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Cartilage Pathology With Concomitant Ankle Instability

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Ankle instability and chondral pathology commonly present together. This review discusses the current evidence for the etiology of chondral lesions, the association between ankle instability and chondral injury, and the optimal method of managing a patient with symptomatic chondral pathology (osteochondral lesions and tibiotalar osteoarthritis) in the setting of an unstable ankle. The literature pool is weak for recommendations on the management of osteophytes and osteoarthritis with ankle instability. Multiple factors do compound dual treatment of osteochondral lesions and instability; however, concomitant surgery addressing both pathologies has been shown to have outcomes comparable to osteochondral surgery alone with similar complication rates.
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Introduction

The recent advances and popularization of ankle arthroscopy has brought the opportunity to treat a broader and forever expanding array of foot and ankle conditions. Scientific literature is not yet available, however, to specifically support certain procedures or combinations of procedures, particularly those that are new and innovative.[1]

The outcomes for ankle instability surgery are well documented and tend to demonstrate good-to-excellent long-term results.[2,3] Equivalent outcome scores are not universally achieved with surgery for osteochondral lesions (OCLs) but remain satisfactory.[4] To date, there is no consensus on the optimal method of managing a patient with the combination of a cartilage lesion and an unstable ankle.[5-9] This article reviews the background to the problem and discuss the pertinent current literature.

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Ankle Instability

Ankle instability is a subjective sensation of the ankle "giving way." It can occur in lateral, medial, torsional, and translational ankle movements. The most common cause is an inversion ankle injury with an associated tear to the lateral ligament complex leading to chronic lateral ligament laxity.[10-13] Ankle sprains occur in an estimated 628,000 Americans every year.[14] Current literature predicts 10%-20% of these patients will develop chronic lateral instability symptoms,[13,15] and 20%-40% are likely to develop chronic pain.[16] In athletes, the figure is substantially worse, with 21%-80% reporting chronic symptoms after an ankle sprain.[13,17,18] The 2 most common symptoms after an ankle sprain in a study of 380 athletes from Hong Kong[18] were pain (30%) and instability (20%). The pathomechanics of ankle instability has been well described by Bonnel et al.[19] The cause for the chronic pain, however, is widely debated. OCLs, osteoarthritis, soft tissue impingement, tendinopathy, and loose bodies are all commonly seen in patients with chronic instability.[9]

Etiology of OCLs

OCL is a broad term that encompasses any injury to the chondral surface. Such injuries include the historical term osteochondritis dessicans as well as osteochondral fracture and

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osteochondral defect. They were initially described by Konig.[20] OCLs of the ankle joint typically present as pain, stiffness, and swelling in the young adult.[21]

Up to 50% of patients have a cartilage injury or a bone bruise after an ankle sprain.[22-24] Does this chondral injury, however, lead to the development of an OCL? A review by McCollum et al[25] noted that the natural history of most bone bruises is benign, but the integrity of the subchondral bone plate is critical to the resolution or possible propagation to an OCL. Previous authors have noted many OCLs develop without any history of trauma.[26-28] In a review of 582 patients with talar OCLs,[28] 66% had a history of ankle trauma, whereas 34% did not. This is similar to the 64% traumatic rate seen in the inaugural OCL series by Wagoner and Cohn.[29] In the study by Tol et al,[28] 56% of the lesions were found on the medial talar dome and 44% on the lateral, with trauma implicated in 94% and 62% of cases, respectively. The literature demonstrates a clear association between OCLs and ankle injuries. The etiology of OCL in patients without a history of trauma (spontaneous), however, remains uncertain.[30] The repetitive microtrauma seen with ankle instability has been postulated to play a role.[31,32]

The evidence indicates that although there does appear to be a correlation between ankle injury and chondral injury, there is a distinct patient group who will have no history of trauma.

Ankle Instability and Chondral Injury

Several studies have noted an association between ankle instability and chondral injury albeit with a wide variation in rates from 23%-95%.[7,8,33] Taga et al[34] demonstrated chondral lesions in 95% of patients undergoing lateral ligament reconstruction. Of these the medial tibial plafond was the most common site (33%), which also saw most higher grade lesions. The suggestion that the medial part of the tibiotalar joint is more susceptible to injury because of instability was reaffirmed by others.[9,33,35] However, Gregush and Ferkel[6] noted lateral lesions (61%) were more common in their study of 31 ankles undergoing lateral ligament reconstruction. Biomechanical data support the increased occurrence of pathology seen on the medial side. For example, lateral ankle ligament disruption has been shown to increase both anterior translation and internal rotation of the talus.[36-38] Theoretically, this should cause medial abutment and abnormal cartilage strain between the talus and the medial malleolus.[39] Bischof et al[40] used a 3-dimensional magnetic resonance imaging model and biplanar fluoroscopy to evaluate in vivo cartilage contact strains in 7 patients with lateral ankle instability. They demonstrated that with an unstable ankle the peak strain increased (21%-29% of body weight) and the location shifted in both anterior (15.5 mm) and medial (12.9 mm) directions.

Taga et al[34] demonstrated a link between the chronicity of the instability and the risk of chondral injury, suggesting a causative association. This prompted the authors to recommend surgical repair of the lateral ligament complex to avoid further damage. Valderrabano et al[41] confirmed the high rate of cartilage lesions (81%) seen at long-term (12 years) follow-up of unstable ankles thereby adding weight to the prophylactic fixation theory of Taga et al.[34] The severity of chondral lesions and the duration of instability were questioned by both Sugimoto et al[42] and Okuda et al.[33] Under arthroscopic examination, Sugimoto et al[42] found no correlation ($P = 0.16$) between the duration of instability symptoms and severity of chondral damage. With no power analysis and a clear trend between increasing severity and duration of symptoms (mean of 41 weeks for the least severe chondral damage and 104 for the most severe), one could question the sensitivity of this study and thus the validity of their conclusion. Okuda et al[33] also found no correlation between instability and severity of chondral lesions in a study of 30 ankles with a mean of 70 months from injury to surgery. However, the time interval between the primary injury and surgery was more than 3 years longer for the patient group found with OCLs than those without. There is stronger evidence supporting the association between the duration of ankle instability and presence of chondral injury.

Does the chondral location or association with trauma affect the outcome in the unstable ankle? Okuda et al[33] demonstrated that focal chondral lesions associated with trauma and instability can, in some instances, do well with ankle stabilization alone. However, whether these OCLs were traumatic or spontaneous in the setting of an unrelated ankle injury is impossible to say. Indeed the role and timing of prophylactic instability surgery is as yet uncertain.

The Management of Ankle Instability With an OCL

Independent of each other, the documented outcomes of surgery for OCLs and instability are different. The outcomes for the many described lateral ankle instability procedures are consistently 90%-95% good to excellent.[43] The same however has not been reported for OCL surgery.[44] The longest follow-up to date (12 years) of talar OCL treated by bone marrow stimulation[21] showed a subjective outcome of "good" to "excellent" in 74%-78% of 50 patients.

To date, no studies have investigated the outcome of staged operations. There are however 5 studies[5-10] that assessed patient outcomes after concomitant ankle instability and OCL treatment (Table). In total, these studies represent data from 155 ankles. They all share similar protocols for the management of the OCL (bone marrow stimulation), but variable techniques for reconstructing the lateral ligament are complex. The postoperative regimes were also similar with all ankles immobilized, and they advised reduced or non–weight bearing for a period of 3-4 weeks. The return to sport duration was commented on by 2 studies[9,6] at 3-4 months and 4-6 months, respectively.

The outcomes from these 5 studies were generally satisfactory and similar to previously published data on outcomes after OCL treatment without the additional lateral ligament reconstruction.[6]

The one anomaly is the article by Choi et al[8] who found 11 of 15 patients had an "unsatisfactory" outcome (Karlsson-Peterson Ankle Score [KPAS] score <90) after lateral ligament reconstruction and bone marrow stimulation. A KPAS score of