

Osteochondral Lesions of the Talus

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Abstract

Osteochondral lesions of the talus can present as a late complication of ankle injuries. As the talus is largely covered by articular cartilage, it has a limited ability for repair. Early and accurate diagnosis is important as talar integrity is required for optimal function of the ankle. The common presentation is chronic ankle pain with a history of ankle trauma. Conservative treatment involving a period of casting and non-weight-bearing is recommended for acute, non-displaced osteochondral lesions. Surgical management is recommended for unstable lesions or failed conservative management.

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Key words: Ankle sprains, Arthroscopy, Cartilage culture, Osteochondral graft

Introduction

Osteochondral lesions of the talus are a known cause of chronic ankle pain. They are frequently found in the active population after ankle sprains. The terminology has its origins in 1856, when Monro¹ first described the presence of cartilaginous loose bodies in the ankle joint. In 1888, Konig² coined the term “osteochondritis dissecans” to describe spontaneous necrosis of subchondral bone and articular cartilage with loose body formation in the knee. In 1922, Kappis³ applied this term to describe similar lesions in the ankle joint. However, such a term implied an inflammatory disease process, leading to confusion. The term was revised in 1959 by Berndt and Harty⁴ who used the term “transchondral fractures of the talus”. Several other terms have been used, including “osteochondral fracture”, and “talar dome fracture”, but currently, “osteochondral lesion of the talus” remains the most inclusive term to describe the problem.

The average age of occurrence of talar osteochondral lesions usually lies between 20 and 30 years of age. They are bilateral in 10% of cases, with a slight preponderance in males. These lesions are seen in 6.5% of ankle sprains.⁵ However, this incidence may be underestimated as many of these lesions may be subclinical, masked by other more obvious associated injuries of the foot and ankle or missed because of limitations in conventional radiological investigations.⁶

Aetiology

The exact cause of osteochondral lesions of the talus remains unclear. Although few studies report an atraumatic cause, most studies report a history of trauma as a likely pathogenesis of talar osteochondral lesions.⁷ Flick and Gould⁸ reviewed the literature in reports of more than 500 patients with these lesions and found that 98% of lateral dome lesions and 70% of medial dome lesions were associated with a history of trauma. The trauma may be a single episode of injury or repetitive microtrauma.⁷ There are 2 common patterns of talar osteochondral lesions, as shown in Figure 1. Anterolateral talar dome lesions result from inversion and dorsiflexion injuries of the ankle where the area impacts against the fibula. These lesions are usually wafer-shaped and shallow due to tangential or shearing forces created by the fibula on the anterolateral

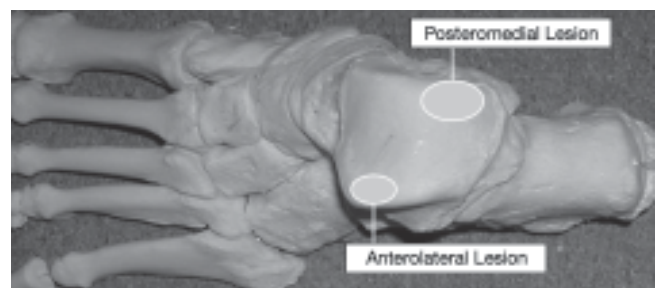


Fig. 1. Common locations of osteochondral lesions of the talus.

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talar dome.^{4,9} Posteromedial lesions result from inversion, plantar flexion and external rotation injuries of the ankle where the area impacts against the tibial ceiling of the ankle joint. These lesions are cup-shaped and deeper due to the posteromedial aspect of the talus impacting perpendicular to the tibial ceiling.^{4,9}

Clinical Evaluation

A typical presentation is of chronic ankle pain persisting after a prior history of an inversion injury of the ankle. Pain is usually experienced at the specific locations of the lesions. Recurrent swelling, weakness, stiffness and catching of the ankle joint is often described. Patients with a history of recurrent ankle sprains often complain of ankle instability. Point tenderness can usually be elicited and should be sought over the common sites of the lesions. Anterolateral lesions can be palpated at the anterolateral talar dome with the ankle in plantarflexion. Posteromedial lesions can often be palpated behind the medial malleolus with the ankle in dorsiflexion. Tests for instability should be performed, including the anterior drawer test as well as the inversion and eversion stress tests. Range of motion of the ankle should be documented and compared with the contralateral side. Examinations to exclude neurological as well as vascular causes of ankle pain should be carried out. In the acute setting, coexisting ligamentous injuries, fractures of the fibula or tibial plafond should also be excluded.

Plain radiographs should be the initial investigation of suspected osteochondral lesions of the talus. Standard views of the ankle should include weight-bearing anteroposterior, lateral and mortise views. Better visualisation of talar dome lesions can be made in ankle radiographs with the foot in 15° pronation and the X-ray beam angled 75°

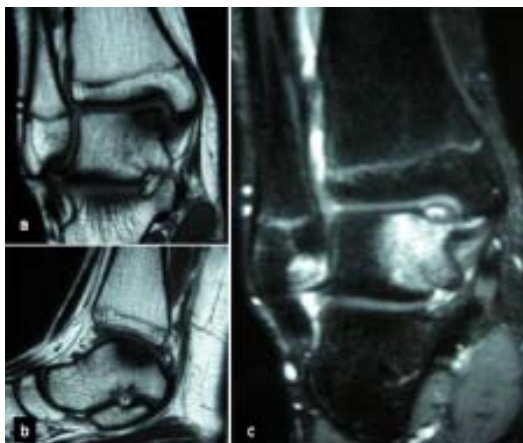


Fig. 2. Magnetic resonance imaging (MRI) of the ankle joint. (a) The oblique coronal MRI demonstrating a lesion in the medial talar dome. (b) The sagittal MRI shows a thin fibrillated cartilage covering the osteochondral lesion in the medial aspect of the talar dome. (c) T-2 weighted oblique coronal MRI demonstrating high signal intensity of oedema surrounding the lesion consistent with grade 2a lesion.

cephalad.¹⁰ Posteromedial talar dome lesions are best visualised in the mortise view of the ankle in plantar flexion. However, plain radiography lacks the ability to detect cartilage defects or undisplaced lesions.⁶ Computed tomography (CT) is useful in determining the site, size, location, shape and degree of displacement of osteochondral fragments.¹¹ However, CT lacks the ability to visualise the articular cartilage, and bone bruises, and detect non-displaced lesions. Bone scintigraphy can be utilised to screen for possible occult osteochondral lesions of the talus in cases where radiographs appear normal.¹² Bone scintigraphy was shown to have a 96% specificity and 94% sensitivity for osteochondral abnormalities.¹³ Magnetic resonance imaging (MRI) has the ability to assess both articular cartilage and subchondral bony lesions, as well as evaluate surrounding soft tissue abnormalities.¹⁴ It has the advantage of being able to detect early subchondral injuries.¹⁴ Figure 2 is the MRI depicting osteochondral lesion of the talar dome. MRI, in detecting osteochondral lesions of the talus, has been found to correlate closely with arthroscopic findings.¹⁵ It has been suggested that in radiological evaluation, plain radiographs should be the initial evaluation of choice in patients with acute ankle injuries.¹⁶ Any detected lesions should be evaluated with CT to determine further details.¹⁶ In cases with persistent ankle pain despite normal plain radiographs, an MRI may be warranted.¹⁶

Classification and Staging

Berndt and Harty⁴ first staged osteochondral lesions of the talus in 1959 based on radiographic findings: stage 1, subchondral bone compression; stage 2, partially detached osteochondral fragment; stage 3, completely detached but undisplaced osteochondral fragment; stage 4, completely detached and displaced osteochondral fragment. Loomer and coworkers¹⁷ later modified the staging system to include stage 5, subchondral cysts. Other staging systems have been developed based on MRI findings. Hepple and

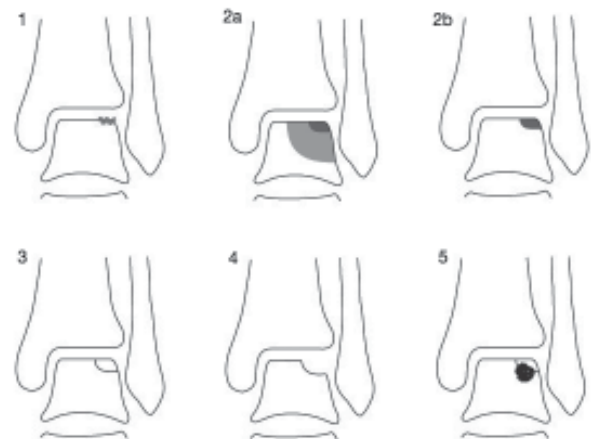


Fig. 3. Classification of osteochondral lesions of the talus using MRI.

colleagues¹⁸ revised the MRI classification in 1999 to resemble Berndt and Harty's original classification. Stage 1 represents articular cartilage damage only. Stage 2a represents articular cartilage damage with underlying fracture and bony oedema. Stage 2b is similar to 2a without bony oedema. Stage 3 represents a detached but undisplaced osteochondral fragment. The fragment is displaced in stage 4 and in stage 5, subchondral cyst formation occurs. Figure 3 illustrates the classification of osteochondral lesions of the talus using MRI.

Pritsch and coworkers¹⁹ were one of the first to grade talar osteochondral lesions with arthroscopic findings according to cartilage quality. Cheng and colleagues²⁰ further developed arthroscopic staging of the lesions. In stage A, the articular cartilage is smooth and intact, but soft. In stage B, the articular cartilage surface is rough. In stage C, fibrillation or fissuring of the cartilage is present. In stage D, an osteochondral flap is present or bone is exposed. In stage E, the osteochondral fragment is detached but undisplaced. In stage F, the osteochondral fragment is detached and displaced. Arthroscopy is useful in staging talar osteochondral lesions, but it is unable to completely assess underlying bony lesions.

Treatment

There exists a wide variety of management strategies for osteochondral lesions of the talus. These range from non-surgical to biological repair and regeneration of cartilage. Authors have suggested that management decisions should be based on the grade of the lesion.^{5,16,21,22} Others based treatment decisions on the size of the lesion: lesions >1.5 cm require surgery.²³

Non-surgical

Opinions vary from treating all lesions to only Berndt and Harty stage 1 lesions non-surgically.^{4,24} Non-surgical treatment involves a combination of rest or restriction from strenuous activity, an initial period of non-weight-bearing with cast immobilisation, and subsequently protected weight bearing with gradual mobilisation thereafter. It has been suggested that Berndt and Harty stage 1 and 2 lesions should be managed non-surgically for up to 1 year to allow for resolution before surgical decisions are made.^{5,16,21,22} Tol and coworkers²⁵ did a meta-analysis of 14 studies with a total of 201 patients who underwent non-operative treatment for mainly stage 1 and 2 talar osteochondral lesions and found a 45% success rate.

Surgical

Surgical treatment is considered with failed conservative management, or Berndt and Harty stage 2 to 5 lesions. Surgical management strategies for osteochondral lesions of the talus include excision with or without techniques for

stimulation of fibrocartilage growth such as microfracture, curettage, abrasion, or transarticular drilling. If the fragment is large enough, it may be secured to the talar dome through retrograde drilling, bone grafting, or internal fixation. Other surgical strategies include cancellous bone grafting and osteochondral transplantation through osteochondral autografts, allografts, or cell cultures. A summary of recent outcome studies examining the various surgical strategies in the treatment of osteochondral lesions of the talus is shown in Table 1.

Debridement, Microfracture and Drilling

Such procedures are reserved for completely detached talar osteochondral lesions that are not amenable by internal fixation. These can be done open or arthroscopically, which entail mainly excision of the osteochondral fragment and curettage of the lesion's surface to remove debris and devitalised tissue. Holes are subsequently drilled, creating vascular access to the underlying subchondral bone which allows marrow elements to migrate into the avascular lesion.^{16,25} This stimulates the formation of fibrin clots and subsequently, fibrocartilage (type I collagen) in the defect.¹⁶ Outcomes of excision and drilling are favourable, with reports of excellent results in 70% to 90% of the patients.^{25,31}

A dilemma arises with intact articular cartilage surface overlying the subchondral lesion. In order to create a path for revascularisation, drill holes may be placed through the transmalleolar/ transarticular method or retrograde/ transtalar approach. The transmalleolar/ transarticular approach involves accessing the lesion from above by drilling through the malleolus and subsequently through the articular cartilage. This technique was studied by Kumai and coworkers,³² which showed both clinical and radiological improvements. The retrograde/transtalar approach involves accessing the lesion from below by drilling through the sinus tarsi. This technique has the advantage of not disrupting the articular surface. Taranow and colleagues³³ evaluated this technique and found good clinical outcome in 81% of patients. Kono and coworkers³⁴ evaluated both techniques and found that retrograde drilling fared better in arthroscopic assessment after 1 year compared to the transarticular approach. However, no significant differences were found in the improvements in the American Orthopaedic Foot and Ankle Society Ankle-Hindfoot Scale (AOFAS) score between the 2 treatment groups after 2 years.

Bone grafting has been commonly coupled with retrograde drilling of talar osteochondral lesions with intact overlying articular cartilage. The drill path supplies a route for revascularisation, whereas the bone graft provides osteoinduction and osteoconduction. This technique is ideal for large subchondral cystic lesions with intact articular cartilage.

Internal Fixation

Internal fixation can be carried out when fragments are large enough with significant amount of subchondral bone attached to the articular cartilage. Internal fixation of

osteochondral lesions may be done using a variety of methods, including screws, pins, fibrin glue, Kirschner wires, and bioabsorbable devices.^{35,36} In the meta-analysis by Tol and coworkers,²⁵ such treatments showed 73%

Table 1. Recent Outcome Studies Examining Surgical Techniques for Treatment of Osteochondral Lesions of the Talus

Technique	Source	n	Follow-up duration (range)	Specific procedures; Outcome measures	Results
Arthroscopic debridement, microfracture, Drilling	Kumai et al (1999)	18	4.6 years (2-9.5)	Arthroscopic drilling. Berndt and Harty criteria. ⁴	72% reported good results. All reported improvement.
	Taranow et al (1999)	16	24 months (19-38)	Retrograde drilling medial talar dome osteochondral lesion without detached cartilage. AOFAS score. ²⁶	Average improvement of 25 points. 88% radiographic healing at 15 months.
	Schuman et al (2002)	38	4.8 years (2-11)	Arthroscopic curettage and drilling. Ogilvie-Harris score. ²⁷	82% reported good to excellent results.
	Kono et al (2006)	30 (11) (19)	32.7 months (24-49) 33.5 months (24-46)	Case control study. Retrograde vs Transarticular drilling. AOFAS score. ²⁶	Average improvement of 26.6 points. Average improvement of 24.4 points.
Internal fixation	Tol et al (2000)	11	-	Meta-analysis of treatment using screws, pins, Kirschner wires.	73% reported good to excellent results.
	Kumai et al (2002)	27	7 years (2-18.8)	Fixation using cortical bone pegs. Berndt and Harty criteria. ⁴	89% reported good results. 81% complete bony union.
	Schuh et al (2004)	20	46 months (18-93)	Kirschner wire fixation of unstable fragments. Ogilvie-Harris score. ²⁷	100% reported good to excellent results.
Allograft	Gross et al (2001)	9	11 years (range 4-19)	Stage IV Berndt and Harty classification. Fresh allograft transplantation.	67% graft survival. 33% resulted in resorption and fragmentation of the graft.
Autografts	Assenmacher et al (2001)	9	9.3 months (range 4-17)	Unstable osteochondral lesions of the talus. MRI follow-up	100% osteointegration of grafts
	Hangody et al (2001)	36	4.2 years (range 2-7)	Large and unstable osteochondral lesions of the talus. Hannover ankle rating score. ²⁸	Good to excellent results in 94%. Normal surfaces with type II articular cartilage biopsies.
	Gautier et al (2002)	11	24 months (range 6-47)	CT scan follow-up	Graft incorporation in 91% of patients.
	Sammarco et al (2002)	12	25.3 months (range 16-41)	Unstable osteochondral lesions of the talus. AOFAS score. ²⁶	Average improvement of 26.4 points (range 3-62)
Autologous chondrocyte transplantation	Koulalis et al (2002)	8	17.6 months (range 8-26)	Finsen score. ²⁹ Arthroscopic re-examination.	Average improvement from 3.4 points to 0.62 points. Hyaline cartilage on histology in 88%
	Giannini et al (2001)	8	24 months	AOFAS score. ²⁶ Biopsy.	Average improvement of 59 points. Hyaline cartilage on histology in all cases
	Whittaker et al (2005)	10	23 months	Modified mazur ankle score. ³⁰ 9 patients had follow-up arthroscopic examination after 1 year.	Mean improvement of 23 points. Defects filled by mostly fibrocartilage with some hyaline cartilage.

success rates. Traditional bone screws have fallen out of favour as it may cause damage to the articular cartilage. Kirschner wire fixation of unstable osteochondral fragments was evaluated by Schuh and colleagues.³⁷ Good to excellent outcome was found over a 4-year follow-up period. Kumai and coworkers³⁸ evaluated the use of cortical bone pegs for fixation of chronic unstable osteochondral fragments of the talus. Over a 7-year evaluation period, fair to good results with radiological improvement was found.

Allografts and Autografts

Osteochondral allografts have been used for osteochondral defects where simple bone grafting may not be appropriate. Allograft transplants are indicated for massive lesions where there is difficulty in obtaining sufficient autologous grafts. The use of allografts has the advantage of restoring articular surface as well as eliminating the risk of donor site morbidity. A few authors have discouraged the use of this form of treatment due to long duration of recovery, and associated complications such as immunogenicity challenges and limited chondrocyte viability.^{39,40} Many would favour the use of osteochondral autografts or mosaicplasty for treatment of large osteochondral defects.⁴¹ This procedure requires harvesting the donor osteochondral plug from non-weight-bearing regions such as the medial or lateral femoral condylar ridge of the ipsilateral knee, or ipsilateral medial or lateral articular facet of the talus. These plugs or grafts are subsequently delivered, compressed and inserted press-fit into the talar defect.

Hangody and colleagues⁴² reported good to excellent results with this treatment in 94% of patients with large (>10 mm) or unstable talar osteochondral lesions over a 2- to 7-year follow-up period. Several other authors also reported good outcomes in terms of pain and functional scores, MRI and arthroscopic evaluation, with no reported problems associated with donor site availability and donor site morbidity.⁴³⁻⁴⁶ Difficulties associated with this procedure include the limited surface area that can be covered, leaving the dead space between grafts to be filled by fibrocartilage. There may be irregularity of the surface due to differing thickness of the cartilage transplant.⁴²

Autologous Chondrocyte Transplantation

Autologous chondrocyte transplantation is a cartilage repair process widely publicised for treatment of knee cartilage defects.⁴⁷⁻⁴⁹ This 2-stage process involves growing the patient's own harvested cartilage cells and reimplanting them at the cartilage defect site. Good results of this treatment of talar osteochondral defects were reported by Koulalis and coworkers.⁵⁰ Arthroscopic examination at 6 months showed complete coverage of defects. The histological examination revealed hyaline cartilage in 7 out

of 8 patients. Similar findings of osteochondral autografts healing with type 2 collagen characteristic of hyaline cartilage were reported by Giannini and colleagues.⁵¹ Theoretically, the advantages of this procedure are that it is not limited by the size of the defect and donor site availability and morbidity. Recently, Whittaker and coworkers⁵² reported favourable results in 9 out of 10 patient over a 4-year follow-up period. Nine patients underwent arthroscopic examination in 1 year and were all shown to have filled defects and stable cartilage. However, biopsies taken from graft sites showed mostly fibrocartilage with some hyaline cartilage.

Other Forms of Treatment

Parenteral administration of Iloprost, a prostacyclin analogue, has been used to treat bone marrow oedema of the talus. Indications for its use include stage 1 lesions with no subchondral fracture or collapse. Aigner and colleagues⁵³ demonstrated complete resolution of oedema in the talus within 3 months on MRI examination with its administration. Iloprost was also successfully used in a pilot study in children with early aseptic osteonecrosis (AON) by Petje and coworkers.⁵⁴ The main pathological mechanism is disturbance of blood supply and is characterised by the death of bone marrow and trabecular bone. It was theorised that Iloprost promotes and regulate blood supply.

The efficacy of such treatments, being mediated by prostaglandin pathways and its relationship with avascular necrosis, questions the aetiological mechanism of early osteochondral lesions. The stage 1 osteochondral lesion could be related to avascular necrosis, traumatic bone bruise, inflammation or a fracture. Future studies are required to further evaluate these subgroups.

Summary

A high index of suspicion is required for early diagnosis of osteochondral lesions of the talus. Chronic ankle pain and functional impairment may result from injuries of the subchondral bone and subsequent degeneration of the articular surface. The aetiology is still unclear but most subscribe to the theory that the lesions arise secondary to ankle trauma. Forceful inversion injuries of the ankle in dorsiflexion are associated with lateral dome lesions, and inversion injuries of the ankle in plantarflexion are associated with medial lesions. Early and stable lesions (stage I or II) may be treated conservatively, while more severe lesions (stage III to V) may require surgical intervention.

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