# **Talar Osteochondral Defects**

# Gokay Gormeli<sup>1</sup>, Yuksel Ersoy<sup>2</sup>, Cemile Ayse Gormeli<sup>3</sup>

#### **Abstract**

Intra-articular ankle injuries to the talar body is one of the common cause of athletic disability that have been estimated to occur in approximately 6.5% of the ankle sprains and commonly affect the dome of the talus. Patients are commonly referred to the clinics with an acute injury to the ankle or with chronic ankle instability. This article describes the clinical presentation and imaging to diagnose of talar osteochondral defects. The treatment options are also reviewed to give consensus on optimal treatment of this condition.

**Key Words:** Ankle injury, imaging of osteochondral defects, osteochondral defect, talus, treatment of osteochondral defects.

(Rec.Date: Feb 06, 2015 Accept Date: Feb 11, 2015)

**Corresponding Author**: Gokay Gormeli, Inonu University, Turgut Ozal Medical Center, Department of Orthopedics and Traumatology; Malatya, Turkey.

E-mail: ggormeli@yahoo.com

<sup>&</sup>lt;sup>1</sup> Inonu University, Turgut Ozal Medical Center, Department of Orthopedics and Traumatology, Malatya, Turkey

<sup>&</sup>lt;sup>2</sup> Inonu University, Turgut Ozal Medical Center, Department of Physical Medicine and Rehabilitation, Malatya, Turkey

<sup>&</sup>lt;sup>3</sup> Inonu University, Turgut Ozal Medical Center, Department of Radiology, Malatya, Turkey

#### Introduction

One of the common cause of athletic disability is intra-articular ankle injuries to the talar body. Osteochondral lesions have been estimated to occur in approximately 6.5% of the 27 million ankle sprains occur per year. Posteromedial lesions are common and deeper in thickness for these injuries. Young population is usually affected with running, jumping or rotating [1]. These injuries commonly affect the dome of the talus. This cartilage injuries would be partial or full thickness or involve bone named as osteochondral defects [2]. The term of "Osteochondritis dissecans" was originally used by Konnig in 1888, to describe loose osteochondral bodies in the knee and hip; and osteochondritis dissecans in the ankle was firstly reported in 1922 by Kappis.

There are some factors causes non-traumatic talar osteochondral lesions such as thromboembolia, corticosteroids or inflammatory diseases (rheumatoid arthritis, ankylosing spondylitis, and systemic lupus erythematosus) [3]. But OCD of the talus is usually seen in athletic population results from acute trauma or chronic lateral ligament instability of the ankle. The traumatic talar OCD are seen in the anterolateral or posteromedial corners. Inversion injury to the plantar flexed food produces shear forces to the lateral talus and also compression forces to the medial talus more ankle dorsiflexion with inversion causes anterolateral lesions of the talus where as more ankle plantar flexion causes posteromedial lesions of the talus [4,5]. These compressive forces could also cause injury to the posteromedial subchondral bone. The overlying articular cartilage may be intact while an injury occurred at the underlying bone.

After injury, edema occurs with softening of the articular cartilage. A partial-thickness defect to articular cartilage has no healing potential as it has no connection with bone marrow which includes mesenchymal and endothelial cells. As the articular cartilage has access to stem cells in marrow in full-thickness defects, they have high potential for fibrocartilage repair that contains Type-1 and Type-2 collagens [6,7]. With weight bearing or lateral ankle instability; the bony defect leads to a fibrous nonunion, cystic lesion, a cartilage defect and loose osteochondral fragment [8].

#### **Examination**

Patients are commonly referred to the clinics with an acute injury to the ankle or with chronic ankle instability. Acute talar OCD cases may have ecchymosis, tenderness, swelling, ankle effusion and synovitis. In the anterior or posteromedial ankle. A drawer or talar tilt test may be positive. A displaced OCD would cause crepitus with ankle movements [9]. In chronic ankle instability, the anterior drawer and talar tilt tests will be positive. Intra-articular loose bodies may cause locking, also crepitus and pain with tibial talar compression may exist [10].

# **Imaging**

Radiographs, computed tomography (CT) and magnetic resonance imaging (MRI) are the main methods for diagnosing talar OCD. Plain radiographs are the first diagnostic test. Weight bearing antero-posterior, lateral, and mortise view are used to identify talar OCD lesions. Radiographs may be negative if the fracture is nondisplaced or at a slight angle. Other x-ray views can also be used for identifying talar OCD lesions. Stress views are useful for detecting ankle ligament laxity. Radiographs of the plantar flexed ankle are good at visualizing posteromedial lesions. The talus may be better visualized with the foot in 15 degrees of pronation and the source tube angled at 75 degrees toward the head [11]. Berndt and Harty classification is the radiographic classification of osteochondral lesions [12]. Stage 1 lesions consist of subchondral trabecular compression with intact overlying cartilage. Stage 2 lesions show incomplete separation of the fragment. Stage 3 lesions are separated but undisplaced. Finally the separated fragment occurs at Stage 4 lesions.

The University of Pittsburgh classification system uses MRI for preoperative evaluation and arthroscopy for the final staging [13]. This system classifies cartilage as viable intact (grade A) or breached and nonviable (grade B). The bone component is described as: Stage 1 is a subchondral compression or bone bruise; Stage 2 lesions are subchondral cysts; Stage 3 lesions are partially separated or detached fragments in situ; and Stage 4 presents displaced fragments (Figure 1).



**Figure 1.** Talar osteochondral defect on T1 weighted images.

CT scanning can be used for the treatment planning of injuries which are visible on plain x-ray radiographs. CT can be used for demonstrating underlying bone cysts. But CT scanning is unable to detect Stage 1 and some Stage 2 injuries; so plain radiographs with good positioning can detect OCD as well as CT [14].

Stage 1 subchondral injuries that would be missed with plain radiographs and CT, so these injuries can be detected by MRI [4]. MRI shows subtle bone changes that may suggest edema or hemorrhage or suspected fractures, which are not visible on x-ray radiographs [15,16]. It can be used to assess talar OCD stability; also the exact site and size of the lesion, especially if it is covered by cartilage. When compared with arthroscopic findings MRI has sensitivity of 95% and specificity of 100% in detecting osteochondral injuries [17]. Contrast can be used as an adjunct to CT and MR arthrography can also be used to assess the stability of talar lesions. MR arthrography had an accuracy of 93% in the evaluation of instability, compared with a 39% accuracy for conventional MR imaging.

Also it can be difficult to visualize the posteromedial corner lesions arthroscopy is the gold standard for determining fragment stability as it gives a clear and certain view of the joint.

### **Treatment Methods of OCD**

## Non-Surgical Treatment

The considerations for treating patients non-operatively were: minor complaints, radiological Berndt and Harty stage I and II, medial stage III lesions or intact cartilage as determined by arthroscopy [18]. These interventions are rest/restriction of (sport-) activities with or without use of non-steroidal anti-inflammatory drugs (NSAID), cast immobilization for 3 weeks to 4 months [19].

**Rest:** This may consist of rest and/or restriction of (sporting) activities with or without treatment of non-steroidal anti-inflammatory drugs (NSAIDs). The aim is to unload the damaged cartilage, so edema can resolve and necrosis is prevented. Another objective could be healing of a (partly) detached fragment to the surrounding bone. Mobilization allowed with non-weight bearing crutches for 3-4 weeks, then progress from partial to full weight bearing up to 4 months [20].

Cast: The aim is similar to the treatment option of rest, but then pursued by below the knee non-weight bearing cast immobilization for at least 4-6 weeks. The main reason to decide for cast immobilization was a Berndt and Harty stage II or III lesion. The typical duration of initial immobilization is four to six weeks. If symptoms are not resolved or radiographic healing is not evident by six weeks, some experts suggest one week of cast or splint removal with physical therapy to decrease the potential for contractions and atrophy followed by a second four to six week period of casting or splinting [21].

**NSAIDs:** Rest and immobilization are the primary treatments for pain in these patients. NSAIDs (eg, ibuprofen) may be used for short-term pain relief during the first three days of treatment [21].

*Exercises:* These type of modalities consist of gait training, weight shifting, BAPS (Biomechanical Ankle Platform System) board, eccentric calf lowering, strengthening and

stretching the lower extremity muscles. The purpose of exercises are to strengthen peroneal musculature, stretch the Achilles tendon and enhance the proprioception.

Swimming and cycling are additional activities that can help building strength and mobility while putting less impact on the knee or ankle [21].

Additional physical modalities, such as continue passive motion (CPM), cryotherapy, joint/soft tissue mobilization, vaso-pneumatic compression, Kinesio taping, balance practices or pulsed electromagnetic fields and also intra articular hyaluronic acid injections could possibly be advantageous for an accelerated and improved outcome, but their exact value has to be further investigated [22].

Resolution of pain and documented healing of the subchondral bone guide the duration of non-operative treatment and determine when the patient can resume daily living/sport activities. Slow return to sports may begin once the patient is asymptomatic, has completed physical therapy and has no pain with running, sprinting or cutting which typically takes six months [21].

## Surgical Treatment

Surgical treatment is preferred for symptomatic OCD of the talus. There is no urgency to treat the lesion in the absence of symptoms as these lesions have slowly progress.

The location, size and depth of the lesion; previous surgery; stage of the disease and the viability of the articular cartilage are the important factors for selecting the surgical approach. We prefer to treat the lesions arthroscopically if possible but when the localization of the lesion is difficult to treat arthroscopically we prefer arthrotomy although it has morbidity when compared with arthroscopy.

A period of rest and immobilization compressive dressings or a brace can be used for Stage 1, Stage 2 and nondisplaced Stage 3 lesions of the talus. To evaluate the progressing regular follow-up and imaging of the ankle should be done. Satisfactory results (92%) were achieved with non-operative treatment of Type-1 or Type-2 lesions by early protection of movement for Stage 1 and immobilization for Stage 2 [23]. Longer conservative period for medial side lesions of the talus is a treatment option as these lesions are more stable, larger and mostly

requires arthrotomy [24]. But patients with displaced Type-3 injuries and Type-4 injuries requires surgical intervention. In our clinic, arthroscopic treatment is the first preferred method for these injuries.

Drilling and micro fracture, bone grafting, chondrocyte implantations are the treatment modalities for OCD lesions. Anterolateral approach is preferred for exposing the lateral lesions of the talus. Combined anterior and posterior arthrotomy or medial malleolus osteotomy can be used for posteromedial lesions which are difficult to treat by only arthroscopy [25].

### **Drilling and Micro fracture**

Arthroscopic drilling for osteochondral lesions of the talus gives good results especially for lesions smaller than 15 mm. Degenerative arthritis, increased age, higher body mass index were negatively affect the outcome scores [26]. Early lesion with the continuity cartilaginous surface and stability of the OCD's fragment are the specific indications for drilling. We prefer retrograde drilling as that gives better results when compared with trans-malleolar drilling [27]. In a systematic review; Kolker et al. concluded that the highest success rate for talar OCD was excision, curettage and drilling. They also concluded that only excision of necrotic tissue or conservative treatment is not recommended [28]. We think that, although micro fracture and drilling is a good treatment option with success rates; the fibrocartilage that is formed after drilling has a different structure from hyaline cartilage. After drilling biological shift to fibro cartilaginous repair tissue exhibiting primarily Type-I collagen at one year. When compared with hyaline cartilage Type-I collagen may degenerate over time as it have different biological and mechanical properties [29]. Drilling is not the preferred treatment method for the defects greater than >15 mm.

Arthroscopic bone-marrow stimulation can be added to drilling and micro fracture for symptomatic osteochondral lesions of the talus that are <15 mm in diameter [29]. Bone-marrow stimulation allows pluripotent mesenchymal stem cells to accumulate into the base of the defect site from the underlying bone marrow [30]. Although several authors reported good results following arthroscopic bone marrow stimulation [31,32]; there are many reports with poorer outcomes, as well [33-35]. Arthroscopic bone-marrow stimulation is a good treatment strategy for small OCD but size guidelines are needed to plan an optimal treatment.

# Autologous Cartilage Replacement

## Autologous Osteochondral Transplantation

Autologous osteochondral transplantation is transplanting the tubular units of cartilage from the non-weight bearing portions of the body. Mosaicplasty is one form of autologous osteochondral transplantation that consists of multiple circular plugs with different sizes. Large primary lesions or cystic lesions usually requires autologous osteochondral transplantation [36]. The inherent advantages of this technique replacing the defect with hyaline cartilage in one session is the important advantage of this technique. Donor site morbidity, incorporation of the grafts, the dead space between grafts, differences in surface curvature between the graft and host tissues and the possible need for an osteotomy are the disadvantages of this procedure.



**Figure 2.** The tubular unit of hyaline cartilage and bone harvested from the ipsilateral knee is transplanted into the defect site of the talus.

Hangody et al. reported 93% of good to excellent clinical results in the talus on the basis of evaluation with the Hannover Scoring System in a study of 98 talar lesions treated with autologous osteochondral transplantation. Also 3% of these patients had donor-site pain in this study [37]. Kennedy et al reported good functional results at a mean follow-up of 28 months in 72 patients treated with autologous osteochondral transplantation [38].

Although osteotomy may be required and donor-site knee pain is a concern, autologous osteochondral transplantation of the talus has good clinical outcomes in the short to mid-term time periods.

## First Generation Autologous Chondrocyte Implantation

This technique uses an injection of chondrocyte cell suspension under a sutured periosteal flap harvested from elsewhere in the body. It is also called as periosteum-covered technique. The limitations of this technique include a technically demanding procedure, the potential for cell differentiation, cell leakage, delamination, and uncontrolled periosteal hypertrophy [39]. In a study by Kwak et al.; 29 patients with talar OCD were followed-up at a mean of 60 months. The authors reported that, the mean AOFAS score significantly improved from 50.1 to 85.9; Tegner activity score improved from 1.6 to 4.3 and also Finsen score showed significant improvement from 13.7 to 5.1 [40]. Giannini et al. evaluated 46 patients with talar OCD. They performed arthroscopy to all patients and clinically evaluated using AOFAS score preoperatively and at 12<sup>th</sup>, 36<sup>th</sup> months and at final follow-up of 87.2±14.5 months. They had significantly improved outcomes and concluded that arthroscopic ACI to repair osteochondral lesions in the ankle joint has satisfactory clinical results after mid-term follow-up [41].

## Second Generation Autologous Chondrocyte Implantation

It is also called matrix-associated autologous chondrocyte implantation (MACI). In this technique, harvested chondrocytes are cultured on a bio absorbable, porcine Type-I/III collagen membrane before implantation into the defect [42]. More cell distribution, performing the technique arthroscopically, preventing the dedifferentiation of chondrocytes. Due to culturing process, avoiding of periosteal graft harvesting are the advantages of MACI technique over the ACI technique.

Anders et al, applied MACI for 22 patients with full-thickness chondral or osteochondral lesions of the talus and concluded that MACI is capable of significant and stable long-term improvement of pain and functional impairment caused by focal full-thickness chondral and osteochondral talar lesions [43]. Dixon et al. evaluated Twenty-seven patients who had talar OCD and treated with MACI and concluded that in patients over 40, the procedure is unlikely to give good pain relief and alternative options should be considered [44]. Magnan et al. evaluated thirty patients with a mean talar lesion size of 2.36 cm2 who were treated with MACI [45]. The mean AOFAS score improved from 36.9 points to 83.9 points at a mean follow-up of forty-five months. But only 50% of the patients returned to their previous sporting activities.

Both ACI and MACI are good treatment options with satisfactory outcomes. No donor-site morbidity occurs with these techniques and periosteum or matrix can also be modified to perfectly fit the defect, therefore, avoiding any potential dead space [46].

## Post-Surgical Rehabilitation

Ideally, the rehabilitation goal is to resume their previous lifestyle/sport activities of patients. Some patients may be encouraged to modify their activity choices, especially if an allograft was used. The goals of the treatment are to help patients keep their pain under control, ensure safe weight bearing and improve strength and range of motion. Patients encouraged to do the exercises as part of an ongoing home program.

Exercises are chosen to help in improving the joint motion and to get the muscles toned and active again. Typically exercises consist of improving the range of motion (ROM), strengthening of the ankle stabilizers (eg, conditioning of the tibialis anterior, peroneus longus, peroneus brevis, and tibialis posterior muscles, and the gastrocnemius and soleus complex) and improving the proprioception (eg, balancing exercises and strengthening of core and gluteus medius muscles) [21].

At first, emphasis is placed on exercising the ankle in positions and movements that don't strain the healing part of the cartilage. As the program evolves, more challenging exercises are chosen to safely advance the knee's strength and function [47].

Immediately after the operation, application of an ankle brace was recommended. For the first 4-6 weeks, walking with non-weight bearing with the use of two crutches is very important. After the removal of the brace, patients encourage to regain weight bearing as tolerated and instruct to perform proprioceptive and complete ankle ROM exercises [48].

For rehabilitation interventions implemented during the immobilization period after surgical fixation, using a removable type of immobilization to enable gentle exercise during this period may reduce activity limitation and pain, and improve ankle range of motion after the treatment period and in the long term. In addition, commencement of weight-bearing during the immobilization period may improve ankle range of motion [22].

Physical therapy improves strength, mobility, and flexibility of the affected joint and is also important to counteract atrophy and contractions associated with immobilization. Physical therapy should improve both active and passive range of motion of the affected joint and should commence once the patient has resolution of pain and evidence of bony healing on imaging [21].

Except of arthroscopic removal of a loose body, patients are instructed to avoid putting too much weight on their foot when standing or walking for up to six weeks after operation. Weight bearing is usually restricted for up to four months after transplant procedures. Patients are strongly advised to follow the recommendations about how much weight is safe. They may require a walker or pair of crutches for up to six weeks to avoid putting too much pressure on the joint when they are up. Return to sporting activities usually permit 12 weeks after the operation [47].

A well-motivated and compliant patient will resume to daily living/professional/sport activities earlier when compared to patients without a strong motivation.

## Overview

The surgical treatment of osteochondral lesions of the talus remains controversial. Depending on lesion size and patient's age drilling and micro fracture, bone marrow stimulation, autologous chondrocyte transplantation or cell-based technologies can be used. Many of these methods have good clinical results with short-term follow-up. But arthritis longer-term

follow-up results with well-designed prospective randomized controlled trials are needed to prevent joints from degeneration.

### References

- 1. Hepple S, Winson IG, Glew D: Osteochondral lesions of the talus: a revised classification, Foot Ankle Int. 1999;20(12):789-93.
- 2. Sorrento DL, Mlodzienski A. Incidence of lateral talar dome lesions in SER IV ankle fractures. J Foot Ankle Surg. 2000;39(6):354-8.
- 3. Schafer DB. Cartilage repair of the talus. Foot Ankle Clin. 2003;8(4):739-49.
- 4. Brunes J, Behrens P: Etiological and pathophysiologic aspects of osteochondrosis dissecans. Arthroskopie. 1998;11:166.
- 5. Ahmad J, Raikin S. Current concepts review: talar fractures. Foot Ankle Int. 2006;27(6):475-82.
- 6. Mankin HJ. The response of articular cartilage to mechanical injury. J Bone Joint Surg Am. 1982;64(3):460-6.
- 7. Shapiro F, Koide S, Glimcher MJ. Cell origin and differentiation in the repair of full-thickness defects of articular cartilage. J Bone Joint Surg Am. 1993;75(4):532-53.
- 8. Scranton PE, McDermott JE. Treatment of type V osteochondral lesion of the talus with ipsilateral knee osteochondral autografts. Foot Ankle Int 2001;22(5):380-4.
- 9. Takao M, Ochi M, Uchio Y, Naito K, Kono T, Oae K. Osteochondral lesions of the talar dome associated with trauma. Arthroscopy. 2003;19(10):1061-7
- 10. Birk GT, DeLee JC. Osteochondral injuries: clinical findings. Clin Sports Med. 2001;20(2):279-86.
- 11. Canale ST, Kelly FB Jr. Fractures of the neck of the talus: long-term evaluation of seventy-one cases. JBJS. 1978;60(2):143-56.
- 12. Berndt AL, Harty M. Transchondral fractures (Osteochon- dritis dissecans) of the talus. JBJS. 1959;41-A:988-1020.
- 13. Taranow WS, Bisignani GA, Towers JD, Conti SF. Retrograde drilling of osteochondral lesions of the medial talar dome. Foot Ankle Int. 1999;20(8):474-80.
- 14. Anderson IF, Crichton KJ, Grattan-Smith T, Cooper RA, Brazier D. Osteochondral fractures of the dome of the talus. J Bone Joint Surg Am. 1989;71(8):1143-52.
- 15. Bencardino J, Rosenberg ZS, Delfaut E. MR imaging in sports injuries of the foot and ankle. Magn Reson Imaging Clin N Am. 1999;7(1):131-49.
- 16. Mintz DN, Tashjian GS, Connell DA, Deland JT, O'Malley M, Potter HG. Osteochondral lesions of the talus: a new magnetic resonance grading system with arthroscopic correlation. Arthroscopy. 2003;19(4):353-9.
- 17. De Smet AA, Fisher DR, Burnstein MI, Graf BK, Lange RH. Value of MR imaging in staging osteochondral lesions of the talus (osteochondritis dissecans): results in 14 patients. AJR Am J Roentgenol. 1990;154(3):555-8.

- 18. Tol JL, Struijs PA, Bossuyt PM, Verhagen RA, van Dijk CN. Treatment strategies in osteochondral defects of the talar dome: a systematic review. Foot Ankle Int. 2000;21(2):119-26.
- 19. Lin CW, Donkers NA, Refshauge KM, Beckenkamp PR, Khera K, Moseley AM. Rehabilitation for ankle fractures in adults. Cochrane Database Syst Rev. 2012:11:CD005595.
- 20. Schachter AK, Chen AL, Reddy PD, Tejwani NC. Osteochondral lesions of the talus. JAAOS. 2005;13:152-8.
- 21. Albert CH, Brian SH. Management of osteochondritis dissecans (OCD). In: UpToDate, Richard GB, ed, UpTo Date, Waltham, MA. (Accessed on January 26, 2015.)
- 22. Maartje Zengerink M, van Dijk CN. Meta-analysis on Therapy. In: van Dijk CN, Kennedy JG, eds, Talar Osteochondral Defects. Diagnosis, Planning, Treatment, and Rehabilitation. Luxembourg: Springer. 2014;83-94.
- 23. Deland JT, Young K. Medial Approaches to Osteochondral Lesions of the Talus Without Medial Malleolar Osteotomy. San Diego: American Orthopaedic Foot and Ankle Society; 2001:75.
- 24. Frost A, Roach R. Osteochondral injuries of the foot and ankle. Sports Med Arthrosc. 2009;17(2):87-93.
- 25. Kumai T, Takakura Y, Higashiyama I, Tamai S. Arthroscopic drilling for the treatment of osteochondral lesions of the talus. J Bone Joint Surg Am. 1999;81(9):1229-35.
- 26. Saxena A, Eakin C. Articular talar injuries in athletes: results of microfracture and autogenous bone graft. Am J Sports Med. 2007;35(10):1680-7.
- 27. Chuckpaiwong B, Berkson EM, Theodore GE. Microfracture for osteochondral lesions of the ankle: outcome analysis and outcome predictors of 105 cases. Arthroscopy. 2008;24(1):106-12.
- 28. Kolker D, Murray M, Wilson M. Osteochondral defects of the talus treated with autologous bone grafting. J Bone Joint Surg Br. 2004;86(4):521-6.
- 29. Zengerink M, Struijs PA, Tol JL, van Dijk CN. Treatment of osteochondral lesions of the talus: a systematic review [Review]. Knee Surg Sports Traumatol Arthrosc. 2010;18(2):238-46.
- 30. 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.
- 31. Van Buecken K, Barrack RL, Alexander AH, Ertl JP. Arthroscopic treatment of transchondral talar dome fractures. Am J Sports Med. 1989;17(3):350-5, discussion:355-6.
- 32. Lee KB, Bai LB, Chung JY, Seon JK. Arthroscopic microfracture for osteochondral lesions of the talus. Knee Surg Sports Traumatol Arthrosc. 2010;18(2):247-53.
- 33. Hunt SA, Sherman O. Arthroscopic treatment of osteochondral lesions of the talus with correlation of outcome scoring systems. Arthroscopy. 2003;19(4):360-7.

- 34. Cuttica DJ, Shockley JA, Hyer CF, Berlet GC. Correlation of MRI edema and clinical outcomes following microfracture of osteochondral lesions of the talus. Foot Ankle Spec. 2011;4(5):274-9.
- 35. Robinson DE, Winson IG, Harries WJ, Kelly AJ. Arthroscopic treatment of osteochondral lesions of the talus. J Bone Joint Surg Br. 2003;85(7):989-93.
- 36. Imhoff AB, Paul J, Ottinger B, W"ortler K, L"ammle L, Spang J, Hinterwimmer S. Osteochondral transplantation of the talus: long-term clinical and magnetic resonance imaging evaluation. Am J Sports Med. 2011;39(7):1487-93.
- 37. Hangody L, V'as'arhelyi G, Hangody LR, S"uk"osd Z, Tibay G, Bartha L, Bod'o G. Autologous osteochondral grafting-technique and long-term results. Injury. 2008;39(Suppl 1):S32-9.
- 38. Kennedy JG, Murawski CD. The treatment of osteochondral lesions of the talus with autologous osteochondral transplantation and bone marrow aspirate concentrate: surgical technique. Cartilage. 2011;2:327-36.
- 39. Murawski C, Kennedy JG. Operative Treatment of Osteochondral Lesions of the Talus. J Bone Joint Surg Am. 2013;95(11):1045-54.
- 40. Kwak SK, Kern BS, Ferkel RD, Chan KW, Kasraeian S, Applegate GR. Autologous chondrocyte implantation of the ankle: 2- to 10-year results. Am J Sports Med. 2014;42(9):2156-64.
- 41. Giannini S, Buda R, Ruffilli A, Cavallo M, Pagliazzi G, Bulzamini MC, Desando G, Luciani D, Vannini F. Arthroscopic autologous chondrocyte implantation in the ankle joint. Knee Surg Sports Traumatol Arthrosc. 2014;22(6):1311-9.
- 42. Jones CW, Willers C, Keogh A, Smolinski D, Fick D, Yates PJ, Kirk TB, Zheng MH. Matrix-induced autologous chondrocyte implantation in sheep: objective assessments including confocal arthroscopy. J Orthop Res. 2008; 26(3):292-303.
- 43. Anders S, Goetz J, Schubert T, Grifka J, Schaumburger J. Treatment of deep articular talus lesions by matrix associated autologous chondrocyte implantation--results at five years. Int Orthop. 2012;36(11):2279-85.
- 44. Dixon S, Harvey L, Baddour E, Janes G, Hardisty G. Functional outcome of matrix-associated autologous chondrocyte implantation in the ankle. Foot Ankle Int. 2011;32(4):368-74.
- 45. Magnan B, Samaila E, Bondi M, Vecchini E, Micheloni GM, Bartolozzi P. Three-dimensional matrix-induced autologous chondrocytes implantation for osteochondral lesions of the talus: midterm results. Adv Orthop. 2012;2012:942174.
- 46. Giannini S, Buda R, Grigolo B, Vannini F. Autologous chondrocyte transplantation in osteochondral lesions of the ankle joint. Foot Ankle Int. 2001;22(6):513-7.
- 47. Ventura A, Terzaghi C, Legnani C, Borgo E. Treatment of post-traumatic osteochondral lesions of the talus: a four-step approach. Knee Surgery, Sports Traumatology, Arthroscopy. 2013;21(6):1245-50.
- 48. A Patient's Guide to osteochondritis dissecans of the Talus. http://www.eorthopod.com/osteochondritis-dissecans-of-the-talus/topic/39. Access date 05.02.2015