VOLUME 69 | ISSUE 2 | APRIL - JUNE 2018

Osteochondral Lesions in Foot and Ankle (current treatment strategies and author's developed technique)

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ABSTRACT

Osteochondral lesion of the talus (OLT) is a broad term used to describe an injury or abnormality of the talar articular cartilage and adjacent bone. A variety of terms have been used to refer to this clinical entity, including osteochondritis dissecans (OCD), osteochondral fracture and osteochondral defect. Whether OLT is a precursor to more generalised arthrosis of the ankle remains unclear, but the condition is often symptomatic enough to warrant treatment. In more than one third of cases, conservative treatment is unsuccessful, and surgery is indicated. There is a wide variety of treatment strategies for osteochondral defects of the ankle, with new techniques that have substantially increased over the last decade. The common treatment strategies of symptomatic osteochondral lesions include nonsurgical treatment, with rest, cast immobilisation and use of nonsteroidal anti-inflammatory drugs (NSAIDs). Surgical options are lesion excision, excision and curettage, excision combined with curettage and microfracturing, filling the defect with autogenous cancellous bone graft, antegrade (transmalleolar) drilling, retrograde drilling, fixation and techniques such as osteochondral transplantation [osteochondral autograft transfer system (OATS)] and autologous chondrocyte implantation (ACI). Furthermore, smaller lesions are symptomatic and when left untreated, OCDs can progress; current treatment strategies have not solved this problem. The target of these treatment strategies is to relieve symptoms and improve function. Publications on the efficacy of these treatment strategies vary. In most cases, several treatment options are viable, and the choice of treatment is based on defect type and size and preferences of the treating clinician.

KEY WORDS: Osteochondral lesions; Osteochondritis dissecans; Talus; Foot and ankle; Cartilage damage; Subchondral bone

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VOLUME 69 | ISSUE 2 | APRIL - JUNE 2018

Introduction

Chondral and osteochondral injuries are relatively common in the weight bearing joints of the lower extremity (**Fig. 1**). The pathology can range from a simple contusion of the articular cartilage and subchondral bone to a fracture involving the cartilage alone or cartilage and underlying subchondral bone together. The mechanism of injury is one of three types of trauma: compaction, shearing, or avulsion. Because the injury is usually subtle and causes little to no dysfunction, the diagnosis of acute injuries is delayed. An osteochondral ankle defect is a lesion of the talar cartilage and subchondral bone mostly caused by a single or multiple traumatic events, leading to partial or complete detachment of the fragment. The defects cause deep ankle pain associated with weight bearing. Impaired function, limited range of motion, stiffness, catching, locking and swelling may be present.

The earliest report of osteochondritis dissecans (OCD) was published in 1888 by Konig, who characterized a loose-body formation associated with articular cartilage and subchondral bone fracture [1]. In 1922, Kappis described this process in the ankle joint [2]. On the basis of a review of all literature describing transchondral fractures of the talus, Berndt and Harty (**Fig. 2**) developed a classification system for radiographic staging of osteochondral lesions of the talus (OLTs) [3]. Their classification system has been the foundation for other systems, yet it remains the most widely used system today. Anatomic studies on cadaver limbs demonstrated the etiological mechanism of transchondral fractures of the lateral border of the talar dome. As the foot is inverted on the leg, the lateral border is compressed against the face of the fibula (stage I), while the collateral ligament remains intact. Further inversion ruptures the lateral ligament and begins avulsion of the chip (stage II), which may be completely detached but remain in place (stage III) or be displaced by inversion (stage IV). Berndt and Harty experimentally proved the traumatic etiology of the lesion; however, non-traumatic lesions also occur. Loomer et al. [4] added a stage V to this system, considering the presence of a subchondral cyst. Ferkel and Sgaglione [5] developed a classification system based on computerized tomography, while Hepple et al. developed an MRI classification system [6].

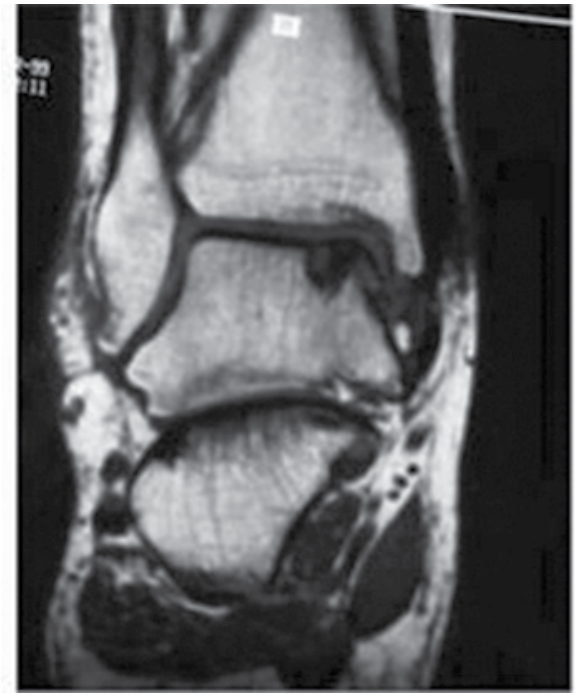

Fig. 1 *MRI image of an osteochondral lesion of the talus*

There is a wide variety of treatment strategies for osteochondral defects of the ankle, with new techniques that have substantially increased over the last decade. The widely considered treatment strategies of symptomatic osteochondral lesions include the non-surgical treatment with rest, cast immobilization and use of NSAIDs, and surgical excision of the lesion, excision and curettage, excision combined with curettage and microfracturing, filling of the defect with autogenous (cancellous) bone graft, antegrade (transmalleolar) drilling, retrograde drilling, fixation and techniques like osteochondral transplantation (osteochondral autograft transfer system, OATS) and autologous chondrocyte implantation (ACI).

Target of these treatment strategies is to relief symptoms and to improve function. Publications on the effectiveness of these treatment strategies vary. In most cases, several treatment options are viable, and the choice of treatment is based on the type and size of the defect and on preferences of the treating clinician.

VOLUME 69 | ISSUE 2 | APRIL - JUNE 2018

Conservative treatment of the osteochondral lesions of talus

Conservative treatment usually consists of immobilization and no weight-bearing, with or without treatment of non-steroidal anti-inflammatory drugs (NSAIDs) for approximately 6 weeks, followed by progressive weight-bearing and physical therapy. This protocol is instituted for Berndt and Harty type I and II lesions and small grade III lesions. Large grade III and any grade IV lesions are generally considered operative candidates. Additionally, grade I and II lesions that fail non-surgical management are also operative candidates [7]. Berndt and Harty [3] reported poor outcomes for nonoperative treatment of OLTs in their original article: good in 16%, fair in 9%, and poor in 75%. A systematic review of treatment strategies for OLT by Verhage et al. [8] in 2003 demonstrated only a 45% success rate for nonoperative treatment. The aim is to unload the damaged cartilage, so edema can resolve and necrosis is prevented. Tol et al. [9] on another review of the literature noted a success rate of only 45% with nonoperative treatment. The duration of symptoms prior to institution of non-operative treatment was either unreported or ranged from sub-acute to acute (<6 weeks) to chronic (>6 weeks). Patients were given the choice between operative and non-operative treatments, and the patient chose non-operative treatment. Conservative treatment consisted of weight bearing as tolerated and reported to be successful in a range 20%-54%.

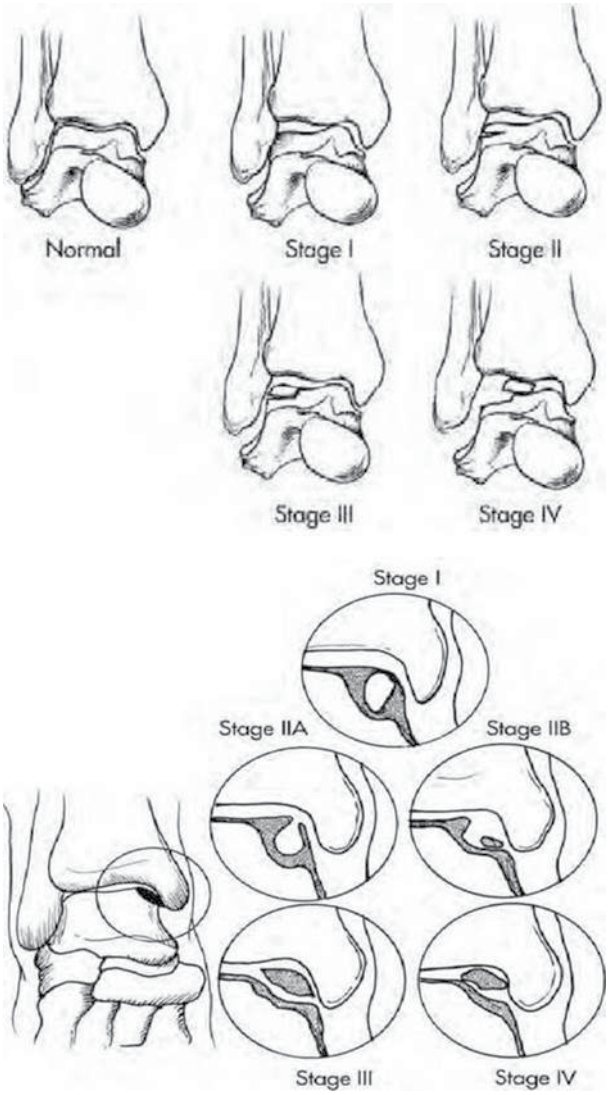

Fig. 2 *Berndt and Hardy classification*

Surgical management of the osteochondral lesions of talus

Retrograde drilling is usually reserved for large OCDs with intact overlying cartilage. This is a technique used for stable primary OCDs when there is more or less intact cartilage with a large subchondral cyst, or when the defect is hard to reach via the usual anterolateral and anteromedial portals, usually Berndt and Harty types I and II. Drilling attempts to bring blood supply to the lesion without disrupting the articular cartilage. It is the treatment of choice when there is a large subchondral cyst with overlying healthy cartilage. For medial lesions, arthroscopic drilling can take place through the sinus tarsi. For lateral lesions the cyst is approached from anteromedial. The aim is to induce subchondral bone revascularization and to stimulate the formation of new bone. Kono et al [10] and Taranow et al. [11] reported success of the treatment in a range of 81–100%.

Transmalleolar antegrade drilling is considered in cases of osteochondral lesions that present difficulty to be approached because of its location on the talar dome. In this technique, a K-wire is inserted about 3 cm proximal to the tip of the medial malleolus and directed across the medial malleolus into the lesion through the intact cartilage. Kono et al.

VOLUME 69 | ISSUE 2 | APRIL - JUNE 2018

[10] and Robinson et al. [12] described the results of this technique that was reported to be successful in 63% of cases.

Surgical treatment includes excision, where the partially detached fragment is excised, and the defect itself is left untreated; excision and debridement, where after excision of the loose body, the surrounding necrotic subchondral tissue is curetted using either an open or arthroscopic technique; excision, debridement and bone marrow stimulation, where after excision and curettage multiple openings into the subchondral bone are created by drilling or by microfracturing. This way, intra-osseous 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, bone marrow cells are introduced in the osteochondral defect, and fibro-cartilaginous tissue is formed. Van Dijk et al [13] in a review of the literature noted a success rate of 54% with the excision technique, where excision was performed for superficial cartilaginous lesions, with mainly intact underlying subchondral bone. Respectively, excision and curettage, reported a successful rate of 77%, where most patients had a Berndt and Harty stage III or IV lesion, although stage II lesions occurred. Finally, the treatment option of excision, debridement and bone marrow stimulation, reported the best rate of success (85%), where most patients often had a Berndt and Harty stage III or IV lesion, although stage I and II lesions occurred, whilst diameter of the lesions usually did not exceed 1.5 cm.

Kouvalchouk et al. [14] studied the filling of the defect with autogenous bone graft. In this technique, the remaining defect after excision and debridement of osteochondral lesions of the dome of the talus with partial necrosis, is filled with autogenous cancellous bone targeting to restore the mechanical properties of the talus. Indications for the treatment were large, often medial lesions exceeding 1.5 cm in diameter.

Larger lesions that fail to improve 6 months after arthroscopy should be considered for osteochondral grafting or autologous chondrocyte implantation. The concept of using restorative cartilage treatment with a osteochondral autograft and allograft has been reported in the literature [15,16]. The results of osteochondral autograft transplantation have been reported at intermediate follow-up with good results. Two related procedures have been developed: mosaicoplasty and OATS. Both are reconstructive bone grafting techniques that use one or more cylindrical osteochondral grafts from the less weight-bearing periphery of the ipsilateral knee and transplant them into the prepared defect site on the talus. This technique's target is to restore the mechanical, structural and biochemical properties of the original hyaline articular cartilage. It is carried out either by an open approach or by an arthroscopic procedure. Indications involve large, often medial lesions, sometimes with a cyst underneath. Osteochondral grafting of defects has yielded 90% to 94% good to excellent results, with Scranton et al. [17] noting 90% satisfaction in 50 patients at 36-month follow-up and Hangody et al. [18] reporting 94% good to excellent results in 36 patients at an average of 4.2 years.

Autologous chondrocyte implantation attempts to regenerate tissue with a high percentage of hyaline-like cartilage. The ACI technique involves placing cultured chondrocytes under a periosteal patch that covers the lesion. It is done for lesions larger than 1 squared cm, in the absence of generalized osteoarthritic changes. Harvesting is first accomplished from either the knee or ankle from the region on the perimeter of the talus lesion. A second procedure is performed after the cells have been cultured for 6 to 8 weeks. An osteotomy of the medial malleolus can be done for medial defects. The damaged articular surface is curetted to a stable border, and a periosteal patch is harvested from the tibia. The patch is sutured to the defect and sealed with fibrin glue. Finally, the cultured chondrocytes are injected under the patch. Whittaker et al [19] reported their results with ACI on 10 patients with a 4-year follow-up: 90% of patients were pleased with the results of the surgery at 24 months, with no change at 48 months.

In Europe and Australasia, matrix-based chondrocyte implantation (MACI) is available. It differs from traditional ACI in that the chondrocytes are not placed under the periosteal patch but are em-

VOLUME 69 | ISSUE 2 | APRIL - JUNE 2018

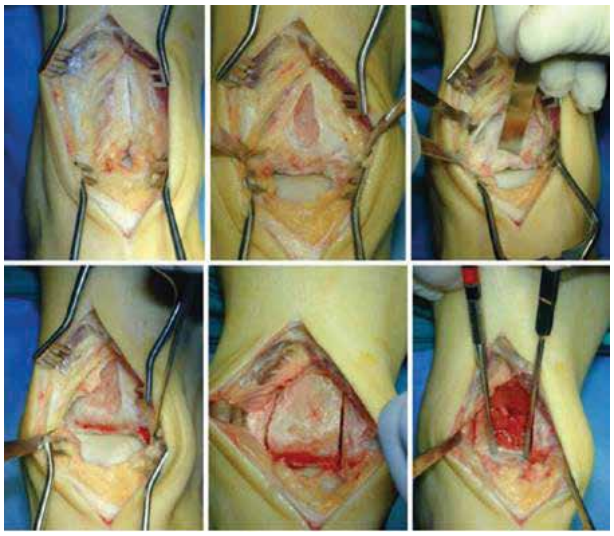

Fig. 3 *Trapezoid wedge shape for tibial osteotomy providing perpendicular access to the recipient site*

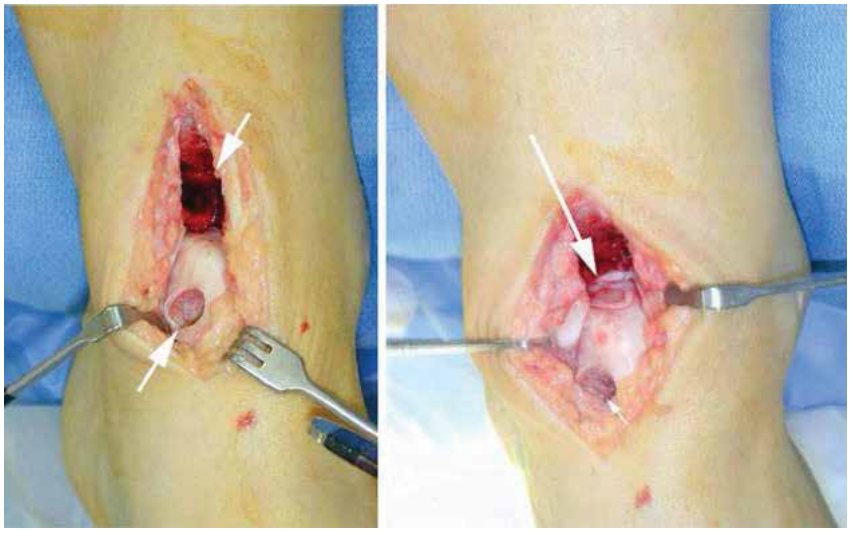

Fig. 4 *Donor medial talar facet recipient site with the local graft inserted*

bedded in a type I/III collagen membrane bilayer. As with ACI, the membrane is placed in the defect, but sutures are not required. The membrane bilayer is secured into place using fibrin sealant. Matrix-based chondrocyte implantation is technically easier than ACI and does not require an osteotomy.

Fixation technique is another treatment option in case of large, loose fragments that can be reattached to the underlying bone. Fixation to the talus may be obtained with headless screws, K-wires, absorbable pins or fibrin glue. Kumai et al [20] reported a success rate of 89% in 24 patients, where osteochondral lesions of stage II,III and IV were elevated, the defect was curetted and drilled, and following alignment of the bone fragment it was secured to the underlying bone with at least two bone pegs from the distal tibia. This type of injury is usually seen in acute injuries, and this technique typically fails in chronic lesions with sclerotic borders.

However, authors' preferred surgical treatment of talar osteochondral lesions is the use of local osteochondral talar autograft. In this surgical procedure, with the patient in supine position and under tourniquet control, an arthrotomy is performed through a 7cm antero-medial or antero-lateral incision, as required. The lesion is approached by removing a bone block from the tibia including the articular surface. To accomplish this, a wedge shaped bone block, 10mm wide, 20mm deep and 30mm in height is made at the distal anterior tibia

VOLUME 69 | ISSUE 2 | APRIL - JUNE 2018

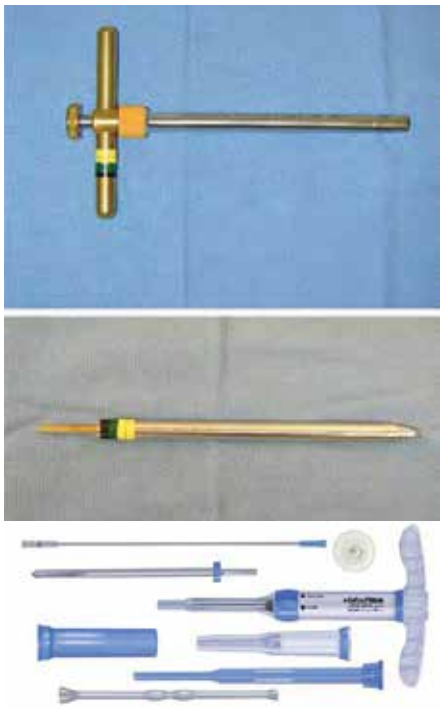

Fig. 5 *Instrumention*

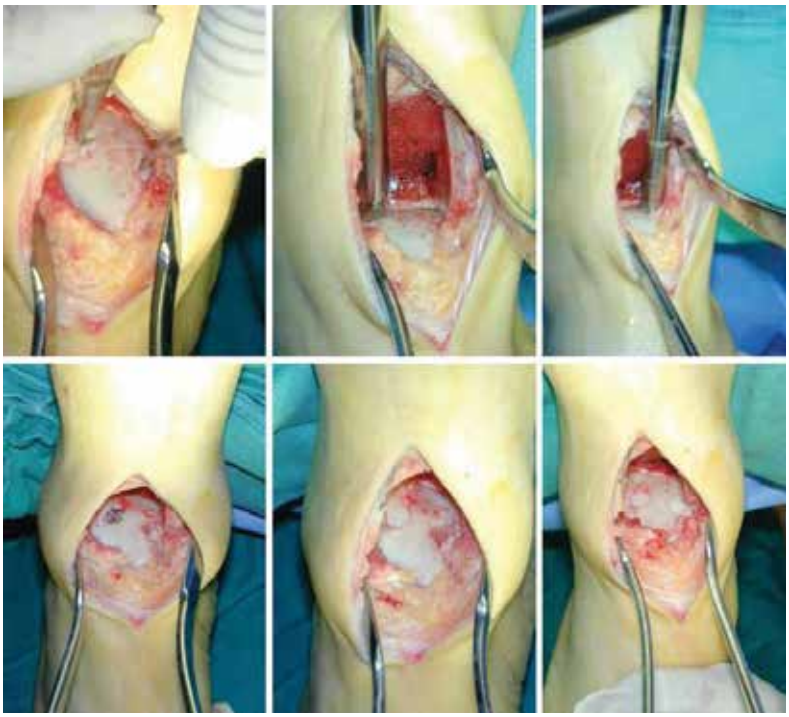

Fig. 6 *Perpendicular access to the recipient site: a case with two lesions*

articular surface on the side of the osteochondral lesion. Vertical parallel saw cuts are made with a high-speed micro oscillating saw by taking care to avoid injuring the uninvolved talar articular surface. Saw is then used to connect the two vertical parallel cuts proximally in the metaphysis. A 10mm wide thin osteotome is then driven from the tip of the transverse cut, inferiorly to the articular surface of the tibial plafond 10 to 20mm deep, depending on the location of the lesion on the talar dome (**Fig. 3**). The tibial fragment is removed and the defect created permits direct access to the lesion, especially by plantar-flexing the ankle. Initial debridement of loosen fragments is followed by drilling and ensuring that drill is perpendicular to the articular surface of the talus, directly over the lesion. Drill sizes are matched to the diameter of the defect 4, 6 or 8mm to the size of the defect, determined from the MRI pre-operative evaluation. The osteochondral graft is harvested from the anterior aspect of the ipsilateral talar articular facet by the same incision, as the tibial osteotomy. The graft is harvested using the core-harvesting device, by positioning the cutter over the talar facet, near to the anterior border and perpendicularly to the articular surface (**Fig. 4**). When the cutter reaches the desired depth, the harvester is removed with the graft and a positioning device is placed perpendicularly to the talar dome, orienting the outer flair of the graft toward the outer edge of the dome (**Figures 5 and 6**). For medial lesions (**Fig. 7**), a Chevron-type medial malleolar osteotomy is performed, that is fixed with two screws at the end of the procedure. The approach to lateral lesions is performed by an anterolateral incision splitting down the ATFL and CFL, followed by a modified Brostrom technique (**Fig. 8**).

The postoperative treatment includes immobilization for four weeks, walker boot for the next four weeks and weight bearing at six weeks. Range of motion exercises are allowed once the surgical incision is healed. In a retrospective study (T.Badekas, N.Souras, paper in process of publication, 2/18) of 121 patients from March 2005 to March 2015, 118 men and 3 women, with an age range of 19–53 (mean 38) years, symptom duration of 65 (range 6–98) months and standard follow-up of 36 months, the result was the significant improvement of an average

VOLUME 69 | ISSUE 2 | APRIL - JUNE 2018

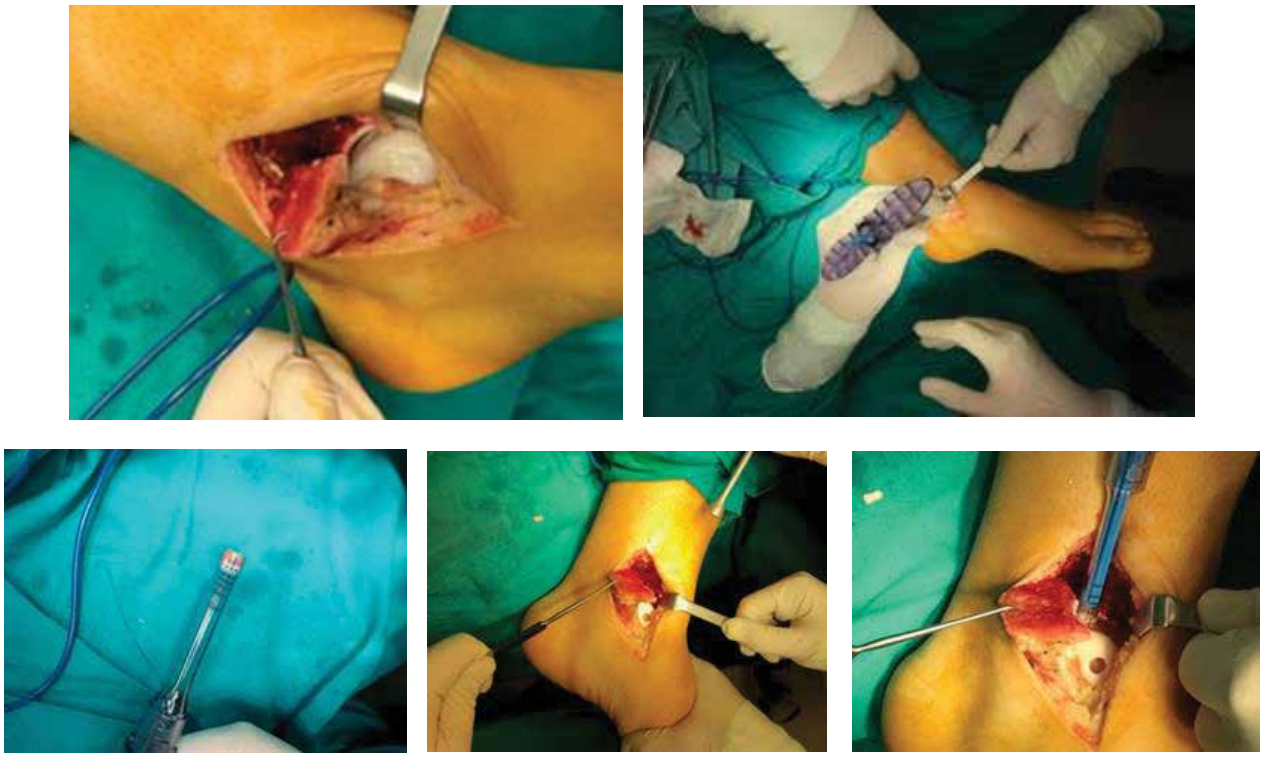

Fig. 7 *Medial malleolus osteotomy for true medial lesions*

preoperative AOFAS score, using the Ankle–Hindfoot Scale, that was 65 in an average postoperative AOFAS score that was 89. Patients younger than 40 years had higher average AOFAS scores postoperatively than patients older than 40. The presence of degenerative arthritis yielded a lower AOFAS score. However, the difference between these small subgroups was not significant. There were no perioperative complications and all patients stated they would undergo the procedure again.

Discussion

The talus is the third most common location of osteochondral lesions behind the knee and the elbow. Patients typically present after a traumatic injury to the ankle (85%) and complain of prolonged pain, swelling, catching, stiffness, and instability. Severe mechanical symptoms such as catching and grinding may indicate a severe OLT and possibly a loose body. A loose body can disrupt normal joint motion secondary to displacement of the fragment and can lead to arthrosis over time. Chronic ankle pain and stiffness without improvement from standard conservative measures should increase the suspicion for OLT.

OCLs of the talus more commonly affect men, in the right ankle, on the medial side. Lateral lesions are traumatic, whereas medial lesions may be atraumatic [3]. It has previously been recognized that medial and lateral lesions differ morphologically with lateral lesions presenting as flat, discoid fragments and medial lesions presenting as more rounded and deeper [21]. MRI studies have demonstrated that medial lesions tend to be deeper and more well defined, while lateral lesions are more superficial and less discrete in location. Lateral lesions are more liable to be displaced and so become symptomatic at an earlier stage. The morphological appearance of medial and lateral lesions can be explained by the different forces that are necessary to produce them. Lateral lesions are produced by a tangential shear force across the talar dome, whereas medial lesions are caused by a more perpendicular force resulting in a deeper lesion which is unlikely to displace from its bed [22].

Treatment strategies for osteochondral defects

VOLUME 69 | ISSUE 2 | APRIL - JUNE 2018

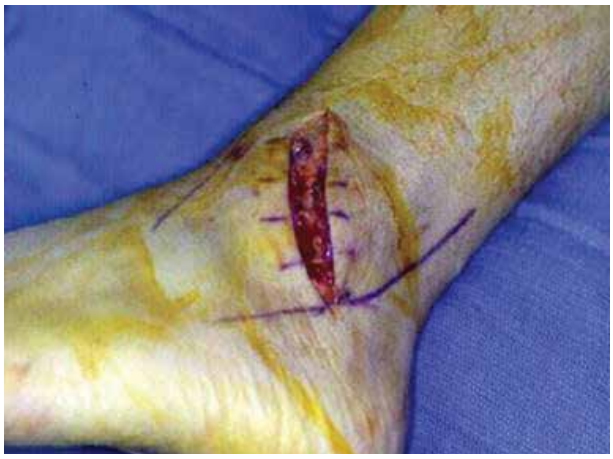

Fig. 8 *Lateral osteochondral lesion of the talus (OLT) approached through an anterolateral incision, with takedown of the anterior tibiofibular ligament (ATFL) and the calcaneofibular ligament (CFL). Reconstruction with modified Broström*

(OCDs) of the ankle have substantially increased over the last decade. The widely published treatment strategies of symptomatic osteochondral lesions include the non-surgical treatment and surgical excision of the lesion, excision and curettage, excision combined with curettage and microfracturing, placement of cancellous bone graft, antegrade (transmalleolar) drilling, retrograde drilling, fixation and techniques like osteochondral transplantation (osteochondral autograft transfer system, OATS) and autologous chondrocyte implantation (ACI).

Retrograde drilling is a technique used for stable lesions with an intact chondral surface (Berndt and Harty types I and II). Drilling attempts to bring blood supply to the lesion without disrupting the articular cartilage. Transmalleolar drilling is performed when a defect is hard to reach because of its location on the talar surface. A disadvantage is that healthy tibial cartilage is damaged.

Primary repair works best for large OCD lesions with healthy-appearing surface cartilage that is attached to a bone fragment. Fixation to the talus may be obtained with headless screws, K-wires, or absorbable pins. This type of injury is usually seen in acute injuries, and this technique typically fails in chronic lesions with sclerotic borders.

Microfracture stimulates subchondral bleeding and development of a fibrin clot. Debridement of diseased cartilage and subchondral cysts prior to microfracture is of paramount importance. Awls or drills are used after sufficient debridement to perforate the base of the lesion (3-4 mm apart) and bring mesenchymal stem cells, growth factors, and healing proteins to the defect. This fibrin clot heals in the defect and eventually becomes fibrocartilage (type I cartilage), which fills the void but lacks the organized structure of hyaline cartilage (type II cartilage). Fibrocartilage possesses inferior wear characteristics to hyaline cartilage, which has led investigators to develop articular cartilage transplantation.

Restoration of articular cartilage can be achieved by osteochondral autograft or allograft transplantation (OATS, mosaicoplasty), autologous chondrocyte implantation (ACI and MACI), and fresh osteochondral allografts (FOCAT).

The most important finding of a recent review of literature of van Djik et al. [13] was that bone marrow stimulation was identified as the best treatment option. In the same review, the results of non-operative treatment were low compared to operative treatment. In spite of this, non-operative treatment should always be the first treatment to be considered.

Nowadays literature on the treatment of osteochondral lesions of the talus involves arthroscopic excision, curettage and bone marrow stimulation, ACI and OATS. ACI is a relatively expensive technique, and OATS gives morbidity from knee complaints in a relevant number of patients. Therefore, is recommended [13] arthroscopic excision, curettage and BMS to be the first treatment of choice for primary osteochondral talar lesions. It is relatively inexpensive, there is low morbidity, a quick recovery and a high success rate. A

Conflict of interest:

The authors declared no conflicts of interest.

VOLUME 69 | ISSUE 2 | APRIL - JUNE 2018

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VOLUME 69 | ISSUE 2 | APRIL - JUNE 2018

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ΠΕΡΙΛΗΨΗ

Η οστεοχόνδρινη βλάβη του αστραγάλου αποτελεί μια ευρεία έννοια που περιγράφει τον τραυματισμό ή την ανωμαλία στην αρθρική επιφάνεια του αστραγάλου. Διάφοροι όροι έχουν χρησιμοποιηθεί αναφορικά με τη συγκεκριμένη κλινική οντότητα, όπως η διαχωριστική οστεοχονδρίτιδα, το οστεοχόνδρινο κάταγμα ή το οστεοχόνδρινο έλλειμμα. Αν η οστεοχόνδρινη βλάβη του αστραγάλου αποτελεί πρόδρομο ανάπτυξης γενικευμένης αρθρίτιδας ή όχι αποτελεί θέμα μελέτης, παραμένει βέβαιο όμως πως συνιστά μια κλινική οντότητα που συνήθως είναι συμπτωματική και χρήζει θεραπείας. Σε περισσότερο από το ένα τρίτο των περιπτώσεων η συντηρητική θεραπεία αποτυγχάνει και χρειάζεται χειρουργική παρέμβαση. Υφίσταται ένα ευρύ φάσμα χειρουργικών επεμβάσεων που αφορούν στην οστεοχόνδρινη βλάβη της ποδοκνημικής άρθρωσης, με αρκετές νέες τεχνικές να βρίσκονται στο προσκήνιο την τελευταία δεκαετία.

Η συνήθης συντηρητική αντιμετώπιση περιλαμβάνει ανάπαυση, ακινητοποίηση και χρήση μη στεροειδών αντιφλεγμονωδών φαρμάκων, ενώ η χειρουργική αντιμετώπιση περιλαμβάνει από την απλή εξαίρεση της βλάβης μέχρι πιο σύνθετες τεχνικές όπως η χρήση αυτόλογου μοσχεύματος από μη φορτιζόμενη επιφάνεια του αστραγάλου. Στόχος της θεραπείας παραμένει η ανακούφιση των συμπτωμάτων και η βελτίωση της λειτουργικότητας της άρθρωσης. Τα συμπεράσματα των μελετών ως προς την αποτελεσματικότητα των διαφόρων τεχνικών διαφέρουν. Στις περισσότερες των περιπτώσεων η απόφαση για την χειρουργική τεχνική που θα χρησιμοποιηθεί για την αντιμετώπιση της βλάβης εξαρτάται από τον τύπο και το μέγεθος της βλάβης, καθώς και από την χειρουργική τεχνική που θα προτιμήσει ο εκάστοτε χειρουργός.

ΛΕΞΕΙΣ ΚΛΕΙΔΙΑ: Οοστεοχόνδρινες Βλάβες, Διαχωριστική Οστεοχονδρίτιδα, Αστράγαλος, Ποδοκνημική Άρθρωση, Χόνδρινη Βλάβη, Υποχόνδριο Οστό