Osteochondral autologous transplantation as a treatment of osteochondral defects of the talus

Sameh S. Alsafty
Shibin Alkom Teaching Hospital, alsaftysameh@yahoo.com

Follow this and additional works at: https://jmisr.researchcommons.org/home

Part of the Medical Sciences Commons, and the Medical Specialties Commons

Recommended Citation
DOI: https://doi.org/10.4103/JMISR.JMISR_49_18

This Original Study is brought to you for free and open access by Journal of Medicine in Scientific Research. It has been accepted for inclusion in Journal of Medicine in Scientific Research by an authorized editor of Journal of Medicine in Scientific Research. For more information, please contact m_a_b200481@hotmail.com.
Osteochondral autologous transplantation as a treatment of osteochondral defects of the talus

Sameh S. Alsafty
Orthopedic Department, Shibir Alkom Teaching Hospital, Shibir Alkom, Menoufia, Egypt

Abstract

Background
For many years, osteochondral defects of the talus has been known as a symptomatic lesion that causes pain, recurrent synovitis, altered joint mechanics, and obstruction from loose bodies. It is a probable precursor of ankle osteoarthritis. Arthroscopic procedures, such as debridement, retrograde drilling, and bone grafting have a great advantage in treating small defects. An open method can be considered for treatment of large osteochondral lesions and unstable osteochondral defect lesions.

Objectives
To present the functional results after autologous osteochondral transplantation in 12 patients using open surgical technique by antromedial approach and antrolateral approach for the ankle according to the site of the lesions, all lesions more than or equal to 1.5 cm the mean age of patients at the time of surgery was 20.5 years (range, 18–23 years), the mean follow up time was 27 months (range, 12–42 months), patient reported outcome measures were taken preoperatively and at final follow up using foot and ankle outcome score.

Results
The mean foot and ankle outcome score improved from 52.2 points preoperatively to 86.2 points postoperatively (range, 71–100 points). Two patients reported donor site knee pain after surgery, and one patient had delayed healing at medial malleolar osteotomy site.

Keywords: Foot and ankle outcome score, osteochondral transplantation, osteochondral defects

INTRODUCTION
Symptomatic osteochondral ankle defects often require surgical treatment. 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. These symptoms place the ability to walk, work, and perform sports at risk [1].

The injury was classified by Berndt and Harty. 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) (Fig. 1). Berndt and Harty experimentally proved the traumatic etiology of the lesion; however, nontraumatic lesions also occur [2,3].

Treatment strategies for osteochondral defects of the ankle have substantially increased over the last decade.

The widely published treatment strategies of symptomatic osteochondral lesions (OCLs) include the nonsurgical treatment with rest or cast immobilization, and surgical excision of the lesion, excision and curettage, excision

combined with curettage and drilling/microfracturing (i.e. bone marrow stimulation), placement of an autogenous (cancellous) bone graft, antegrade transmalleolar drilling, retrograde drilling, fixation, and newer techniques like osteochondral transplantation (osteochondral autograft transfer system, Osteochondral Autograft Transplant System), and the autologous chondrocyte implantation (ACI). The last two techniques focus on the replacement and regeneration of the hyaline cartilage, respectively [1,4] (Fig. 2).

The goal of these treatment strategies is to diminish symptoms like pain and swelling, and to improve function. The choice of treatment is based on the type, stage, and size of the defect [5].

Patients and methods

This study including 12 skeletally mature patients undergoing ankle arthrotomy ipsilateral knee (as a donor site) to the ankle (as a recipient site) autologous osteochondral grafts for treatment of OCLs of the talus more than or equal to 1.5 cm in diameter. All types of four classification according to Berndt and Harty and their location as antrolateral, antromedial, computed tomography (CT), and MRI were also used to assist the classification and evaluation of the integrity of lateral ankle ligaments.

Foot and ankle outcome score (FAOS) scale of FAOS applied preoperatively and at outpatient follow up. The location and the stage of the injuries were considered subjective. Satisfaction questionnaire were also applied one year after the procedure, considering the following responses: very satisfied, satisfied, regular, or dissatisfied.

Between the years of 2009 and 2015: 12 patients (four males, eight females) underwent autologous osteochondral transplantation for an OCL of talus.

All patients were assessed using FAOS and general health questionnaire.

The mean patient age at the time of surgery was 20.5 years (range, 18–23 years) the mean follow up time was 27 months (range, 12–42 months).

The talar dome postromedial lesions were nine patients.
The talar dome antrolateral lesions were three patients.

All lesions more than or equal to 1.5 cm in diameter.

Three patients were at left ankle.
Nine patients were at right ankle.

Grafts were fixed by K wire 1.5 mm.
(1) Post slabs were applied for 2 weeks postoperative.
(2) 2 weeks postoperative sutures removal.
(3) 3 weeks postoperative K wire removal.
(4) Below knee casts were applied for 6 weeks.
(5) After 9 weeks ankle brace and physiotherapy, partial weight bearing.

After 12 weeks complete weight bearing.

For postromedial lesions of the talus, medial malleolus osteotomy was done.

Diagnosis

The suspected diagnosis of OCLs of the talus starts with complaints of pain related to physical activities, usually with a history of previous trauma. Joint swelling, sensation of instability, joint blockage, or extremely painful clamping may occur [6,7].

Despite the great chance of false-negative diagnosis, simple ankle radiographs in anteroposterior, lateral, and oblique are the first imaging to be obtained in the diagnostic process of OCLs of the talus [8].

The most common finding in simple radiology is the presence of poorly defined radiolucent area in the talar dome, in the place where the pathological process has become installed [9].

MRI provides information, allowing for the assessment of articular cartilage and presence of subchondral inflammatory changes, as well as for the identification of the depth of the chondral lesion. It is therefore regarded as the gold standard in the diagnosis of OCLs [10,11].

The most widespread classification for OCLs of the talus is that proposed by Berndt and Harty; it is based on the degree of displacement of the osteochondral fragment and has four stages: stage I – small focal subchondral trabecular compression area;
stage II – partially loose fragment (incomplete fracture); stage III – loose fragment (complete fracture), but not displaced; and stage IV – loose fragment (complete fracture) and displaced from its bed [3,12–14].

Mintz and colleagues report combined arthroscopic observations with MRI to design their rating for OCLs of the talus, following the same dynamics of the other classifications. Six different stages are possible: stage 0 – normal cartilage; stage I – hypersignal cartilage on MRI, but normal arthroscopic appearance; stage 2 – fibrillation and cracks that do not reach the bone; stage 3 – presence of cartilage flap, with exposure of the subchondral bone; stage 4 – loose fragment, nondisverted; stage 5 – diverted fragment.

Surgical treatment: surgical treatment of osteochondral ankle injuries can be divided into five main groups of procedures. (1) Reduction and fixation of osteochondral fragments (2) Bone marrow stimulation. (3) Articular cartilage replacement. (4) Regenerative cell therapy.

Autologous osteochondral graft: the osteochondral autologous grafting system known as mosaicplasty involves obtaining cylindrical cartilage and bone/grafts, most commonly originating from the lateral femoral condyles, and transferring them to areas of osteochondral lesion in the loading surface of the talar dome. This procedure presents encouraging results. The indications for osteochondral grafting for lesions larger than 1.5 cm, recurrent or refractory to more conservative treatment methods, and especially lesions associated with subchondral cysts [15,16].

Mosaicplasty has technical standards that must be followed in order to achieve better results: (1) the donor area can never be a load bearing region; (2) osteochondral cylinders must be inserted perpendicularly to the receiving surface; (3) the cartilage portion should have the shape and curvature as close to the receiving zone as possible; (4) the cylinder must be at least 15 mm in length for chondral lesions and 25 mm in the presence of subchondral cysts; (5) the cartilage plug must remain perfectly leveled with the edges of the receiving region.

Autologous chondrocyte implantation: this procedure begins by obtaining viable chondrocytes through resection of a small fragment of the healthy cartilage tissue from the joint or from another joint from the same individual. The chondrocytes are isolated and cultured for 3 to 6 weeks in order to multiply. The second part of the procedure is the preparation of the receiving area and the implantation of the cultured cells. Curettage and debridement of the base and edges of the injury until they establish the limits of healthy cartilage [17].

Mesenchymal stem cells

Stem cell therapy is based on two mechanisms of action. At first, the cells differentiate and mimic the final cells of tissues and organs; then, there is the production of substances cytokines and growth factors that favorably influence the angiogenesis and the reduction of cell apoptosis, and induce the endogenous regeneration [14,18,19].

Classifications

The original radiographic classification system for osteochondral lesions of talus (OLT) was developed by Berndt and Harty and determined only by plain radiographs. At the time they referred to it as an arbitrary classification, which was developed to aid understanding of the etiological mechanism of the fracture and to help determine the appropriate treatment. Although radiographs are now rarely used to stage OLTs, the original classification system by Berndt and Harty is still the most commonly used classification system [3,20] (Tables 1 and 2).

The initial staging classification from CT scans was introduced by Ferkel and colleagues (Table 3). Since that time it has remained the predominant CT staging system. Although useful for defining bony anatomy, it has not been shown to be correlated with patient outcomes [21,22] (Table 4).


| Table 1: Radiographic classification of transchondral fractures of the talus |
|---|---|
| Stages | Definition |
| I | Compression fracture with intact overlying cartilage |
| II | Complete avulsion of an osteochondral fragment |
| III | Complete avulsion of an osteochondral fragment without displacement |
| IV | Avulsed fragment displaced into the joint |

Reference [34].

| Table 2: MRI staging system for osteochondral lesion of the talus |
|---|---|
| Stages | Definition |
| 1 | Articular cartilage damage only |
| 2a | Cartilage injury with underlying fracture and surrounding bony edema |
| 2b | Stage 2a without surrounding bony edema |
| 3 | Detached but nondisplaced fragment |
| 4 | Detached and displaced fragment |
| 5 | Subchondral cyst formation |


| Table 3: Computed tomographic staging system for osteochondral lesions of the talus |
|---|---|
| Stages | Definition |
| I | Cystic lesion with intact roof |
| IIA | Cystic lesion with communication to talar dome surface |
| IIB | Open articular surface lesion with overlying nondisplaced fragment |
| III | Nondisplaced fragment with lucency |
| IV | Displaced fragment |

Computed tomographic classification of osteochondral lesions of talus by Ferkel et al. [20].
Surgical technique

Medial talar dome OCL. A medial talar lesion is most commonly located in a central or posterior position. The less commonly seen anteromedial lesion may be visualized with a standard arthroscopy and allows adequate exposure of the anteromedial talar dome to remove and replace an anteromedial lesion. Typically, however, the size and location of the lesion demand a medial malleolar osteotomy [23]. For central and posterior lesions, the osteotomy is created to provide adequate exposure of the talar dome. This requires angulating the osteotomy cut. A provisional K wire can be used to fluoroscopically visualize the osteotomy cut. This is typically made at ~30° relative to the long axis of the tibia but can vary depending on the exact approach required. Once this has been established, it is important to predrill the osteotomy fixation holes [24].

A provisional K wire is drilled, fluoroscopically visualize the medial malleolar osteotomy. A chevron-type cut is then made in the medial malleolus.

For lateral talar dome OCL: An antrolateral approach to the ankle.

Incision for the anterolateral to the ankle. Make a 15-cm slightly curved incision on the anterolateral aspect of the ankle. Begin ~5 cm proximal to the ankle joint and 2 cm anterior to the anterior border of the fibula. Curve the incision downward to cross the ankle joint 2 cm medial to the tip of the lateral malleolus, and continue into the foot, ending about 2 cm medial to the fifth metatarsal (Fig. 3).

Identify the peroneus tortius and the extensor digitorum longus muscles and incise down to bone lateral to them in the upper half of the wound.

Retract the extensor musculature medially to expose the anterior aspect of the distal tibia ankle joint. Identify the origin of the extensor digitorum brevis (Figs 4–9).

Determining graft size

Several commercial systems are available to harvest the graft and donor site. All systems use a similar ‘apple core’ technique by taking a tubular-shaped unit of bone and cartilage from each respective site. Currently utilize the Osteochondral Autograft Transplant System [25,26]. The typical size of a lesion utilizes an 8-mm core, but this should be individualized and based on the size of each lesion. Core sizes increase from 6 mm up to 8 and 10 mm and 12 mm. These should be ‘nested’ to reduce the amount of fibrocartilage between the grafts (Fig. 10). Two cylindrical grafts can be placed side by side to produce a figure-of-eight configuration, thereby allowing fibrocartilage to fill in the nonadjacent space of the graft. This is a potential site for synovial fluid inflow that could theoretically undermine the graft over time. By placing the grafts in a nested position, this potential is therefore reduced [27]. The damaged cartilage and bone are removed from the talus. The base of the graft recipient site is then overdrilled by a further 2 mm, yielding a standard graft depth of 12 mm [28,29].

Results

Our study including 12 patients, all underwent ankle minimal arthroscopy for mosaicplasty from antrolateral nonarticular surface of ipsilateral knee for OCL of the talus.

The mean FAOS improved from 71.4 point preoperatively to 93.2 points postoperatively (range, 82.3 points).

With degree of very satisfaction (75%) and satisfaction (16.6%) and regular (8.4%).

Two patients reported donor site knee pain after surgery, relieved later by NSAIDS and physiotherapy and one patient had delayed healing at osteotomy site (Tables 5 and 6).

Table 4: Arthroscopic staging system for osteochondral lesions of the talus

<table>
<thead>
<tr>
<th>Grades</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Smooth, intact, but soft cartilage</td>
</tr>
<tr>
<td>B</td>
<td>Rough cartilage</td>
</tr>
<tr>
<td>C</td>
<td>Fibrillations or fissures</td>
</tr>
<tr>
<td>D</td>
<td>Flap present or bone exposed</td>
</tr>
<tr>
<td>E</td>
<td>Loose, nondisplaced fragment</td>
</tr>
<tr>
<td>F</td>
<td>Displaced fragment</td>
</tr>
</tbody>
</table>

Cheng/Ferkel arthroscopic staging system of osteochondral lesions of talus Cheng et al.
In 1870 Paget described a pathological process that he termed ‘quiet necrosis,’ which resulted in the creation of loose, necrotic osteochondral fragments from articular surfaces. This report was followed in 1888 when Konig first coined the term ‘osteochondritis dissecans’. Konig theorized that inflammation of the bone and cartilage, followed by spontaneous necrosis led to a fragment dissecting away, hence the term. Such early descriptions were generally cases involving the knee, which accounts for ~75% of all osteochondritis cases. In 1932, Rendu reported an intra-articular fracture of the talus that appeared similar to osteochondritis dissecans. This report led to the publication by Roden in 1953 of 55 osteochondritis dissecans like lesions of the talus [30–32]. In 1959, Berndt and Harty demonstrated that both medial and lateral lesions of the talus, previously considered to be osteochondritis dissecans, were actually osteochondral fractures caused by trauma with viable bone rather than the necrotic bone found with osteochondritis dissecans. Simultaneously, it was shown that there was no associated inflammatory component to osteochondritis dissecans. Therefore the name was incorrect, and there was a strong evidence for a traumatic etiology [33,34].

This report led to the publication by Roden in 1953 of 55 osteochondritis dissecans like lesions of the talus [30–32].

In 1870 Paget described a pathological process that he termed ‘quiet necrosis,’ which resulted in the creation of loose, necrotic osteochondral fragments from articular surfaces. This report was followed in 1888 when Konig first coined the term ‘osteochondritis dissecans’. Konig theorized that inflammation of the bone and cartilage, followed by spontaneous necrosis led to a fragment dissecting away, hence the term. Such early descriptions were generally cases involving the knee, which accounts for ~75% of all osteochondritis cases. In 1932, Rendu reported an intra-articular fracture of the talus that appeared similar to osteochondritis dissecans. This report led to the publication by Roden in 1953 of 55 osteochondritis dissecans like lesions of the talus [30–32].

In 1959, Berndt and Harty demonstrated that both medial and lateral lesions of the talus, previously considered to be osteochondritis dissecans, were actually osteochondral fractures caused by trauma with viable bone rather than the necrotic bone found with osteochondritis dissecans. Simultaneously, it was shown that there was no associated inflammatory component to osteochondritis dissecans. Therefore the name was incorrect, and there was a strong evidence for a traumatic etiology [33,34].

The lack of understanding of the specific pathological process describing this condition has led to a plethora of terms, including: osteochondrosis dissecans, osteochondral fractures, transchondral

**DISCUSSION**

In 1870 Paget described a pathological process that he termed ‘quiet necrosis,’ which resulted in the creation of loose, necrotic osteochondral fragments from articular surfaces. This report was followed in 1888 when Konig first coined the term ‘osteochondritis dissecans’. Konig theorized that inflammation of the bone and cartilage, followed by spontaneous necrosis led to a fragment dissecting away, hence the term. Such early descriptions were generally cases involving the knee, which accounts for ~75% of all osteochondritis cases. In 1932, Rendu reported an intra-articular fracture of the talus that appeared similar to osteochondritis dissecans. This report led to the publication by Roden in 1953 of 55 osteochondritis dissecans like lesions of the talus [30–32].

In 1959, Berndt and Harty demonstrated that both medial and lateral lesions of the talus, previously considered to be osteochondritis dissecans, were actually osteochondral fractures caused by trauma with viable bone rather than the necrotic bone found with osteochondritis dissecans. Simultaneously, it was shown that there was no associated inflammatory component to osteochondritis dissecans. Therefore the name was incorrect, and there was a strong evidence for a traumatic etiology [33,34].

The lack of understanding of the specific pathological process describing this condition has led to a plethora of terms, including: osteochondrosis dissecans, osteochondral fractures, transchondral
A positive bone scan with uptake in or near the talus should be followed up with a CT scan or MRI for definitive diagnosis and localization. In a study with 92 patients Loomer and colleagues reported that a CT scan gave the correct diagnosis in 98% of the cases. The radiolucent lesion (grade V) accounted for an unexpected 77% of the lesions, which is surprising since this majority had gone unrecognized until the grade V lesion was introduced by Loomer and colleagues. The grade V lesion was mainly diagnosed through bone scans and CT scans following normal radiograph [37–39].

Historically, surgical arthroscopy has been the treatment of choice. Arthroscopic procedures, however, are now accessible for specific lesions. The procedure usually involves curettage and drilling, and when present, removal of the fragment. If the fragment is large enough, fixation of the fragment to the site of separation may be possible [37,40].

Our open technique allow direct access to either sides of the ankle even if the lesion is located on the posterior third of the dome of the talus.

We debride the lesion 2 mm more in width by reamer and press fit the graft. Our results were excellent even with little instruments.

**Conclusion**

We suspect OLT to patient presented with history of multiple repetitive ankle trauma and pair related to physical activities, so we request further investigations if the diagnosis is clear for the lesions more than 1.5 cm in width at the dome of the talus. Autogenous osteochondral transplantation is reproducible and is the primary treatment strategy for OLT.

**Financial support and sponsorship**

Nil.

**Conflicts of interest**

There are no conflicts of interest.

**References**


