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Osteochondral lesion of the distal tibial plafond in an adolescent soccer player: a case report

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Introduction: *Osteochondral lesions of the tibial plafond account for approximately 2.6% of osteochondral lesions in the ankle. There are few cases describing this lesion in the literature, with little information on mechanism of injury, history/physical findings or recommendations for management.*

Case Presentation: *A 17-year-old male competitive soccer player presented with a 6-7 month history of medial ankle pain after an inversion sprain. He presented with locking and giving way of the ankle with weight-bearing and pushing off the foot to the contralateral side. Radiographs were negative for fracture or osteochondral involvement. Magnetic resonance imaging revealed an osteochondral lesion of the tibial plafond with no injury to the talar dome.*

Summary: *This case discusses the clinical presentation, imaging findings, management and outcomes of this osteochondral lesion of the distal tibial plafond.*

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KEY WORDS: chiropractic, osteochondral lesion, tibial plafond, athletic injury

Introduction : *Les lésions ostéochondrales du plateau tibial comptent pour environ 2,6 % des lésions ostéochondrales de la cheville. Dans la littérature, on trouve peu de cas de ce type de lésion et peu de renseignements sur le mécanisme de la blessure, les antécédents, les observations physiques et les recommandations thérapeutiques.*

Présentation du cas : *Un joueur de soccer de haut niveau de 17 ans s'est plaint d'une douleur à la cheville médiane apparue il y a 6 ou 7 mois à la suite d'une entorse en inversion. On a observé un blocage et une instabilité de la cheville en position sur l'articulation portante et un écartement du pied controlatéral. Les radiographies n'ont révélé aucune fracture ni aucune atteinte ostéochondrale. L'examen par imagerie par résonance magnétique a révélé la présence d'une lésion ostéochondrale au dôme talien.*

Résumé : *Le présent cas présente le tableau clinique, les variations observables, la prise en charge et l'évolution de cette lésion ostéochondrale du plateau tibial distal.*

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MOTS CLÉS : chiropratique, lésion ostéochondrale, plateau tibial, blessure sportive

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Introduction

The majority of osteochondral lesions (OCLs) of the ankle occur in the talus.[1,2] Approximately 2.6% of isolated OCLs of the ankle occur in the distal tibial plafond (OCLTP), equivalent to a 14:1 or 20:1 ratio of talar:tibial OCLs in the literature.[3–5] There is no clear explanation why talar OCLs are more common than distal tibial plafond lesions, however, it has been suggested that the tibial cartilage is stiffer than talar cartilage making it less susceptible to damage and that the concave shape of the distal tibial surface may predispose it to less stress than the convex talar dome.[2,5,6] Approximately 16-23% of OCLs in the ankle occur in both the talus and the tibia, and while the talus is more susceptible to osteochondral injury, only approximately 2.6% of OCLTP demonstrate adjacent injury to the talus (kissing lesion).[4,5] In addition, there appears to be no significant difference in functional outcome between kissing lesions and isolated OCLTP.[7]

Currently, there is no systematic classification specific to OCLTP, however, both Anderson's and Elias' classification systems have been used to describe the degree and location of OCLs, respectively.[5,8–11] These classifications have typically been used to characterize OCLs of the talus (OCLT), describing the degree of osteochondral involvement and the location of the lesion.[5,8–11] In 1959, Berndt and Harty described a classification system for OCLs on plain radiograph, which Anderson referenced to create magnetic resonance imaging (MRI) stages and associated findings (Table 1).[8,10] Elias *et al*.[11] originally proposed an anatomical nine-zone grid scheme to the articular surface of the talus, to more easily describe the location of an OCL, and have also applied this to the articular surface of the distal tibial plafond (Figure 1).

Few cases and retrospective studies describe OCLTP with little consensus on mechanism of injury, description of symptomatology, physical findings or recommendations for management. The purpose of this case report is to describe the history and physical findings of this rare injury that has not been reported in chiropractic and to emphasize the specific findings that led to the diagnosis of OCLTP. In addition, we propose the use of the Anderson[9] classification system to grade the severity of OCLTP in a similar fashion as is used for OCLT.

Table 1.
Summary of radiograph and MRI classification systems of OCLs, modified from Dipaola et al.[10]

	Berndt & Harty (Radiograph)	**Anderson (MRI)**
Stage 1	• Compression lesion. • No visible fragment.	• Thickening of articular cartilage. • Low signal changes.
Stage 2	• Fragment attached.	• Articular cartilage breached. • Low signal rim behind fragment indicating fibrous attachment.
Stage 3	• Nondisplaced fragment without attachment.	• Articular cartilage breeched. • High signal changes behind fragment indicating synovial fluid between fragment and underlying subchondral bone.
Stage 4	• Displaced fragment.	• Loose body.

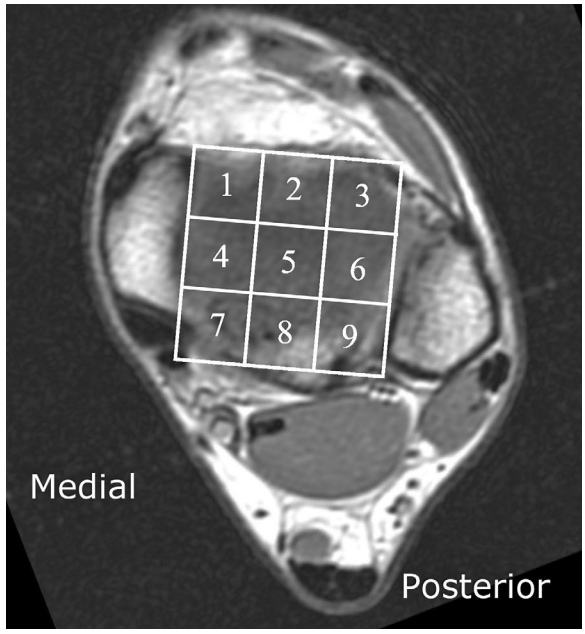

Figure 1.
Axial PD MRI of current patient's OCLTP with overlay of Elias et al's localization grid. This grid indicates the OCL is located in zones 8 and 9.

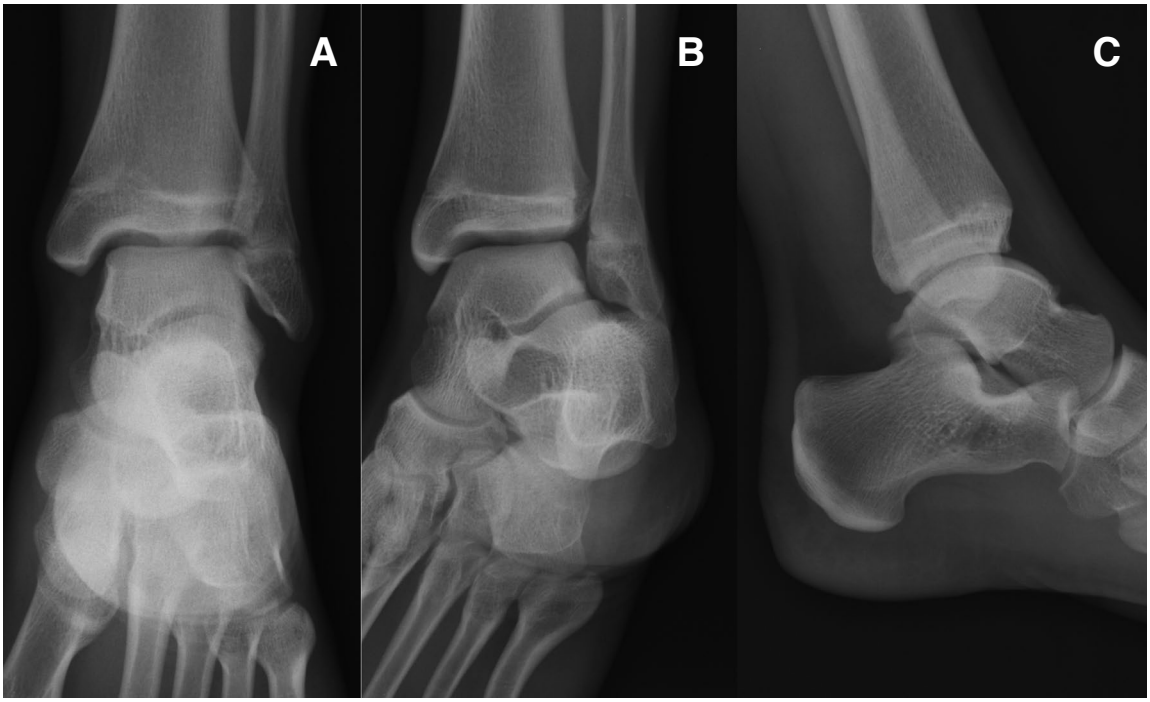

Figure 2. *Radiographs taken on November 23, 2016 reveal no finding indicating OCL in the ankle. (A) AP ankle; (B) Medial oblique ankle; (C) Lateral ankle*

Case Presentation

History

A 17-year-old competitive male soccer player presented to a Sports Specialist chiropractor (Fellow of the Royal College of Chiropractic Sports Sciences (Canada)) with chronic left medial ankle pain of six to seven months duration. It began after an inversion ankle sprain for which he received no medical assessment, imaging or structured rehabilitation. However, the patient reported seeing a physiotherapist who was "working on his cuboid" intermittently. The patient reported locking and giving way of the ankle, especially with weight-bearing on that side and when pushing off the foot to the contralateral side. His pain was aggravated in soccer training and competition. He reported taking two weeks' rest from his regular schedule, which involved training four to five times per week, but the pain returned after resuming his normal training schedule. Additional complaints included the plantar aspect of the right forefoot and chronic bilateral hip stiffness. His Lower Extremity Functional Scale (LEFS) was 51/80, with the most difficulty on sharp turns while running, hopping, and running on even or uneven ground.

Physical Examination

Upon observation, there was no evidence of ongoing soft tissue swelling or other abnormalities. Walking did not cause pain but single leg squats, hopping or weight-bearing with inversion reproduced the chief complaint. Palpation revealed mild to moderate tenderness over the anterior talofibular ligament, and restrictions in the subtalar and navicular joints. Knee-to-wall test revealed a mild limitation in dorsiflexion of the left ankle, while all other ranges of motion and muscle strength testing were within normal limits.

Diagnosis and Management

A working diagnosis of chronic ankle impingement syndrome was provided with a differential diagnosis of OCL. It was agreed that manual care, including ankle mobilization, adjustment, and mobilization with movement would be performed along with soft tissue therapy and balance exercises. Radiographs were also ordered to investigate the possibility of an OCL.

Radiographs of the left ankle were unremarkable with no evidence of an OCL (Figure 2). Four treatments over a one-month period were provided with symptomatic relief and increased mobility in the ankle, demonstrated with

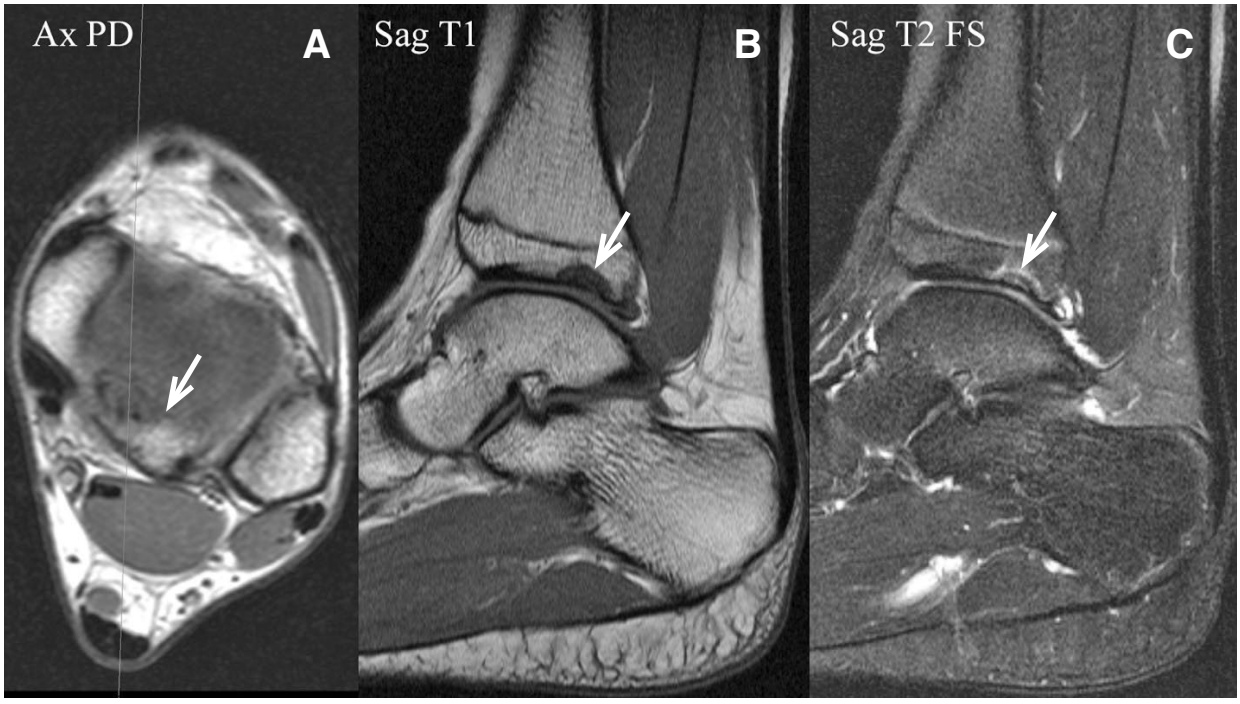

Figure 3.

Axial PD (A), sagittal T1 (B) and T2 fat-sat (C) sequences of the right ankle 6 weeks after initial visit reveal a cup-shaped OCL in the posteromedial tibial plafond demarcated completely by a thin hypointense T1 and T2 line and minimal marrow edema (hypointense T1 and hyperintense T2 FS signals) deep to the lesion. Arrows indicate OCL. (A) Axial MRI Proton Density; (B) Sagittal MRI T1-weighted; (C) Sagittal MRI Fat-suppressed

a knee-to-wall test within normal limits and reduction of restrictions in the midfoot. The patient reported that treatment was helping, however, activities were being modified as part of the treatment and this coincided with the holiday season with an associated decrease in training and competition volume. Following a trial of practice sessions, the symptoms of locking and giving way returned.

The patient was instructed to rest and refrain from soccer and high-impact exercise while waiting to see a sports physician to provide further management options and possibly an orthopaedic referral. One month later, the sport physician agreed with continued rest and made recommendations for an air cast for bracing and immobilization. An MRI was obtained six weeks after the patient initially presented to the chiropractor in January 2017 (Figure 3). The talar dome cartilage was preserved with no defect or OCL. However, a 1.1 x 1.1 cm shallow cup-shaped OCL was present at the posteromedial aspect of the tibial plafond demarcated by a thin crescent-shaped hypointense T1 and T2 line with minimal marrow edema in the osteochondral fragment and surrounding medullary space. This was associated with a small tibiotalar joint effusion. Resolution of the images did not allow for assessment of the integrity of overlying cartilage. There were no other abnormalities of the tibiotalar joint. No articular abnormality was observed in the hindfoot and midfoot. There was no synovitis or ganglion cyst.

The impression provided by the radiologist was an existing completely detached OCL at the posteromedial aspect of the tibial plafond without displacement and a small joint effusion. A follow-up CT scan was ordered to assess for further changes in the defect and to assess for loose bodies, which confirmed the MRI findings (images unavailable). The patient continued to experience symptoms when resuming normal soccer activities despite the bracing and reduced activities. The patient was referred

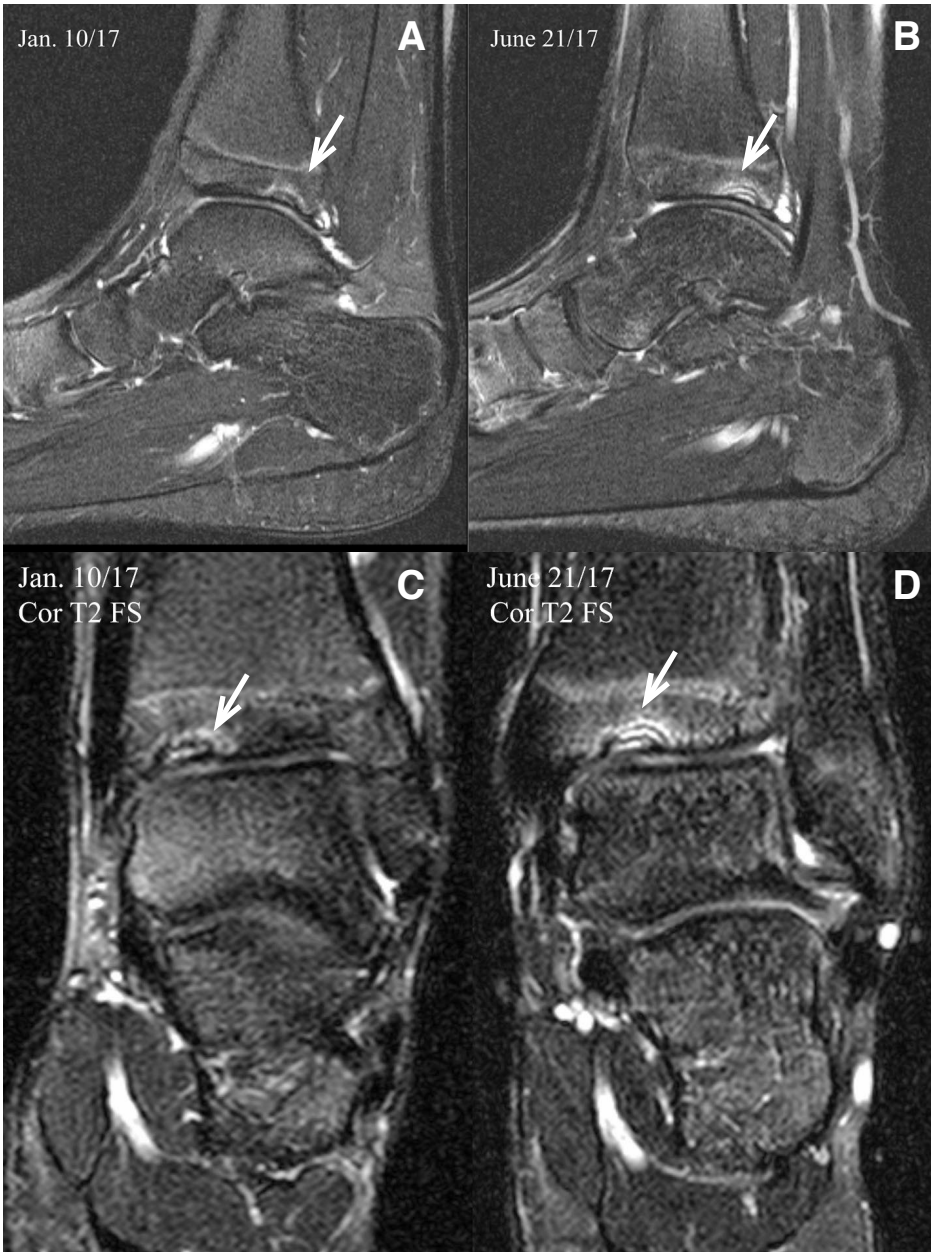

Figure 4.
Comparison of sagittal (A, B) and coronal (C, D) T2 fat-saturation images of the left ankle showing separation of the osteochondral fragment from the posterior tibial plafond with a fluid cleft and increased surrounding marrow edema at follow-up (June 21, 2017).

for an orthopeadic consult and follow up MRI. The second MRI in June 2017 demonstrated separation of the osteochondral fragment from the posterior tibial plafond with a fluid cleft and increased surrounding marrow edema, suggesting progression of the lesion since previous imaging (Figure 4). Most recently the patient was referred to an orthopaedic foot and ankle specialist.

Discussion

The current literature is unclear with regards to a specific mechanism of injury that may lead to an OCLTP. Even in the larger pool of literature regarding OCLTs, there is no consensus on mechanism of injury, although the majority occur due to trauma.[12] It has been suggested that OCLTs may occur by three types of trauma; compaction, shearing

or avulsion.[13] However, there are also reports of atraumatic causes.[13–15]

Due to the lack of literature characterizing OCLTP, we believe there is reason to highlight findings relating to OCLT, the reciprocal and more common OCL in the ankle. OCLT are often subtle, and may cause little to no dysfunction.[13] However, if symptomatic, pain is often described as non-specific, deep ankle pain that occurs with weightbearing.[12,13,16] As such, function is often affected especially during weight-bearing or high impact activities, such as sport participation.[12,13,16] Further findings include limited range of motion, stiffness, catching, locking or generalized swelling.[12,13,16]

There are no specific physical examination tests or findings that can accurately assess or diagnose OCLT.[16] However, the physical examination should include assessment of range of motion, orthopedic tests, and palpation.[16] The patient may complain of tenderness or pain in the ankle that is poorly localized and may not always correspond with the location of the OCL.[16] Assessment of hindfoot malalignment, joint flexibility and laxity is also recommended.[16] In a retrospective study of 41 OCLT cases, the authors found associations with pes planus in 42%, lateral ankle instability in 32% and arthritis in 27% of ankles.[13] Therefore, there is value in performing anterior drawer and inversion maneuvers to identify any concurrent lateral ankle instability or associated factors.[16]

OCLTP are difficult to identify by conventional radiographs, and can be missed in up to 50% of cases.[16] Therefore diagnosis is often delayed or missed, where in some cases surgery is delayed on average by 22 months.[4] It is important to have a high level of suspicion in patients with persistent ankle pain with a history of ankle injuries.[16] If visible, they often appear radiolucent, suggested by a loss of sharp cortical line and are best seen on AP images. They are generally less evident on lateral images.[3] Due to the difficulty in diagnosing OCL via radiographs, we suggest follow-up MRI or computed tomography (CT) in cases involving a history of ankle injury with persistent limitations or no resolution at six to eight weeks.

CT has an excellent ability to detect OCLT, with a sensitivity of 0.81 and specificity of 0.99, however it lacks the ability to assess cartilage involvement[16], or bone marrow edema. MRI has a sensitivity and specificity of 0.97 and is the recommended imaging technique to assess for OCLs.[3,16] OCLs can be identified by low signal intensity on T1-weighted images and high signal intensity on T2-weighted images with adjacent bone marrow edema, which is evident in this case.[3] In the current case, a region of subchondral bony sclerosis with surrounding edema and small joint effusion were identified by MRI which led to the diagnosis of OCLTP.

Elias *et al*. proposed the use of a localization scheme to classify OCLTPs, standardizing how they are reported on the joint surface.[5,11] As such, the location of the OCLTP in this case is located in zones 8 and 9, or the posteromedial aspect of the tibial plafond. In addition, we propose that the classification system by Anderson *et al*.[9] be applied to characterize the severity of OCLTPs imaged by MRI going forward. As per the MRI findings, the OCLTP in this case is a stage III lesion. Grading of a lesion may be important in determining the management of the patient, where first line management of grade I, II, and small grade III lesions can be treated conservatively, and grade III or IV lesions are treated surgically.[13,15,16] However, literature on OCLT is unclear as to whether grading systems lead to changes in clinical decision-making or whether there is any correlation between grade of the lesion and clinical outcomes.[13,16]

There is no consensus on how OCLTPs should be managed. Conservative care in one study suggests non-weight bearing immobilization in a controlled ankle movement boot for 3-4 weeks, dorsi- and plantar-flexion exercises, and progression to weight-bearing by 10% every day for 4 weeks.[7] When conservative management fails, arthroscopic surgery is often performed. The average return to activity ranges from 20.9 months in one study[2], with excellent or good results in 82% of patients at 44 months in another[4], and significant improvement in foot outcome scales and quality of life at 44 months in a third[7].

Due to the reciprocal nature of the talus and the distal tibial plafond, one can hypothesize that similar treatments may be effective in their management, although composition and anatomical differences may result in different outcomes. In asymptomatic or mildly symptomatic OCLT in pediatric populations, resolution may occur without treatment, however spontaneous healing in adults in uncommon.[14] The goal of treatment for OCLs are to relieve symptoms and swelling, and improve function by unloading the damaged cartilage; allowing edema to decrease and prevent necrosis.[13] Conservative management of OCLT involves immobilization, non-weight bearing

and activity modification, with or without non-steroidal anti-inflammatory drugs (NSAIDs) for approximately 6 weeks.[13,15] However, this management can continue for up to 5 months.[16] Progressive weight-bearing and physical therapy follow immobilization, including but not limited to closed chain balance and proprioception exercises, peroneal strengthening, and neuromuscular control of the ankle.[13,15] Ankle supports, such as tape or orthoses can be used in the short-term management, but have limited usefulness in the long-term treatment.[15] Success of the intervention is determined by symptomatic complaints rather than size of the lesion, as symptoms may persist with conservative treatment even if the size of the lesion decreases, and vice versa.[14,15]

The success rate of non-operative management is only approximately 45-53%, with success of surgical intervention ranging from 54-85% depending on the type of intervention.[13,14] Reparative procedures are generally used for lesions <15 mm in diameter or 150 mm^2 in area, whereas replacement procedures are used for larger lesions or failed repairs.[16] A systematic review by Zengerink *et al*.[12] in 2010 identified debridement and bone marrow stimulation (BMS) as the best surgical treatment options, with an 85% success rate. Autologous chondrocyte implantation had a 76% success rate, and while osteochondral transplantation had an 86% success rate, there was a high incidence (36%) of knee complaints associated post-operatively.[12] Overall, treatment is determined by the size and location of the lesion, as well as the activity level and preference of the patient.[14]

There are limited studies investigating prognostic factors for OCLTP. In a study by Ross *et al*. they found that age was negatively correlated with pre- to post-operative changes in function measured by the SF-12, where older age was associated with a worse functional outcome.[7,17] There was no significant association with the location or size of the lesion.[7] In addition, they found that score on the magnetic resonance observation of cartilage repair tissue was not associated with functional outcomes.[7] The literature regarding the prognosis of talar OCLs is equally limited. There is no consensus regarding location of the lesion and prognosis, except for uncontained lesions of the talar shoulder which have worse clinical outcomes after BMS.[14]

The strength of this case report is the presentation of a rare condition to a chiropractor, and the description of history and physical findings leading to further imaging of this patient. Additionally, multiple images are available to identify and follow-up on the progression of this lesion, especially with lack of compliance to reduced weight-bearing and aggravating activities. The limitations include the lack of literature regarding OCLTP, and therefore the application of OCL of the talus to this case. Unfortunately, as this case is ongoing, full management and resolution of symptoms is not available.

Summary

In summary, we presented a case of a 17-year old male competitive soccer player who sustained an inversion ankle sprain. He presented to the clinic six to seven months later complaining of non-specific ankle pain, aggravated by weight-bearing. He also described catching and locking during sporting activities. Plain radiographs were negative, while MRI revealed a grade III OCL of the distal tibial plafond in zones 8 and 9. The athlete in this case was managed conservatively, including rest, immobilization and modified activities, in-line with the current recommendations for the management of OCLT. While symptomatic relief occurred as a result of chiropractic treatment, returning to full activities and competition remained problematic even after medical referral.

References

1. Bauer KL, Polousky JD. Management of osteochondritis dissecans lesions of the knee, elbow and ankle. Clin Sports Med. 2017;36(3):469–487.
2. Cuttica DJ, Smith WB, Hyer CF, Philbin TM, Berlet GC. Arthroscopic treatment of osteochondral lesions of the tibial plafond. Foot Ankle Int. 2012;33(8):662–668.
3. Bui-Mansfield LT, Kline M, Chew FS, Rogers LF, Lenchik L. Osteochondritis dissecans of the tibial plafond: Imaging characteristics and a review of the literature. Am J Roentgenol. 2000; January:167–172.
4. Mologne TS, Ferkel RD. Arthroscopic treatment of osteochondral lesions of the distal tibia. Foot Ankle Int. 2007;28(8):865–872.
5. 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):524–529.
6. Athanasiou KA, Niederauer GG, Schenck RC. Biomechanical topography of human ankle cartilage. Ann Biomed Eng. 1995;23(5):697–704.
7. Ross KA, Hannon CP, Deyer TW, Smyth NA, Hogan M,

Do HT, *et al*. Lesions of the distal tibial plafond. 2014;1708–1715.
8. You JY, Lee GY, Lee JW, Lee E, Kang HS. An osteochondral lesion of the distal tibia and fibula in patients with an osteochondral lesion of the talus on MRI: Prevalence, location, and concomitant ligament and tendon injuries. Am J Roentgenol. 2016;206(2):366–372.
9. 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 Sep;71(8):1143-1152.
10. Dipaola JD, Nelson DW, Colville MR. Characterizing osteochondral lesions by magnetic resonance imaging. Arthroscopy. 1991;7(1):101–104.
11. 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):154–161.
12. Zengerink M, Struijs PAA, Tol JL, van Dijk CN. Treatment of osteochondral lesions of the talus: A systematic review. Knee Surgery, Sport Traumatol Arthrosc. 2010;18(2):238–246.
13. Badekas T, Takvorian M, Souras N. Treatment principles for osteochondral lesions in foot and ankle. Int Orthop. 2013;37(9):1697–1706.
14. Savage-Elliott I, Ross KA, Smyth NA, Murawski CD, Kennedy JG. A current concepts review and evidence-based treatment paradigm. Foot Ankle Spec. 2014;7(5):414–422.
15. Shimozono Y, Yasui Y, Ross AW, Kennedy JG. Osteochondral lesions of the talus in the athlete: up to date review. Curr Rev Musculoskelet Med. 2017;10(1):131-140.
16. Gianakos AL, Yasui Y, Hannon CP, Kennedy JG. Current management of talar osteochondral lesions. World J Orthop. 2017;8(1):12.
17. Bauer M, Jonsson K, Linden B. Osteochondritis dissecans of the ankle. J Bone Joint Surg Br. 1987;69(1):93-96.