sports Med.* 2009;37(2):274-284.
52. Nehrer S, Spector M, Minas T. Histologic analysis of tissue after failed cartilage repair procedures. *Clin Orthop Relat Res.* 1999;365:149-162.
53. Niemeyer P, Salzmann G, Schmal H, Mayr H, Sudkamp NP. Autologous chondrocyte implantation for the treatment of chondral and osteochondral defects of the talus: a meta- analysis of available evidence. *Knee Surg Sports Traumatol Arthrosc.* 2012;20(9):1696-1703.
54. Pettine KA, Morrey BF. Osteochondral fractures of the talus: a long-term follow-up. *J Bone Joint Surg Br.* 1987; 69(1):89-92.
55. Pritsch M, Horoshovski H, Farine I. Arthroscopic treatment of osteochondral lesions of the talus. *J Bone Joint Surg Am.* 1986; 68(6):862-865.
56. Raikin SM. Fresh osteochondral allografts for large-volume cystic osteochondral defects of the talus. *J Bone Joint Surg Am.* 2009;91(12):2818-2826.
57. Robinson DE, Winson IG, Harries WJ, Kelly AJ. Arthroscopic treatment of osteochondral lesions of the talus. *J Bone Joint Surg Br.* 2003;85(7):989-993.
58. Rothrauff BB, Tuan RS. Cellular therapy in bone-tendon inter- face regeneration. *Organogenesis.* 2014;10(1):13-28.
59. Sammarco GJ, Makwana NK. Treatment of talar osteochondral lesions using local osteochondral graft. *Foot Ankle Int.* 2002; 23(8):693-698.
60. Savva N, Jabur M, Davies M, Saxby T. Osteochondral lesions of the talus: results of repeat arthroscopic debridement. *Foot Ankle Int.* 2007;28(6):669-673.
61. Saxena A, Eakin C. Articular talar injuries in athletes: results of microfracture and autogenous bone graft. *Am J Sports Med.* 2007;35(10):1680-1687.
62. Schneider TE, Karaikudi S. Matrix-induced autologous chondrocyte implantation (maci) grafting for osteochondral lesions of the talus. *Foot Ankle Int.* 2009;30(9):810-814.
63. Schuman L, Struijs PA, van Dijk CN. Arthroscopic treatment for osteochondral defects of the talus: results at follow-up at 2 to 11 years. *J Bone Joint Surg Br.* 2002;84(3):364-368.
64. Scranton PE Jr, Frey CC, Feder KS. Outcome of osteochondral autograft transplantation for type-V cystic osteochondral lesions of the talus. *J Bone Joint Surg Br.* 2006;88(5): 614-619.
65. Shepherd DE, Seedhom BB. Thickness of human articular cartilage in joints of the lower limb. *Ann Rheum Dis.* 1999; 58(1):27-34.
66. Tol JL, Struijs PA, Bossuyt PM, Verhagen RA, van Dijk CN. Treatment strategies in osteochondral defects of the talar dome: a systematic review. *Foot Ankle Int.* 2000;21(2): 119-126.
67. Valderrabano V, Leumann A, Rasch H, Egelhof T, Hintermann B, Pagenstert G. Knee-to-ankle mosaicplasty for the treatment of osteochondral lesions of the ankle joint. *Am J Sports Med.* 2009;37(suppl 1):105S-111S.
68. Valderrabano V, Miska M, Leumann A, Wiewiorski M. Reconstruction of osteochondral lesions of the talus with autologous spongiosa grafts and autologous matrix-induced chondrogenesis. *Am J Sports Med.* 2013;41(3):519-527.
69. Verhagen RA, Maas M, Dijkgraaf MG, Tol JL, Krips R, van Dijk CN. Prospective study on diagnostic strategies in osteochondral lesions of the talus: is MRI superior to helical CT? *J Bone Joint Surg Br.* 2005;87(1):41-46.
70. Waterman BR, Belmont PJ, Cameron KL, Deberardino TM, Owens BD. Epidemiology of ankle sprain at the United States Military Academy. *Am J Sports Med.* 2010;38(4): 797-803.
71. Whittaker JP, Smith G, Makwana N, et al. Early results of autologous chondrocyte implantation in the talus. *J Bone Joint Surg Br.* 2005;87(2):179-183.
72. Williams SK, Amiel D, Ball ST, et al. Prolonged storage effects on the articular cartilage of fresh human osteochondral allografts. *J Bone Joint Surg Am.* 2003;85A(11): 2111-2120.
73. Zinman C, Wolfson N, Reis ND. Osteochondritis dissecans of the dome of the talus: computed tomography scanning in diagnosis and follow-up. *J Bone Joint Surg Am.* 1988;70(7): 1017-1019.