Management of Osteochondral Disorders of the Ankle

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1 Introduction

Osteochondral lesions of the talus (OLT) are common lesions. They can be found in isolation or in combination with other ankle pathology such as ankle instability [1]. The majority are thought to be post traumatic in nature in older patients [2], while in younger patients, these lesions may have a bone developmental origin [3]. They may also be vascular in nature and may have a genetic predisposition to occur. If they remain symptomatic and do not resolve with non-operative treatment, then surgery can be performed. However, many patients are younger compared to the average patient in a foot and ankle practice and have an expectation to return to sport. This occurs in 88% of surgical cases but sometimes at a reduced level [4].

Some patients may fail to get adequate function despite following the guidelines outlined by ICRA [5].

Patients therefore need to be aware of the potential for not being able to return to the sport of their choice or to the level that they wish to achieve.

Patients with malalignment that is overloading the osteochondral defect may require realignment surgery [5], and associated ankle instability needs to be both diagnosed and treated [6]. If both instability and malalignment are treated, then outcomes can be optimized [7]. The incidence of osteochondral lesions occurring in patients with ankle instability treated surgically was approximately 30% in one series [8].

The international consensus group on cartilage repair of the ankle meeting in Pittsburgh in 2017 has assisted the medical community in trying to standardize care with regards to investigation, non-operative treatment, operative intervention, and the use of cartilage or bone graft into the defect [9]. This has been very helpful in trying to codify the treatment of these challenging lesions.

The resources available to treat osteochondral ankle lesions in different centers may determine the treatment. Allograft may or may not be available. Bone graft substitute, cartilage products, and cell products may or may not be affordable.

Arthroscopic procedures are more likely to result in a shorter recovery and maintenance of range of motion [10]. As a result, primary procedures are often arthroscopic, and open procedures are reserved for revision procedures or large initial defects. Some of these procedures are maximally invasive requiring iliac crest or knee harvest sites and tibial osteotomies to gain vertical access to the defect site.

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2 Pathology

Failed debridement of OLT results in increased levels of inflammatory mediators in the synovial fluid [11]. Histological analysis of the cartilage in the osteochondral defects show changes suggestive of cartilage degeneration, compared to no degeneration in normal cartilage from allograft donors. Similarly there were raised levels of tumour necrosis factor (TNF) alpha, Interleukin (IL)-2, 6, 10, 13, Matrix Metalloproteinases (MMP) 1, 2, 3, 9 and 10. Medial lesions are more likely to occur than lateral lesions as reported in most papers, and were 60% of all lesions in one systematic review [12]. The location of lesions are mapped out using a grid system as outlined by Elias et al. for the talus [13] and tibia [14]. The grid consists of nine zones of the tibia and talus, with division into each plane into thirds. Zone 1 is anterior and medial, zone 3 anterior and lateral, zone 7 posterior and medial, and zone 9 posterior and lateral.

On the talus the lesions are most commonly on the medial central location [13], and these lesions are deeper and longer than the lateral lesions. The lesions located on the tibia more often medial, with posterior medial being slightly less common than the Centro medial location [14]. The inter and intra observer reliability of this system was reviewed and shown to be reproducible and reliable [15].

The formation of the cyst through a breakdown of the subchondral bone and the subchondral oedema remains debatable. Access of joint fluid may be a precipitating event [16].

Bruns, Maier and Fraissler in separate studies have shown that the OLT lesions are associated with vitamin D deficiency.

3 Terminology

The international cartilage society published their recommendations for terminology in discussing osteochondral lesions [17]. This assists surgeons in understanding the literature with regards to outcomes. They recommended using the term subchondral bone lesion (SBL) if only the bone is affected. This is a rare occasion.

An isolated cartilage lesion has no bone involvement. There was no acronym recommended for classifying this lesion.

A combined cartilage and bone lesion is an osteochondral lesion of the talus, abbreviated to OLT.

Bone marrow stimulation was considered the correct term to cover all procedures in which the defect was debrided, and the bone stimulated via an awl or drill.

Bulk grafts were termed autologous osteochondral transplant (AuOT) or allograft transplant (AlOT).

Subchondral oedema is an increase in fluid in the subchondral bone diagnosed by MRI. The size is also measured by MRI in three planes to calculate a volume of involvement.

Cysts are defined as a lesion under the subchondral bone and characterized by consistency (loculation), communication with the joint, depth, and size, walled or not, and location.

Oedema is classified by size, depth, location, total volume and whether there was communication with the joint.

Acute is defined as under 1 month, sub-acute 1–6 months, and chronic over 6 months since the onset of symptoms [17].

4 Clinical Assessment

Clinical assessment should focus on the key elements of disability. The international consensus group on cartilage repair of the ankle identified assessment of deficit in ADL's and sporting activities, duration of symptoms, history of trauma, mechanism of injury, localization of the pain, mechanical symptoms such as locking and instability, previous treatment and swelling as the core information to obtain in determining treatment options [18].

For physical examination, the physician should inspect for weight bearing alignment, ankle range of motion (compared to the normal side), stability of the lateral and medial collateral ligaments, swelling, and tenderness to palpation [18]. Location of tenderness can be determined by clinical examination, which studies have

shown correlates well with the radiotracer uptake on SPECT [19].

If a cartilage lesion is present, deep ankle pain aggravated by activity may be the hallmark symptoms [18].

5 Investigations

Standing AP, lateral and mortise views of the ankle are required for the initial investigation [18] (Fig. 1). Often the defect can be visualized, and the alignment of the ankle can be appreciated. Additional views including a calcaneal axial view, long leg alignment view, as well as AP and lateral views of the foot can be obtained to fully appreciate the associated weight-bearing pathology. Standing radiographs allow for the assessment of alignment and associated lesions such as osteophytes, cystic changes, degenerative change, and size of the OLT plus location. CT allows better estimation of cyst location and size, as well as osteophyte formation and bony impingement (Fig. 2), while MRI allows assessment of cartilage delamination,

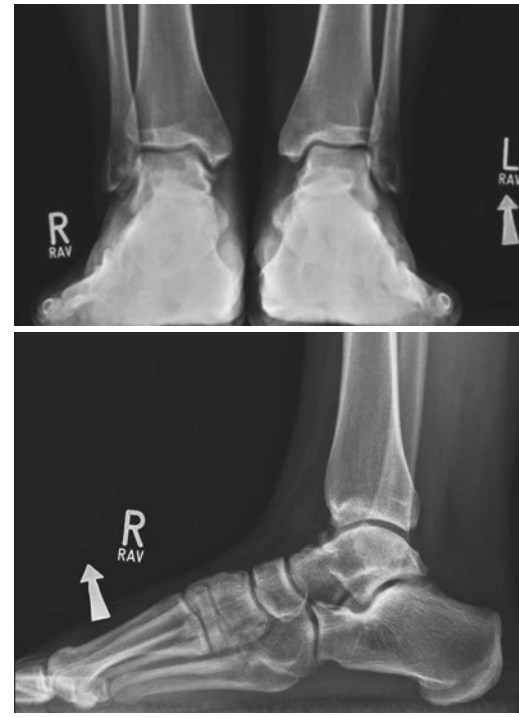

Fig. 1 Standing AP and lateral views showing an osteochondral injury. Long leg alignment views and a calcaneal axial view can also be of value

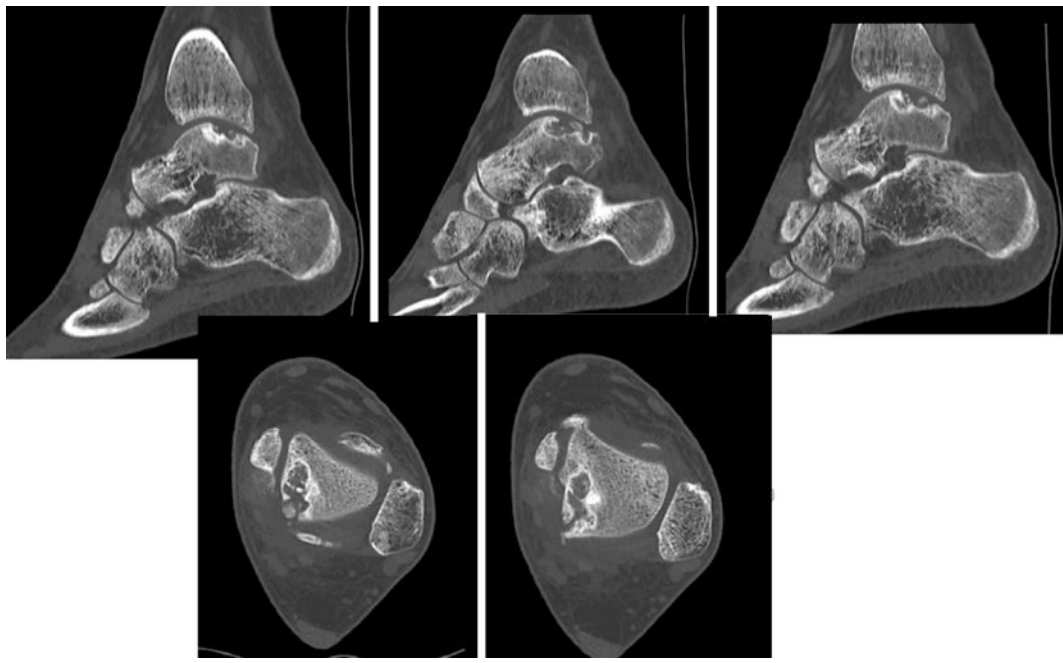

Fig. 2 Assessment of a large cystic defect on the medial talar dome using CT. This lesion was asymptomatic until the subchondral bone fractured (not the same patient as Fig. 1)

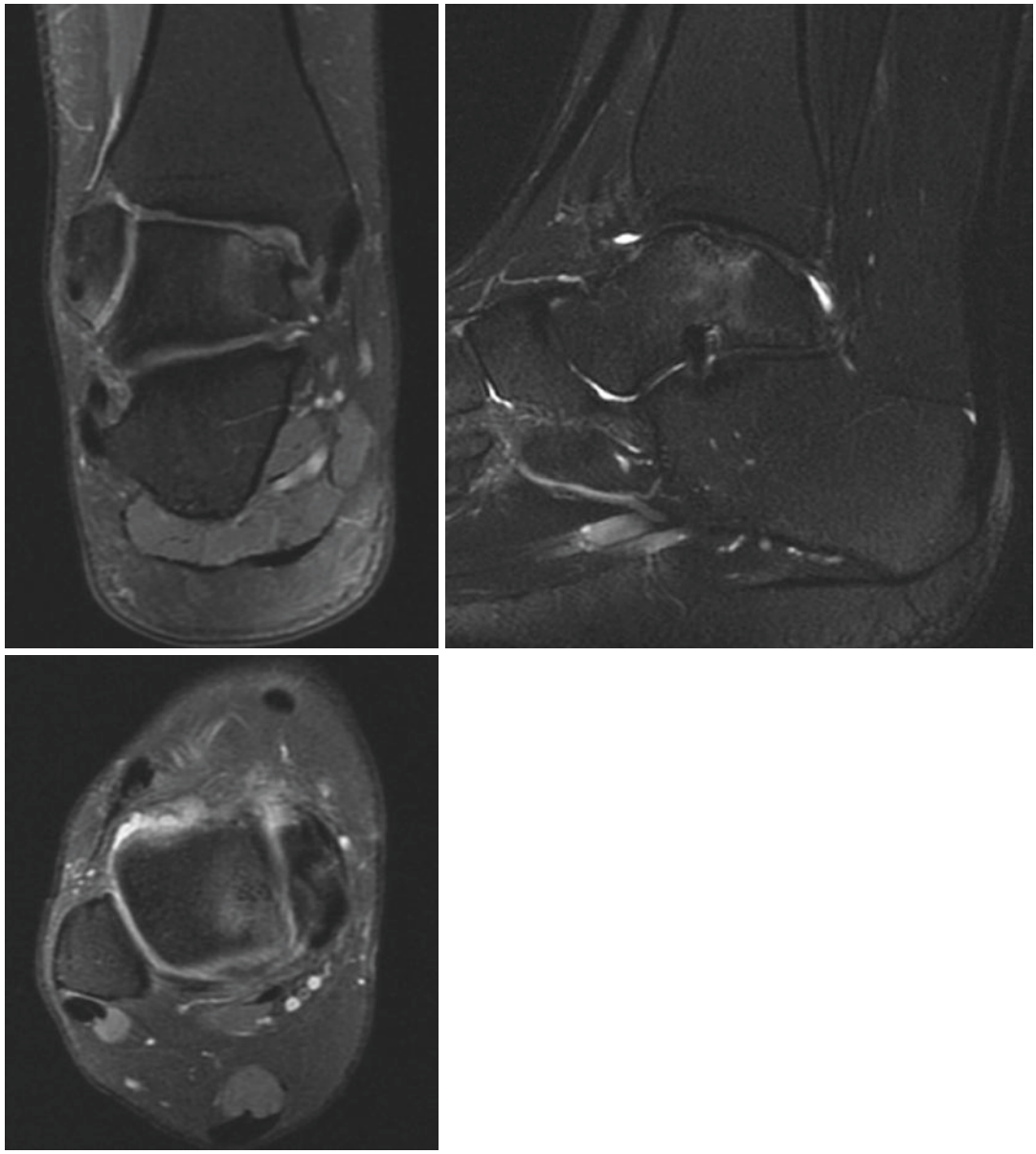

Fig. 3 MRI of the same lesion in Fig. 1

bone marrow oedema, and soft tissue impingement [18] (Fig. 3).

Assessment of sensitivity and specificity shows that plain X-ray is specific (0.91) but not sensitive (0.59) (i.e., it may fail to diagnose an osteochondral defect that is present). The CT scan is more sensitive (0.99) and specific (0.81) than radiographs for OLT identification but not as good as MRI which is reported to be sensitive and specific with a value of 0.96. [20]

As a result, three-dimensional imaging is usually required to understand the full size and location of the defect. MRI remains the principal tool for advanced investigation because it is more sensitive and specific and gives a better estimation on cartilage damage. The bone marrow oedema

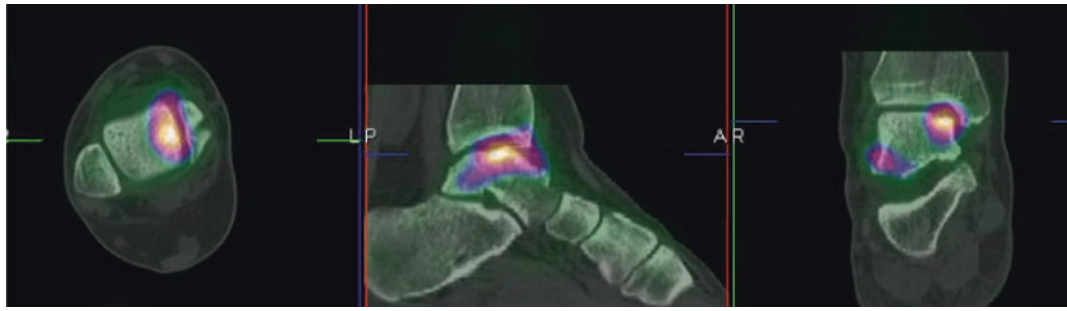

Fig. 4 SPECT of the same patient in Fig. 1 showing bone activity in the osteochondral lesion

indicates the extent of the soft cartilage. Finally, deficiency of the lateral and medial collateral ligaments, the presence of peroneal tendon pathology, and other cartilage lesions or impingement lesions can often be present and assessed via MRI.

The combination of bone scan and CT scan (SPECT) allows visualization of the defect with determination of bone scan uptake superimposed on the CT scan views. Activity of the lesion and other areas of radiotracer uptake such as anterior impingement can also be seen (Fig. 4).

6 Treatment

6.1 Conservative Treatment

Conservative treatment is recommended initially in all patients with asymptomatic lesions, such as incidental findings, non-displaced acute bone and cartilage injuries, older age with lower functional status, adjacent joint arthritis, or a skeletally immature patient [21].

The response to non-operative treatment is affected by patient age, body mass index (higher is worse), acuity of the lesion, size of the lesion (larger is worse), location of the lesion, presence of cystic change (cysts are worse), ankle instability, loose bodies, functional status, associated cartilage injury, medical comorbidities, and progression on imaging [21].

If the patient has an acute non-displaced OLT, ankle immobilization with touch down weight bearing is recommended for 6 weeks. Bone stimulators are unlikely to be beneficial [21].

The goal of non-operative treatment is for a full return to function with occasional pain. Low impact sports are recommended. An MRI should be obtained if there is no improvement in symptoms or radiographs by 3 months. Follow up should be every 6 months until resolution of symptoms. Deterioration may indicate the need for surgery. Concentrated bone marrow aspirate or platelet rich plasma (PRP) may be considered as an adjunct treatment, but there is little evidence to support the use of PRP in this setting [21].

Non-operative treatment can result in symptom resolution in some patients. Bracing, physiotherapy, NSAID medication are the initial treatments. Night splints may be helpful in patients with tight heel cords [21].

6.2 Operative Treatment

Operative treatment is directed at the bone defect and associated pathology.

6.3 Treatment of the Osteochondral Defect

The goal of treatment of the osteochondral defect is to try and get the bone and cartilage void to heal. Appropriate treatment of the primary lesion depends on the location, the residual cartilage condition, and size of the defect [22].

Treatment of the bone defect can be through marrow stimulation, bone graft substitute, autograft, or allograft (Fig. 5). The graft may be can-

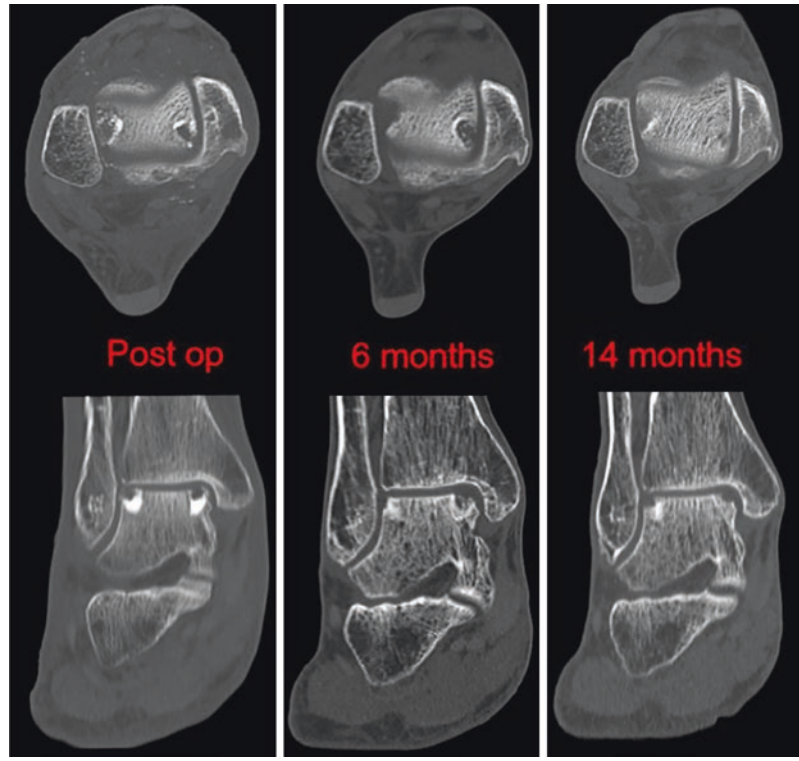

Fig. 5 Treatment of recurrent osteochondral defect on the medial and lateral side of the talus with PDGF and tricalcium sulphate. Gradual healing over time is seen

cellous or bulk such as an OATS. Grafting can be done through the defect by way of joint access or retrograde through the extra-articular bone.

Retrograde grafting can be performed for talar and tibial defects if the cartilage surface is intact. This is a rare case.

If bone is placed in a subchondral defect, it is helpful to seal it from the joint to prevent egress of bone growth factors that may negatively impact the cartilage or the synovium. Fibrin glue over the exposed bone can be used to achieve this seal.

7 Debridement, Curettage, and Bone Marrow Stimulation

Debridement of the osteochondral lesion is routinely performed arthroscopically. For lesions anterior to the 12 o'clock apex of the talus, the lesion can be approached anteriorly. A plantar flexion X-ray can assist in determining if the lesion can be reached [23]. Plantar flexion and occasional limited tibial osteotomy can allow the access to the lesion. Calf tourniquets used during surgery will result in compression of the calf muscles and can limit access to the talus by restricting muscle movement.

Anterior arthroscopy is performed using anterior medial and anterior lateral portals. Care is taken to avoid the superficial branch and deep branch of the peroneal nerve, using blunt dissection and superficial skin incisions.

The author also uses the posterior portals in the supine position as described by Acevedo [24]. This allows access posteriorly to posterior pathology as well as synovitis, loose bodies, or visualization in a tight ankle.

The portal is made just posterior to the medial malleolus and passing behind the tibialis posterior tendon. Because the neurovascular bundle is in this region the deep dissection is blunt, and any shaver should be visualized in the joint before being used. Alternatively, a posterior lateral portal can be made behind the peroneal tendons, a blunt obturator passed through the joint and a switching stick technique used.

Debridement of the osteochondral defect requires removal of all unstable cartilage, and removal of any loose bone. The bone in regions of bone marrow oedema is usually soft secondary to the biologic effects of synovial fluid and needs to be removed. After debridement, the defect should be free of loose cartilage and curetted back to solid bone [25].

Lesions amenable to debridement as a definitive procedure include partial thickness chondral lesions, acute lesions such as those found after ankle fractures, and lesions secondary other disease such as PVNS [22].

Size guidelines exist for the success of debridement. Based on the studies of Choi and Kennedy, smaller lesions do well with debridement, while larger lesions fail [26]. The threshold is debatable, but lesions classified as small should be under 10 mm in diameter, under 100 mm^2 in area, and under 5 mm in depth [27] (Fig. 6). Bone marrow stimulation alone is unlikely to work for lesions greater than 15 mm in diameter [27].

Other factors affecting the success of debridement include the presence of ankle instability, bone marrow oedema, joint malalignment in addition to lesion size, lesion location, cysts, revision procedures, and uncontained lesions [22].

After debridement, an awl or 2 mm drill can be used to penetrate the base of the lesion to allow egress of bone marrow cells to create a new bone base [22]. Releasing the tourniquet can confirm successful penetration by bone bleeding. Two to 3 penetrations should suffice.

The addition of biologics may assist in the healing of the bone and cartilage in the defect. Adjuncts include concentrated bone marrow aspirate, mesenchymal stem cells, platelet rich plasma, and hyaluronic acid [28].

For acute unstable lesions bone marrow stimulation is appropriate, and if the unstable lesion is saucer shaped and large enough that it can be preserved and reattached [29]. Fixation can be achieved using poly-L-lactide pins as described by Nakaska et al. [30, 31], who also showed that fixation was superior to excision and BMS for lesions under 100 mm^2. Alternatives include bone plugs or 2 mm screws. A lesion smaller than 10 mm length or 3 mm depth is better excised than transfixed [32].

Repeat bone marrow stimulation for failed debridement can be considered for cases where the debridement may be incomplete, or when the patient is unwilling to undergo a more invasive open procedure.

BMS has been reported to result in osteoarthritis in 33–34% of patients.

7.1 Post-operative Protocol

Historically non-weightbearing was advocated for 6 weeks. However more recently outcomes have been demonstrated to be similar with 2 weeks non-weightbearing. Range of motion is initiated at 2 weeks with avoidance of shear loading. Various protocols can be used. MRI can be

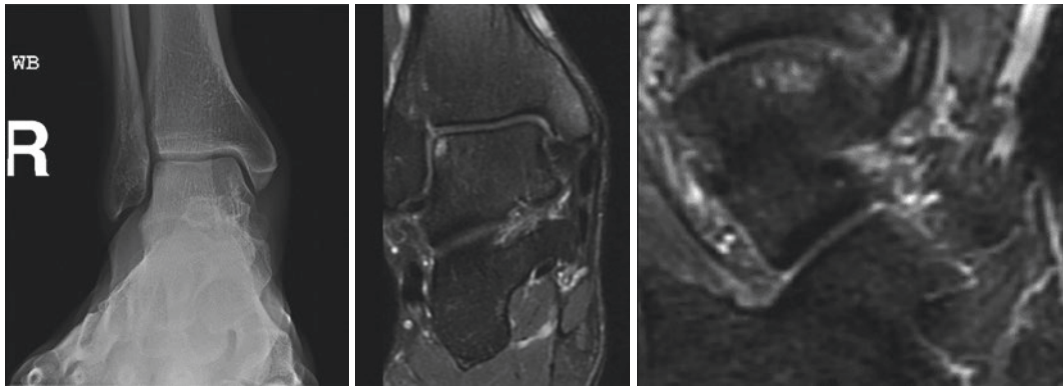

Fig. 6 A lesion of appropriate size and location for bone marrow stimulation (BMS)

used to determine if increased activity can be tolerated. The concomitant procedures can determine the recovery protocol such as lateral ligament reconstruction or calcaneal osteotomy.

After remobilization it is usual to contain shear by using a walker boot or brace.

8 The Cartilage Defect

The cartilage defect is managed by removal of all loose fragments and the filling the defect. In many cases the cartilage defect can be left unfilled, particularly in smaller lesions (Fig. 6). Typically, the defect becomes filled with hyaline cartilage which works well for small defects but may shear with larger defects (Figs. 7 and 8). Cartilage substitutes can be used of various descriptions and various costs; however they may not provide any better graft than marrow stimulation [33]. Cartilage graft can include extracellular cartilage matrix [34–36], juvenile cartilage [37, 38], scaffold [39] or amplified cartilage cells [40] in a single stage [41, 42] procedure, or a two stage procedure.

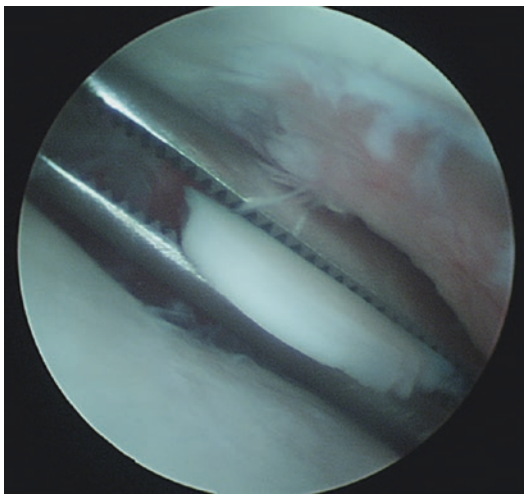

Fig. 7 The same lesion with the cartilage and subchondral bone defect removed

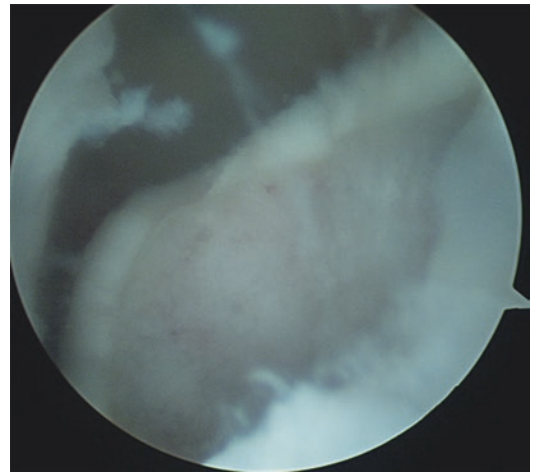

Fig. 8 The same lesion after debridement of loose cartilage and loose bone

9 Larger Defects: Bulk Grafts and Oats

Larger defects may require a bulk graft which can be a fresh frozen graft OATS, a packaged allograft Osteochondral Allograft Transfer (OATS), or an autograft OATS [43]. Mosaicplasty is a little different in technique as the grafts are smaller and defects exist in the cartilage surface [44].

9.1 Procedure

To perform an OATS procedure on the talus, a vertical approach should be obtained through the tibia to gain access to the cartilage surface. Depending on the location of the defect, this usually requires an osteotomy.

9.2 Medial Defects

Medial defects of the talus usually require a medial malleolar osteotomy. They are approached via a high oblique medial malleolar osteotomy [45, 46]. A longitudinal incision is made, and the

anterior and posterior joint line are outlined. This needs to be performed carefully as an incorrect osteotomy will compromise the approach. The usual error is to enter the joint too medially. To ensure a correct approach a k wire is placed, and its position confirmed on c arm. Predrilling the screw holes for fixation prior to the osteotomy is helpful to maintain articular joint surface congruity. Sometimes full plantar flexion and limited palatoplasty can allow access to the lesion [47].

Before starting the saw cut, retractors are placed anteriorly and posteriorly. The posterior retractor should be placed under the posterior tibial tendon to ensure it does not get cut.

Once performed the anterior and posterior capsule may have to be released to allow the talus to be pulled into valgus hinged on the deep deltoid ligament to improve the visualization. The lesion can then be sized. Because the lesion is usually over 10 mm in size one or two 10 mm grafts are required (Fig. 7).

The guide wire should be carefully placed over the defect. The wire has to be vertically oriented over the joint surface. The wire should end up pointing at the lateral corner of the lateral process of the talus, which is the central axis of rotation of the talus on the lateral view. On the AP view the wire should be perpendicular to the top surface of the talus.

Reaming is performed carefully to avoid heat and irrigation used. A depth of 15 mm should be achieved. The harvested graft should also be planned to match the talar defect as this will often have a shoulder area and may be still a little eccentric in position.

After harvesting the autograft or allograft, the end of the graft is trimmed to be bullet shaped and at the depth of the defect. If desired, platelet rich plasma (PRP) can be placed into the depth of the defect [48, 49].

The graft is carefully placed onto the defect and rotated to best match the defect. It should push in and be flush.

Nested grafts are two or more grafts that are used to fill a defect. They should overlap the adjacent graft by a quarter of the diameter. After one is placed, the second drill hole is prepared, and the second graft placed.

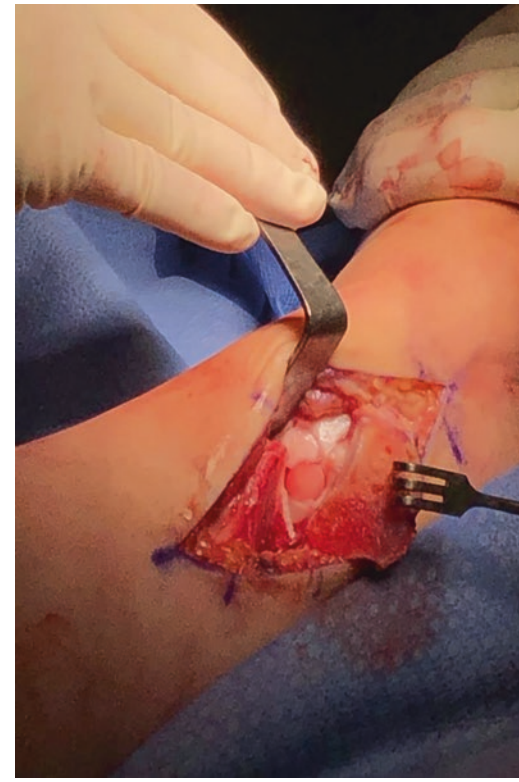

Fig. 9 A medial malleolar osteotomy and double 10 mm OATS autograft for a failed prior debridement of a larger posterior medial lesion

The tibial osteotomy site is reduced and held with screws and or a plate and screws in a stable position (Figs. 9 and 10).

9.3 Lateral Defect

The lateral defect can be approached by an extensile lateral approach. This is performed by sectioning the lateral collateral ligaments. Anterior translation of the talus will often allow vertical access to the defect. If this cannot be achieved after release of the capsule, then a lateral malleolar osteotomy will need to be performed (Fig. 11). This will need to be proximal enough to allow vertical access (Figs. 12, 13, and 14). A transverse osteotomy can be used with external rotation of the fibula, oblique osteotomy with external rotation, or distal rotation similar to the Zimmer ankle approach. Fixation is either with plates or a

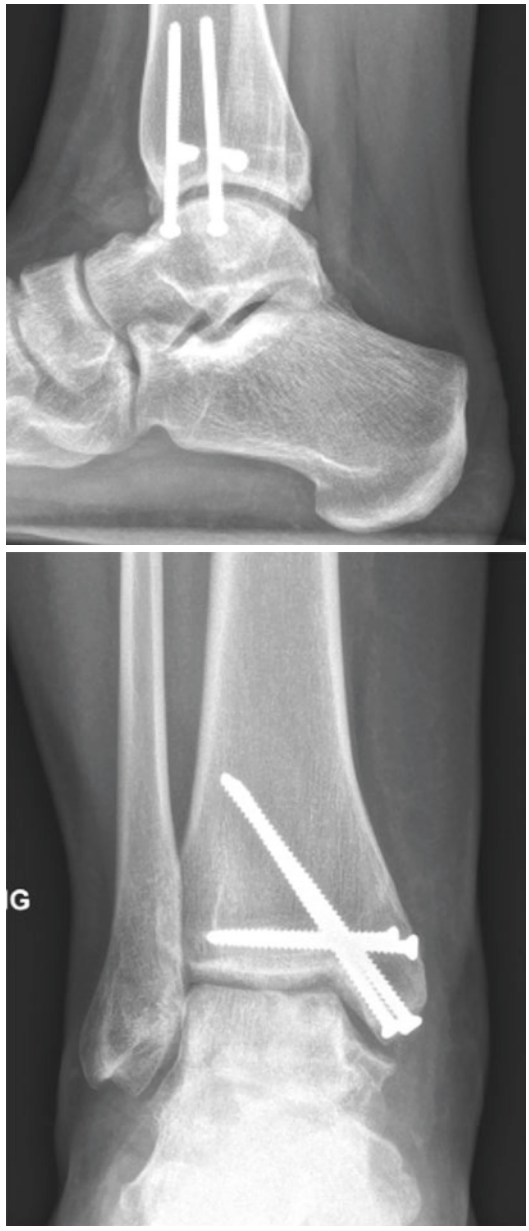

Fig. 10 The postoperative X-ray of the case in Fig. 9 showing screw configuration and osteotomy site

fibular nail. A Chaput osteotomy can also improve access and may allow vertical access to the defect.

The consensus meeting reviewed the role of osteochondral autografts. The recommended indications include cystic lesions more than 1 cm in diameter and revision of failed bone marrow stimulation procedures over 1 cm in size.

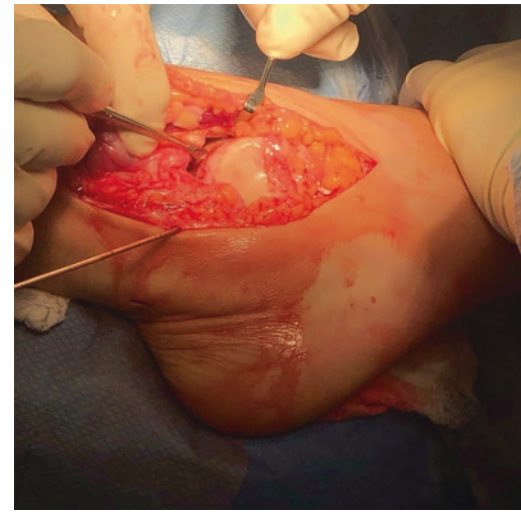

Fig. 11 A lateral malleolar osteotomy to expose a lateral osteochondral defect

Similar prognostic factors exist to bone marrow stimulation, such as size and location. Unconstrained lesions can be treated by OATS. The graft should be congruent and a depth of 12–15 mm used. Two or less grafts have a similar outcome, and three grafts or more may result in a poor outcome.

Cysts may occur after OATS and can be prevented by careful drilling, to avoid thermal injury to bone, a press fit construct, and the use of biologic. The relevance of the cysts are debated [50] (Fig. 15).

The preferred donor site for autograft is the lateral femoral condyle [51]. Donor site morbidity can be reduced by reducing damage to the cartilage, avoiding a tight lateral closure, reducing the soft tissue manipulation, and perform early mobilization of the donor site [52]. Ideally the defect should be backfilled by a plug.

9.4 Postoperative Protocol OATS Procedure

Because of the extensive incision and either the lateral ligament repair, or the medial malleolar osteotomy the ankle is splinted for 2 weeks postoperatively. The medial malleolar osteotomy should be stable when fixed, and therefore mobi-

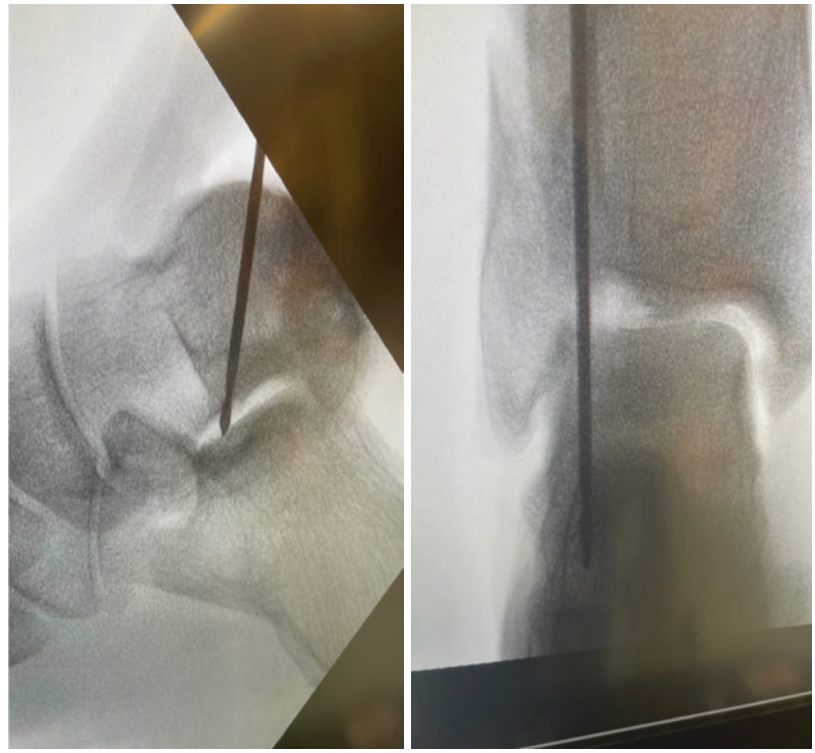

Fig. 12 The correct angle for the OATS drill. The guide wire should be vertical to the joint surface on all planes. On the lateral view the tip of the guide wire should point down to the tip of the lateral process of the talus

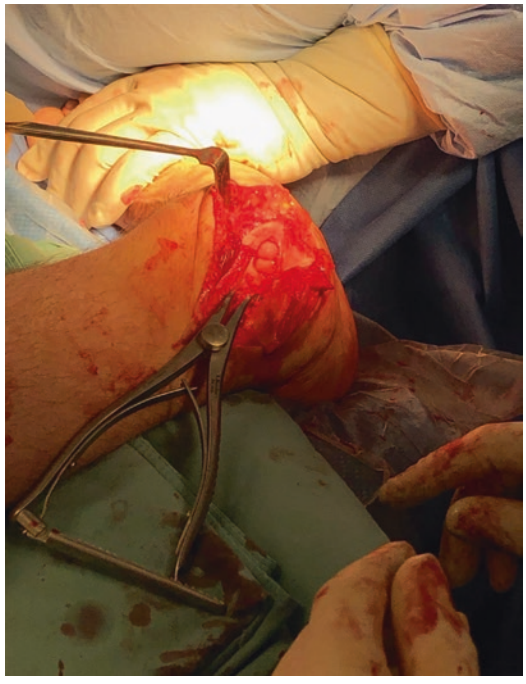

Fig. 13 The final grafts in place

lization can be faster. After suture removal and assuming wound healing, the ankle is mobilized with range of motion. Weight-bearing can be initiated depending on stability of the osteotomy at 2–6 weeks postoperative. Range of motion can be initiated using a stationary bicycle. At 12 weeks proprioception can be initiated.

9.5 Revision Treatment of the Osteochondral Defect

Revision of an osteochondral defect follows the principles of the primary lesion. A thoughtful history and physical is required to ensure that the cause of ongoing pain is correctly identified.

Factors considered important in choice of revision procedure include imaging appearance, mechanical factors such as stability, patient age, presence of other cartilage pathology, presence of a cyst, lesion progression, size of lesion, and type of initial procedure [32].

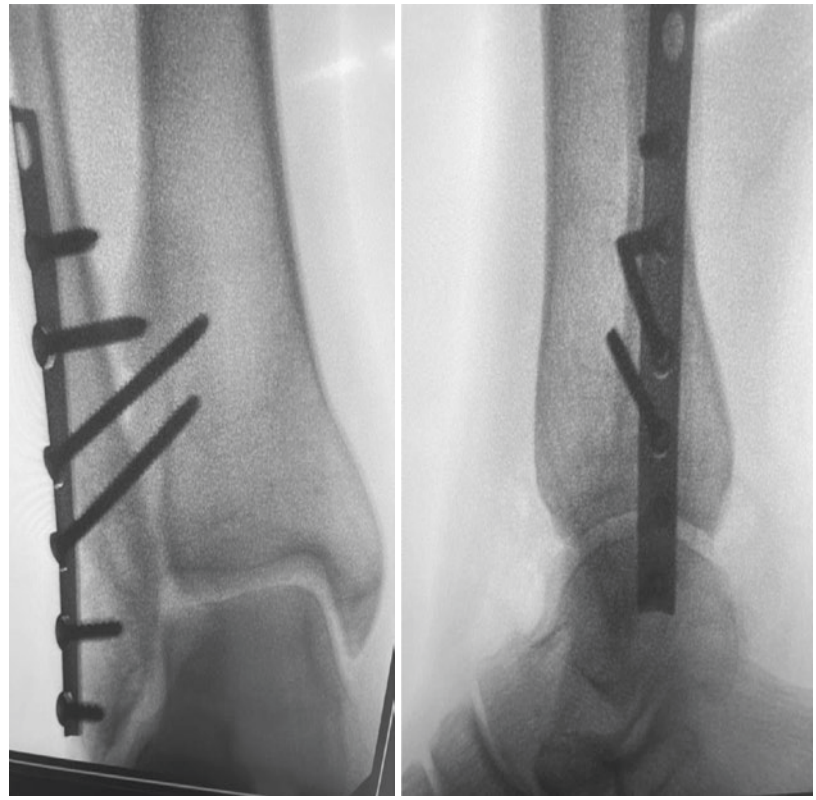

Fig. 14 Fixation of the fibular osteotomy

Revision of the cartilage procedure is contra indicated if the patient has infection, extensive degenerative disease, inflammatory arthropathy, severe stiffness, discrepancy between clinical symptoms and imaging findings, unrealistic expectations for the outcome of the revision procedure, and patient non-compliance [32].

If the primary procedure was a BMS procedure, then an OATS procedure may be the best salvage [53].

A systematic review of five papers showed a success rate for revision BMS at 61%, so revision BMS may not achieve a desirable result and should be considered carefully [54].

Revision may be more successful with an OATS or bulk graft. Park et al. compared primary OATS procedures against revisions and found similar survivorship and outcome scores. Larger lesions were the cause of failure in both groups (over 225 mm^2 on preoperative MRI) [55]. This is not the case of BMS [54]. Yoon et al. also outlined that OATS was a better treatment compared to BMS for failed debridement [53].

Ettinger and Maiorano have advocated a titanium hemicap as a revision device [56, 57].. However only ten lesions were treated in 7 patients in Ettinger's study and 12 patients in the other study. Ettinger observed high body mass index (BMI) as a risk factor for failure. This is unlike Koh et al. that found similar results if longer procedure times for patients over a BMI of 25 [58]. A BMI of 25 is a low threshold for many patient populations.

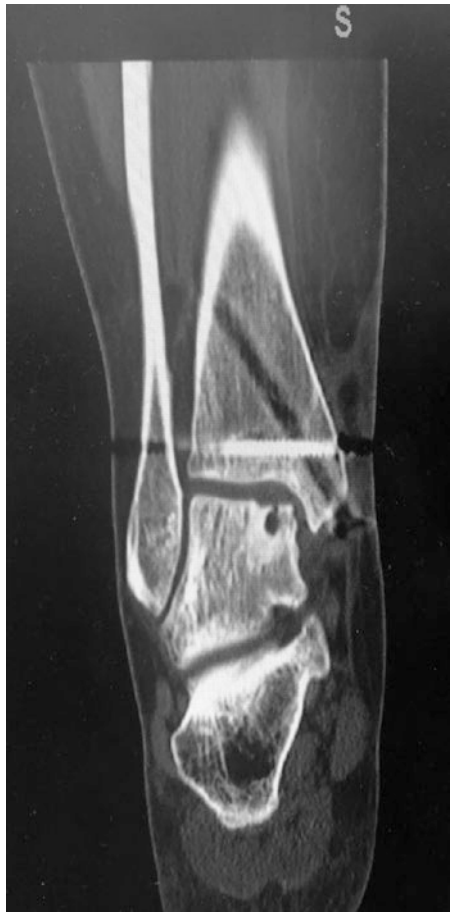

Fig. 15 Cyst defect in an OATS graft

10 Treatment of Bone Marrow Oedema Alone

Subchondroplasty, the injection of Calcium Phosphate into the bone, has been advocated as a treatment for symptomatic subchondral oedema if the overlying cartilage is intact. Concern has been raised about the risk of AVN of the talus from the injection due to over pressurization at the time of injection so caution should be used, and currently the recommendation is to use a low volume of graft (1.5 cc's) [59].

11 Treatment of Lesions of the Tibia

Tibial lesions have a slightly different pathology: The defect is often smaller and the marrow signal greater. The treatment is also challenging as accessing the lesion through the joint is technically challenging. The defect is therefore best addressed retrograde.

Smaller tibial lesions can be treated with debridement and BMS as described by Ferkel and the German registry [60, 61].

Our preferred treatment is to use a tibial targeting device from the knee or biotenodesis set. The lesion is targeted and a k wire from the biotenodesis screw set used (Figs. 16 and 17). The appropriately sized reamer is then used to ream up to but not through the subchondral bone (Fig. 18). The bone is then reamed to a size to allow bone healing. Typically, a 6–10 mm reamer is used. Care is taken not to create heat during the reaming, and so irrigation or reaming with the tourniquet down will achieve this.

Cancellous autograft or allograft is then packed into the defect (Figs. 19 and 20), and platelet derived growth factor at the discretion of the surgeon can be added to stimulate bone healing.

An alternative is to place an OATS graft. However, matching the graft to the defect is difficult to do, and a vertical approach to the joint line is difficult to achieve. An osteotomy can be used to access the tibia to perform the graft [62].

The German registry had a total of 15 cases with over 1 year follow up in a registry of 844 OLT's with a total of 47 being tibial lesions (the majority not having over 1 year follow up). This amounts to 6% of all ankle osteochondral lesions. The majority were treated with BMS [60].

Ferkel reported on arthroscopic treatment of tibial lesions in 17 patients with 14 of 17 doing well with arthroscopic debridement. If there was

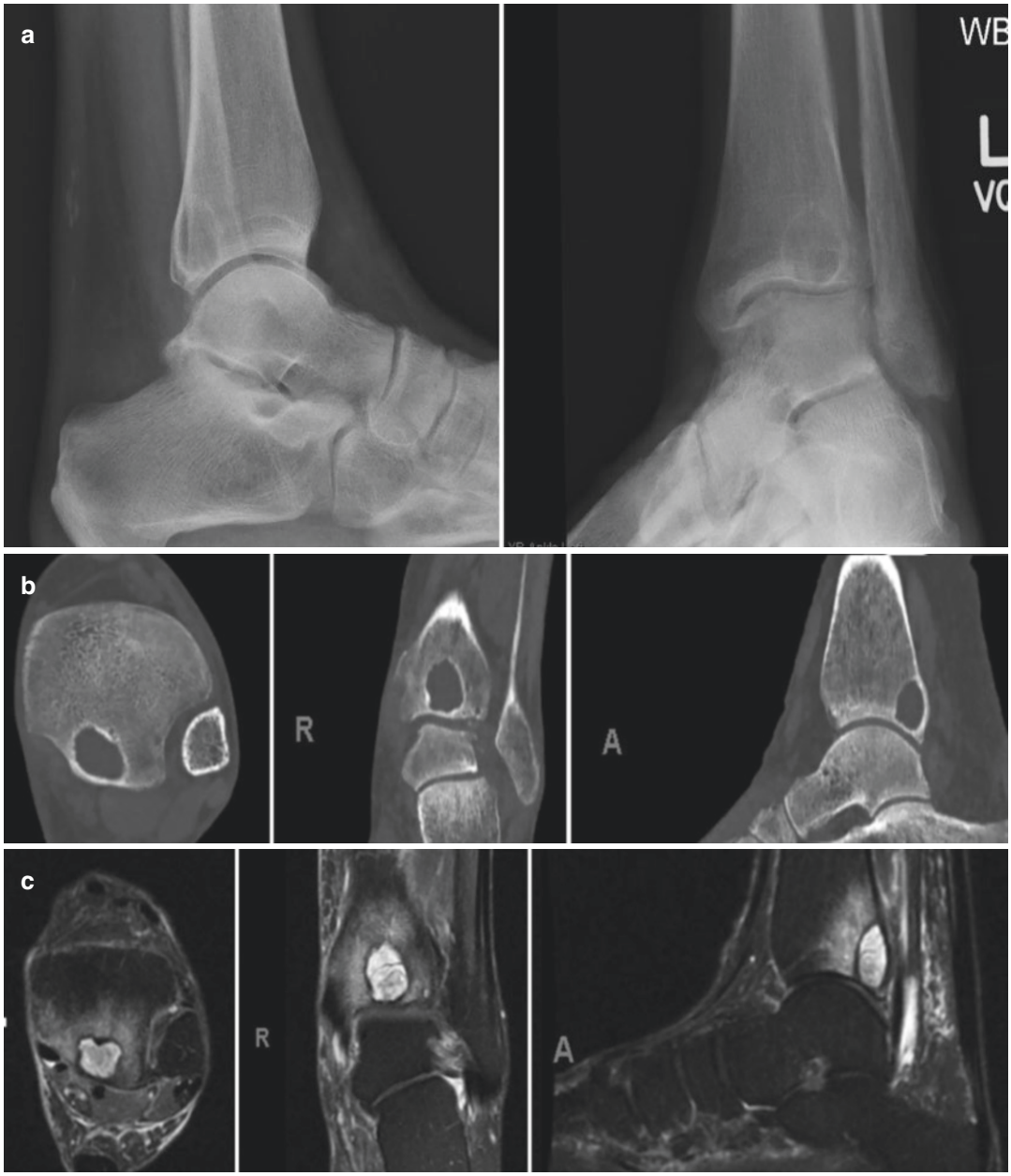

Fig. 16 A large posterior medial cystic defect in the tibia. (**a**) Plain X-rays (**b**) ct appearance (**c**) MRI preop

a cystic component or bone defect, iliac crest graft was added to the defect [61].

Another alternative has been to use injected bone graft made from Calcium phosphate in a subchondroplasty procedure. Some concern has been raised about the risk of AVN. This may however represent a radiographic artefact. As a result, a small amount of graft may be placed and can be of use in isolated bone marrow oedema.

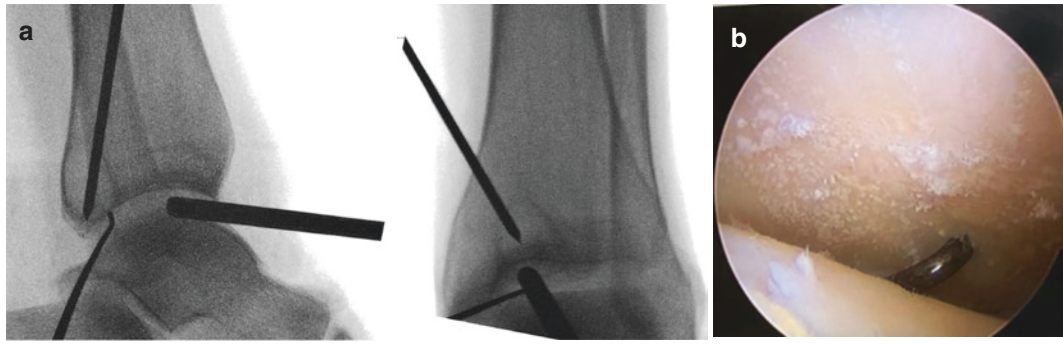

Fig. 17 (**a**) Targeting the lesion using an anterior medial, posterior medial portal, and incision over the distal tibia. The reamer for the biotenodesis screw set was used to access the defect and debridement of the cyst wall was performed arthroscopically. (**b**) Intra-articular view

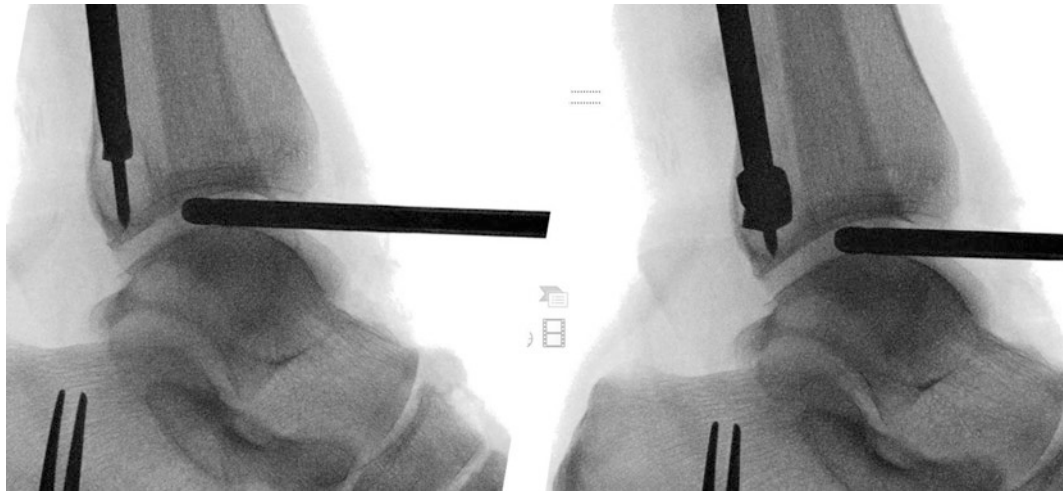

Fig. 18 Reaming to the cyst and staying outside the subchondral bone

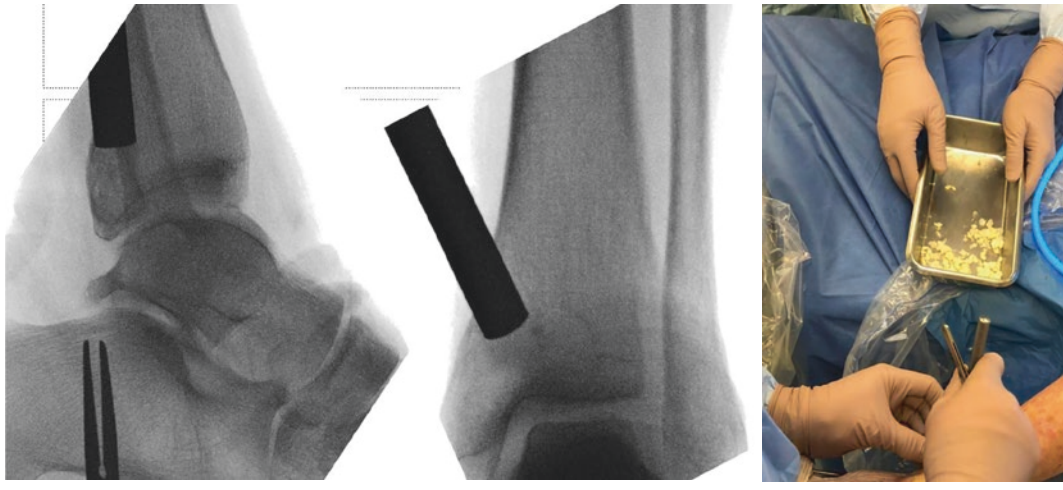

Fig. 19 Grafting the defect with allograft with a 10 mm tamp

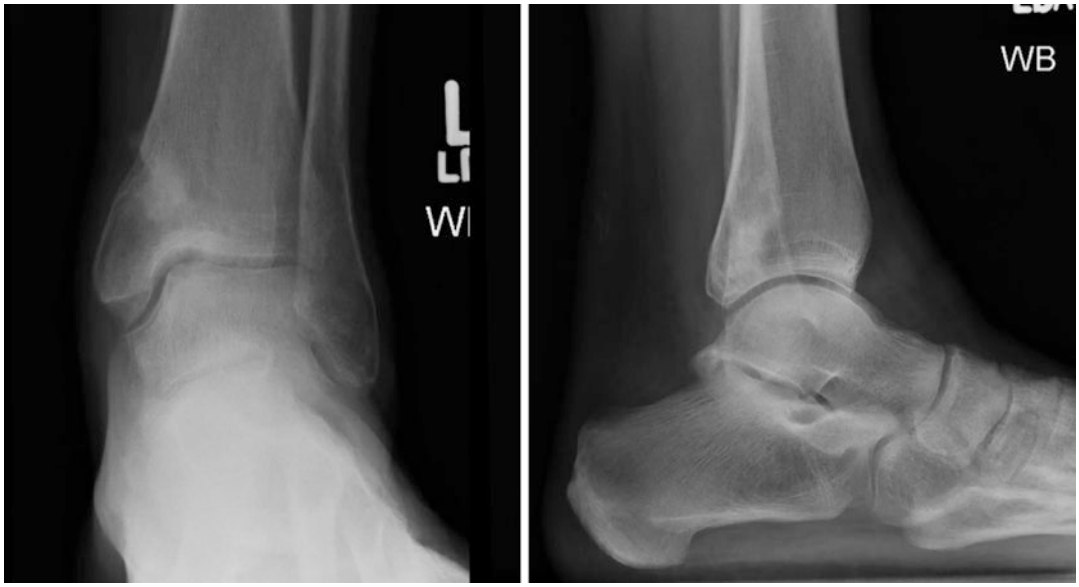

Fig. 20 Postoperative X-rays at 6 months. The patient is symptom free and has unrestricted activity

11.1 Postoperative Recovery After Subchondral Graft Procedures

If bone graft is used, remobilization can be achieved with weight bearing at 2 weeks. If there is concern about shear on a more unstable grafted OCD, delay may be considered.

Return to sport should be delayed until there is clear healing on X-ray and if required CT and MRI. Impact activity should be avoided if there is a residual bone marrow oedema signal.

11.2 Salvage: Fusion or Replacement

In some cases, the treatment of the osteochondral defect is unsuccessful despite a number of strategies and surgeries. In these cases, it may be better to consider fusion of the ankle joint to ensure return of weight bearing function for the patient.

The Consensus meeting recommended fusion or replacement if the failed cartilage procedure cannot be reasonably addressed by a revision procedure, or if there is progressive arthritis in the joint [32].

11.3 Outcomes

In a systematic review by Zengerink et al. in KSST in 2010, they quoted a success rate of 84% for transplantation procedures, 82% if the fragment could be transfixed, 76% for BMS and MFX, and 71% for debridement alone.

However, may factors can change the outcome including lesion size, patient weight, gender, location, vitamin d deficiency [63–65].

Outcomes are hard to interpret because of the scientific quality of the papers. Few are high quality, and the enrolment criteria, surgical procedures, documentation of demographics, outcomes used are all variable [66].

Toal et al. did a systematic review of BMS in 2019 and demonstrated reasonable short-term results [67]. Imaging findings continued to be present potentially indicating deterioration in time.

For OATs, a systematic review showed good to excellent results in 87.4%, 3.6% had donor site morbidity [68] [68]. Mosaicplasty is different than OATS so results should be considered separately.

Return to sport remains a critical outcome measure [69]. With microfracture all ten national

basketball players returned to play [69]. In a systematic review 86% of patients returned to play at an average of 4.5 months [70]. However lesion size is critical in this reporting [71].

11.4 Associated Pathology

Osteochondral defects are associated with a number of pathologies. These include lateral ankle instability, hindfoot varus [72], tibial varus [73], anterior medial osteophytes, and peroneal tendinopathy.

Some may require treatment for the success of the osteochondral defect management. In the case of ligamentous instability in the presence of an osteochondral defect, the surgeon may consider performing a lateral ligament reconstruction at the same time of the OLT treatment [74]. This will help treat the osteochondral defect as it prevents shear of the joint surface. It is recommended by the author that any ankle instability present should be managed at the same time as the OLT.

The surgeon may also consider treatment of the hindfoot varus or tibial varus at the same time. This may involve a tibial osteotomy, a calcaneal osteotomy or a combination of both.

Anterior medial impingement and synovitis should also be treated at the time of arthroscopy.

Arthroscopy also allows the assessment of the remaining cartilage. This may assist in future management should the joint remain symptomatic.

11.5 Authors Preferred Technique/ Algorithm

Based on the current research and information the author of this chapter prefers to perform an arthroscopic debridement if there is any uncertainty about the pathology before performing an open or osteotomy procedure. If the lesion is small, no bone graft or cartilage substitute is used. If the lesion is over 5 mm deep or 10 mm long bone grafting using cancellous graft ± platelet derived growth factor is used. This is sealed with fibrin glue, and lyophilized cartilage placed on top.

If the lesion is larger, cystic, or is a revision of a known well debrided and well visualized defect then an OATS autograft is performed.

All associated pathology is treated at the time of the primary procedure with a low threshold to perform a lateral ligament reconstruction, a calcaneal osteotomy (90% of the time lateralizing and done percutaneously), or a tibial osteotomy if needed.

If the OATS procedure fails then I will perform a second look arthroscopy and if there is more diffuse cartilage damage offer an ankle fusion in younger patients or a replacement in older patients.

Bibliography

1. Kerkhoffs GM, Kennedy JG, Calder JD, Karlsson J. There is no simple lateral ankle sprain. Knee Surg Sports Traumatol Arthrosc. 2016;24(4):941–3.
2. Hintermann B, Regazzoni P, Lampert C, Stutz G, Gachter A. Arthroscopic findings in acute fractures of the ankle. J Bone Joint Surg Br. 2000;82(3):345–51.
3. Hannon CP, Smyth NA, Murawski CD, et al. Osteochondral lesions of the talus: aspects of current management. Bone Joint J. 2014;96-B(2):164–71.
4. Steman JAH, Dahmen J, Lambers KTA, Kerkhoffs G. Return to sports after surgical treatment of osteochondral defects of the talus: a systematic review of 2347 cases. Orthop J Sports Med. 2019;7(10):2325967119876238.
5. Wiewiorski M, Werner L, Paul J, Anderson AE, Barg A, Valderrabano V. Sports activity after reconstruction of osteochondral lesions of the talus with autologous spongiosa grafts and autologous matrix-induced chondrogenesis. Am J Sports Med. 2016;44(10):2651–8.
6. Korner D, Ateschrang A, Schroter S, et al. Concomitant ankle instability has a negative impact on the quality of life in patients with osteochondral lesions of the talus: data from the German Cartilage Registry (KnorpelRegister DGOU). Knee Surg Sports Traumatol Arthrosc. 2020;28(10):3339–46.
7. Ahrend MD, Aurich M, Becher C, et al. Preexisting and treated concomitant ankle instability does not compromise patient-reported outcomes of solitary osteochondral lesions of the talus treated with matrix-induced bone marrow stimulation in the first postoperative year: data from the German Cartilage Registry (KnorpelRegister DGOU). Knee Surg Sports Traumatol Arthrosc. 2022;30(4):1187–96.
8. Hadeed MM, Dempsey IJ, Tyrrell Burrus M, et al. Predictors of osteochondral lesions of the talus in

patients undergoing Brostrom-Gould ankle ligament reconstruction. J Foot Ankle Surg. 2020;59(1):21–6.
9. Murawski CD, Hogan MV, Thordarson DB, Stone JW, Ferkel RD, Kennedy JG. Editorial. Foot Ankle Int. 2018;39(1_Suppl):1S–2S.
10. Medda S, Al'Khafaji IM, Scott AT. Ankle arthroscopy with microfracture for osteochondral defects of the talus. Arthrosc Tech. 2017;6(1):e167–74.
11. Danilkowicz RM, Allen NB, Grimm N, et al. Histological and inflammatory cytokine analysis of osteochondral lesions of the talus after failed microfracture: comparison with fresh allograft controls. Orthop J Sports Med. 2021;9(10):23259671211040535.
12. van Diepen PR, Dahmen J, Altink JN, Stufkens SAS, Kerkhoffs G. Location distribution of 2,087 osteochondral lesions of the talus. Cartilage. 2021;13(1_Suppl):1344S–53S.
13. Elias I, Zoga AC, Morrison WB, Besser MP, Schweitzer ME, Raikin SM. Osteochondral lesions of the talus: localization and morphologic data from 424 patients using a novel anatomical grid scheme. Foot Ankle Int. 2007;28(2):154–61.
14. Elias I, Raikin SM, Schweitzer ME, Besser MP, Morrison WB, Zoga AC. Osteochondral lesions of the distal tibial plafond: localization and morphologic characteristics with an anatomical grid. Foot Ankle Int. 2009;30(6):524–9.
15. Martin KD, McBride TJ, Horan DP, et al. Validation of 9-grid scheme for localizing osteochondral lesions of the talus. Foot Ankle Orthop. 2020;5(3):2473011420944925.
16. Shimozono Y, Brown AJ, Batista JP, et al. Subchondral pathology: proceedings of the international consensus meeting on cartilage repair of the ankle. Foot Ankle Int. 2018;39(1_Suppl):48S–53S.
17. Murawski CD, Jamal MS, Hurley ET, et al. Terminology for osteochondral lesions of the ankle: proceedings of the international consensus meeting on cartilage repair of the ankle. J ISAKOS. 2022;7(2):62–6.
18. van Bergen CJA, Baur OL, Murawski CD, et al. Diagnosis: history, physical examination, imaging, and arthroscopy: proceedings of the international consensus meeting on cartilage repair of the ankle. Foot Ankle Int. 2018;39(1_Suppl):3S–8S.
19. Agrawal K, Swaroop S, Patro PSS, Tripathy SK, Naik S, Velagada S. Comparison of bone SPECT/CT and MRI in detection of pain generator in ankle and foot pain: a retrospective diagnostic study. Nucl Med Commun. 2021;42(10):1085–96.
20. van Bergen CJ, Gerards RM, Opdam KT, Terra MP, Kerkhoffs GM. Diagnosing, planning and evaluating osteochondral ankle defects with imaging modalities. World J Orthop. 2015;6(11):944–53.
21. Dombrowski ME, Yasui Y, Murawski CD, et al. Conservative management and biological treatment strategies: proceedings of the international consensus meeting on cartilage repair of the ankle. Foot Ankle Int. 2018;39(1_Suppl):9S–15S.
22. Hannon CP, Bayer S, Murawski CD, et al. Debridement, curettage, and bone marrow stimulation: proceedings of the international consensus meeting on cartilage repair of the ankle. Foot Ankle Int. 2018;39(1_Suppl):16S–22S.
23. Brohard J, Chin KM. Maximum plantarflexion lateral ankle radiograph for preoperative planning for the arthroscopic treatment of osteochondral lesions of the talus. Foot Ankle Orthop. 2022;7(2):24730114221102453.
24. Acevedo JI, Busch MT, Ganey TM, Hutton WC, Ogden JA. Coaxial portals for posterior ankle arthroscopy: an anatomic study with clinical correlation on 29 patients. Arthroscopy. 2000;16(8):836–42.
25. Murawski CD, Foo LF, Kennedy JG. A review of arthroscopic bone marrow stimulation techniques of the talus: the good, the bad, and the causes for concern. Cartilage. 2010;1(2):137–44.
26. Hunt KJ, Lee AT, Lindsey DP, Slikker W 3rd, Chou LB. Osteochondral lesions of the talus: effect of defect size and plantarflexion angle on ankle joint stresses. Am J Sports Med. 2012;40(4):895–901.
27. Ramponi L, Yasui Y, Murawski CD, et al. Lesion size is a predictor of clinical outcomes after bone marrow stimulation for osteochondral lesions of the talus: a systematic review. Am J Sports Med. 2017;45(7):1698–705.
28. Hwang YG, Lee JW, Park KH, Hsienhao C, Han SH. Intra-articular injections of hyaluronic acid on osteochondral lesions of the talus after failed arthroscopic bone marrow stimulation. Foot Ankle Int. 2020;41(11):1376–82.
29. Reilingh ML, Murawski CD, DiGiovanni CW, et al. Fixation techniques: proceedings of the international consensus meeting on cartilage repair of the ankle. Foot Ankle Int. 2018;39(1_Suppl):23S–7S.
30. Nakasa T, Ikuta Y, Ota Y, Kanemitsu M, Adachi N. Clinical results of bioabsorbable pin fixation relative to the bone condition for osteochondral lesion of the talus. Foot Ankle Int. 2019;40(12):1388–96.
31. Nakasa T, Ikuta Y, Sumii J, Nekomoto A, Kawabata S, Adachi N. Clinical outcomes of osteochondral fragment fixation versus microfracture even for small osteochondral lesions of the talus. Am J Sports Med. 2022;11:3019.
32. Mittwede PN, Murawski CD, Ackermann J, et al. Revision and salvage management: proceedings of the international consensus meeting on cartilage repair of the ankle. Foot Ankle Int. 2018;39(1_Suppl):54S–60S.
33. Ettinger S, Gottschalk O, Kostretzis L, et al. One-year follow-up data from the German Cartilage Registry (KnorpelRegister DGOU) in the treatment of chondral and osteochondral defects of the talus. Arch Orthop Trauma Surg. 2022;142(2):205–10.
34. Shieh AK, Singh SG, Nathe C, et al. Effects of micronized cartilage matrix on cartilage repair

in osteochondral lesions of the talus. Cartilage. 2020;11(3):316–22.
35. Hansen OB, Eble SK, Patel K, et al. Comparison of clinical and radiographic outcomes following arthroscopic debridement with extracellular matrix augmentation and osteochondral autograft transplantation for medium-size osteochondral lesions of the talus. Foot Ankle Int. 2021;42(6):689–98.
36. Ahmad J, Maltenfort M. Arthroscopic treatment of osteochondral lesions of the talus with allograft cartilage matrix. Foot Ankle Int. 2017;38(8):855–62.
37. Manzi J, Arzani A, Hamula MJ, Manchanda K, Dhanaraj D, Chapman CB. Long-term patient-reported outcome measures following particulated juvenile allograft cartilage implantation for treatment of difficult osteochondral lesions of the talus. Foot Ankle Int. 2021;42(11):1399–409.
38. Giza E, Delman C, Coetzee JC, Schon LC. Arthroscopic treatment of talus osteochondral lesions with particulated juvenile allograft cartilage. Foot Ankle Int. 2014;35(10):1087–94.
39. Albano D, Martinelli N, Bianchi A, Messina C, Malerba F, Sconfienza LM. Clinical and imaging outcome of osteochondral lesions of the talus treated using autologous matrix-induced chondrogenesis technique with a biomimetic scaffold. BMC Musculoskelet Disord. 2017;18(1):306.
40. Buda R, Vannini F, Castagnini F, et al. Regenerative treatment in osteochondral lesions of the talus: autologous chondrocyte implantation versus one-step bone marrow derived cells transplantation. Int Orthop. 2015;39(5):893–900.
41. Bai L, Guan S, Liu S, et al. Clinical outcomes of osteochondral lesions of the talus with large subchondral cysts treated with osteotomy and autologous chondral grafts: minimum 2-year follow-up and second-look evaluation. Orthop J Sports Med. 2020;8(7):2325967120937798.
42. Lee KT, Kim JS, Young KW, et al. The use of fibrin matrix-mixed gel-type autologous chondrocyte implantation in the treatment for osteochondral lesions of the talus. Knee Surg Sports Traumatol Arthrosc. 2013;21(6):1251–60.
43. Hannon CP, Baksh N, Newman H, Murawski CD, Smyth NA, Kennedy JG. A systematic review on the reporting of outcome data in studies on autologous osteochondral transplantation for the treatment of osteochondral lesions of the talus. Foot Ankle Spec. 2013;6(3):226–31.
44. 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):556–60.
45. Hu Y, Yue C, Li X, et al. A novel medial malleolar osteotomy technique for the treatment of osteochondral lesions of the talus. Orthop J Sports Med. 2021;9(3):2325967121989988.
46. Padiolleau G, Amouyel T, Barbier O, et al. Safety of malleolar osteotomies in surgery for osteochondral lesions of the talus. Orthop Traumatol Surg Res. 2021;107(8S):103070.
47. Murawski CD, Kennedy JG. Operative treatment of osteochondral lesions of the talus. J Bone Joint Surg Am. 2013;95(11):1045–54.
48. Yasui Y, Ross AW, Kennedy JG. Platelet-rich plasma and concentrated bone marrow aspirate in surgical treatment for osteochondral lesions of the talus. Foot Ankle Clin. 2016;21(4):869–84.
49. Shimozono Y, Yasui Y, Hurley ET, Paugh RA, Deyer TW, Kennedy JG. Concentrated bone marrow aspirate may decrease postoperative cyst occurrence rate in autologous osteochondral transplantation for osteochondral lesions of the talus. Arthroscopy. 2019;35(1):99–105.
50. Savage-Elliott I, Smyth NA, Deyer TW, et al. Magnetic resonance imaging evidence of postoperative cyst formation does not appear to affect clinical outcomes after autologous osteochondral transplantation of the talus. Arthroscopy. 2016;32(9):1846–54.
51. Hurley ET, Stewart SK, Kennedy JG, Strauss EJ, Calder J, Ramasamy A. Current management strategies for osteochondral lesions of the talus. Bone Joint J. 2021;103-B(2):207–12.
52. Shimozono Y, Seow D, Yasui Y, Fields K, Kennedy JG. Knee-to-talus donor-site morbidity following autologous osteochondral transplantation: a meta-analysis with best-case and worst-case analysis. Clin Orthop Relat Res. 2019;477(8):1915–31.
53. Yoon HS, Park YJ, Lee M, Choi WJ, Lee JW. Osteochondral autologous transplantation is superior to repeat arthroscopy for the treatment of osteochondral lesions of the talus after failed primary arthroscopic treatment. Am J Sports Med. 2014;42(8):1896–903.
54. Dahmen J, Hurley ET, Shimozono Y, et al. Evidence-based treatment of failed primary osteochondral lesions of the talus: a systematic review on clinical outcomes of bone marrow stimulation. Cartilage. 2021;13(1_Suppl):1411S–21S.
55. Park KH, Hwang Y, Han SH, et al. Primary versus secondary osteochondral autograft transplantation for the treatment of large osteochondral lesions of the talus. Am J Sports Med. 2018;46(6):1389–96.
56. Ettinger S, Stukenborg-Colsman C, Waizy H, et al. Results of HemiCAP((R)) implantation as a salvage procedure for osteochondral lesions of the talus. J Foot Ankle Surg. 2017;56(4):788–92.
57. Maiorano E, Bianchi A, Hosseinzadeh MK, Malerba F, Martinelli N, Sansone V. HemiCAP(R) implantation after failed previous surgery for osteochondral lesions of the talus. Foot Ankle Surg. 2021;27(1):77–81.
58. Koh DTS, Tan MWP, Zhan X, et al. Association of elevated body mass index and outcomes of arthroscopic treatment for osteochondral lesions of the talus. Foot Ankle Orthop. 2022;7(2):24730114221103263.
59. Hanselman AE, Cody EA, Easley ME, Adams SB, Parekh SG. Avascular necrosis of the talus after subchondroplasty. Foot Ankle Int. 2021;42(9):1138–43.

60. Gottschalk O, Korner D, Aurich M, et al. Descriptive analysis and short-term follow-up clinical results of osteochondral lesions of the distal tibia based on data of the German Cartilage Register (Knorpelregister((R)) DGOU). Arch Orthop Trauma Surg. 2021;143:809.
61. Mologne TS, Ferkel RD. Arthroscopic treatment of osteochondral lesions of the distal tibia. Foot Ankle Int. 2007;28(8):865–72.
62. Chen W, Tang K, Yuan C, Zhou Y, Tao X. Intermediate results of large cystic medial osteochondral lesions of the talus treated with Osteoperiosteal cylinder autografts from the medial tibia. Arthroscopy. 2015;31(8):1557–64.
63. Malhotra K, Baggott PJ, Livingstone J. Vitamin D in the foot and ankle: a review of the literature. J Am Podiatr Med Assoc. 2020;110(3):Article_10.
64. Oberti V, Sanchez Ortiz M, Allende V, Masquijo J. Prevalence of hypovitaminosis D in patients with juvenile osteochondritis dissecans. Rev Esp Cir Ortop Traumatol (Engl Ed). 2021;65(2):132–7.
65. Telleria JJM, Ready LV, Bluman EM, Chiodo CP, Smith JT. Prevalence of vitamin D deficiency in patients with Talar osteochondral lesions. Foot Ankle Int. 2018;39(4):471–8.
66. Yasui Y, Ramponi L, Seow D, et al. Systematic review of bone marrow stimulation for osteochondral lesion of talus - evaluation for level and quality of clinical studies. World J Orthop. 2017;8(12):956–63.
67. Toale J, Shimozono Y, Mulvin C, Dahmen J, Kerkhoffs G, Kennedy JG. Midterm outcomes of bone marrow stimulation for primary osteochondral lesions of the talus: a systematic review. Orthop J Sports Med. 2019;7(10):2325967119879127.
68. Shimozono Y, Hurley ET, Myerson CL, Kennedy JG. Good clinical and functional outcomes at midterm following autologous osteochondral transplantation for osteochondral lesions of the talus. Knee Surg Sports Traumatol Arthrosc. 2018;26(10):3055–62.
69. Sheu C, Ferkel RD. Athletic performance in the National Basketball Association after arthroscopic debridement of osteochondral lesions of the talus. Orthop J Sports Med. 2021;9(1):2325967120970205.
70. Hurley ET, Shimozono Y, McGoldrick NP, Myerson CL, Yasui Y, Kennedy JG. High reported rate of return to play following bone marrow stimulation for osteochondral lesions of the talus. Knee Surg Sports Traumatol Arthrosc. 2019;27(9):2721–30.
71. Lee KT, Song SY, Hyuk J, Kim SJ. Lesion size may predict return to play in young elite athletes undergoing microfracture for osteochondral lesions of the talus. Arthroscopy. 2021;37(5):1612–9.
72. Easley ME, Vineyard JC. Varus ankle and osteochondral lesions of the talus. Foot Ankle Clin. 2012;17(1):21–38.
73. Li X, Zhu Y, Xu Y, Wang B, Liu J, Xu X. Osteochondral autograft transplantation with biplanar distal tibial osteotomy for patients with concomitant large osteochondral lesion of the talus and varus ankle malalignment. BMC Musculoskelet Disord. 2017;18(1):23.
74. Ackermann J, Casari FA, Germann C, Weigelt L, Wirth SH, Viehofer AF. Autologous matrix-induced chondrogenesis with lateral ligament stabilization for osteochondral lesions of the talus in patients with ankle instability. Orthop J Sports Med. 2021;9(5):23259671211007439.